Exhibit 482

Scott Richard Keller v. United States of America

U.S. District Court for Eastern District of NC, Southern Division
Case No. 7:23-cv-01501

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

Prepared by

Dean W. Felsher, M.D., Ph.D.
269 Campus Drive
CCSR Building
Stanford, CA 94305-5151

Contents

I. EDUCATION AND QUALIFICATIONS

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

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

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

I received my Bachelor of Arts in Chemistry from the University of Chicago, followed by both an M.D. and a Ph.D. in Molecular Biology with a specialization in cancer immunology from the University of California, Los Angeles. I completed my residency in internal medicine at the Hospital of the University of Pennsylvania and a fellowship in hematology-oncology at the University of California, San Francisco, where I conducted postdoctoral 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 medical and scientific literature and used a weight-of-evidence approach to evaluate causation in this case. I integrated my analysis of mechanistic, preclinical, and epidemiological studies and considered the Bradford Hill considerations, using methods that any scientist and doctor with my training would utilize and that are commonly utilized by other scientists, governmental agencies, and public organizations such as the EPA, NTP, ATSDR, and IARC, in their analysis of similar considerations. I have also reviewed, and to varying degrees, relied upon general causation reports from Morris Maslia, Kelly Reynolds, Kate Gilbert, and my own general causation report in this case.

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

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

To perform a differential etiology generally, I first compiled a list of demographic and risk factors for NHL based both on my experience with hematopoietic cancers and on information from the same scientific

sources referenced herein. For completeness, I include demographic and risk factors, even if I do not conclude there is sufficient evidence to consider them as risk factors for NHL, if there is any scientific literature suggesting an association with NHL.

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

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

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

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

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

III. SUMMARY OF OPINIONS

I conclude with a reasonable degree of medical certainly that it is more likely than not that exposure to volatile organic compounds (VOCs) including benzene, can generally be a cause of hematopoietic cancers including non-Hodgkin's lymphomas (NHL).

I conclude as to Mr. Keller specifically, with a reasonable degree of medical certainly, that his exposure to benzene in the water at Camp Lejeune is more likely than not a substantial contributing cause of his NHL.

I conclude as to Mr. Keller specifically, with a reasonable degree of medical certainly, that his development of kidney disease is more likely than not caused by his treatment for NHL.

I conclude as to Mr. Keller specifically, with a reasonable degree of medical certainly, that his significant worsening of cardiomyopathy is more likely than not caused by his NHL treatment.

I conclude as to Mr. Keller specifically, with a reasonable degree of medical certainty, that his NHL treatment, kidney disease, and cardiomyopathy more likely not will increase the likelihood of his premature death.

IV. HEMOTOPOETIC CANCERS AND NON-HODGKIN'S LYMPHOMA

Hematopoietic cancers are cancers often derived from hematopoietic cells and stem cells (Bryder 2006, Filipek-Garzala 2024)[1,2]. There are many types and subtypes of hematopoietic stems cells that, when they acquire sufficient genetic changes such as mutations, deletions and chromosomal translocations, can become cancerous (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024). This includes erythroid, myeloid, and lymphocytes amongst other lineages. Most types of hematopoietic cancers initially arise in immature stem, progenitor cells and/or less differentiated cells that can in some circumstances further mature to become more or fully differentiated (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).

There are a multitude of associated hematopoietic diseases, including hematopoietic cancers commonly deriving from immature cells as well as less or more mature cells (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).[3–6] For example, diseases causing a disruption of the bone marrow function can be a myeloproliferative disorder such as chronic myelogenous leukemia (CML) or essential thrombocytopenia (ET) or a disease called myelodysplasia. Alternatively, when the disease involves solid immune organs such as lymph nodes or the spleen, then the disease is often described as a lymphoma. Additionally, hematopoietic cancers are usually considered leukemias when they are associated mostly with abnormal increased numbers of leukocytes that have become abnormal cancerous cells seen in excess in the blood. Hematopoietic cancers are usually considered lymphomas when they are associated with abnormal increased normal of lymphocytes in hematopoietic or lymphoid organs such as bone marrow, lymph nodes and/or spleen. However, all types of hematopoietic cancers, whether designated by a name of leukemia or lymphoma, generally are both in the blood and the hematopoietic organs. While each of these types and subtypes have different naming conventions, they are all hematopoietic cancers due to the types of cells from which they arise, and many of these cancers begin and/or are initiated in the same common hematopoietic stem cells even if the final disease at diagnosis for a patient corresponds to a more specific hematopoietic lineage and differentiative state, or subtype of that lineage or state, as has been generally accepted for decades (Greaves 1986, Kuppers 2005, Greaves 2003, Weigert 2012, Weniger 2024).

