Exhibit 107

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

Leukemia & Non-Hodgkin Lymphoma

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This report provides a medical expert opinion regarding three key issues related to water contamination at Camp Lejeune. First, it examines whether a causal relationship exists between exposure to trichloroethylene (TCE), tetrachloroethylene (PCE), benzene, and vinyl chloride (VC) in the water at Camp Lejeune and the development of leukemia and NHL. Second, because I conclude that a causal relationship is established for trichloroethylene (TCE), tetrachloroethylene (PCE), and benzene, the report generally assesses the likely exposure-response relationship at concentrations of the chemicals in the Camp Lejeune water. Finally, it considers the impact of combined exposures to TCE, PCE, benzene, and VC on health outcomes.

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,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.

My opinions consider whether there is at least as likely as not a causal relationship between the chemical carcinogens described in this report and cancer, and in particular, blood cancers. 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.

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

Based on my education and experience as a physician and scientist for over 30 years and review of the medical literature using the generally-accepted methodology including integrative analysis of mechanistic, preclinical, and epidemiological evidence, as well as considering the weight of evidence and the Bradford Hill Considerations, I conclude that there is more likely than not a causal relationship between exposure to contaminated drinking water at Camp Lejeune and NHL and leukemia.

IV. DISCUSSION OF OPINIONS

A. General Considerations of Carcinogenesis

Cancer is a multi-step pathological process caused by a variety of insults acting upon normal cells. It arises when a susceptible cell undergoes spontaneous and/or environmentally associated genetic changes in the context of a permissive host, supporting microenvironment, and/or immunologic state (for a general review, see Hanahan and Weinberg, 2011; Hanahan, 2022; Weinberg et al., 2023). These insults lead to genetic and epigenetic alterations, including DNA mutations, deletions, rearrangements, chromosomal aberrations, and changes in chromatin and gene regulation. These alterations activate oncogenes or inactivate tumor suppressor genes, which drives cancer progression.

The medical and scientific communities widely recognize that environmental exposures and individual habits contribute to cancer development. Known causes include exposure to electrophilic agents (chemicals that directly interact harmfully with cells), chemicals metabolized into electrophilic agents, and genotoxic agents (which damage DNA). Additional contributors include agents that disrupt cellular processes, such as epigenetic regulation, endocrine systems, inflammation, oxidative redox programs, and immune surveillance. Therapeutic and environmental radiation are also recognized causes.

Certain chemicals, including benzene, TCE, vinyl chloride, PCE, ethylene oxide, and asbestos, are known carcinogens. Carcinogens are external factors that contribute to cancer by causing genetic and epigenetic changes. They act through mechanisms such as direct chemical reactions with DNA (genotoxicity), disruption of cellular processes, and suppression of immune surveillance. For example, benzene, TCE,

vinyl chloride, and asbestos contribute to cancer by inducing mutations, epigenetic changes, and oxidative stress while promoting immune evasion and tumor growth. Social exposures like tobacco use and alcohol consumption, and infectious agents, such as specific viruses and microbes, are also linked to cancer. Decades of scientific evidence (Peto, 1991; Flanders, 2003; Frank, 2007; Korchevskiy and Korchevskiy, 2022; Smith et al., 2020) demonstrate that environmental exposure to carcinogens contributes to cancer, even in individuals with hereditary risk factors, which often serve as susceptibility factors enhancing the effects of carcinogens.

Cells have specialized DNA repair mechanisms that protect against both endogenous and exogenous DNA damage. These systems are critical for addressing damage caused by, among other things, radiation, oxygen radicals, and routine DNA replication errors. However, sustained or intermittent exposure to carcinogens like benzene and TCE can overwhelm these mechanisms. When DNA repair fails, genotoxicity occurs, resulting in mutations and chromosomal damage that activate oncogenes, inactivate tumor suppressor genes, and drive cancer progression. Mutations caused by exposure to carcinogens that are not repaired by our DNA mechanisms may result in the development of cancer.

Carcinogenesis is the "process by which normal cells are transformed into cancer cells" (NIH, https://www.cancer.gov/publications/dictionaries/cancer-terms/def/carcinogenesis). Carcinogenesis involves three stages: initiation, promotion, and progression:

- **Initiation** is the first stage of carcinogenesis, characterized by rapid, irreversible changes caused by carcinogens structurally modifying DNA, leading to mutations in growth-regulating genes (oncogenes). Once a cell is mutated, it becomes susceptible to tumor promotion.

- **Promotion** is the second step in cancer development, triggering the multiplication of abnormal cells (neoplastic development). Promoters are carcinogens that activate dormant oncogenes. They may act by killing normal cells that suppress adjacent oncogenes or by inhibiting suppressor genes, which normally prevent uncontrolled cell growth. When suppressor genes are inactivated, oncogenes drive tumor formation.

- **Progression** is the final stage of carcinogenesis, involving the growth of neoplastic cells and their spread through invasion and metastasis. Invasion refers to local movement into adjacent tissues, whereas metastasis involves distant spread. These processes reflect a physiological system out of control. Tumor promoters drive the clonal expansion of mutated cells, inducing proliferation and preventing apoptosis, preserving the potential for further genetic and epigenetic changes.

Carcinogens can be classified by their role in carcinogenesis (initiators or promoters) or by their characteristics (Smith, 2020), including genotoxicity, epigenetic changes, oxidative stress, and immune suppression. Many carcinogens exhibit one or multiple key characteristics that are sufficient to initiate and promote cancer.

The relationship between exposure and cancer risk can be highly complex. The influence of a carcinogen can be dependent on the duration of exposure, route of exposure, combination of exposure, genetics, and age of the host, among other factors. Duration can be more critical than concentration, with risk often increasing exponentially with prolonged exposure (Peto, 1991; Flanders, 2003; Frank, 2007; Korchevskiy and Korchevskiy, 2022). Carcinogens can show many different relationships with dosage, including linear, and supralinear. Some carcinogens display supralinear dose-response relationships,

where lower exposures may pose greater risks than higher exposures because of metabolic or toxic saturation effects. (IRIS, 2016; Korchevskiy and Korchevskiy, 2022). At higher exposures, there can be a plateau effect, such that increasing dosage is not associated with any or much increase in risk. (Kyle, 2004)

Understanding dose-response relationships requires extensive statistical analyses and integration of epidemiological and preclinical data. Studies often examine exposure as a continuous variable that accounts for confounding factors, lag times, and individual heterogeneity. Although population-level risk estimates provide valuable insights, they may underestimate individual risks given the wide variability in genetic and environmental susceptibility.

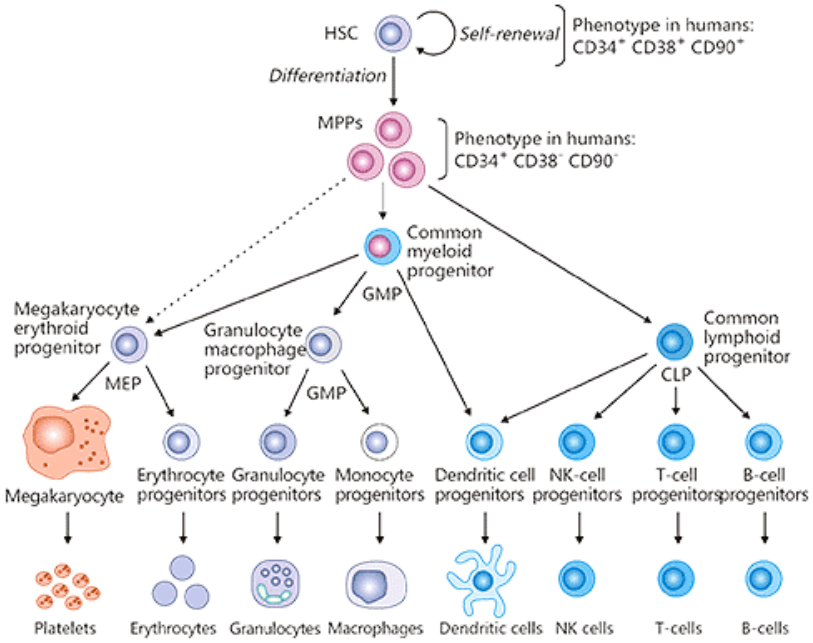
Cancer evolves from a single clonal cell to an invasive disease over years or decades. During this progression, cancer cells undergo genetic and epigenetic changes, whereas pre-cancerous cells may remain dormant for long periods. Host factors, including age, immune function, and genetic susceptibility, influence cancer progression. Carcinogens such as TCE, VC, PCE, and benzene contribute at multiple stages, affecting both tumor cells and the host microenvironment.

B. Hematopoietic Cancers

1. Hematopoietic Cancers and Subtypes

In general, hematopoietic cancers can be divided in various ways, but one way is into red blood cells, megakaryocytes/platelets, myeloid cells, and lymphocytes. Lymphocytes that become cancerous can cause what are generally referred to as lymphohematopoietic cancers. Acute lymphocytic leukemia, multiple myeloma, NHL, and Hodgkin lymphoma are examples of hematopoietic cancers that derive from lymphocytes.

They are all derived from the hematopoietic stem cells that then give rise to specific lineages of differentiated cells, including the cells that become RBCs, platelets, macrophages/monocytes, granulocytes, neutrophils, eosinophils, basophils, and lymphocytes (see figure below from Seita and Weissman, 2010). Lymphocytes are a group of cells that can be B-cells (can make antibodies), T-cells (have a T-cell receptor), or NK cells. Lymphocytes are the type of hematopoietic cell that can give rise to lymphoid-related leukemias and lymphomas (both Hodgkin lymphoma and NHL). However, all of these cancers are part of the blood system. The specific subtypes of cancer can be distinguished by doctors and scientists by specific phenotypic features, as they are generally the malignant expansion of cellular lineages of hematopoietic cells (they can look different under the microscope, and we can measure different proteins on the surface of the various cell types). There are some genetic events that are quite common to all hematopoietic cancers, but some are associated with specific subtypes. There are common risk factors among blood cancers, while some are specific to certain subtypes. At bottom, however, they are all hematopoietic cancers. Furthermore, often the first or one of the first steps in the formation of cancer of hematopoietic cells begins in hematopoietic stem cells, and then further steps can occur in more mature lineages (Greaves, 1986; Bowman, 2018).



The words “lymphoma” and “leukemia” are descriptions given by physicians. The word lymphoma tends to be used for a cancer more in the lymph organs, like the lymph nodes and spleen, whereas leukemia includes hematopoietic cancers that can be found in the blood and can include a variety of hematopoietic cancers, including lympho-hematopoietic (lymphocytes) as well as myeloid (macrophages, monocytes, and related cell types). Importantly, leukemic states can exist in hematopoietic cancers that are described as “leukemia,” such as acute or chronic myeloid leukemia (AML, CML) and acute and chronic lymphocytic leukemia (ALL, CLL), but can also include diseases of the hematopoietic system, such as NHL or Hodgkin lymphoma, that can present with a “leukemia.” In many circumstances, diseases are referred to as both leukemia and lymphoma, such as small lymphocytic lymphoma (SLL) and chronic lymphocytic leukemia (CLL).

Patients with these types of hematopoietic cancer have cancerous lymphocytes that can be found in the blood, bone marrow, and other locations. Lymphocytes include either B-cells, which are antibody-producing cells, or T-cells, which have a T-cell receptor. Acute lymphocytic leukemia is a type of cancer that arises from lymphocytes, more commonly B-cells but also T-cells, usually in less mature pre-B or pre-T-cells—lymphocytes that have not completed the formation of antibodies or T-cell receptors, respectively. This disease occurs in infants, children, and, less commonly, adults. The disease is often located in the blood but can also be located in the bone marrow and other hematopoietic and lymphoid organs.

Multiple myeloma is a type of cancer that arises from B-cell lymphocytes that produce antibodies, also known as plasma cells. B-cells are the type of cells that normally help fight infections through antibody production. This type of cancer occurs in adults, is generally more common in males than females, and occurs in approximately 0.7% of people during their lifetime. The mean age at diagnosis was 60 years.

NHL is a group of cancers affecting either B or T-cells at different stages of differentiation, although it more commonly arises from mature B-cells. Clinically, NHL can be divided into high-grade, fast-growing, and low-grade, slow-growing lymphomas. These are further subdivided based on their phenotypic features,

observed through histology, characterization of cell surface markers, and genomic features. Risk factors for NHL include age, family history, autoimmunity, immune suppression, and exposure to carcinogens such as chemicals such as benzene and TCE.

Marginal zone lymphoma (MZL) has been described as extra-nodal, nodal, and splenic (also called MALT) and has been associated in some cases with infection by *Helicobacter pylori* (the bacteria linked to peptic ulcers), infection by hepatitis C, autoimmune diseases, and a family history of lymphoma. MZL is slow-growing, generally responds initially to chemotherapy and/or rituximab, but is not typically considered a curable disease.

Mantle cell lymphoma is a type of NHL with intermediate-aggressive clinical behavior, most commonly seen in middle-aged adults, and is characterized by a distinct B-cell lymphocyte phenotype.

Hodgkin lymphoma is a disease of lymphocytes that characteristically feature Reed-Sternberg cells and abnormal binucleated lymphocytes that are very distinctive in appearance.

Acute lymphocytic leukemia (ALL) includes B-cell or T-cell subtypes (NLM, 2023). B-cell ALL can be further divided based on the differentiative state of the lymphocyte. B-cell ALLs can also include Burkitt's type leukemia, and Burkitt's can also be called a lymphoma. ALL occurs slightly more frequently in males than in females and three times as frequently in white versus Black populations. Patients with ALL typically present with symptoms related to anemia, thrombocytopenia, and neutropenia due to tumor-invasion of the bone marrow. Clinical symptoms include fatigue, bruising/bleeding, infections, fever, night sweats, and unintentional weight loss. Hepatomegaly, splenomegaly, and lymphadenopathy can be seen in up to half of adults on presentation. Central nervous system (CNS) involvement is common and can be accompanied by cranial neuropathies or symptoms, predominantly meningeal, related to increased intracranial pressure. ALL is thought to occur after DNA damage causes uncontrolled growth and spread of lymphoid cells throughout the body. Splenomegaly and hepatomegaly occur due to the sequestration of platelets and lymphocytes in the spleen and liver. Environmental factors have been implicated in the etiology of ALL, including exposure to benzene, as well as other chemical carcinogens such as ionizing radiation or previous exposure to chemotherapy/radiotherapy. ALL can be associated with activating mutations in a variety of genes, including ARD5B, IKZF1 (the gene encoding Ikaros), and CDKN2A, and less commonly PAX5, ETV6, and particularly p53. ALL is not generally considered familial. ALL is seen in about 4,000 people each year, most of whom are under the age of 18, and is the most common cancer of childhood. The peak age at diagnosis is between two and 10 years. ALL is more common in children with Trisomy 21 (Down syndrome), neurofibromatosis type 1, Bloom syndrome, and ataxia-telangiectasia. Genomic studies have noted that somatic, polymorphic variants of ARD5B, IKZF1 (the gene encoding Ikaros), and CDKN2A are associated with an increased risk of ALL (odds ratio 1.3 to 1.9) (NLM, 2023). Other rare germline mutations in PAX5, ETV6, and particularly p53 can also strongly predispose individuals to leukemia. Prognosis is worse in infants and better in older children. The MLL gene at chromosome 11q23 is associated with poor prognosis. ALL occurs in approximately 3.3 cases per 100,000 children. Survival rates for ALL are greater than 85%.

Acute myeloid leukemia (AML) is a subgroup of hematopoietic cancers that is also classified as myeloproliferative disorders. AML is characterized by the clonal proliferation of undifferentiated myeloid precursors, known as blasts, within the bone marrow compartment. Extensive research, both past and ongoing, investigates the communication pathways of these cells within bone marrow. However, this

proliferation primarily stems from the accumulation of diverse genomic and cytogenetic abnormalities. The clinical manifestations of this process result in ineffective erythropoiesis, megakaryopoiesis, and bone marrow failure. Patients with myeloproliferative neoplasms, including myelofibrosis, essential thrombocythemia, polycythemia vera, and chronic myeloid leukemia, may also progress or evolve into a higher-grade myeloid neoplasms, such as AML. AML also includes patients who have previously received chemotherapy for other malignancies. Patients who have been exposed to alkylating agents or radiation can develop MDS/AML with chromosome 5 or 7 abnormalities 5–7 years after exposure. Other chemotherapeutic agents, such as topoisomerase inhibitors, can lead to AML, which is known as therapy-related MDS/AML. Environmental exposures associated with AML include radiation, tobacco smoke, benzene, other volatile organic compounds, and carcinogenic exposures. The annual incidence of new cases in both men and women is approximately 4.3 per 100,000 population, with over 20,000 cases per year recorded in the United States. The median age at diagnosis was 68 years. AML is more common in non-Hispanic whites. AML is more common in males than females, with a ratio of 5:3. AML is a highly heterogeneous disease that can be genetically categorized into favorable, intermediate, or high-risk groups based on the criteria outlined in the ELN 2022 guidelines. Genetic abnormalities of favorable-risk disease include chromosomal translocations t(8;21)(q22;q22.1) or inv(16)(p13.1q22). Patients who lack FLT3-ITD (internal tandem duplication) mutations without mutated NPM1 or with CEBPA (bZIP in-frame) mutations are also categorized as favorable risk. NPM1 mutations are present in up to 35% of patients with AML. Intermediate-risk AML is diagnosed in the presence of any FLT3-ITD mutation or t(9;11)(p21.3;q23.3), or MLL rearrangement. High-risk AML can be diagnosed in the presence of several cytogenetic or molecular aberrancies, which notably include monosomy 5/del 5q or 7/deletion 7q, other monosomal or complex karyotype (≥3 unrelated abnormalities), or mutations in ASXL1, EZH2, SRSF2, or TP53. Runt-related transcription factor (RUNX1) is an essential component of hematopoiesis and is also known as AML1 protein or core-binding factor subunit alpha-2 (CBFA2). RUNX1 is located on chromosome 21 and is frequently translocated with the ETO (Eight Twenty One)/RUNX1T1 gene located on chromosome 8q22, creating an AML-ETO or t(8;21)(q22;q22) AML, which is observed in approximately 12% of AML cases. These mutations are associated with trisomies 13 and 21 and are resistant to standard induction therapy. Mutations in isocitrate dehydrogenase (IDH) are oncogenic and present in 15%–20% of all AML cases and 25%–30% of patients with cytogenetically normal AML, with a higher prevalence in older individuals. Additionally, TP53 mutations are associated with poor prognosis and chemotherapy resistance. The administration of multiagent induction chemotherapy can induce complete remission. However, allogeneic stem cell transplantation is the only established curative therapy. Despite existing therapeutic approaches, the prognosis remains poor, especially in older individuals.