All hematopoietic cancers, including those that are derived from erythroid, myeloid cells, B-cell or T-cell lymphocytes, arise when an otherwise normal cell acquires a complement of genetic events that activate oncogenes and inactivate tumor suppressor genes (Weissman, Blood, 2008).[7] These genetic events can occur spontaneously but occur more frequently when bone marrow cells, myeloid cells or lymphocytes are exposed to environmental agents, such as carcinogens that are known to initiate, promote, accelerate cancer formation and can reduce the sensitivity of cancer cells to therapy. [3–6]

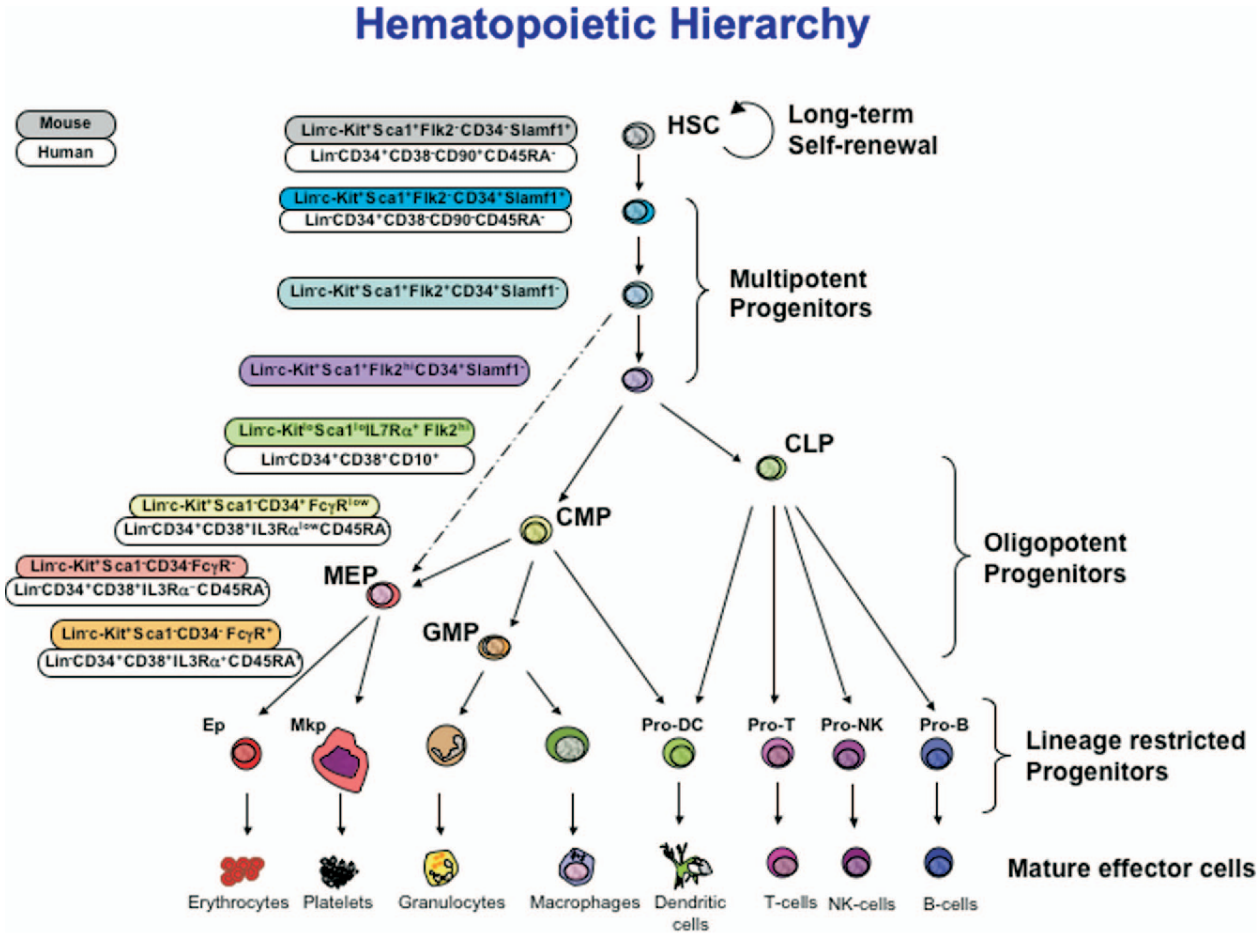

Figure 1. Schematic of hematopoietic development indicating intermediates in the hierarchy of hematopoietic differentiation. Surface markers used for isolation are indicated at left for human (top) and mouse (bottom) for each stem and progenitor cell. HSC indicates long-term reconstituting, self-renewing;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