All hematopoietic cancers, including leukemias, are caused by genetic events that activate oncogenes and/or inactivate tumor suppressor genes. Carcinogens that increase the likelihood of such genetic events also increase the risk of these cancers. Although the causes of hematopoietic cancers are generally similar, treatments are tailored to each type, reflecting their phenotypic and clinical differences.

2. Shared Mechanisms in Hematopoietic Cancer

As described in more detail above, hematopoietic cancers are a group of neoplastic diseases of the blood-forming cells (Greaves, 1986; Bowman, 2018). They share that they are all derived from common hematopoietic stem cells (HSCs). The specific subtypes of the diseases refer to cancers of specific

different degrees of maturation of hematopoietic cells, but they are all blood cells, and they all derive from a common precursor cell (i.e., HSCs). Lymphoid-related hematopoietic cancers, also referred to as lympho-hematopoietic cancers, are all derived from lymphocytes and can give rise to both non-Hodgkins and Hodgkins lymphomas (NHL and HL) and Multiple Myeloma (MM), other plasma cell neoplasms, and leukemias that include acute lymphocytic leukemia and acute lymphoblastic leukemia (ALL) and chronic lymphocytic leukemia (CLL), natural killer cell leukemia (NKL), as well as numerous other lymphoid-related diseases. Notably, even myeloid and erythroid neoplastic diseases are all derived from common HSCs. Further, the first steps in hematopoietic tumorigenesis are thought to commonly occur in HSCs, and the final neoplasia seen at diagnosis reflects the multi-step trajectory of cancer such that the final expanded cancer is a discrete, often more mature stage of differentiation. Given these similarities, my report employs a combined analysis by evaluating blood cancers (lymphoma and leukemia) as one group.

C. Carcinogens at Camp Lejeune

1. Specific Carcinogens

(a) TCE

TCE is a well-known carcinogen recognized by multiple reputable scientific and governmental agencies using methodologies typically employed by medical scientists and physicians. These agencies concluded that TCE exposure can cause cancer, including leukemia and NHL.

The International Agency for Research on Cancer (IARC), which is part of the World Health Organization (WHO) and is highly regarded for its expertise in evaluating carcinogenic risks, classified TCE as carcinogenic to humans. In its 2012 assessment, IARC placed TCE in Group 1, which is the highest category used for agents where there is sufficient evidence of carcinogenicity in humans. IARC also notes that an agent may be included in this group when there is sufficient evidence from animal studies and strong evidence that the agent acts through relevant mechanisms of carcinogenicity in exposed humans and noted a positive association with NHL (IARC, 2012).

In the 14th Report on Carcinogens (2016), the National Toxicology Program (NTP) elevated TCE classification from “reasonably anticipated” to a “known” carcinogen based on substantial epidemiological evidence in humans, including two meta-analyses indicating an increased risk of NHL, supported by mechanistic and animal studies. In its 2021 report, NTP reaffirmed that TCE exposure is linked to cancer, including leukemia, based on sufficient evidence of carcinogenicity (NTP, 2021).

The Agency for Toxic Substances and Disease Registry (ATSDR), an agency of the U.S. Department of Health and Human Services, which focuses on the public health impacts of hazardous substances, concluded that exposure to TCE can lead to cancer in humans. In its 2017 assessment, the ATSDR specifically noted that there is “equipoise and above evidence” that TCE exposure is linked to all adult leukemias (ATSDR, 2017). The ATSDR also concluded that there is sufficient evidence for causation for TCE and NHL.

Additionally, the U.S. Environmental Protection Agency (EPA) has taken significant actions concerning TCE. The EPA recently proposed a rule to ban the manufacture, processing, and distribution of TCE for all

uses, with a timeline for compliance and workplace controls for certain industrial and commercial uses until full prohibitions take effect. This action aims to protect workers, consumers, and bystanders from the harmful health effects of TCE (EPA, 2023).

A final revised risk determination for TCE was released last year by the EPA, which superseded previous findings from 2020. In this updated evaluation, the EPA concluded that TCE presents an unreasonable risk to human health as a whole chemical substance. The EPA identified multiple health risks associated with TCE, including developmental toxicity, reproductive toxicity, liver and kidney toxicity, immunotoxicity, neurotoxicity, and cancer, from both inhalation and dermal exposure (EPA, 2023). Of the 54 conditions of TCE use evaluated, the EPA found that 52 contributed to the unreasonable risk to human health, which underscores the extensive and serious nature of TCE's impact.

I have reviewed these agencies' conclusions about TCE and the sources that underlie their conclusions and I conclude that there is a clear and consistent understanding that TCE is a potent carcinogen with well-established links to cancers, particularly leukemia and NHL. The comprehensive evidence from these trusted sources, frequently relied upon by scientists like myself, reinforces the need for strict regulation of TCE to protect public health.

(b) Benzene

According to the IARC review of Benzene, "Benzene causes acute myeloid leukemia (AML)/acute non-lymphocytic leukemia. Additionally, a positive association has been observed between exposure to benzene and acute lymphocytic leukemia (ALL), chronic lymphocytic leukemia (CLL), multiple myeloma (MM), and non-Hodgkin lymphoma (NHL)" (IARC Monograph on Benzene, 2012). IARC has classified benzene as a human carcinogen (Group 1).

In its 2017 report, the ATSDR concluded that there is "sufficient evidence for causation for benzene and NHL" and "sufficient evidence for benzene and all leukemia types, i.e., ALL, CLL, AML, and CML."

The EPA also classifies benzene as a known human carcinogen, primarily based on evidence linking it to leukemia. The EPA notes that benzene exposure increases the risk of developing AML and may also be linked to other blood-related cancers, including ALL, CLL, and NHL. The agency emphasizes that, even at low levels, presents a significant risk to human health due to its carcinogenic properties. The EPA has set strict regulatory limits on benzene in air and water to minimize public exposure and reduce the risk of cancer (EPA IRIS Benzene, 2023).

(c) Vinyl Chloride

Vinyl chloride is recognized as a carcinogen that has been linked to certain blood cancers, particularly leukemia and lymphoma, according to key health and regulatory agencies.

IARC classifies vinyl chloride as Group 1: Carcinogenic to humans based on sufficient evidence that it causes cancer in humans. While IARC is best known for linking vinyl chloride exposure to liver cancer, it also acknowledges associations between exposure to vinyl chloride and hematopoietic cancers, including leukemia and lymphoma. These associations have been observed in workers with prolonged exposure to vinyl chloride, typically in industrial settings (IARC Monograph on Vinyl Chloride).

The ATSDR has also linked vinyl chloride as a potential cause of leukemia and lymphoma. The ATSDR reports that chronic exposure to vinyl chloride, particularly via inhalation or ingestion, is linked to an increased risk of developing leukemia and other blood cancers. The agency emphasizes that vinyl chloride exposure disrupts the bone marrow's ability to produce healthy blood cells, which may contribute to the development of these cancers (ATSDR Toxicological Profile for Vinyl Chloride, 2006).

The U.S. EPA similarly classifies vinyl chloride as a known human carcinogen and has identified a potential link between exposure to vinyl chloride and certain hematopoietic cancers, such as leukemia and lymphoma. The EPA's Integrated Risk Information System (IRIS) notes that evidence from occupational studies shows an association between vinyl chloride exposure and an increased risk of blood cancers. (EPA IRIS Vinyl Chloride, 2020).

(d) PCE

PCE, widely used as a solvent in dry cleaning and industrial settings, has been extensively studied for its possible carcinogenic effects. Several studies have suggested that PCE exposure is associated with an increased risk of bladder cancer, multiple myeloma, and non-Hodgkin's lymphoma. Additionally, preclinical models have shown that PCE can cause liver, kidney, and hematopoietic cancers.

IARC classifies PCE as Group 2A: Probably carcinogenic to humans. This classification is based on limited evidence of carcinogenicity in humans but with sufficient evidence from animal studies in which PCE caused liver, kidney, and blood-related cancers (IARC Monograph on PCE).

The ASTDR has also reported on the potential cancer risk associated with PCE exposure. For example, in its 2017 assessment, the ATSDR highlighted that meta-analyses, including those conducted by Vlaanderen et al. (2014), found an association between PCE exposure and an increased risk of bladder cancer. ATSDR's 2017 report found equipoise and above evidence for causation for PCE and NHL and below equipoise evidence for causation for PCE and leukemias; however, as explained throughout this report, studies since the ATSDR evaluation now support at least as likely as not a causal relationship between PCE exposure and leukemia. ATSDR's evaluation considers both epidemiological studies and evidence from toxicological data, further supporting the concern over PCE's potential to cause cancer.

The U.S. EPA currently classifies PCE as "likely to be carcinogenic to humans by all routes of exposure" based on sufficient evidence from animal studies and suggestive evidence from human studies. The EPA's IRIS evaluation emphasizes that PCE exposure poses a potential risk for several types of cancer, particularly bladder and hematopoietic cancers, as observed in occupational and environmental settings. The EPA has set regulatory limits on PCE exposure in drinking water and air to mitigate the risks (EPA IRIS PCE, 2023).

2. Concentration levels present at Camp Lejeune

The ATSDR water modeling tables document detailed monthly mean contaminant concentrations over time for Tarawa Terrace, Hadnot Point, and Holcomb Boulevard, which were also provided to me in Appendices H1, J, and K of October 25, 2024, Expert Report by Morris L. Maslia. I have used these values for my opinions in this case.

Tarawa Terrace PCE concentrations reached a maximum monthly average value of 183 micrograms per liter (µg/L) compared to a one-time maximum measured value of 215 µg/L and exceeded USEPA's current maximum contaminant level (MCL) of 5 µg/L during the period November 1957–February 1987. At Hadnot Point, reconstructed TCE concentrations reached a maximum monthly average value of 783 µg/L during the period August 1953–December 1984, with a maximum measured value of 1,400 µg/L. Hadnot Point also provided contaminated drinking water to the Holcomb Boulevard housing area continuously prior to June 1972, when the Holcomb Boulevard water treatment plant came online (maximum reconstructed TCE concentration of 32 µg/L) and then intermittently during the period June 1972–February 1985 (maximum reconstructed TCE concentration of 66 µg/L). (See Maslia Report, p. 20).

D. Literature Review

1. TCE and Hematopoietic Malignancy

The association between NHL and exposure to TCE has been investigated in multiple studies in several countries that collectively suggest that there is an association between TCE and hematopoietic cancers, including lympho-hematopoietic cancers such as NHL (IARC, 2014; ATSDR, 2017; NTP, 2015; NTP, 2021).

The NTP (2015) determined that TCE exposure elevated the risk of NHL across all three studies they deemed to have moderate to high utility for assessing NHL risk (Cocco, 2013; Hansen, 2013; Radican, 2008). Among these, the pooled InterLymph analysis conducted by Cocco stands out as the most detailed, as it analyzed NHL subtypes and incorporated a robust exposure assessment. Both the NTP and ATSDR recognized this study as highly significant. The study identified an increased risk of NHL (all types) among workers with a high likelihood of TCE exposure (OR=1.4, 95% CI: 0.9, 2.1) and also observed elevated risks for specific NHL subtypes, including follicular lymphoma (FL) (OR=1.6, 95% CI: 0.7, 3.4) and CLL (OR=1.8, 95% CI: 0.9, 3.6). Notably, the analysis highlighted associations with exposure duration, intensity, and frequency.

ATSDR agrees with the EPA's conclusion that occupational epidemiological studies offer compelling evidence supporting a causal relationship between TCE and NHL.

Several meta-analyses demonstrated an elevated risk of NHL with TCE exposure (Mandel, 2006; Scott, 2011; Karami, 2013; Odutola, 2021).

Mandel (2006): The meta-analysis found a nearly 30% increased risk of NHL in the total cohort (RR = 1.29; 95% CI, 1.0–1.66). In seven studies specifically examining TCE exposure, the risk was even higher (RR = 1.59; 95% CI, 1.21–2.08). Three cumulative exposure studies revealed a higher risk of NHL (RR = 1.8; 95% CI, 0.62–5.26) in the lowest exposure category and a slightly lower risk in the highest exposure category (RR = 1.41; 95% CI, 0.61–3.23).

Karami (2013): Analysis showed no evidence of between-study heterogeneity or publication bias for cohort studies, while the EPA meta-analysis reported minimal heterogeneity in studies focusing on higher TCE exposure levels. Hence, multiple studies showed an association, and several of the better studies showed a statistically significant association. Karami's analysis indicated an elevated risk of NHL in both cohort and case-control TCE exposure studies (RR = 1.32; 95% CI, 1.14–1.54 for case-control studies) (RR = 1.52; 95% CI, 1.29–1.79).

Scott (2011): The meta-analysis conducted for the US EPA revealed an increased association between TCE exposure and NHL (RR = 1.23; 95% CI, 1.07–1.42), with an even higher risk in the highest exposure group (RR = 1.43; 95% CI, 1.13–1.82).

Odutola (2021): The meta-analysis found an association between TCE and an elevated risk of follicular lymphoma, a subtype of NHL (RR = 1.35; 95% CI, 1.09–1.68).

Alexander (2006): Alexander (and other colleagues from Exponent) meta-analysis of 8 cohort of case-control studies of TCE with 131 cases in an exposed populations and concluded no association between MM or leukemia. They reported for MM (SRRE = 1.05, 95% CI: 0.80-1.38; P value for heterogeneity = 0.94) or leukemia (SRRE = 1.11, 95% CI: 0.93-1.32; P value for heterogeneity = 0.50), based on TCE-exposed subgroup meta-analyses. Study-specific RR estimates for MM ranged between 0.57 and 1.62. RRs for leukemia ranged between 1.05 and 1.15 in five studies, while one study reported a 2-fold increased RR, and another study reported an inverse association of 0.60. All confidence intervals (CIs) for study-specific estimates included 1.0.

In contrast, Karami (2013) did an analysis of TCE for 9 cohort studies and one case-control, including 188 cases, concluding an sRR of 1.1 (0.94, 1.28).

Other studies are described in more detail here:

Hardell (1994: This studied 105 cases of NHL among Swedish workers exposed to solvents, including TCE, finding more than a 700% increased risk of NHL in these workers, albeit with a wide confidence interval (RR = 7.2; 95% CI, 1.3–42). Additionally, brief exposures (1 to 17 days) were associated with an elevated NHL risk for all organic solvents, including TCE (RR = 6.5; 95% CI, 2.1–18). There was also an almost 300% increase in lymphocytic NHL (RR = 2.9; 95% CI, 1.3–6.6).

Hansen (2001): Hansen analyzed Danish workers exposed to TCE, revealing a more than threefold increased risk among exposed male workers (RR = 3.5; 95% CI, 1.5–6.9).

Cocco (2013): This was conducted before IARC reclassified TCE as a Group 1 carcinogen, showed an increased risk of NHL overall (RR = 1.4; 95% CI, 1.0–2.1) and nearly a twofold risk for chronic lymphocytic leukemia (CLL) (RR = 1.8; 95% CI, 0.9–3.6). The data on CLL risk ranged from near null to a more than 360% increase. Among Finnish, Swedish, and Danish workers, there was a more than 50% increase in NHL risk (RR = 1.55; 95% CI, 1.06–2.20). The authors also noted that even modest TCE exposures were associated with reduced major lymphocyte blood counts, thus providing biological plausibility of TCE immunotoxicity as a contributing factor in NHL development.

Persson (1989, 1993); Persson & Fredrikson (1999): Two case-control studies with similar designs were pooled to investigate risk factors for NHL. Exposure was assessed using information reported in a questionnaire posted to the subjects including 199 cases and 479 controls. Exposure to TCE in workers resulted in an odds ratio stratified by age and sex of 1.2 (95% CI, 0.5–2.4).

Miligi (2006): A population-based case-control study of lympho-hematopoietic tumors included all new cases of NHL from 1991 to 1993 in Italy. The participation rate was 85% among cases of NHL (n = 1428) and 73% among controls (n = 1530). Cases were identified from hospital and pathology departments. Controls were randomly selected from the general population. Face-to-face interviews were performed

to collect information on occupational history, and exposure was assessed by an industrial hygienist. Odds ratios were adjusted for age, sex, education, and area. When combining NHL, the odds ratios associated with occupational exposure to trichloroethylene were 0.8 (95% CI, 0.5–1.3) for exposure at very low or low levels, or 1.2 (95% CI, 0.7–2.0) at medium or high levels. No association was found with duration of exposure. A non-statistically significant increased risk was observed for DLBCL (OR, 1.9; 95% CI, 0.9–3.7; 13 exposed cases), but not for other types of NHL.

Seidler (2007): A case-control study in Germany included patients with NHL (n = 710; participation rate, 87%) and controls (matched by sex, region, and age) recruited from population registers (n = 710; participation rate, 44%). Interviewers collected a complete history of all jobs held for more than 1 year and specific job tasks. On the basis of job task-specific questionnaires, a trained occupational physician assessed exposure to chlorinated hydrocarbons, including TCE. Highest exposure to TCE (> 35 ppm-years) had an increased risk of NHL (OR, 2.1; 95% CI, 1.0–4.8), but no significant dose-response relationship was observed (P for trend, 0.14). Estimated increased risks for workers in the category of highest exposure to TCE were also observed for B-cell NHL, T-cell NHL, HL, DLCL, FL, and MZ lymphoma, but most precise for the B-cell NHL (n = 554; OR, 2.3; 95% CI, 1.0–5.3).

Wang et al. (2009): In the USA, a case-control study investigated the association between NHL and occupational exposure to solvents among women. For subjects between 1996 and 2000, it included 601 cases (participation rate, 72%) and 717 controls selected by random-digit dialing (participation rate, 69%) or random selection from Medicare service files. Exposure assessment was based on occupational questionnaire and expert assessment using a job-exposure matrix. An increased risk of NHL in workers ever exposed to trichloroethylene was found (OR, 1.2; 95% CI, 0.9–1.8), after adjustment for age, family history of hematopoietic cancers, alcohol consumption, and race. Compared with non-exposed subjects, the risk was elevated at a medium-to-high intensity of exposure (OR, 2.2; 95% CI, 0.9–5.4) but was still not statistically significant compared with that in the group with a low level of exposure (OR, 1.1; 95% CI, 0.8–1.6; P for trend, 0.06).

Deng et al. (2012): An analysis was done to determine whether the association between NHL and solvent exposure was influenced by genes of the immune system. Samples of blood or buccal cells were collected from 518 patients and 597 controls, and the interaction between exposure to trichloroethylene and IL12A (rs582054) genotype was assessed. Among women with AT/AA genotypes who had been exposed occupationally to TCE, there were increased risks observed for NHL overall (OR, 2.09; 95% CI, 1.28–3.42), DLBCL (OR, 2.66; 95% CI, 1.42–4.96), and follicular lymphoma (FL) (OR, 1.71; 95% CI, 0.78–3.77). In contrast, among women who carried the IL12A (rs582054) TT genotype and who had been exposed occupationally to trichloroethylene, the risk estimates were below one and not statistically significant.

Cocco et al. (2010, 2013): A multicentric case-control study on occupational exposure to TCE and NHL, the Epilymph study was conducted in the Czech Republic, France, Germany, Ireland, Italy, and Spain from 1998 to 2004. The study included 2348 cases of lymphoma and 2462 controls (hospital and population-based; matched by age, sex, residence in Germany and Italy). The overall participation rates were 88% for cases, 81% for hospital controls, and 52% for population controls. Face-to-face interviews were conducted to collect data on occupational history, and exposures were assessed by an industrial hygienist. Odds ratios were adjusted for age, sex, education level, and study center. No association was found between occupational exposure to TCE and any subtype of NHL.

However, in the study they reported subsequently (Cocco et al., 2013), the strongest evidence for an association was between TCE exposure and NHL for the InterLymph pooled analysis (P = 0.004 for combined analysis of duration, frequency, and intensity of exposure among individuals with the highest probability of exposure) and the two meta-analyses (mRR =1.23, 95% CI 1.07 to 1.42, Scott and Jinot, 2011; mRR 1.32, 95% CI 1.14 to 1.54, Karami et al., 2013). The risk of NHL increased with increasing level or duration of exposure in the InterLymph pooled analysis. The authors specifically described that for follicular (FL), but not NHL overall or other subtypes, increased by probability (p=0.02) and intensity level (p=0.04), and with the combined analysis of four exposure metrics assumed as independent (p=0.004). After restricting the analysis to the most likely exposed study subjects, they found that the risk of overall NHL, FL, and chronic lymphocytic leukemia (CLL) were elevated and increased by duration of exposure (p=0.009, p=0.04 and p=0.01, respectively) and with the combined analysis of duration, frequency, and intensity of exposure (p=0.004, p=0.015 and p=0.005, respectively). They explained that although based on small numbers of exposures, the risk of all major NHL subtypes, namely diffuse large B-cell lymphoma, FL, and CLL, showed increases in risk ranging 2-3.2-fold in the highest category of exposure intensity. Finally, they noted significant heterogeneity in risk was detected by major NHL subtypes or by study.

Purdue et al. (2011): In one of its component studies, authors examined the association between NHL and TCE using cases came from four Surveillance, Epidemiology, and End Results (SEER) registry areas in the USA (Iowa, Los Angeles County, Seattle, Detroit) who were diagnosed between July 1998 and June 2000 (n = 1189; participation rate, 76%). Controls were recruited from the general population (n = 982; participation rate, 52%). Subjects were interviewed to obtain a job history. Occupational exposure to TCE was assessed by an industrial hygienist. After adjustment for age, sex, study center, race and education, workers who had an estimated average weekly exposure of > 150 ppm-hours had an odds ratio of 2.5 (95% CI, 1.1–6.1; P for trend, 0.02). An increased risk of NHL was identified for cumulative exposure exceeding 112,320 estimated ppm-hours (OR, 2.3; 95% CI, 1.0–5.0; P for trend, 0.08).

Christensen (2013): A case-control study from Montreal included 215 men diagnosed with non-Hodgkin lymphoma (NHL) and 2341 population and cancer controls. The criteria for inclusion of cancer controls were contiguous sites that were excluded as controls for the index cancer series; cancer of the lung was excluded; and subsamples were constituted such that no cancer site constituted more than 20% of any series of controls. The cancer controls were selected for any solvent site association. The odds ratio associated with exposure TCE was 1.2 (95% CI, 0.5–2.9; seven exposed cases) for any exposure, and 1.0 (95% CI, 0.3–3.5; three exposed cases) for substantial exposure, after adjustment for age, income, education, ethnicity, questionnaire respondent, and smoking.

Several studies reported associations between exposure to TCE and hematopoietic malignancies other than NHL. **Persson (1993)** reported a non-statistically significant increased risk of NHL (crude OR, 2.0; no P-value or confidence intervals provided) related to exposure to TCE in Sweden. The study by **Seidler (2007)** in Germany reported a non-significant increased risk of HL (OR, 2.0; 95% CI, 0.4–10.5) in the category of highest exposure. The international **Epilymph study (Cocco et al., 2010)** did not find any association between occupational exposure to TCE and HL, CLL, or MM. A study by **Gold (2011)** found an association between (cumulative) occupational exposure to TCE and MM. **Seidler** found no association between occupational exposure to TCE and MM or CLL. **Nordström (1998)** reported a non-significant association (OR, 1.5; 95% CI, 0.7–3.3) between exposure to trichloroethylene and hairy cell leukemia in a study in Sweden.

Childhood studies also have evaluated exposure to TCE and leukemia. **Lowengart et al. (1987); McKinney et al. (1991)**: Paternal exposure to trichloroethylene. In McKinney et al. (1991), during the preconception, periconception and gestational, and postnatal periods risks were 2.27 (95% CI, 0.84–6.16), 4.40 (95% CI, 1.15–21.01), and 2.66 (95% CI, 0.82–9.19), respectively. Lowengart et al. (1987) noted a two-fold non-significant increase in risk of leukemia with paternal exposure TCE (OR, 2.0; P = 0.16), during pregnancy (OR, 2.0; P = 0.16), and after delivery (OR, 2.7; 95% CI, 0.64–15.60; P = 0.07). There were no associations with maternal exposure to TCE.

2. Benzene and Hematopoietic Malignancy

Benzene exposure has been widely recognized as a cause of hematopoietic cancers, including leukemia and lymphoma (IARC, 2012; ATSDR, 2017; Loomis, 2017). More recent studies, including several occupational and environmental studies, have provided additional notable evidence that benzene exposure is linked to NHL **(Francisco, 2023; Rana, 2021)**, with a nonlinear relationship between increased risk and dose, even at low levels (Vermeulen, 2023; Lin, 2023).

Human epidemiological studies also suggest that low ambient levels of environmental benzene exposure—studied in over 393,000 participants—can contribute to cancer risk in humans with "no threshold" **(Wang, 2024; Villenueve, 2024)**, including risks for respiratory cancer (HR, 1.29; 95% CI, 1.25–1.34), lung cancer (HR, 1.29; 95% CI, 1.24–1.34), and hematopoietic malignancies. These include leukemia (HR, 1.27; 95% CI, 1.15–1.39), multiple myeloma (HR, 1.45; 95% CI, 1.30–1.62), and NHL (HR, 1.27; 95% CI, 1.16–1.39).

Occupational benzene exposure studies also confirm an association with NHL, including 134,449 participants with "ever exposure," to NHL and MM **(Bassig, 2024)**, a Shanghai Workers study examining leukemia, lung, and stomach cancers **(DeMoulin, 2024)**, and Swedish Oil Refinery workers (Anderson, 2024). Collectively, these studies demonstrate that benzene exposure, even at environmental levels, can contribute to cancer, including hematopoietic cancers like leukemia, MM, and NHL.

Several meta-analyses have evaluated the relationship between benzene exposure and NHL.

The 2008 meta-analysis by **Steinmaus** identified an increased summary risk ratio (sRR) after analyzing 16 case-control and 6 cohort studies. The sRR increased from 1.22 (95% CI: 1.03, 1.46) to 2.12 (95% CI: 1.11, 4.02) when the analysis was limited to studies that avoided self-reported exposure data. This meta-analysis also sought to address potential bias from the healthy worker effect, which can influence standardized mortality ratios (SMRs). By using a mortality odds ratio—comparing NHL cases to other causes of death from benzene exposure—this method assumes a consistent healthy worker effect bias across groups. A slight increase in the sRR for the six studies, excluding self-reported exposures, from 2.12 (95% CI: 1.11, 4.02) to 2.26 (95% CI: 1.29, 3.97) was reported.

Two additional meta-analyses were conducted after the 2009 IARC workgroup meeting (IARC, 2012):

The **Kane (2010)** meta-analysis included two studies based on occupational information from cancer registries and death certificates, which are better categorized as surveillance studies. This analysis reported a summary RR of 1.11 (95% CI: 0.94, 1.30) from 24 studies.

Meanwhile, the **Vlaanderen (2011)** meta-analysis found an increased sRR when limited to six cohort studies with quantitative exposure assessments (sRR=1.27, 95% CI: 0.90, 1.79). Limiting the analysis to these six studies also eliminated the between-study heterogeneity seen when broader datasets were included. However, a limitation of this meta-analysis was the reliance on mortality data, despite NHL's relatively high 5-year survival rate of approximately 70%. Vlaanderen (2011) was summarized by ATSDR (2017):

#	
217	AML
217	Quantitative & qualitative exposure assessment: mRR=1.68 (1.35, 2.10) 21 studies
108	Quantitative to some industrial hygiene sampling: mRR=1.73 (1.26, 2.38) 10 studies
95	Quantitative or semi-quantitative estimates: mRR=1.82 (1.25, 2.66) 9 studies
71	Quantitative exposure assessment: mRR=2.32 (1.55, 3.47) 6 studies (2 incidence, 4 mortality)
47	ALL
47	Quantitative & qualitative exposure assessment: mRR=1.44 (1.03, 2.02) 17 studies
11	Quantitative to some industrial hygiene sampling: mRR=1.26 (0.50, 3.16) 4 studies
11	Quantitative or semi-quantitative estimates: mRR=1.26 (0.50, 3.16) 4 studies
5	Quantitative exposure assessment: mRR=2.80 (0.27, 29.2) 1 incidence study
111	CLL
111	Quantitative & qualitative exposure assessment: mRR=1.14 (0.78, 1.67) 18 studies
61	Quantitative to some industrial hygiene sampling: mRR=1.38 (0.71, 2.69) 8 studies
53	Quantitative or semi-quantitative estimates: mRR=1.54 (0.72, 3.31) 7 studies
43	Quantitative exposure assessment: mRR=2.44 (0.88, 6.75) 4 studies (1 incidence, 3 mortality)
76	CML
76	Quantitative & qualitative exposure assessment: mRR=1.23 (0.93, 1.63) 17 studies
29	Quantitative to some industrial hygiene sampling: mRR=1.44 (0.82, 2.53) 6 studies
29	Quantitative or semi-quantitative estimates: mRR=1.44 (0.82, 2.53) 6 studies
18	Quantitative exposure assessment: mRR=1.68 (0.74, 3.84) 3 studies (2 incidence, 1 mortality)

*Three major cohort studies published after Vlaanderen's (2011**)** analysis examined the incidence of NHL and found higher risks are associated with benzene exposure compared with the earlier meta-analysis* (Linet, 2015; Stenehjem, 2015; Bassig, 2015 – discussed in more detail below.)

Linet (2015): Analyzed a cohort of 73,789 benzene-exposed workers, showing increased risks for a broad range of myeloid and lymphoid neoplasms. The findings include significantly elevated risks for non-Hodgkin lymphoma (RR = 3.9, 95% CI = 1.5, 13) and acute myeloid leukemia (RR = 2.7, 95% CI = 1.2, 6.6)

Stenehjem (2015): Here researchers utilized a semi-quantitative job-exposure matrix (JEM) and reported monotonic exposure-response trends for B-cell NHL linked to exposure duration and cumulative benzene exposure. The study also demonstrated dose-related risk patterns for AML based on cumulative exposure.

Bassig (2015): The study observed non-monotonic exposure-response trends for benzene and NHL. Bassig's methodology was notably rigorous, utilizing both industry and occupational job-exposure matrices (JEMs) calibrated with short-term area air measurements from Shanghai factories. Significantly, women ever exposed to benzene showed a higher risk of developing NHL, with a hazard ratio of 1.87 (95% CI: 1.19, 2.96).

Liu and Wang (2022): This more recent meta-analysis considered 14 articles in its meta-analysis, including 9 cohort studies and 5 case-control studies with NOS scores between 5 and 9. Four of the articles were at low risk of bias, and 10 were at moderate risk of bias. Of the 492,719 people exposed to benzene, 1,994 developed NHL. The OR of NHL in the benzene-exposed population compared with the non-exposed population was 1.23 (P=0.03), with moderate heterogeneity (I2=63.47%). High exposure to

benzene was associated with a higher risk of NHL (OR =1.81), and there was a low degree of heterogeneity (I2=27.56%). The risk of benzene exposure in China (OR =2.48) was higher than that in Europe (OR =1.19), the United Kingdom (OR =1.07), and the United States (OR =1.24). They concluded that exposure to benzene was positively associated with NHL.

Wong and Raabe, 2000: This was one notable negative study was that of over 308,000 petroleum workers. They found 506 deaths due to NHL, with 561.68 expected, with an SMR of 0.90 (95% CI 0.82–0.98). However, the study has problems (Goldstein, 2009). This makes the study unable to address whether benzene can be a cause of lymphoproliferative cancer. Importantly, the overall cohort was not reported to have an increase in AML, however, this is the hallmark cancer caused by benzene. This suggests that the cohort is not representative and would be less likely to detect any cancers associated with benzene. As Goldstein illustrates for example, there is questionable relevance to a study looking at whether cigarette smoking causes NHL when the study's average smoking rates are so low. Hence, at most what can be concluded by Wong and Raabe is that petroleum workers in this study were not at increased risk of NHL as a result of their exposure to benzene. But in the apparent absence of an increase in the risk of AML, one cannot conclude this study demonstrates that benzene exposure cannot cause lymphoma. Further, Goldstein notes that the Wong and Raabe study actually suggests that it supports the relationship between benzene and NHL. Hence, if one looks at their different worker groups within this large study of petroleum workers, it reports what appears to be a statistically significant higher risk of NHL mortality among refinery workers than among distribution workers, who possibly have lower exposure to benzene than do those at the refinery (US refinery workers SMR 0.96; 95% CI 0.86–1.07; non-US refinery workers SMR 1.12; 95% CI 0.90–1.37; global distribution workers SMR 0.64; 95% CI 0.50–0.82).

3. PCE and Hematopoietic Malignancy

The relationship between NHL and PCE exposure has been explored in numerous studies, with mixed results. Some investigations suggest a connection to hematopoietic cancers, including lympho-hematopoietic cancers such as NHL, whereas others have not (**EPA, 2012; ATSDR, 2017; EPA IRIS, 2020**). Among the dry-cleaning worker studies, **Calvert et al. (2011)** and **Selden et al. (2011)** reported elevated NHL risks (SIR 2.05; 95% CI 1.30–3.07) in male workers. Conversely, other studies (**Blair et al., 2003; Morton et al., 2014**) found no significant elevation or risks near the null, while **Lynge et al. (2006)** observed no elevated risk except in workers employed for ≤1 year or with unknown employment duration. **t'Mannetje et al. (2015)** reported an OR of 1.29 (95% CI: 0.74, 2.23) for workers with over 10 years of employment, despite finding no elevated risk for those "ever exposed."