Hematopoietic cancers include Lympho-Hematopoietic cancers that are derived from lymphocytes that include: acute lymphocytic or lymphoblastic leukemia (ALL), multiple myeloma (MM), non Hodgkin's lymphoma (NHL) and Hodgkin's lymphoma (HL). Patients with these types of hematopoietic cancer have cancerous lymphocytes that can be found in the blood and bone marrow and other locations. Lymphocytes can be either B-cells that are the antibody producing cells or T-cells that are the cells that have a T-cell receptor. B-cells are the type of cell that when they become mature are normally in the human body helps fight infections through the production of antibodies. T-cells are the type of cell that when they become mature are normally in the body to help fight infections and also help prevent cancer

through their T-cell receptor that can result that help the function other immune cells including B-cells. ALL is a type of cancer that arising from lymphocytes, more commonly B-cells but also T-cells and usually in less mature pre-B or pre-T-cells or lymphocytes that have not completed the formation of their antibodies or T-cell receptor respectively. MM is a type of cancer arising in B-cell lymphocytes that are antibody producing. B-cell lymphocytes make antibodies that are secreted, and they are called plasma cells.

NHL is a group of cancers that occur more commonly in B cells but also occurs in T cells and rarely in other types of lymphocytes, that occur in different stages of their maturation. (Rohit 2020, Leval 2020, Pasqualucci 2024).[8–10] Mr. Keller has a B-cell lymphoma. NHL of mature B-cells is most common in humans. NHL can be further divided clinically into the high-grade, fast-growing lymphomas and low grade, more slow growing lymphomas and then further subdivided into many other subtypes mainly based upon their phenotypic features as observed by a pathologist under the microscopy through histology as well as through characterization of cell surface markers. (Rohit 2020, Leval 2020, Pasqualucci 2024).[8–10] The World Health Organization (WHO) consensus classification of hematologic malignancies uses an updated version of the Revised European American Lymphoma (REAL) classification for lymphoid neoplasms (de Leval, Cancer Journal, 2020).[8,11] In the Working Formulation, NHL can be subdivided into many specific subtypes that can be differentiated based upon their morphologic features, surface antigen phenotype, biological behavior, immunoglobulin rearrangement status, oncogenic activation as well as other parameters (Rohit 2020, Leval 2020, Pasqualucci 2024).

Diffuse large cell lymphoma is a type of NHL that can be divided into other subtypes including Immunoblastic lymphoma (Kurz 2023).[12] In turn, this subtype is classified with lymphoblastic lymphoma and small noncleaved-cell lymphoma as a high-grade non-Hodgkin lymphoma (NHL) The International Lymphoma Study group uses all available information, morphology, immunophenotype, genetic features, and clinical features, to define a disease entity. Immunoblastic lymphoma (IBL), can also be called a diffuse histiocytic lymphoma, is a malignant disorder of the B-cell. Immunoblastic lymphoma is fatal disease if left untreated. But is curable with intensive chemotherapy. Treatment success has been found to relate to the stage of the disease, presence of B symptoms, the initial therapeutic choice, and the treatment response In the United States this corresponds to about 9% of all NHL. Of these, 70% are of B-cell origin and 25% are of T-cell origin. No difference exists among races or between the sexes. However, although immunoblastic lymphoma can appear in persons of any age, it presents most commonly in persons who are middle-aged or older, and it is commonly observed in patients of any age who are immunocompromised.

In general, there are certain demographics and many risk factors generally associated with lympho-hematopoietic cancers and/or NHL (Koff 2015).[13]

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

Family History: There is some suggestion that a family history of a hematological malignancy can be associated with increased risk (Cerhan, Blood, 2015).[11]

Familial predisposition syndromes: Familial predisposition syndromes are associated with increased lymphoma such as Ataxia Telangectasia, Klinefelter's Syndrome, Wiskott Aldrich Syndrome, Chediak-

Higashi Syndrome amongst many others often associated with defects in DNA repair, aging mechanisms and/or immune deficiencies. (Szymd 2021).[14]

Autoimmune Disorders: Many autoimmune disorders are associated with a risk of lymphoma, including rheumatoid arthritis, systemic lupus erythematosus, sjogren's syndrome and Crohn's disease. (Smedby 2008).[13,15]

Immune Suppression: Congenital, infectious or iatrogenic immune suppression including patients with HIV, transplant patients in immune suppression. (Gruhlich 2007).[16] Some infectious agents can be associated with increased risk such as EBV, HTLVIII, Herpes 8, heliobacter pylori, hepatitis C and tuberculosis. (Engels 2007).[17]

Exposure to Radiation and Chemotherapy: Prior treatment with chemotherapy or radiation therapy and radiation exposure can increase the risk of NHL. (Harbon 2020).[18]

Exposure to chemical carcinogens: The exposure to chemical carcinogens, such as benzene (Smith 2007, Vlaanderen 2010, Steinmaus 2015,Rana 2021, Ge 2024, Bassig 2024) [19–24], trichloroethylene (also for benzene and TCE see as described in detail further elsewhere in this report) (see for example NTP 2015[25]), formaldehyde[26] (see for example Catalina 2019), glyphosate (see for example Zhang 2021, Davoren 2018)[27,28] and ethylene oxide (see for example: IRIS 2016)[29], as well as many other chemical agents, such as pesticides(Cavalier 2021), [30] or other environmental exposures can cause NHL. (Francisco 2023).[31]

Other risk factors include the use of some types of breast implants (Kricheldorff 2018)[32]; obesity (Lichtman 2010, Skibola 2007, Willet 2008)[33–36]; diabetes (for example: Xu 2019)[37] and NHL survival (Han 2023). The exposure to tobacco smoke may be associated with NHL, but evidence is conflicting. (for example: Diver 2012, Taborelli 2017).[37,38]

The VA recognizes herbicide exposure during military service as a risk factor for CLL. The VA also recognizes exposure to Agent Orange, despite the fact that to date epidemiology studies do not necessarily support a causal relationship.[38]

Importantly, many of these risk factors are predisposition factors but not direct causes of tumorigenesis and thus not independent causal risk factors. Thus, aging can be a risk factor because there is simply more time for tumorigenesis to occur. Gender may influence tumorigenesis through other factors such as occupational and environmental exposures (Radkiewicz 2023).[39] Familial predisposition syndromes for cancer have been when examined shown to increase susceptibility to environmental exposures that are carcinogenic (as reviewed by Carbone 2020).[40] Finally, many cancers will have more than one substantial risk factor and/or more than one significant contributing cause, since cancer formation requires many steps that occurs over a long period of time and involves initiation, progression and therapeutic resistance.

A major consideration is that many, but not all, of these risk factors are generally relevant to hematopoietic and/or lympho-hematopoietic cancers. Some risk factors are specific to subtypes (for discussion see both Wang 2010; Wild 2020).[41,42] Indeed, it is generally accepted for purposes of evaluating potential risks for NHL and/or lympho-hematopoietic cancers, that these cancers be grouped together, as is accepted scientific methodology for epidemiological studies, as well as for analyses

performed and reported by, for example, scientists at the EPA, IARC and other scientific and government organizations that assess carcinogenesis and carcinogenic risk.

V. SCIENTIFIC EVIDENCE UNDERLYING OPINON

I incorporate by reference my general causation report, including all of the opinions contained therein and the materials considered list, submitted in this case on December 9, 2024.

I have performed an integrative analysis to examine and conclude based upon the weight of evidence and examination of epidemiological, preclinical and mechanistic data that exposure to benzene can be a cause of hematopoietic cancers including leukemia, lympho-hematopoietic cancers and NHL (see Felsher, General Causation Report, Leukemia and NHL). Examination of medical and scientific literature establishes that benzene exposure is a carcinogen. Further, the exposure to benzene is a known cause of NHL.[23,31,43,44]

VI. SCOTT RICHARD KELLER MEDICAL DIAGNOSIS AND CAUSATION

A. Medical Summary

1. Medical History

Mr. Scott Richard Keller is a 58-year-old white male veteran of the United States Marine Corps stationed at Camp Lejeune, North Carolina, for over 500 days between November 1985 and January 7, 1988. While serving in the US Marine Corps at Camp Lejeune, Mr. Keller was exposed to toxic chemicals including Benzene, which unfortunately led to a diagnosis of Non-Hodgkins Lymphoma in 1998. Following his departure from Camp Lejeune, Mr. Keller was transferred to California where he was stationed at 29 Palms and Camp Pendleton, until he was honorably discharged at age 30 on January 25, 1997, after twelve years of active-duty service.