Cohort studies have also contributed evidence regarding PCE exposure. Four cohort studies (**Antilla et al., 1995; Radican et al., 2008; Lipworth et al., 2011; Silver et al., 2014**) and two case-control studies (**Seidler et al., 2007; Christensen et al., 2013**) identified elevated NHL risks. Notably, the **Seidler et al. (2007)** study found increased NHL and B-cell NHL risks only among workers in the 90th percentile of cumulative PCE exposure.

In its review, the **EPA (2012)** concluded that findings from PCE cohort studies and some dry-cleaning worker cohort studies support a link between PCE and NHL. However, the **IARC (2014)** noted inconsistent findings and the small number of exposed cases in many studies, especially mortality studies, given the 70% 5-year survival rate for NHL. The lack of consistent findings may partly result from

non-differential exposure misclassification. ATSDR agrees with the EPA that the positive findings are unlikely to be explained by confounding due to lifestyle factors, as these are not strong risk factors for NHL or typically associated with PCE exposure. ATSDR concludes that the epidemiological evidence, though weak, supports a classification of equipoise for the association between PCE and NHL.

No meta-analyses have evaluated PCE exposure and leukemia. A cohort study of dry-cleaning workers (**Blair et al., 2003**) and a second combining dry-cleaning and laundry workers (**Selden et al., 2011**) found risks near the null. Two case-control studies observed slightly elevated risks for CLL/SLL (**Morton et al., 2014; t'Mannetje et al., 2015**), with Morton reporting an OR of 1.10 (95% CI: 0.15, 8.12) for ALL. Other studies reported mixed results: a cohort mortality study (**Silver et al., 2014**) found risks near the null for all leukemias, and a case-control study (**Talibov et al., 2014**) found no elevated risk for AML at higher cumulative exposures. PCE-contaminated drinking water has also been linked to leukemia. See discussion above regarding **Aschengrau et al. (1993)** and **Cohn et al. (1994)**.

4. VC and Hematopoietic Malignancy

VC has been shown to be carcinogenic in humans through many epidemiologic studies with particular evidence for liver and angiosarcoma (**EPA IRIS, 2000; ATSDR, 2006; IARC, 2012; NTP, 2016; ATSDR, 2017**). To date, there is some suggestion that there could be an association between VC exposure and cancer of the hematopoietic and lymphohematopoietic systems (**Simonato, 1991; Weber, 1981**). The observed increases in other studies fell below statistical significance, most likely because of the small numbers of these types of cancers (**Tabershaw and Gaffey, 1974**). Hence, at this time, there is only suggestive epidemiologic evidence that VC could be a cause of hematopoietic cancers. **Boffetta (2003)** conducted a study of VC with 53 cases and reported an SMR of 0.91 (95% CI: 0.69, 1.21).

E. Water Contamination Studies

Numerous studies illustrate the associations between exposure to TCE, PCE, and elevated risks of cancer, including leukemia. These studies include the following:

Cohn (1994) investigated the association between TCE/PCE exposure and the incidence of both leukemia and NHL. While ecologic studies have inherent design limitations, they can still offer valuable insights at an aggregate level. The ecologic study involving water contamination by TCE and the incidence of NHL provides support for meta-analyses results, particularly in a more closely related exposure scenario than most occupational studies.

A water contamination study conducted in New Jersey in the 1990s examined 75 communities with TCE and PCE in their drinking water (Cohn, 1994). The study found positive associations between TCE and PCE exposure levels—similar to or lower than those at Camp Lejeune—and NHL incidence. The highest assigned TCE level was 67 ppb, the highest assigned PCE level was 14 ppb, and the highest assigned total non-THM VOC level was 92.9 ppb (Cohn, 1994 at 557).

NHL incidence among women was associated with the highest TCE exposure (>5 ppb), with an RR = 1.36 (95% CI: 1.08–1.70). For diffuse large cell NHL and non-Burkitt's high-grade NHL among females, the RRs were 1.66 (95% CI: 1.07–2.59) and 3.17 (95% CI: 1.23–8.18), respectively. Among males, the RRs were 1.59 (95% CI: 1.04–2.43) and 1.92 (95% CI: 0.54–6.81), respectively. PCE was associated with the

incidence of non-Burkitt's high-grade NHL among females, but collinearity with TCE made it difficult to assess their relative influences. The results suggest a link between TCE/PCE and leukemia/NHL incidence.

The Cohn (1994) study also examined the association between TCE/PCE and leukemia. They found "a statistically significant association between leukemia incidence among females and residence in towns in the highest exposure stratum (>20 ppb) of total [volatile organic compounds]" (Cohn, 1994 at 558). This suggests that drinking water containing at least 20 ppb of organic compounds can cause leukemia. The TVOC levels at Camp Lejeune often exceeded 20 ppb, which supports the potential for leukemia in Camp Lejeune.

The study specifically analyzed TCE and PCE exposure. For TCE, females exposed to at least 5 ppb had a 1.43 (95% CI: 1.07–1.90) risk compared to those with lower exposure, while males showed a 1.10 (95% CI: 0.84–1.43) risk. For PCE, females with at least 5 ppb exposure had a 20% increased risk of leukemia (Cohn 1994 at 560). The authors noted that "the carcinogenic activity of TCE and PCE may be compounded by joint exposure because TCE and PCE appear to share toxic metabolic pathways" (Cohn, 1994 at 561).

This New Jersey ecology study is unique, as it specifically analyzed TCE exposure via drinking water. It also explored combined TCE and PCE exposures, both present in Camp Lejeune's Hadnot Point water supply. Importantly, TCE levels in the New Jersey study were often lower than predicted levels at Camp Lejeune. Among all studies reviewed, the New Jersey study most closely aligns with Camp Lejeune's exposure levels, even though New Jersey's water had only two of the four carcinogens present at Camp Lejeune. Interestingly, Frank Bove – one of the New Jersey study authors –later joined the ATSDR and became lead author of the Camp Lejeune epidemiological studies. In fact, Bove et al. (2014) underscored this connection, noting that the maximum TCE levels detected in Camp Lejeune's Hadnot Point drinking water exceeded those observed in the New Jersey towns (Bove et al., 2014b at 11).

Aschengrau (1993) studied cancer risk among individuals exposed to PCE in Cape Cod, Massachusetts, after contamination was found in the local drinking water. Years after the contamination was discovered, the Massachusetts Department of Public Health observed elevated cancer mortality in the Upper Cape Cod area, particularly for leukemia. Specifically, there was a 1.72-fold increase in the crude relative risk of leukemia among exposed individuals (95% CI: 0.50-4.71), which increased to 5.78 (95% CI: 0.98-22.97) for those with exposure above the 90th percentile. After adjusting for confounding factors, the relative risks rose to 1.96 (95% CI: 0.71-5.37) for all exposed individuals and 5.84 (95% CI: 1.37-24.91) for those with the highest cumulative exposure.

Typical PCE concentrations ranged from 1.5–80 µg/L at medium- and high-use sites, reaching up to 1,600–7,750 µg/L at low-use (dead-end) sites. The 1.5–80 µg/L concentrations at medium- and high-use sites were comparable to those found in the Hadnot Point and Tarawa Terrace water systems at Camp Lejeune.

To estimate total PCE intake, researchers developed a measure called Relative Delivered Dose (RDD), with cumulative exposure at the 90th percentile ranging from 27.1 to 44.1 mg. Individuals at Camp Lejeune could have experienced similar exposure levels. The maximum simulated PCE concentration at Camp Lejeune's Tarawa Terrace Water Treatment Plant reached 183 µg/L in March 1984, with levels around 170 µg/L in preceding months. According to ATSDR estimates, "A marine in training at Camp Lejeune consumes an estimated 6 liters of water per day for three days per week and 3 liters per day the

rest of the week" (ATSDR 2017). Using this average water intake of 4.28 liters per day, a marine's daily PCE intake would be approximately 0.7276 mg (0.170 mg/L × 4.28 L). At this rate, cumulative exposure would reach 44.1 mg after just 61 days, placing the marine in the top 10% of cumulative PCE exposure observed in the Aschengrau (1993) study.

Even with lower PCE concentrations, cumulative exposure could still fall within the 90th percentile range observed in Aschengrau (1993). For example, if PCE levels were 40 µg/L (0.040 mg/L), a marine would consume about 0.1712 mg of PCE per day. To reach the lower end of the 90th percentile, 27.1 mg, the marine would need approximately 158 days (about 5.3 months) of exposure; to reach the upper end, 44.1 mg, roughly 257 days (around 8.6 months). This demonstrates that even during periods of lower PCE contamination, extended exposure at Camp Lejeune could result in cumulative levels similar to those in the Aschengrau (1993) study.

The study authors concluded that there was "evidence for an association between PCE-contaminated public drinking water and leukemia" (Aschengrau, 1993 at 291).

Fagliano (1990) was an ecological study to explore the relationship between volatile organic compounds (VOCs), such as TCE and PCE, and leukemia incidence. They grouped towns in New Jersey by VOC contamination levels into three ranges: 37-72 ppb, 5-12 ppb, and <1-3 ppb. These levels are comparable to those observed at Camp Lejeune and, in many cases, are considerably lower than the contamination levels documented there.

The study found that females in towns with high VOC exposure—defined here as 37-72 ppb—had a relative risk of leukemia of 1.53 (95% CI: 1.02-2.21), indicating a statistically significant 53% increase in risk. While the authors noted that "a statement of causal inference cannot be made with confidence," this study provides evidence that VOC exposure levels similar to, or even lower than, those at Camp Lejeune are linked with increased leukemia risk. Specifically, the findings indicate that VOC exposure in the range of 37-72 ppb is sufficient to significantly raise the risk of leukemia.

A series of **Woburn, Massachusetts studies** have examined the cancer cluster following water contamination from industrial activities in the late 1970s and 1980s, primarily from tanning and degreasing operations. The two contaminated wells, G and H, contained 267 ppb of TCE and 21 ppb of PCE (Fagliano, 1986 at 583). The state of Massachusetts noted an "overall cancer mortality rate in Woburn [that] was significantly higher than those of the state and six adjacent communities," with reports of multiple childhood leukemia cases in a small area (Parker and Rosen, 1981 at 2). A 1981 study confirmed a childhood leukemia cluster in Woburn (Costas, 2002 at 1).

Parker and Rosen (1981) found that childhood leukemia incidence was significantly elevated in Woburn, particularly in its eastern section, where 12 cases were observed versus an expected 5.3 (Parker and Rosen, 1981 at 31, 3). These findings suggest that TCE and PCE levels in Woburn—at tens and hundreds of ppb—are sufficient to increase leukemia risk.

Lagakos (1986) confirmed "a positive association between [Well] G and H exposure and the incidence rate of childhood leukemia" (Lagakos, 1986 at 587). Both cumulative and "none-some" exposure to these wells correlated with higher leukemia rates, which declined "after the wells were shut down" (Lagakos, 1986 at 591). Between 1964 and 1983, 20 cases of leukemia were diagnosed, where only 9.1

were expected (Lagakos, 1986 at 591). This supports that TCE and PCE levels in Woburn's water were sufficient to induce leukemia.

Cutler (1986) investigated the connection between environmental hazards and childhood leukemia in Woburn, focusing on the period 1969-79. In one census tract supplied by the contaminated wells, the leukemia rate was "7.5 times the expected number" (Cutler, 1986 at 201). This further indicates that TCE and PCE levels in these wells were linked to elevated leukemia risk.

Lastly, the **Costas (2002)** study followed up on the Woburn cancer cluster, finding that children whose mothers likely consumed water from wells G and H during pregnancy had a significantly higher leukemia risk, with those exposed to the most contaminated water showing a relative risk exceeding 14.0, representing an increase of several thousand percent (Cutler, 2002 at 31). A dose-response relationship was also noted, where greater exposure led to higher leukemia risks (Cutler, 2002 at 30). This study underscores the hazardous impact of TCE and PCE at Woburn's contamination levels, demonstrating that exposure to these chemicals can lead to leukemia.

F. Camp Lejeune Studies

Evidence indicates that TCE, PCE, and benzene are linked to an increased risk of leukemia, and this risk has been demonstrated at chemical concentrations similar to those found at Camp Lejeune. What makes this case unique is the availability of epidemiological studies specifically examining the Camp Lejeune population—something rarely seen in contamination events. Typically, researchers must rely on data from other exposure incidents, but in this case, there is direct data on the specific exposure levels and their associated leukemia risk. The epidemiological studies based on the Camp Lejeune population provide strong evidence that the chemical levels present were sufficient to increase leukemia risk. As to NHL in the Bove studies, particularly the 2024 Cancer incidence study, the data demonstrated that the levels of chemicals present at Camp Lejeune were sufficient to increase the risk of NHL.

It should be noted that Dr. Bove, the author of the Camp Lejeune studies, has testified that there were potentially several limiting factors which could have biased the results of his studies towards the null value. These factors include exposure misclassification (Bove, 2014; Bove, 2018; Bove, 2024); small sample size and/or low participation rate (Bove, 2018); inability to verify outcomes (Bove, 2018); inadequate follow up for blood cancers with early onset (Bove 2024); and inadequate follow up for blood cancers with late onset (Bove, 2024) (See Frank Bove Deposition Transcript vol. 1, pp. 295-297; Frank Bove Deposition Transcript vol. 2, pp. 98-101, 114-117).

The **Bove 2014a Marine Mortality study** was a retrospective cohort mortality study focused on Marine and Navy personnel who began service between 1975 and 1985 and were stationed at either Camp Lejeune or Camp Pendleton, during this period. While Camp Lejeune's drinking water was contaminated with toxic chemicals, Camp Pendleton's water was considered uncontaminated, thus serving as a comparison group. Mortality follow-up extended from 1979 to 2008. Standardized Mortality Ratios (SMRs) were calculated using U.S. mortality rates as the reference, and survival analysis was employed to compare mortality rates between the Camp Lejeune cohort (N = 154,932) and the Camp Pendleton cohort (N = 154,969).

On average, each person in the Camp Lejeune cohort stayed on base for 18 months (Bove, 2014a at 3). The hazard ratio for leukemia among Camp Lejeune marines was 1.11 (0.75-1.62) compared to those at Camp Pendleton, suggesting an 11% higher risk of mortality of leukemia linked to the Camp Lejeune location. These results strongly suggest that the chemical exposure at Camp Lejeune contributes to an elevated risk of leukemia, with no significant differences between the two populations other than the toxic exposure. Notably, marines at Camp Lejeune experienced a higher mortality rate from leukemia, indicating a link between the contaminated water and leukemia risk.

The authors further analyzed leukemia risk based on cumulative chemical exposure levels. Estimated monthly mean contaminant concentrations in the water system serving an individual's residence, along with their occupancy dates, were used to calculate cumulative exposures ("µg/L-months") to each contaminant and the total volume of these contaminants (TVOC). Only the location of residence was factored into cumulative exposure calculations. For instance, if a marine resided at Hadnot Point from January 1976 to March 1976, and the ATSDR data indicate mean TCE concentrations of 227, 317, and 323 µg/L for those months, the cumulative TCE exposure would total 867 µg/L-month. This calculation method was similarly applied for other contaminants and TVOCs.

Cumulative exposure categories for each chemical were as follows (Bove, 2014a at Table 6):

- **TCE**: Low (1-3,100 µg/L-month), Medium (3,100-7,700 µg/L-month), High (7,700-39,745 µg/L-month)
- **PCE**: Low (1-155 µg/L-month), Medium (155-380 µg/L-month), High (380-8,585 µg/L-month)
- **Benzene**: Low (2-45 µg/L-month), Medium (45-110 µg/L-month), High (110-601 µg/L-month)
- **Vinyl Chloride**: Low (1-205 µg/L-month), Medium (205-500 µg/L-month), High (500-2800 µg/L-month)
- **TVOC (Total Volatile Organic Compounds)**: Low (1-4,600 µg/L-month), Medium (4,600-12,250 µg/L-month), High (12,250-64,016 µg/L-month)

The study found that compared to Camp Pendleton personnel, leukemia risk in Camp Lejeune personnel increased (Bove, 2014a Suppl File 1 at Table S-3):

- For TCE exposure at cumulative levels from 1 to 39,745 µg/L-month, there was a 40% increased leukemia risk (RR = 1.40; 95% CI: 0.93-2.12).
- For PCE exposure at cumulative levels from 1 to 8,585 µg/L-month, there was a 37% increased leukemia risk (RR = 1.37; 95% CI: 0.90-2.08).
- For benzene exposure at cumulative levels from 2 to 601 µg/L-month, there was a 47% increased leukemia risk (RR = 1.47; 95% CI: 0.97-2.23).
- For vinyl chloride exposure at cumulative levels from 1 to 2,800 µg/L-month, there was a 37% increased leukemia risk (RR = 1.37; 95% CI: 0.90-2.08).

- For TVOC exposure at cumulative levels from 1 to 12,250 µg/L-month, there was a 45% increased leukemia risk (RR = 1.45; 95% CI: 0.97-2.17).

No elevated risk was observed among those with minimal or no exposure (below 1 µg/L-month of TCE, PCE, or TVOC, or below 2 µg/L-month of benzene). The authors interpreted this finding as showing that the higher leukemia rates among Camp Lejeune personnel were directly tied to cumulative chemical exposures (Bove, 2014a at 9).

The study also examined the risk of leukemia within the Camp Lejeune group by comparing individuals exposed to higher chemical levels with those exposed to lower levels (Bove, 2014a Suppl. Materials at Table S-1).

- For TCE exposure, the low exposure group (1-3,100 µg/L-month) demonstrated the highest leukemia risk, with an approximately two-fold increase (RR = 2.0; 95% CI: 1.0-4.0), with elevated risks also observed at higher exposure levels.

- For benzene exposure, the low exposure group (2-45 µg/L-month) showed the highest leukemia risk, with an estimated two-and-a-half-fold increase (RR = 2.54; 95% CI: 1.27-5.08), with elevated risks also observed at medium and high exposure levels.

- For TVOC exposure, the low exposure group (1-4,600 µg/L-month) exhibited the highest leukemia risk, with an estimated two-and-a-half-fold increase (RR = 2.50; 95% CI: 1.24-5.03), with elevated risks also observed at medium and high exposure levels.

In conclusion, the Bove (2014a) study provides strong evidence linking Camp Lejeune's contaminated water to increased leukemia risk.

One further consideration is that these findings may underestimate the overall risk of leukemia in some cases. Some types of leukemia are more likely to occur from initial exposure to diagnosis after a shorter exposure to contaminants; hence, those leukemia cases would not be included in these studies because they included a 10-year lag between exposure and diagnosis. This would be particularly problematic for comparing more aggressive hematopoietic cancers, such as acute myeloid leukemia and some acute lymphocytic leukemias, that can have a latency period of less than 10 years. Notably, whereas for other hematopoietic cancers the longer latency periods of years to decades are more likely, for some cases a much shorter latency period can be observed, meaning some cases may not have been captured at all by these studies. This would also be true for exposures that occur in utero, to a fetus, and are then associated with early child onset of leukemia, all of which are well known to have a much shorter latency, as short as 1-3 years.

As to NHL, Dr. Bove's Mortality Study involving Marines and Navy personnel did not reveal an increased risk of death due to NHL among the Camp Lejeune cohort (HR = 0.81; 95% CI: 0.56–1.18). That said, as previously mentioned, the same cohort did show elevations in mortality for hematopoietic cancers generally (HR = 1.05; 95% CI: 0.82–1.33), as well as for two B-cell malignancies: multiple myeloma (HR = 1.68; 95% CI: 0.76–3.72) and Hodgkin's lymphoma (HR = 1.47; 95% CI: 0.71–3.06) (see Table 5).

This study suffered from several limitations, including exposure misclassifications (e.g., errors in base assignments, unit locations, family housing, and uncounted time away from the base), which would bias

the hazard ratios toward the null value. Additionally, the fact that most of the cohort was under the age of 55 at the end of the follow-up period would also bias the mortality hazard ratios toward the null.

In my professional opinion, the limitations above—to which Dr. Bove also testified during his deposition—would bias both the NHL hazard ratios and the leukemia hazard ratios toward the null.

The **Bove 2014b Civilian Mortality Study** was a retrospective cohort analysis examining cancer mortality among 4,647 full-time civilian employees at Camp Lejeune from 1973 to 1985 exposed to contaminated drinking water. A comparison group of 4,690 civilian employees stationed at Camp Pendleton, where water was considered uncontaminated, served as a control. Mortality follow-up for both groups extended from 1979 to 2008. Cause-specific standardized mortality ratios were calculated using U.S. mortality rates by age, sex, race, and calendar period as a reference. Survival analysis compared mortality rates between Camp Lejeune and Camp Pendleton workers and assessed cumulative contaminant exposures within the Camp Lejeune cohort. Groundwater contaminant fate, transport, and distribution models provided monthly contaminant level estimates in Camp Lejeune drinking water, with confidence intervals (CIs) indicating precision for effect estimates.

Because "virtually all civilian workers at Camp Lejeune resided off-base," their exposure to contaminated drinking water was considered to have occurred only during work hours on base. This setup makes the study valuable in assessing the effects of even periodic exposure to contaminated water (Bove, 2014b at 2). The median employment duration in the study was approximately 2.5 years (Bove, 2014b at 2).

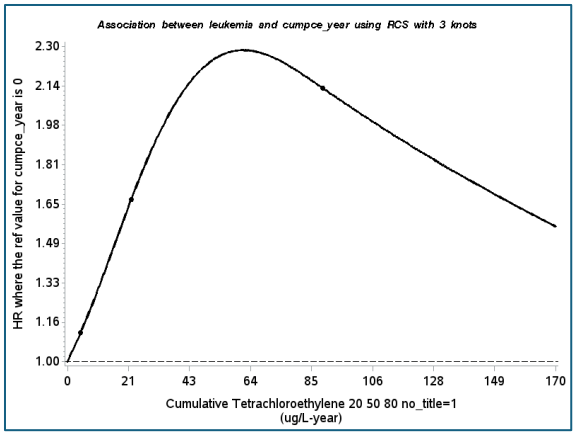
Compared to Camp Pendleton workers, Camp Lejeune civilian workers showed an increased leukemia risk, with a mortality hazard ratio (HR) of 1.59 (95% CI: 0.66-3.84), indicating a 59% higher risk of leukemia. This finding suggests that contaminant concentrations at Camp Lejeune were sufficient to elevate leukemia risk, even for workers residing off-base and exposed intermittently.

Within the Camp Lejeune cohort, monotonic exposure-response relationships were observed for leukemia, associated with cumulative exposure to vinyl chloride and PCE. The following hazard ratios further highlight elevated leukemia risk in the high exposure categories:

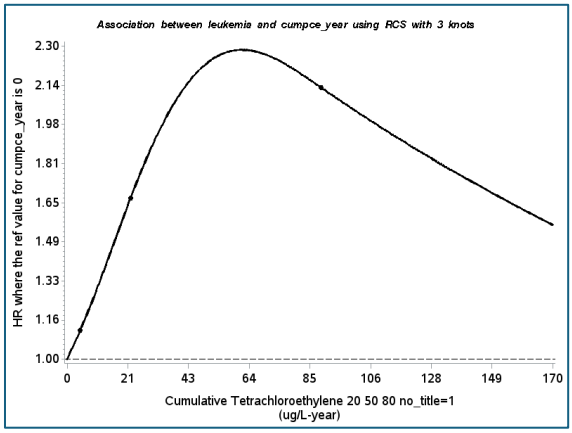
- Vinyl Chloride: HR = 1.72 (95% CI: 0.33-8.83)
- PCE: HR = 1.82 (95% CI: 0.36-9.32)

The study also examined cumulative exposure across Camp Lejeune personnel, finding a clear exposure-response trend for leukemia as PCE and vinyl chloride exposure levels increased [Bove 2014b at 9]. Additionally, the authors used "splines"—graphical models that display relative leukemia risk in relation to cumulative PCE and vinyl chloride exposure.

The splines illustrated that leukemia rates begin to rise at approximately 5 µg/L-years of PCE exposure, with relative risk reaching 1.65 at 21 µg/L-years. By around 60 µg/L-years, the relative risk exceeded 2.0, underscoring a strong exposure-response relationship.



Similar trends were observed for vinyl chloride:



As to NHL, some of the exposure misclassification issues outlined in the military study were also present in this civilian study. Although the average age of the civilian cohort (most under the age of 65) was approximately 10 years older than that of the military personnel study, only 14% of the civilian population had died by the end of the study.

The study revealed decreased mortality from NHL at Camp Lejeune (HR = 0.83; 95% CI: 0.26–2.67). However, the p-value of 0.76 suggests that the hazard ratio for NHL is very likely due to chance. Comparatively, the p-value for leukemia was 0.30, indicating that the hazard ratio for leukemia is less likely to be due to chance. In fact, the p-value for NHL was the highest among all cancers studied, except for colon cancer, which had a p-value of 0.98 (see Table 4).

ATSDR (2018) Morbidity Study included 214,970 Marines and Navy personnel stationed at Camp Lejeune between April 1975 and December 1985, along with a sample of 50,000 Marines from Camp Pendleton who were not stationed at Camp Lejeune during this period. It also included 8,085 civilians employed at Camp Lejeune from October 1972 to December 1985, and 7,236 civilians employed at Camp Pendleton during the same timeframe who had no employment history at Camp Lejeune. The 2018 morbidity study, conducted by ATSDR, aimed to assess the potential health impacts of

contaminated drinking water at Camp Lejeune. Data from this survey supported the morbidity study to evaluate links between Camp Lejeune water contamination and specific diseases, including leukemia.

The relative risk for leukemia in civilian employees at Camp Lejeune versus those at Camp Pendleton was 1.10, indicating a 10% increase in risk. These findings suggest that exposure to contaminated water at Camp Lejeune between 1972 and 1985 could increase leukemia risk. The authors further analyzed leukemia risk by comparing Camp Pendleton civilians to Camp Lejeune Marines with varying levels of chemical exposure, using "water distribution models" and "residential locations and durations" to estimate cumulative and average exposure to each contaminant.

For civilians with medium combined exposure to TCE/PCE (between 10,868 and 50,563 ppb-months of TCE or 457 and 2,118 ppb-months of PCE), the relative risk was 1.41, indicating a 41% higher risk of leukemia compared to Camp Pendleton civilians. In those with high TCE/PCE exposure, the relative risk increased to 1.32, indicating a 32% greater risk of leukemia.

Finally, the study examined leukemia rates among Camp Lejeune personnel exposed to higher versus lower levels of TCE and PCE. Civilians with medium exposure (10,868–50,563 ppb-months of TCE or 457–2,118 ppb-months of PCE) had a relative risk of 1.69, reflecting a 69% increase in leukemia risk. Those with even higher exposure had a relative risk of 1.58, representing a 58% increased risk. These findings indicate that cumulative exposure to at least 457 ppb-months of PCE or 10,868 ppb-months of TCE is sufficient to elevate leukemia risk among those exposed at Camp Lejeune.

In this survey, NHL was grouped with Hodgkin's lymphoma and "unspecified" lymphoma as a single category due to the inability to confirm the specific type of lymphoma using the medical confirmation process employed. For lymphomas, Camp Lejeune Marines had a 6% higher risk compared to Camp Pendleton Marines (HR = 1.06; 95% CI: 0.75–1.50). Civilians at Camp Lejeune, however, had a 28% increased risk of lymphomas compared to civilians at Camp Pendleton (HR = 1.28; 95% CI: 0.59–2.79). Civilians with high workplace exposures had an even greater risk for lymphoma (HR = 1.71; 95% CI: 0.46–6.39).

While the survey does have limitations, as outlined by Dr. Bove in his deposition, the overall findings are consistent with elevated hazard ratios observed in a New Jersey study involving water contaminated with TCE and PCE (Cohn, 1994).

Bove 2024b Cancer Incidence Study was conducted on Marines, Navy personnel, and civilian workers stationed at Camp Lejeune (154,821 service members; 6,494 civilians) and Camp Pendleton (163,484 service members; 5,797 civilians) between 1975 and 1985. Unlike Camp Lejeune, Camp Pendleton's water was not considered contaminated. Researchers collected data on primary cancers cancer cases diagnosed from 1996 to 2017 across 54 US cancer registries. Using proportional hazards regression, they calculated adjusted hazard ratios (aHRs) to compare cancer incidence between the two groups, adjusting for factors like sex, race, education, rank, and job type, with age as the time variable. The precision of these aHRs was assessed using the 95% confidence interval ratio (CIR).

The study conducted a detailed analysis of cancer risk based on total chemical exposure at Camp Lejeune, comparing it with Camp Pendleton. To assess cumulative exposure, "duration of assignment" was used as a surrogate for Marines and Navy personnel, while "duration of employment" was used for civilian workers. For military personnel, duration at Camp Lejeune between April 1975 and December

1985 was categorized into low (1–6 quarters), medium (7–10 quarters), and high (>10 quarters) exposure levels. Civilian employment at Camp Lejeune from October 1972 to December 1985 was classified as low/medium (1–21 quarters) and high (>21 quarters) exposure.

The study conducted a detailed analysis of cancer risk based on total chemical exposure at Camp Lejeune, comparing it with Camp Pendleton. To assess cumulative exposure, "duration of assignment" was used as a surrogate for Marines and Navy personnel, while "duration of employment" was used for civilian workers. For military personnel, duration at Camp Lejeune between April 1975 and December 1985 was categorized into low (1–6 quarters), medium (7–10 quarters), and high (>10 quarters) exposure levels. Civilian employment at Camp Lejeune from October 1972 to December 1985 was classified as low/medium (1–21 quarters) and high (>21 quarters) exposure.

In comparisons between Camp Lejeune and Camp Pendleton, Marine and Navy personnel at Camp Lejeune showed an adjusted hazard ratio (aHR 1.38; 95% CI, 1.03–1.85) for acute myeloid leukemia (AML), indicating a 38% increased risk of AML compared to personnel at Camp Pendleton. For leukemia in general, the aHR was 1.07 (95% CI, 0.91–1.25), suggesting a 7% increased risk for leukemia overall. (2024 CIS at Table 3).

Among civilian workers, the aHR for AML was 1.35 (95% CI, 0.59–3.09) when compared to Camp Pendleton civilians (2024 CIS at Table 4), indicating a 35% increased risk of AML.

For Marines with low-duration exposure (1–6 quarters on base), the relative risk of leukemia was 1.11 (95% CI, 0.92–1.33), indicating a 11% increased risk of leukemia. For AML in the same group, the aHR was 1.36 (95% CI, 0.97–1.90), suggesting a 36% increased risk for AML. (2024 CIS at Table 5). These findings suggest that spending even 1–6 quarters on base between 1975 and 1985—or being exposed to the chemical levels typical of that timeframe—is associated with an elevated risk of leukemia.

In the high-exposure Marine group, the aHR for leukemia was 1.15 (95% CI, 0.86–1.55), suggesting a 15% increased risk of leukemia, while for AML, the aHR was 1.90 (95% CI, 1.12–3.21), indicating a 90% increased risk of AML for Marines with high-duration exposure. (2024 CIS at Table 5).

For civilian workers with high-duration exposure, the study identified the most elevated risks, with an aHR of 1.43 (95% CI, 0.72–2.86) for leukemia, indicating a 43% increased risk of leukemia. For AML, the aHR was 2.53 (95% CI, 0.76–8.37), indicating a 153% increased risk, and for chronic myeloid leukemia (CML), the aHR was 1.61 (95% CI, 0.29–8.91), or a 61% increased risk. (2024 CIS at Table 6).

For NHL, the study related to Marines demonstrated that exposure to the water at Camp Lejeune created moderately to significantly increased risks for several subtypes of NHL all of which originate in the B cells. These include Mantle Cell Lymphoma, which showed an aHR of 1.26 (95% CI, .73-2.19); Burkitt's Lymphoma, which showed an aHR of 1.53 (95% CI, .71-3.3); Marginal Zone B-Cell, which showed an aHR of 1.45 (95% CI, .92-2.28). CLL revealed a positive association with an aHR of 1.02 (95% CI, .79-1.32), while Follicular lymphoma had an aHR of 1.07 (95% CI, .84-1.36). (2024 CIS at Table 3).

Civilians exposed to the water at Camp Lejeune also demonstrated an increase in certain subtypes of B cell lymphomas including Follicular lymphoma, which had an aHR of 1.41 (.63-3.17) and Diffuse Large B-Cell, with an aHR of 1.48 (95% CI, .81-2.70). (2024 CIS at Table 4).

Among civilian workers, the study demonstrated a 19% risk of NHL based upon an aHR of 1.19 (95% CI, .83-1.71). (2024 CIS at Table 4). Overall NHL risk, without subtypes, was only slightly elevated for Marines at an aHR of 1.01 (95% CI, .90-1.14). (2024 CIS at Table 3).

Overall, the 2024 CIS is one piece of a larger epidemiological review which suggests that exposure to TCE and/or Benzene creates an increased risk of hemopoietic cancers of several types. The 2024 CIS also points towards increased cancer risks at levels of exposure that are measured in the parts per billion range. The 2024 CIS is one more study which strongly suggests that even at low levels, TCE and Benzene target B cells in the immune system. B Cells play an important role in cancer immunity and disease prevention, such that a depletion of B cells increases risks for various types of cancer and other diseases.

The **Bove 2024b Mortality Study** was conducted of Marines/Navy personnel who began service between 1975 and 1985 and were stationed at Camp Lejeune (N=159,128) or MCB Camp Pendleton, California (N=168,406), as well as civilian workers employed at Camp Lejeune (N=7,332) or Camp Pendleton (N=6,677) between October 1972 and December 1985. Camp Pendleton's drinking water was not considered contaminated with industrial solvents. Mortality follow-up occurred between 1979 and 2018. Proportional hazards regression was used to calculate adjusted hazard ratios (aHRs) comparing mortality rates between Camp Lejeune and Camp Pendleton cohorts.