Mr. Keller's social history reflects that he is a married father of two, a nonsmoker and a social/casual drinker with a negative history of substance abuse. He did report heavier alcohol intake when he was younger. Mr. Keller reported eight years of exposure to RF radiation in his job in the military but was never required to wear a radiation badge to gauge potential radiation exposure or report to medical for radiation exposure evaluation. He otherwise denies any known exposure to radiation or unusual chemicals. He and his family live in Walla Walla, Washington. He denies any history of blood transfusions or history or any other known HIV risk factors. His medical history is significant for diagnoses of non-Hodgkin's lymphoma, chronic kidney disease stage IV, diabetes mellitus type 2, hypocalcemia, obesity, hypertension, hyperlipidemia, asthma, gastroesophageal reflux disease (GERD), gout, anemia in chronic kidney disease, obstructive sleep apnea, neuropathy, congestive heart failure, albuminuria, secondary hyperparathyroidism, atrial fibrillation with AV block, atrial flutter, basal cell carcinoma, adenomatous colon polyps, Barrett's esophagus without dysplasia, diabetic retinopathy, macular degeneration, GI bleed, duodenal angiectasia, kidney failure, nephropathy, arthritis, and myocardial infarction. His surgical history was significant for vasectomy, stem cell transplant, umbilical hernia repair, esophageal dilation, cardioversion, heart catheterization with stent placement, and pacemaker insertion (x2).

In November 1997, Mr. Keller presented to his dentist, Dr. Rietz, with complaints of gum swelling. An x-ray obtained at that time was interpreted as normal. Surgical exploration of the jaw revealed nothing

abnormal. After the exploration, swelling decreased initially, but later reappeared at the end of December. Mr. Keller was referred to Dr. Sundberg and repeat x-rays of the mandible showed 2 mm of bone loss compared to the November x-ray films. Dr. Sunberg then performed surgical exploration which revealed nothing abnormal. Some dead tissue was debrided and the involved tooth removed as it was felt it could be hiding an abscess. Mr. Keller presented to the Emergency Department at Providence St. Mary Medical Center on January 27, 1998, with complaints of pain in the medial right clavicle. X-rays of the right clavicle were interpreted as normal. An x-ray of the skull obtained at Providence St. Mary Regional Medical Center on January 27, 1998, was interpreted as revealing 4 mm bone loss in the jaw.

A February 2, 1998, pathology report by Dr. Abbas Sameh of soft tissue and tooth from the left mandible was interpreted as showing diffuse large cell anaplastic malignant lymphoma. Dr. Sameh further opined "This is a rare manifestation of malignant lymphoreticular process" and recommended a lymph node biopsy to assist with precise classification of the cellular type and grading. Mr. Keller was seen by Dr. Jeanne Berretta and Dr. Ed Berretta who referred Mr. Keller to the Medical Oncology Department at Providence St. Mary Cancer Center.

February 5, 1998, Dr. Stephen J. Iacoboni of the St. Mary Regional Cancer Center saw Mr. Keller in consultation at the request of Dr. Berretta for treatment of his newly diagnosed lymphoma. Dr. Iacoboni advised Mr. Keller he had a "worrisome, high-grade cancer" that required a "fairly intensive chemotherapy with curative intent." His prognosis was dependent on staging. Due to the type of cancer, immediate treatment was recommended. Staging would consist of CT scans of the neck, chest, abdomen, and pelvis, as well as a bone marrow biopsy. A CT of the soft tissues of the neck obtained February 6, 1998, showed mild lymphoid hyperplasia at the level of the Waldeyer's ring not necessarily pathologic by size criteria. Clinical correction and direction examination and/or biopsy was felt to be indicated. A CT of the chest dated February 6, 1998, showed no evidence of adenopathy within the thorax, while a CT of the abdomen and pelvis identified mild splenomegaly with no evidence of adenopathy. Labs revealed an increased uric acid level for which Mr. Keller was prescribed Allopurinol 100 mg QID.

On February 6, 1998, Mr. Keller underwent a bone marrow biopsy of the left iliac crest, which was interpreted by Dr. Abbas Sameh as showing (1) hypercellular marrow, with no malignant lymphoreticular single cell infiltrate (negative for lymphoma); (2) myeloid cell series with normal maturation; (3) erythroid cell series with normoblastic maturation; and (4) megakaryocytes present in normal to increased number.

February 9, 1998, Mr. Keller was admitted to Providence St. Mary Cancer Center for treatment of high-grade lymphoma of the mouth with a CHOP chemotherapy regimen. On day one, he received Decadron, Adriamycin, Oncovin, and Cytoxan. Adriamycin was then repeated. He was placed on Basaljel and Allopurinol. On day two, Mr. Keller received Adriamycin 40 mg in divided doses along with Cytoxan 700 mg and Decadron 12 mg. He tolerated the therapy well and was discharged home on February 10, 1998, with three days of oral Decadron to follow at home.

February 13, 1998, Dr. Stephen J. Iacoboni saw Mr. Keller in follow-up to review the CT scan and bone marrow results and evaluate his response to the first cycle of chemotherapy. CT scans were discussed and revealed no sign of metastatic disease. The bone marrow biopsy was also negative for metastatic disease. The bone scan, however, did show positivity in several areas, including the ribs, clavicle, and skull, as well as the area in the mandible. An x-ray of the right clavicle was obtained and interpreted as

revealing a large, jagged fracture through the proximal clavicle. Dr. Iacoboni opined the lesion in the clavicle was also due to cancer, which would unfortunately make the NHL a Stage IV and possibly change his treatment. "I would say that the likelihood of him indeed having cancer in the clavicle or other bones is probably in the 90% range already." An x-ray of the head showed two ill-defined areas of subtle lucency over the right parietal bone. Mr. Keller was administered a dose of Neupogen in the office and prescribed self-injections of Neupogen daily to protect him against myelosuppression that occurs after chemotherapy. The next cycle of chemotherapy was planned.

On February 19, 1998, Dr. Julie Rose saw Mr. Keller in follow-up, noting he was status post first cycle of CHOP regimen at day 11 and feeling relatively well. Mr. Keller complained of significant knee achiness and hip tenderness while taking Neupogen injections, reporting he had been off the injections for two days and was feeling much better. Examination revealed mild right clavicular tenderness on palpation. On February 26, 1998, Mr. Keller was once again seen by Dr. Rose, at which time he reported feeling good with improvement in his mouth pain. Dr. Rose felt he had an excellent response to chemotherapy and wanted to keep on with dose intensity with treatment scheduled the following day. Labs revealed an increased uric acid level of 8.5, an increased basophil count of 1.6, and decreased lymphocytes of 19.

February 27, 1998, Cycle 2 of CHOP was initiated and concluded on March 2, 1998. Once again, Mr. Keller was administered Decadron, Adriamycin, Oncovin, and Cytoxan, with three days of oral Decadron to follow. An echocardiogram obtained on February 27, 1998, was interpreted as normal with an ejection fraction of 60% with no evidence of valvular abnormality. On March 1, 1998, he was diagnosed with bronchiolitis. In a March 2, 1998, progress note, Dr. Iacoboni outlined his plan for Decadron twice daily for two days, Neupogen four times daily, complete course of Allopurinol, and Baclofen as needed.

March 9, 1998, Mr. Keller reported resolution of his right clavicle pain at a follow-up visit with Dr. Iacoboni but continued to complain of achiness from the Neupogen, as well as complaints of insomnia. He reported a mild dry cough reporting a cold was going through his house. Examination revealed the swelling in the neck and mandible had resolved. Dr. Iacoboni stated it seemed quite clear Mr. Keller had bone metastases, including the mandible, clavicle, and some spots on his skull that were seen on the bone scan.

On March 13, 1998, Mr. Keller had an RN check with complaints of a cough and sore throat. His Neutrophil count was noted to be 3400. Dr. Iacoboni started Mr. Keller on Bactrim DS twice a day and advised him to return on March 16th for labs and another check. Labs obtained March 16, 1998, showed improvement with a neutrophil count of 2400. Chemotherapy was ordered to continue and Mr. Keller returned to St. Mary Regional Cancer Center for Cycle 3 of the CHOP regimen on March 17, 1998, where he once again was administered Cytoxan, Adriamycin, Vincristine, and Decadron.