The study showed that Navy/Marine personnel at Camp Lejeune had a hazard ratio of 1.13 (95% CI: 0.89–1.43) compared to personnel at Camp Pendleton, indicating that those exposed at to the water at Camp Lejeune had a 13% higher risk of death from leukemia than their military counterparts at Camp Pendleton. For AML specifically, the risk increased to 1.21 (95% CI: 0.94–1.56). (Suppl. Table 3). For CML, the risk increased to 1.73 (95% CI: 0.47–6.42). (Suppl. Table 7). This study demonstrates that the chemical levels present at Camp Lejeune were sufficient to be a cause of leukemia.

The study authors also evaluated leukemia risk based on exposure, as measured by time spent on base. For Marines/Navy personnel, they found an increased risk even at low duration, i.e., 1–2 quarters on base—RR=1.17 (95% CI: 0.84–1.64). (Bove, 2024 Suppl. Table S6). At medium duration—2–7 quarters on base—the RR increased to 1.19 (95% CI: 0.86–1.64). These results demonstrate that 1–2 quarters on base during 1972–1985 were sufficient to cause leukemia. The same applies to individuals who were present on base at different times but who received a cumulative dose equivalent to spending that duration on base during those years.

Rosenfeld 2023 employed cancer-slope factors and other health-assessment methodologies to assess cancer risks for Marines stationed at Camp Lejeune from 1953 to 1987. According to Rosenfeld et al. (2023 at 10), even a single month of work on the base between 1980 and 1984 resulted in a risk greater than the 1 in a million de minimis threshold (Rosenfeld, 2023 at 10). For individuals who remained on base for six months, the estimated cancer risk increased up to six times compared to the one-month exposure scenario (Rosenfeld, 2023 at 11). The authors concluded that Camp Lejeune's water contamination was substantial enough to pose a significant health threat to Marines living and working there (Rosenfeld, 2023 at 13).

Although the limitations of cancer-slope calculations are acknowledged, the broader epidemiological evidence suggests that the risks were substantial enough to be detectable, even with coarse-grained analyses. As such, the exact numerical increases in risk provided by these assessments should not be interpreted as definitive. However, the Rosenfeld (2023) study aligns with existing literature in affirming

that as little as one month of exposure could create a meaningful risk of developing hematopoietic cancers.

G. Mechanistic and Preclinical Evidence of Carcinogenesis

1. TCE

The evidence that TCE is toxic and can be a cause of cancer is considerable (**IARC, 2014; NTP, 2015; Lash, 2024; Zhu, 2024**). A risk evaluation by the EPA as a result of the Toxic Substances Control Act in 2022 led to the conclusion that TCE poses an unreasonable health risk to human health resulting in the proposal to eliminate the use of TCE (**EPA Federal Register, 2023**). There is extensive mechanistic, preclinical, and epidemiological literature (described above) on the role of TCE in human carcinogenesis.

Mechanistic data demonstrates that exposure to TCE and its metabolites contributes to carcinogenesis through both genotoxic and nongenotoxic mechanisms, genetic and epigenetic changes, and alterations in the tumor microenvironment and host immune system (**Bassig, 2013; Bassig, 2016; Lash, 2024; Zhu, 2024**). Specifically, exposure to TCE and its metabolites can cause DNA damage, mutagenesis, oxidative stress, epigenetic modifications, changes in the tumor microenvironment, and immunotoxicity.

In vitro, TCE metabolites are also genotoxic, clastogenic and/or mutagenic in vitro (IARC, 2014) as seen in bacteria (**Nestman, 1980**) and human lymphocytes (**Varshney, 2013**) and induce DNA breaks in human HepG2 (**Zhang, 2012**).

In vivo in animals, TCE is associated with micronucleus induction in bone marrow but not peripheral erythrocytes after a single inhalation exposure (**Kligerman et al., 1994**). In the mouse, some other studies found micronucleus induction in the bone-marrow erythrocytes (**Duprat and Gradiski, 1980; Hrelia et al., 1994; Kligerman et al., 1994; Shelby et al., 1993**). TCE metabolites also elicit genotoxicity **in vivo** (**Bhunya, 1987**) and in some studies DNA breaks (**Hassoun, 2010; Nelson, 1988; Nelson, 1989**).

In vivo in humans, statistically significant increases in chromosomal aberrations but not SCE was found among 15 metal degreasers exposed to trichloroethylene for >20 hours per week (**Rasmussen, 1988**). SCE was seen in 6 exposed workers; no increase was reported in a larger study of TCE exposed workers in Japan (**Nagaya et al., 1989**).

Animal studies further support TCE's role as a carcinogen through its effects on the immune system causing autoimmunity (**Chiu, 2013**). Autoimmunity is known to be a risk factor for lympho-hematopoietic cancers including multiple myeloma (**Brown, 2008; McShane, 2014**) and non-Hodgkin lymphoma (**Onishi, 2013; Zhang, 2013**) through multiple mechanisms (**McShane, 2014**).

More recently, TCE has been shown to influence DNA methylation in humans who were exposed—even at low exposure levels and in a dose-dependent manner—in genes associated with autoimmunity and cancer (**Phillips, 2019**).

Preclinical studies confirm that TCE can contribute to carcinogenesis in animal bioassays. An increased incidence of lymphoma (female mice) and leukemia (female rats) was reported in experimental animals exposed to TCE (**NTP, 1990, 2011**).

Consideration of mechanistic and preclinical data along with the epidemiological data further confirms that TCE exposure can cause cancer (including blood cancers), including evidence from cohort, case–control, and ecological studies of environmental exposures (see below). These study designs, which cover exposure by both dermal and inhalation routes in a variety of settings, complement one another in terms of their strengths and limitations.

2. PCE

PCE exposure is toxic and can be carcinogenic with evidence based upon mechanistic and preclinical as well as epidemiologic studies. **ATSDR (2017), EPA (2020).** The EPA considers PCE to be "likely to be carcinogenic to humans by all routes of exposure." The **2017 ATSDR assessment**, based on a meta-analysis conducted by **Vlaanderen et al. (2014)**, indicates that PCE exposure can increase the risk of bladder cancer. The **EPA Risk Assessment (2020)** has determined circumstances where PCE is a reasonable versus unreasonable risk, and this includes the consumer use of PCE is considered an unreasonable risk (**EPA, 2020** at 42-44). Mechanistic studies suggest that PCE could contribute to carcinogenesis through multiple mechanisms including immunotoxic, hematotoxic and genotoxic based upon examination of in vitro, animal and/or human studies.

Animal studies have shown that PCE is immunotoxic and may elicit autoimmunity. PCE in animal studies can exacerbate autoimmunity (**Wang, 2017**), reduce immune cell B-, T and NK- cell activity (**Schlichtling, 1992**) and increase death when exposed to bacteria (**Aranyi, 1986**) but no effects were seen in other studies (**Boverhof, 2013**). PCE can cause general effects on hematopoiesis, anemia and/or bone marrow suppression (**Marth, 1985; Seidel, 1992; Ebrahim, 2001**), but this is not seen in all studies (**Boverhof, 2013**).

In human studies, PCE also has been associated with hematologic toxicity and immune toxicity. PCE can cause hematotoxicity in humans with reduced RBC and hemoglobin (**Emara, 2010**). PCE exposure has been associated with autoimmunity including Sjorgren's syndrome, rheumatoid arthritis, connective tissue disease, systemic sclerosis and anti-neutrophil antibody (ANCA) related disease (**Chaigne, 2015; Li, 2008; Beaudreuil, 2005; Garabrant, 2003; Lacey, 1999; Goldman, 1996; Lundberg, 1994**). More recent studies have shown that women who work at dry cleaners and are exposed to PCE have markers of oxidative stress and pro-inflammatory cytokines in their blood (**Changhao, 2024**).

Some studies have identified increased relative risks with PCE exposure up to 4.07 for autoimmune diseases. Two of these studies are statistically significant, with an overall risk of 2.64 for Sjogren's syndrome associated with semi-quantitative measures of PCE exposure (**Chaigne et al., 2015**) and increased frequency of sclerosis with self-reported PCE exposure and work in the dry cleaning industry ($p < 0.001$) (**Goldman, 1996**). A meta-analysis of 14 case-control studies (6 with TCE and/or PCE exposure analysis) identified an overall risk 2.03, but it was not statistically significant. An overall risk of 2.0 for ANCA-related disease has been found but that was not statistically significant (**Beaudreuil, 2005**). Finally, there are also case reports of PCE exposure being associated with scleroderma (**EPA, 2012**).

PCE metabolites have been shown to be genotoxic in multiple studies in vitro and in vivo in animal and human studies (IARC, 2014; ATSDR, 2019; EPA, 2020). PCE itself is not likely genotoxic or mutagenic but its oxidative and conjugated metabolites appear to be both. Notably, PCE after glutathione conjugation is mutagenic in vitro such as in the Ames assay of TCVG after metabolic activation (**Vamvakas, 1989;**

Dreessen, 2003). Oxidative metabolites of PCE are mutagenic in some assays but not others (**EPA, 2012**). PCE metabolites are also mutagenic in log phase in S. typhimurium (**Demarini, 1994; Reichert, 1983**) and can elicit in log phase growing S. cerevisiae gene conversion, mitotic recombination and reverse mutation (**Koch, 1988; Bronzetti, 1983**) and aneuploidy (**Koch, 1988**). PCE alone does not cause DNA damage (**Deferme, 2015**).

Preclinical animal studies show some evidence that PCE is carcinogenic (**ATSDR, 2017; EPA, 2020**). PCE has been associated with inducing hematopoietic tumors. Mononuclear Cell Leukemia (MCL) was observed in male and female F344/N rats exposed to PCE by inhalation at concentrations up to 400 ppm for 103 weeks, with decreased time to onset in exposed females (**NTP, 1986a; Jisa, 1993**). One limitation of these studies was the high background (36-56%, **NTP, 1986**; 20-22%, **Jisa, 1993**).

Integration of mechanistic, preclinical and epidemiologic evidence supports that PCE is a human carcinogen.

3. Benzene

Benzene is toxic and a carcinogen based upon integrated examination of mechanistic, preclinical and epidemiologic data. Benzene elicits hazards to the health of those exposed even at low doses of exposure (**Cao, 2023; Wang, 2023**). Benzene has been shown in mechanistic, preclinical and epidemiological studies to be a cause of cancer.

Benzene is easily absorbed, widely distributed throughout the body, and extensively metabolized, leading to the production of reactive electrophiles through multiple metabolic pathways, including those in the bone marrow (**Linet, 2020; Loomis, 2017**). In humans exposed to benzene, its metabolic activation results in oxidative stress, epigenetic alterations (**Fenga, 2016**), cytotoxicity (**Mozzoni, 2023**), gene mutations (**Zhang, 2016**), telomere length changes (**Ren, 2020**), and mitochondria copy number changes (**Ji, 2021**). Benzene is both genotoxic and immunosuppressive (**McHale, 2011**) both contributing to hematotoxicity (**Sun, 2018**) and combined with other mechanisms can contribute to carcinogenesis including resulting hematopoietic tumorigenesis (**ATSDR, 2017**) including leukemia (**Au, 2010; Loomis, 2017; Yi, 2020**) and lymphoma (**Loomis, 2017; Wang, 2023; Bassig, 2024**).

In experimental studies, benzene induces genomic instability including inhibiting topoisomerase II, modulating receptor-mediated effects related to the aryl hydrocarbon receptor, and triggering apoptosis. In humans, benzene forms epoxide-protein and benzoquinone-protein adducts in the blood, contributing to its toxic effects (**Wang, 2023**). In the bone marrow of experimental animals exposed to benzene, evidence points to the formation of DNA adducts, chromosomal aberrations, and micronuclei. Studies performed in in vitro systems using human and animal cells also show that benzene and its metabolites induce DNA adducts, DNA damage, and chromosomal aberrations (**Farris, 1996; Farris, 1997; Hirabayashi, 2004; Recio, 2005**).

In studies of occupationally exposed humans, benzene has been shown to induce various forms of oxidative DNA damage, including DNA strand breaks, gene mutations, chromosomal aberrations, and the formation of micronuclei (**Vigliani, 1969; Forni, 1971; Forni, 1996**). Specific cytogenetic changes observed in humans include aneuploidy, chromosomal translocations, and other structural chromosome alterations (**Zhang, 1996; Zhang, 2002**).

Many preclinical studies support that in animal bioassays benzene is carcinogenic. Further preclinical studies by the NTP and others demonstrate that benzene is a carcinogen in long term bioassays, confirming dose-response relationship and association with the cause of many types of cancer including hematopoietic and lympho-hematopoietic cancers (**Snyder, 1980; Cronkite, 1986; Huff, 1989; Maltoni, 1989; Farris, 1993; NTP, 2007; Kawasaki, 2009**).

Based upon integrated analysis of mechanistic, preclinical studies and epidemiologic studies (see above) benzene is a carcinogen and can be a cause of many types of cancer including hematopoietic and lympho-hematopoietic cancers (**Goldstein, 2010**).

4. Vinyl Chloride

Vinyl chloride (VC) is toxic, and a carcinogen based upon integrated consideration of mechanistic, preclinical and epidemiologic evidence (**EPA IRIS, 2000; ATSDR, 2006; IARC, 2012; NTP, 2016; ATSDR, 2017**). The NTP describes VC as "known to be a human carcinogen." The NCI as of June 13, 2024, describes: "Vinyl chloride exposure is associated with an increased risk of a rare form of liver cancer (hepatic angiosarcoma), as well as primary liver cancer (hepatocellular carcinoma), brain and lung cancers, lymphoma, and leukemia." The EPA is considering VC amongst 5 chemicals as an unreasonable hazard (**EPA, July 24, 2024**). VC is metabolized by cytochrome P450 enzymes, primarily in the liver, to form chloroethylene oxide (CEO), which can undergo spontaneous rearrangement to produce chloroacetaldehyde (CA). Both of these metabolites are highly reactive and can chemically bind to DNA, leading to genetic damage. A major DNA adduct, 7-(2'-oxoethyl)guanine, has been identified along with four minor adducts, known as etheno adducts. These etheno adducts are particularly harmful, as they can cause mutations, primarily base pair substitutions but also frameshift mutations.

VC is genotoxic and mutagenic based upon studies in vitro and in vivo in animals and humans. VC caused genetic damage in bacteria, yeast, insects, cultured human and other mammalian cells, and rodents exposed in vivo, and in exposed humans (**IRIS, 2000**). The genetic damage included mutations, DNA damage, micronucleus formation, chromosomal aberrations, and sister chromatid exchange. VC causes mutations in bacteria with or without metabolic activation (addition of rodent liver microsomes to simulate mammalian metabolism) (**Bartsch, 1975; Rannug, 1974; Walles, 1988**); however, its metabolites CEO and CO are more potent mutagens. Hence, VC likely requires metabolic activation in order to cause genetic damage (**Giri, 1995**). VC forms DNA adducts that include etheno-DNA adducts that may be responsible for its genotoxic effects (**Swenberg, 1992**) because they have been associated with mismatches (**Cheng, 1991**), but this remains to be identified. Mutations caused by VC exposure have been detected in critical genes involved in cancer development, such as the p53 tumor-suppressor gene and the ras oncogene. These mutations commonly occur at base pairs (**Kielhorn et al., 2000**) and can occur in a dose-dependent manner (**Smith, 1988**).

VC is genotoxic in humans. Thus, in a study of 52 nonsmokers exposed to VC and 41 nonsmoking controls those exposed to 1.3-16.7 ppm (high-exposure group) and 0.3-7.3 ppm (low-exposure group), with an average duration of 17 years were observed to have increased SCEs and micronuclei, and this increase was correlated with exposure levels (**Sinues, 1991**). In another study, it was found that there were increased chromosome aberrations in peripheral lymphocytes that correlated with exposure duration in a cohort of 57 VC workers compared with 19 on-site controls and 5 off-site controls, with an average exposure was 5 ppm; but, excursions up to 1,000 ppm were reported (**Purchase, 1978**). VC workers

exposed to 25 ppm were found to have chromosome aberrations in lymphocytes which were elevated relative to controls at the initial sampling but not after exposure ceased (**Hansteen, 1978**).

VC has been found to be carcinogenic in preclinical animal studies (**NTP, 2000**). Multiple studies have reported increased incidence of many tumor types including liver, lung, and breast tumors (Suzuki, 1978; Maltoni, 1980; Maltoni, 1981; Hong, 1981; Suzuki, 1983; Maltoni, 1984).

These mechanistic and preclinical, as well as epidemiological studies (see above) strongly support the conclusion that VC is a carcinogen in humans.

V. Considerations of Exposure and Duration

As further described above, there are many considerations in evaluating chemical exposure and the risk of cancer, briefly, the concentration and route of exposure, the duration of exposure, intensity of exposure, and combination of exposures. Also, the host context must be considered (ATSDR, 2017), including the age, comorbidities, social and family history, susceptibilities and comorbidities. In general, each individual has a different susceptibility to carcinogenesis that is based upon individual factors including: genetic, social and biologic parameters (age) and hence exhibit what scientists describe as a heterogeneity in response. As a result, because there is also an individualized difference in a risk to exposure to a carcinogen, a general estimation of risk of a population only approximates the risk for any given individual that can be the same, higher or lower than the predicted population-based risk. Further, an individual who has been diagnosed with a medical condition such as cancer *has* the cancer, and so, by definition, that person's risk is now a 100%; estimates post hoc of what was the individual's risk can only give an approximation of his/her risk prior to disease. Individuals who have a disease such as cancer, have the disease, and considerations of potential contributing causes are necessarily different from predicting whether an individual may get a disease subsequently. Evaluating whether one would not have gotten a disease if he/she had not been exposed to a particular carcinogen is similarly scientifically illogical, because no expert can predict what could have happened, and what might happen, such as other risk factors and exposures. What one can evaluate is whether a particular contributing risk factor was more or less likely to have a causal relationship.