On March 25, 1998, Mr. Keller again returned for follow-up at which time he reported having nausea, dry heaves, cotton mouth, tightness in his throat, and a gritty feel going down. In addition, he was noted to have had bronchitis which was felt to be contributory. He continued to take the previously prescribed Cipro. White blood cell count was noted to be 13.2, with a hemoglobin of 12.2 and platelets at 252. Discussion regarding a stem cel transplant took place at which time it was decided they would pursue the treatment at the University of Washington in Seattle.

March 27, 1998, revealed Mr. Keller was feeling better but still had a lingering cough. Mr. Keller reported being scheduled with the University of Washington in Seattle on April 8, 1998. Dr. Iacoboni felt he could

administer another round of chemotherapy before his trip to Seattle in hopes they could possibly harvest his stem cells during the Neupogen phase of that cycle. A fourth cycle of the CHOP regimen was scheduled and Mr. Keller completed four days of chemotherapy consisting of Decadron, Adriamycin, Oncovin, and Cytoxan beginning April 3, 1998, and ending April 6, 1998.

April 8, 1998, Dr. Douglas McNeel, in conjunction with Dr. Stephen H. Petersdorf of University of Washington Medical Center, saw Mr. Keller in consultation. In a History and Physical report authored by Dr. McNeel, he reports the slides accompanying Mr. Keller were briefly reviewed by the Hematopathology Department, who felt that the morphology may be more consistent with an immunoblastic phenotype. Further studies, including immunohistochemistry, were ordered. It was Dr. McNeel's impression that Mr. Keller's underlying diagnosis was a little uncertain with regard to the exact phenotype of his lymphoma. Dr. McNeel also felt the stage of his disease was questionable as well, as no head CT was performed at the time of his initial diagnosis, and therefore the extent of the disease at the time of his initial treatment was not entirely known. Additional imaging studies were ordered, and Mr. Keller's case was placed in line for review by the biweekly Lymphoma Conference scheduled for April 17, 1998. Mr. Keller subsequently was apheresed with 100 million cells collected over two days and ordered a conditioning regimen in the form of high dose Cytoxan and VePesid.

April 21, 1998, pathology slides from the original biopsy of the gum with tooth extractions were reviewed by pathologist Dr. Rodney Schmidt and Dr. D.A. Hansen of University of Washington Medical Center. In their pathology report, Dr. Hansen concludes that the expression of CD45, CD138, kappa light chain, and EMA, along with the high Ki-67 fine proliferative rate and large cell morphology was consistent with immunoblastic lymphoma.

On the April 13, 1998, follow-up visit, Dr. Iacoboni noted Mr. Keller had a rough last week with chemotherapy but had recovered nicely. Judicious use of Neupogen was decided upon due to the adverse side effects Mr. Keller had been experiencing, so administration was held off with plans to reinitiate when his counts dropped. Long-term treatment was deferred pending recommendations from the University of Washington. In an April 20, 1998, progress note, Dr. Iacoboni noted he spoke with Dr. Petersdorf from the University of Washington, who had reported they had reviewed the case at the lymphoma board and determined Mr. Keller had a very high grade, 100% proliferative index, immunoblastic/plasmacytoid lymphoma. Mr. Keller reported feeling well at the visit, but did report complaints of numbness and tingling in his fingertips. Labs revealed a neutrophil count of 2700. The plan was to await direction from Dr. Petersdorf.

On April 24, 1998, Mr. Keller underwent a multigated acquisition (MUGA) scan which was interpreted as showing normal left ventricular wall motion with an ejection fraction of 53%, within normal limits. Pulmonary function testing was also performed April 24, 1998, and interpreted as being essentially normal (in the setting of mild anemia caused by lymphoma).

On April 29, 1998, Mr. Keller was admitted to Providence St. Mary Cancer Center for chemo immobilization and placement of a Hickman catheter in the right subclavian vein. He received Cytoxan, Neznil, Etoposide, and Dexamethasone. He tolerated the medications well with only some nausea and vomiting on day one which was controlled with Zofran. Mr. Keller was discharged on May 2, 1998, on GCSF 1260 mcg subcutaneously every day as well as Ciprofloxacin 500 mg twice daily with diagnoses of immunoblastic non-Hodgkin's lymphoma status post CED for stem cell mobilization.