Importantly, even what may be perceived as very low-level environmental exposures to carcinogens, and in particular combinations of carcinogens, can be an increased risk of cancer. The most sensitive methodologies that exist have found that even at the lowest doses measurable for the limitations of each method, there is evidence for mutagenesis. First, genetic damage does not necessarily, standing alone, lead to cancer in a given individual, but it is the antecedent of cancer and is a common driving mechanism by which an agent can be a contributing cause of cancer. In any event, and as demonstrated above, setting this minimum threshold is not necessary where, as here, the data supports that the levels of contaminants at Camp Lejeune were sufficient to cause cancer. Second, assays that estimate the relationship between an amount of exposure and the risk of cancer are only capable of making such a distinction between this dosage and exposure within the limits of the size of the study and its ability to detect such a relationship, this is referred to statistically as the power of the study. Also, there are limitations to our current methodologies are not sensitive enough to measure the genetic changes that we know are occurring based upon our understanding mechanisms, and hence there are changes we may not be able to see or detect but we know are indeed contributing to the cause of cancer. Thus, it is simply impossible to sample the DNA of every cell in any human for a genetic lesion that can contribute

to the cause of a cancer, but we know that cancer arises from a single cell that clonally evolves to a clinically measurable cancer that at the time of diagnosis may take years or decades to become billions to more typically trillions of cancer cells. Moreover, and as discussed throughout this report, any population-based studies, including ecology studies and epidemiological studies, are generally not sufficiently powered to provide, with reliable confidence, such as through a confidence interval, a quantitative level at which a particular chemical is capable of causing a disease such as cancer. Hence studies that find a relative risk with the 95% confidence interval overlapping 1 are appropriately deemed not statistically significant. However, this lack of statistical significance is often erroneously considered evidence of no risk. That is not the case; one can only say that when one considers other factors to estimate the extent to which the study is able to identify a level of risk. For example, a study may be not statistically significant, with a confidence interval overlapping 1, but if the study is not powered enough to detect amount of increase in cancer risk that would be considered medically capable of identifying a clinically important consequence. Another way to describe this is that a study has to be powered such that it can provide a statistically significant increase over what is medically and scientifically considered the relevant effect size (Stuart, NEJM, 2016). This is particularly a limitation when one subdivides types of diseases such as cancer into very specific subtypes like the various subtypes of hematopoietic cancers. When this is done, the number of test cases is reduced, and the power to detect a meaningful association is further reduced. This is further complicated by the fact that ecological studies do not have exposure data and multiple other potential confounds. Epidemiological studies for carcinogens are more often than not worker studies that have limitations including the healthy worker effect, individuals more susceptible can leave employment, exposures may be above a level that there is a "plateau effect", the exposure duration can be limited, and follow up limited so there is not time for the disease to occur or mortality points to be reached, and the population doesn't include groups that are highest at-risk, among other problems.

In consideration of these caveats, it is notable that some studies address these concerns by performing intergroup analysis that reduces the healthy worker effect, including a lag time to enable a chance for disease occurrence and mortality endpoints. Further statistical analyses, such as Cox analysis and modeling of data such as consideration of exposure as a continuous variable, can be done. When such considerations are performed, for many carcinogens such as benzene, it has been found that there has yet to be measured a level of exposure where there is no risk. Risk at lower levels of exposure -- particularly the more typical environmental exposures as opposed to worker levels of exposure -- is relatively "supralinear." This can lead to incorrect conclusions because at a very high level of exposure you do not see additional risk of cancer that the agent may be carcinogenic. Even regarding the most potent carcinogens, there is never the expectation that in every person exposure will always or even more often than not lead to cancer. But their exposure remains a known risk of cancer, and for those who are diagnosed with cancer, as stated above, the risk is now 100%.

With the above-described considerations in mind, I examined for the Camp Lejeune associated VOCs the efforts made by the EPA to quantify the risk of cancer through exposure through UREs that are reported. Further, for studies that have defined mechanistic influences of Camp Lejeune VOCs, there are descriptions for the exposures at which genotoxic and other carcinogenic outcomes were observed. For all the preclinical studies, the dosages have been described where carcinogenic effects have been described and this can give some general notion of the amount of exposure associated with the cause of cancer, within the limitations of these animal bioassays that involve small numbers of animals observed for relatively shorter amounts of time. Historically, as methods have become more sensitive, our ability

to detect carcinogenic effects has improved. Hence, using a flow cytometry based PIGa assay (Cao, 2023), it has been shown that inhalation of benzene can cause mutagenesis in lymphocytes of the peripheral blood of exposed humans that can be measured at exposures at 70 PPB and presumably even lower. (Cao, 2023).

Further, one must consider what duration of exposure is sufficient to contribute to carcinogenesis. In general, duration of exposure is more important than the concentration of exposure in consideration of dosage, as I have described already (Peto, 1991; Flanders, 2003; Frank, 2007; Korchevskiy and Korchevskiy, 2022). ATSDR (2017) considered in detail what would be the minimal duration of exposure that would be sufficient to increase the risk of cancer. I agree with the analysis of the ATSDR (2017). They describe that they considered this and based upon their review concluded that in many cases 1-3 months of exposure was sufficient to increase the risk of cancer. This finding is consistent with the elevated levels of TCE, PCE, benzene and vinyl chloride in Camp Lejeune's drinking water systems, which far exceeded their respective MCLs.

The ATSDR Public Health Assessment (PHA, 2017) provided a summary of the MCLs for these contaminants:

- **TCE**: 5 ppb
- **PCE**: 5 ppb
- **Vinyl chloride**: 2 ppb
- **Benzene**: 5 ppb

These limits were established in 1989 for TCE, vinyl chloride, and benzene, while the MCL for PCE was introduced in 1992.

As another way of illustrating the potency of carcinogenesis, I point out that for VOCs at a concentration of even 1 PPB or 1PPT this would involve an individual breathing in through a single breath on average over around 30 trillion or 30 billion respectively, molecules (there are around 30 trillion-billion, or 10x10^23, molecules inhaled per breath). Recognize that is just one breath. For cancer to initiate, progress, or become therapeutically less responsive, even a single mutation in a single cell can be contributory to drive cancer. Now not every molecule of a VOC will cause a mutation. Not every mutation will be in a gene. Not every mutated gene will be in a manner that contributes to increased cancer. But when there are exposures that would be estimated to be in the 100s of trillions of molecules of a carcinogenic VOC that can be genotoxic that would include TCE, benzene, PCE and VC, it is scientifically and medically hard to imagine how such exposures would not cause genotoxic events including mutations in humans. Certainly, not all mutations will cause cancer, but these mutations can cause cancer, and individuals with such mutations are at greater risk of getting cancer than if they had not been exposed. Moreover, pre-cancers will be more likely to mutate to become cancers, and existing cancers are likely to become more aggressive.

As noted above, there is no way to evaluate in a human if there are any mutations in every one of the many 100s of trillions of cells in the human body (you simply cannot remove every cell from a human body and sequence the DNA of every cell) – one obviously cannot take every cell one by one out of a

human and sequence every cell to see if there is a cancer associated mutation, so there is at least no current way to examine this for these individual genetic lesions. But the science predicts medically, much more likely than not, that for even brief exposures to carcinogenic agents, this is the case. Individuals exposed to VOCs through the water supply at Camp Lejeune more likely than not will have 100s of trillions of cells in their body and permanent genetic changes that were caused by exposure to carcinogenic VOCs from Camp Lejeune that otherwise would not be present. Hence as a population this group is more likely than not to be at an increased risk of genetic events that can be a cause of cancer, and therefore an increased risk of cancer. Indeed, the evidence to date is already consistent with the conclusion that residents at Camp Lejeune have a higher incidence and mortality to some cancers.

VI. BRADFORD HILL'S CONSIDERATIONS

I used a methodology to conclude that VOCs (TCE, Benzene, VC, and PCE) exposure can generally cause cancer by considering the weight of evidence approach, which integrates an analysis of mechanistic, preclinical, and epidemiological data from published scientific and medical studies. I am using the same methods that have been similarly employed by many scientists and physicians with similar medical and scientific training and experience.

I also used an accepted methodology, known as the Bradford Hill methodology, that provides nine considerations to evaluate whether causation specific to blood cancers can be deduced: (i) strength of association; (ii) consistency of association; (iii) specificity of association; (iv) temporality; (v) biological gradient, or dose-response relationship; (vi) biological plausibility; (vii) coherence, or consistency of evidence; (viii) experiment; and (ix) analogy, or structure-activity relationship.

Notably, these are not mandatory considerations, despite many people commonly referring to these factors as "criteria." An analysis need not satisfy all factors to demonstrate causation; rather, the weight of the evidence applied across these factors should be considered. Sir Austin Bradford Hill himself (for whom these "considerations" were first described) outlined a set of factors that practitioners should "consider" to evaluate the strength of the evidence for a causal relationship between an exposure and an outcome. Hill made clear that the considerations are not intended to be a checklist or a rigid set of rules but rather are meant to serve as a guide for evaluating the strength of the evidence for a causal relationship. Hill expressed that compelling biological evidence may serve as sufficient evidence for cause. In particular, Hill was critical of the over-reliance on statistics as a consideration for defining causality. Modern textbooks of epidemiology likewise emphasize that none of these considerations are necessary for establishing causality, with the exception of temporality (i.e., the exposure precedes the disease) (Lash et al. 2021). Hill considered that biological considerations, such as a mechanism, were particularly compelling considerations.

As discussed in detail above, exposure to TCE, Benzene, and PCE have generally been demonstrated to be more likely a contributing factor to the cause of cancer. I summarize the causal relationship briefly in the context of the Hill considerations below for each chemical and its causal relationship to hematopoietic cancers, and more specifically leukemia and NHL:

A. Benzene and Hematopoietic Cancers

Strength: There are multiple compelling studies suggesting a strong association between benzene exposure and NHL, and specifically demonstrating that Benzene is a cause of hematopoietic cancers including generally and specifically, NHL and leukemia.

Consistency: There is consistency amongst studies that benzene can be a cause of hematopoietic cancers.

Specificity: Benzene has been shown to specifically be a cause of hematopoietic cancers, including NHL and leukemia. However, because Benzene does not cause only NHL or Leukemia, it does not meet the criteria of specificity for either disease.

Temporality: Benzene exposure has been associated with hematopoietic cancers after a latency that is consistent with our knowledge of the trajectory of these cancers.

Biological gradient: Benzene exposure has been shown to cause cancer in some cases consistent with a dose response and has been shown in some studies to be related to duration, frequency and intensity of exposure.

Plausibility: Benzene has a mechanism of action such that it causes hematotoxicity, immunotoxicity and genotoxicity (after metabolism), all of which are known mechanisms and causes of cancer.

Coherence: Benzene has mechanistic, preclinical and epidemiological evidence that it is a cause of hematopoietic cancer.

Experiment: Benzene has been shown experimentally to cause hematotoxicity, immunotoxicity and genotoxicity in animal bioassays and to be a cause of cancer including hematopoietic, lympho-hematopoietic cancers.

Analogy: Benzene, like other VOCs, is genotoxic, carcinogen. and a cause of hematopoietic cancers.

Benzene, based on an integrated analysis, weight of evidence, and Bradford Hill considerations, is more likely than not a cause of hematopoietic cancers, including NHL and leukemia.

B. TCE and Hematopoietic Cancers

Strength: TCE has been shown in many studies to have a strong association with hematopoietic cancers and increases the risk of these cancers.

Consistency: TCE has been shown through multiple strong and independent studies to be a cause of hematopoietic cancers.

Specificity: TCE is associated with specific types of cancers, including hematopoietic cancers such as NHL and leukemia. However, because TCE does not only cause NHL or Leukemia, it does not meet the criteria of specificity for either disease.

Temporality: TCE exposure has been associated with hematopoietic cancers after an expected and reasonable latency of onset.

Biological gradient: TCE exposure has been shown in some cases to exhibit a dose response in its ability to induce cancer.

Plausibility: TCE has been found to have effect on metabolism, to be immunotoxic, and to elicit autoimmunity – both mechanisms that can result in hematopoietic cancers. Many studies suggest that TCE is genotoxic, albeit most of these studies are not in hematopoietic cells. As cancer science has evolved, biological plausibility is extremely important as it is unethical to test potential carcinogens on humans. The link between TCE and hematopoietic cancers in animal models and mechanistic data not only reinforces what has been shown in the epidemiological studies, but gives scientists like myself a clear picture of how TCE causes said cancers.

Coherence: TCE has been shown in experimental studies and epidemiological studies to be a cause of hematopoietic cancers.

Experiment: TCE has been shown to experimentally elicit immunotoxicity, changes in immune cells, and induces in some cases hematopoietic cancers.

Analogy: TCE, similar to related VOCs, is genotoxic, a carcinogen, and a cause of cancer and hematopoietic cancers.

TCE, based on an integrated analysis, weight of evidence, and Bradford Hill considerations, is more likely than not a cause of hematopoietic cancers, including NHL and leukemia.

C. PCE and Hematopoietic Cancers

Strength: There are some studies that suggest that PCEs can increase the risk of hematopoietic cancers, albeit not as strong as either Benzene or TCE.

Consistency: There are independent studies that support that PCE can cause hematopoietic cancers, but I don't believe that the results are as consistent as either Benzene or TCE.

Specificity: PCEs are associated specifically with hematopoietic cancers but do not exclusively cause hematopoietic cancers. As such, the specificity consideration is not met.

Temporality: PCE exposure is associated with hematopoietic cancers with a reasonable latency of onset.

Biological gradient: There is some suggestion that there is dose response to exposure toxicity and the occurrence of hematopoietic cancers.

Plausibility: PCE has been shown, through multiple mechanisms, to contribute to hematopoietic carcinogenesis including hematotoxicity, immunotoxicity, autoimmunity, and genotoxicity.

Coherence: There is some evidence that PCE can cause hematotoxicity, and autoimmunity in humans, in particular, autoimmune diseases that are associated with hematopoietic cancers such as Sjogren's syndrome, scleroderma/sclerosis and rheumatoid arthritis.

Experiment: PCE can cause cancer in animal bioassays.

Analogy: PCE, similar to other VOCs, is carcinogenic, genotoxic, and can be a cause of hematopoietic cancers.

PCE, based on an integrated analysis, weight of evidence, and Bradford Hill considerations, is at least as likely as not a cause of hematopoietic cancers, including NHL and leukemia.

A proper application of Hill's considerations demonstrates that TCE, Benzene, and PCE exposure can be a cause of hematopoietic cancer in humans. I note that these same conclusions were reached by the EPA, IARC, and/or the ATSDR, among many other reputable organizations worldwide. As discussed above, applying Hill's considerations also demonstrates that there is at least as likely as not, and in some cases more likely than not, a causal relationship between TCE, Benzene, PCE, and hematopoietic cancer, including NHL and Leukemia. For VC, while there is some evidence suggesting a potential causal relationship with cancer, the current data are less robust and do not yet provide sufficient support to conclude that VC exposure alone is at least as likely as not causal.

VII. ADDITIVITY AND SYNERGISTIC EFFECTS

Carcinogenic agents are thought to often work in concert to elicit tumorigenesis (Weinberg, 2013). As described above, carcinogens have been described as initiating and promoting agents for carcinogenesis (Stewart Chapter 11, IARC 2019). Combinations of low-dose mixtures of carcinogens may work in concert (Goodson, 2015; Miller, 2016; Smith, 2020). Our understanding of the key characteristics of carcinogenesis (Smith, 2020) and the hallmarks of cancer (Hanahan and Weinberg, 2011) suggests a framework for understanding that carcinogenic agents using identical or different mechanisms of carcinogenesis could be expected to have additive and, in some cases, synergistic effects on tumorigenesis. Thus, agents that cause genotoxicity, immunotoxicity, autoimmunity, or hematotoxicity would be expected to work in concert to elicit tumorigenesis. Agents that induce genotoxicity through different mechanisms would be additive or synergistic, and a combination of agents could cooperate to elicit mechanisms of carcinogenesis. The exact relationship between the interactions of TCE, benzene, PCE, and VC is not known, but based on what we know about their mechanisms of carcinogenesis, they would be expected to be at least additive and, in some cases, synergistic (Bove, 2024).

An additive and/or synergistic approach to the Camp Lejeune mixture makes sense, where all four compounds are known or probable carcinogens. Three of the four chemicals in the drinking water (TCE, PCE, and VC) are related compounds with several shared genotoxic metabolites, at least between TCE and PCE. Additionally, two of the chemicals, TCE and benzene, and/or their metabolites, are also known to be immunotoxic. Given the role of the immune system and its relationship to blood cancers, an additive effect would seem to be conservative. (Hendrikson, 2007; Veraldi, 2006). Even the US EPA and ATSDR utilize additive models for certain chemical mixtures when studying health effects. (US Environmental Protection

Agency. Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures. 2000; Agency for Toxic Substances and Disease Registry. Interaction Profiles for Toxic Substances. 2024.)

VIII. CONCLUSIONS

My opinions use methods that would be generally accepted by similar physicians and medical scientists with my expertise. I reviewed and integrated analyses of medical and scientific literature, and I considered the mechanistic, preclinical, and epidemiological studies. I considered the weight of evidence in addition to the Bradford Hill Considerations. After employing this methodology, I conclude that:

(1) There is more likely than not a causal relationship between TCE exposure and hematopoietic cancers;

(2) There is more likely than not a causal relationship between benzene exposure and hematopoietic cancers;

(3) There is at least as likely as not a causal relationship between PCE exposure and hematopoietic cancers;

(4) There is at least an additive, and possibly synergistic, effect associated with exposure to multiple of these VOCs;

(5) These causal relationships are observed at contaminant levels similar to those found at Camp Lejeune.

Exposure to VOCs in the water at Camp Lejeune, such as TCE, PCE and benzene, is associated with an increased risk of hematopoietic cancers, including leukemia and non-Hodgkin lymphoma. Studies discussed above have identified specific exposure levels and durations that correlate with these risks. Water containing **20 ppb of total volatile organic compounds (TVOCs)** has been linked to these cancers (Cohn et al., 1994). Similarly, exposure to water containing **5 ppb of TCE** (Cohn et al., 1994) or **5 ppb of PCE** (Cohn et al., 1994) has shown a significant association. Cumulative exposure to **10,868–50,563 ppb-months of TCE** or **457–2,118 ppb-months of PCE** has also been deemed hazardous (ATSDR, 2018). Prolonged residence or employment on contaminated bases, such as living on base for **1–6 quarters between 1975 and 1985** (Bove et al., 2024) or working there for **2.5 years** (Bove et al., 2014), has been shown to increase cancer risks. For leukemia specifically, cumulative exposure to **27–44 mg of PCE** (Aschengrau et al., 1993), **2–45 µg/L-months of benzene**, or 1**–4,600 µg/L-months of TVOCs** has been associated with heightened risk (Bove et al., 2014a). Notably, water containing **267 ppb of TCE** or **21 ppb of PCE** (Woburn) has been reported as hazardous. These findings underscore the dangers posed by prolonged or significant exposure to contaminants commonly found in drinking water.

I conclude that it is medically more likely than not that the cancer risk for any individual is heterogeneous, varying based on multiple parameters, including the concentration and duration of exposure, the age of the host (e.g., embryo > baby > 2 years > child > adolescent > adult), comorbidities, genetic susceptibilities, immune status, and other personal, social, and family risk factors, as well as unique considerations from the individual's medical history.

I declare under penalty of perjury under the laws of the United States that the foregoing is true and correct. This declaration was executed by me on December 9, 2024, in Stanford, California.

Dean W. Felsher, M.D., Ph.D.
Professor of Medicine-Oncology and Pathology
Stanford University School of Medicine

Appendix A

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

<u>Graduate Student Committees</u>

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-	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.

5. Goodglick, L. A., Felsher, D. W., Mehran, N., and Braun, J. A novel octamer regulatory element in the VH11 leader exon of B-1 cells. Journal of Immunology, 154(9): 4546, 1995.

6. Felsher, D. W., and Bishop, J. M. Transient excess of MYC activity can elicit genomic instability and tumorigenesis. Proceedings of the National Academy of Sciences, 96(7): 3940, 1999.

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

8. Felsher, D. W., Zetterbert, A., Zhu, J., Tlsty, T., and Bishop, J. M. Overexpression of MYC causes p53-dependent G2 arrest of normal fibroblasts. Proceedings of the National Academy of Sciences, 97(19): 10544, 2000.

9. Johansen, L. M., Iwama, A., Lodie, T. A., Sasaki, K., Felsher, D. W., Golub, T. R., and Tenen, D. G. c-Myc is a critical target for C/EBP & in Granulopoiesis. Molecular and Cellular Biology, 21(11): 3789, 2001.

10. Jain, M., Arvanitis, C., Chu, K., Dewey, W., Leonhardt, E., Trinh, M., Sundberg, C. D., Bishop, J. M., and Felsher, D. W. Sustained loss of a neoplastic phenotype by brief inactivation of MYC. Science, 297(5578): 102, 2002. Also see: I. Bernard Weinstein, Addiction to oncogenes—The achilles heal of cancer. Science, 297: 63, 2002.

11. Karlsson, A., Giuriato, S., Tang, F., Fung-Weier, J., Levan, G., and Felsher, D. W. Genomically complex lymphomas undergo sustained regression upon MYC inactivation unless they acquire chromosomal translocations. Blood, 101(7): 2797, 2003. Also see K. Shannon, Bypassing the requirement for MYC in lymphoma progression. Blood, 101(7): 2451, 2003.

12. Karlsson, A., Deb-Basu, D., Cherry, A., Turner, S., Ford, J., and Felsher, D. W. Defective double-strand DNA break repair and chromosomal translocations by MYC overexpression. Proceedings of the National Academy of Sciences, 100(17): 9974-9979, 2003. Also see: E. Greenwood, Oncogenes: One-man band, 3: 714, 2003.

13. Yang, Y, Contag, C. H., Felsher, D. W., Shachaf, C. M., Cao, Y, Herzenberg, L. A., Herzenberg, L. A., and Tung, J. W. The E47 transcription factor negatively regulates CD5 expression during thymocyte development. Proceeding of the National Academy of Sciences, 101(11): 3898-3902, 2004.

14. Baron, B. W., Anastasi, J., Montag, A., Huo, D., Baron, R. M., Karrison, T., Thirman, M. J., Subudh, S. K., Chin, R. K., Felsher, D. W., Fu,Y., McKeithan, T. W., and Baron, J. M. The human BCL6 transgene promotes the development of lymphomas in the mouse. Proceeding of the National Academy of Sciences, 101(39): 14198-14203, 2004.

15. Beer, S., Zetterberg, A., Ihrie, R. A., Yang, Q., Bradon, N., Arvanitis, C., Attardi, L. D., Ruebner, B., Cardiff, R. D., and Felsher, D. W. Developmental context determines latency of MYC induce tumorigenesis. PLOS Biology, 2(11): e332, 2004.

16. Shachaf, C. M., Kopelman, A., Arvanitis, C., Beer, S, Karlsson, A., Mandl, S., Bachmann, M. H., Borowsky, A. D., Ruebner, B., Cardiff, R. D., Yang, W., Bishop, J. M., Contag, C. H., and Felsher, D. W. MYC inactivation uncovers pluripotent differentiative properties and induces a state of tumor dormancy in hepatocellular cancer. Nature, 431(7012): 1112-7, 2004. Also see: E. Hutchinson, Nature Reviews Cancer 7:834, 2004.

17. Sander, S., Bullinger, L., Karlsson A., Giuriato S., Hernandez-Boussard, T., Felsher, D. W., and Pollack J. R. Comparative genomic hybridization on mouse cDNA microarrays and its application to a murine lymphoma model. Oncogene, 24(40): 6101-7, 2005.

18. Beverly, L. J., Felsher, D. W., and Capobianco, A. J. Suppression of p53 by notch in lymphomagenesis: implications for initiation and regression. Cancer Research 65(16): 7159-68, 2005.

19. Kwon, H., Ogle L., Benitez, B., Bohuslav, J., Montano M., Felsher, D. W., and Greene, W. C. Lethal cutaneous disease in transgenic mice conditionally expressing type I human T cell leukemia virus Tax. Journal of Biological Chemistry, 280(42): 35713-22, 2005.

20. Deb-Basu, D., Aleem, E., Kaldis, P., and Felsher, D. W. CDK2 is required by MYC to induce apoptosis. Cell Cycle, 5(12): 1342-7, 2006.

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37. Atibalentja DF, Deutzmann A, Felsher DW. A Big Step for MYC-Targeted Therapies. Trends in Cancer, Trends Cancer. 2024 Apr 4:S2405-8033(24)00058-X. doi: 10.1016/j.trecan.2024.03.009.

Abstracts: (total of 59)

1. Felsher, D. W., Dennis, K. A., Weiss, D., Ando, D. T., and Braun, J. A murine model of CD5+ B-cell lymphomagenesis in immune compromised hosts. UCLA Symposia: B-cell Development, 1988.

2. Felsher, D. W., Ando, D. T., and Braun, J. Independent rearrangement of lambda light chain in CD5+ B-cells. Western Conference Immunology. Asilomar, CA, 1988.

3. 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.

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

5. Felsher, D. W., and Braun, J. A murine model of CD5+ B-cell lymphomagenesis. Western Conference of Immunology. Asilomar, CA, 1990.

6. Goodglick, L. A., Felsher, D. W., Anderson, M., Hassett, T., and Braun, J. B-cell specific binding to VH11 leader sequence. FASEB. Atlanta, GA, 1991.

7. Felsher, D. W. Defining when inactivation of the MYC oncogene is sufficient to results in sustained regression of lymphoma. FOCIS, June 1992.

8. Felsher, D. W., and Bishop, J.M. Hematopoietic tumorigenesis by MYC using a conditional transgenic model system. ASH, December 1999.

9. Felsher, D. W., and Tang, F. Song, SS., Beer, S. MYC inactivation in hematopoietic tumors that have lost p53 still regress, but subsequently relapse. ASH, San Francisco CA, December 2000.

10. Felsher, D. W., and Zetterberg, A., Zhu, JY., Tlsty T., Bishop, J. M. Over-expression of MYC causes p53-dependent G2 arrest of normal fibroblasts. ASCB, San Francisco CA, December 2000.

11. Felsher, D. W., and Tang, F., Sundberg, C., Karlsson, A., Giuriato, S. Defining when MYC-induced lymphomagenesis is reversible. ASH, Orlando, FL, December 2001.

12. Karlsson, A., Fung-Weier, J., Pedersen, R., and Felsher, D. W. Genetically complex hematopoietic tumors undergo sustained regression upon MYC inactivation. SALK/EMBL, San Diego, CA, August 2001.

13. Jain, M., Arvanitis, C., Chu, K., Dewey, W., Leonhardt, E., Trinh, M., and Felsher, D. W. Brief cessation of MYC over-expression results in the abrogation of a neoplastic phenotype. SALK/EMBL, San Diego, CA, August 2001.

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15. Felsher, D. W., Arvanitis, C., Beer, S., Deb-Basu, D., Feng, C., Giuriato, S., Karlsson, A., Shachaf, C., Sundberg, C., Tang, F., and Yang, Q. Defining when MYC-induced tumorigenesis is reversible. SALK/EMBL, San Diego, CA, August 2001.

16. Deb-Basu, D., Karlsson, A., and Felsher D. W. Restoration of p27 function prevents MYC from inducing genomic instability and apoptosis. AACR, San Francisco, CA, April 2002.

17. Arvanitis, C., Jain, M., Chu, K., Dewey, W., Leonhardt, E., Trinh, M., and Felsher, D. W. Brief loss of MYC over-expression results in the suppression of a neoplastic phenotype. AACR, San Francisco, CA, April 2002.

18. Tang, F., Sundberg, C. D., Giuriato, S., and Felsher, D. W. The loss of p53 function prevents MYC inactivation from causing sustained tumor regression. AACR, San Francisco, CA, April 2002.

19. Giuriato, S., Tang, F., Drago, K., Sundberg, C. D., and Felsher, D. W. Cooperation between MYC and RAS in the induction and maintenance of hematopoietic tumorigenesis. AACR, San Francisco, CA, April 2002.

20. Karlsson, A., Fung-Weier, J., Pedersen, R., and Felsher, D. W. Genetically complex hematopoietic tumors undergo sustained regression upon MYC inactivation. AACR, San Francisco, CA, April 2002.

21. Felsher, D. W. Defining when inactivation of the MYC oncogene is sufficient to result in sustained regression of lymphoma. FOCIS, San Francisco, CA, June 2002.

22. Shachaf, C. and Felsher, D. W. Threshold levels of MYC expression required to maintain a neoplastic phenotype is modulated by cell cycle regulatory genes. FOCIS, San Francisco, CA, June 2002.

23. Deb-Basu, D., Karlsson, A., and Felsher, D. W. Restoration of p27 function MYC from inducing genomic instability and apoptosis. SALK, San Diego, CA, June 2002.

24. Shachaf, C. and Felsher, D. W. Targeting MYC inactivation for the treatment of lymphoma. SALK, San Diego, CA, June 2002.

25. 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.

26. Giuriato, S., Passegue, E., Fan, A., Tang, F., and Felsher, D. W. Defining the genetic contexts when MYC inactivation induces sustained regression of hematopoietic tumors. ASH, San Diego, CA, December 2003.

27. Rabin, K., Giuriato, S., Ray, S., and Felsher, D. W. MYC inactivation induces tumor regression through the recovery of a functional DNA damage response. ASH, San Diego, CA, December 4-7, 2004.

28. Fan, A.C., Giuriato, S., Feng, C., Padua, R. A., and Felsher, D. W. Cooperation between MYC and BCL2 to induce lymphoma is uncovered in an adult context. ASH, San Diego, CA, December 4-7, 2004.

29. Shachaf, C.M., Bendapudi, P.K., Bradon, N., Yang, Q., Borowsky, A.D., Ruebner, B., and Felsher, D.W. Characterization of tumor dormancy and the liver cancer stem cell uncovered upon myc inactivation in hepatocellular cancer. AACR, Maui Hi, March 22-26, 2005.

30. Fan, A.C., Giuriato, S., Karlsson, A., Padua, R.A., Felsher, D.W. Two oncogenic hits are required to initiate lymphomagenesis in adult, but not neonatal hosts. ASH, Atlanta, GA, December 10-13, 2005.

31. Fan, A.C., Giuriato, S., Karlsson, A., Bachireddy, P., Bendapudi, P., Rakhra, K., Padua, R.A., Felsher. D.W. MYC or RAS, but not BCL2 expression induces reversible lymphomagenesis. AACR, Washington DC, April 1-5, 2006.

32. Fan, A.C., Voehringer, D., Deb-Basu, D., Gossett, J., O'Neill, O., Felsher, D.W. Nanoliter-scale western-blot-like BCL-2 analysis of lymphoma fine needle aspirates. AACR, Washington DC, April 1-5, 2006.

33. Fan, A.C., Voehringer, D., Deb-Basu, D., Gossett, J., O'Neill, R., Felsher, D.W. MYC quantification in lymphoma fine needle aspirates using, firefly, a novel nanofluidic protein analysis instrument. AACR, Washington DC, April 1-5, 2006.

34. Bachireddy, P., Fan, A., Rakhra, K., Zeiser, R., Kopelman, A., Negrin, R. S., Contag, C.H., Felsher, D.W. The effects of host immune status on the consequences of oncogene inactivation. AACR, Cambridge Massachusetts, October 25, 2006.

35. Riggelen, J. v., Wu, N., Felsher, D. W. The impact of epigenetics on tumor regression upon MYC oncogene inactivation. AACR, Cambridge Massachusetts, October 25, 2006.

36. Fan, A. C., Deb-Basu, D., Horoschak, M., Shirer, A., Voehringer, D., O'Neill, R., Felsher, D. W. Nano-fluidic detection of oncoprotein signaling in preclinical and patient lymphoma samples. ASH, Orlando, Florida, December 10, 2006.

37. Deb-Basu, D., Fan, A., Voehringer, D., Ferrante, J., Bhamidipatil, A., Gossett, J., O'Neill, R., Felsher, D.W. Measurement of oncoproteins in preclinical and clinical specimens using a non-fluidic high throughput approach. ASCB, San Diego, CA, December13, 2006.

38. Wu, N., Riggelen, J.v., Yetil, A., Felsher, D. W. Cellular senescence programs are an important mechanism of tumor regression. AACR, Los Angeles, CA, April 14-18, 2007.

39. Deb-Basu, D., Fan, A. C., Voehringer, D., Felsher, D. W. Monitoring drug impact on signaling pathways in precious samples in primary hematopoietic malignancies. AACR, Los Angeles, CA, April 14-18, 2007.

40. Choi, P. S., Rabin, K., Giuriato, S., Ray, S., Yang, Q., Felsher, D. W. Loss of ATM or H2AX accelerates MYC-induced tumorigenesis and prevents sustained tumor regression. AACR, Los Angeles, CA, April 14-18, 2007.
41. Fan, A., Deb-Basu, D., Gotlib, J., Voehringer, D., Felsher, D. W. Monitoring changes in signaling proteins upon oncogene inactivation in hematopoietic tumors using a nano-immunoassay system. AACR, San Diego, CA, April 12-16, 2008.

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.

Appendix B

Dean W. Felsher, MD Ph.D. Prior Testimony 2020-2024

Testimony Date	Court	Testimony	Case Name	Case Number
2022	Circuit Court of Cook County, Illinois	Deposition & Trial	*Kamuda, et al. v. Sterigenics US., LLC, et al.*	2018-L-010475
2022	Circuit Court of Cook County, Illinois	Deposition & Trial	*Fornek v. Sterigenics US., LLC, et al.*	2018-L-010744
2022	Circuit Court 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

Appendix C

Fee Schedule

Dean W. Felsher, M.D., Ph.D.
Professor and Associate Chief of Medicine-Oncology and Pathology

Center for Clinical Sciences Research (CCSR) Building
269 Campus Drive
Stanford, CA 94305-5151

My rates:

- 1000/hour: review and preparation
- 1500/hour: trial and depo, patient interview