May 8, 1998, Mr. Keller's wife called at 3 a.m. reporting her husband had a fever of 100.5° with no other symptoms. He was admitted by Dr. Iacoboni with a diagnosis of neutropenic fever on May 8, 1998, and was empirically treated with vancomycin and Primaxin, as well as Neupogen. He received a red blood cell transfusion after his hemoglobin dipped to 8.0 on May 10th and was subsequently discharged on May 11, 1998, with final diagnoses of neutropenic fever (culture negative); chemotherapy-induced anemia; chemotherapy-induced thrombocytopenia; headache associated with fever; and immunoblastic lymphoma in remission status post recent chemotherapy.

At a May 18, 1998, follow-up visit, Dr. Iacoboni found he was doing well with a little bit of anemia. Labs revealed a white blood cell count of 19.5, hemoglobin of 9.1, and platelets of 115. Plans were for Mr. Keller to return to Seattle in 14 days for high-dose radiation therapy and chemotherapy followed by stem cell rescue. At a May 27, 1998, follow-up visit with Dr. Iacoboni, Mr. Keller reported feeling well. He reported plans to travel to Seattle that weekend to start radiotherapy on June 1st followed by chemotherapy. Labs showed a white blood cell count of 3.8, Hemoglobin of 10.8, platelets of 313, and neutrophils of 1600. Dr. Iacoboni noted some neutropenia post-pheresis and post all the Neupogen. Plans continued for his transplant at the University of Washington.

June 1, 1998, Dr. Stephen H. Petersdorf of University of Washington Medical Center saw Mr. Keller in consultation in preparation for a June 2nd admission for peripheral blood stem cell transplant for high grade non-Hodgkin's lymphoma. Mr. Keller had previously undergone stem cell collection where he had more than 145 million cells collected. It was Dr. Petersdorf's plan to start treatment the following day with total body irradiation twice a day from June 2nd through June 5th, followed by high dose Etoposide and high dose Cytoxan with Mesna, followed by the stem cell transplant. A chest x-ray obtained June 1st was interpreted as showing no evidence of acute cardiopulmonary disease or pneumonia.

June 2, 1998, Mr. Keller was admitted to the University of Washington Medical Center where he received total body irradiation twice per day beginning June 2nd and continuing daily through June 4th, after which he received VePesid on June 5th and Cyclophosphamide on June 7th. He also received a lumbar puncture with intrathecal methotrexate. A peripheral blood stem cell transplant with CMV-negative radiated leukocyte-poor blood products was performed on June 9, 1998. Mr. Keller became neutropenic on transplant day number zero. On transplant day two, anemia and thrombocytopenia developed, and Mr. Keller required transfusions of packed red blood cells and platelets. He was noted to have a fever of 39.5° C on transplant day two. He was pan cultured and started on Ceftazidime. He continued to spike fevers and on transplant day 3 was started on vancomycin secondary to some mild erythema at a previous Hickman insertion site. A chest x-ray obtained on June 11, 1998, was interpreted as showing new opacities in the lung bases, suggesting atelectasis, edema, or bronchitis. Cardiomegaly was unchanged. Fevers persisted and blood cultures remained negative, so amphotericin was started on transplant day five. Neutropenia finally resolved on transplant day nine.

June 23, 1998, Mr. Keller developed scrotal swelling and pain, which was felt to be secondary to the total body irradiation as well as volume overload. A CT scan of the abdomen was obtained to rule out an abscess. It was interpreted as showing no focal abscess, but did reveal edema of the anterior abdominal wall and of the root of the penis with scrotal swelling which was felt to be concerning for infection or Fornier's gangrene. Aggressive diuresis was performed which resulted in hypernatremia with his sodium level peaking at 153.

A June 29, 1998, CT scan of the abdomen and pelvis was interpreted as being unremarkable and Mr. Keller was discharged home June 29th with instructions to follow-up with Dr. Iacoboni. Discharge diagnoses included non-Hodkin's lymphoma; status post total body irradiation, VP16, and Cytoxan conditioning regiment for peripheral blood stem cell transplant; chemotherapy induced myelosuppression; neutropenic fevers; mucositis; hypernatremia; scrotal swelling; and fluid overload.

On June 29, 1998, Dr. Iacoboni reported receiving a call from Dr. Petersdorff advising Mr. Keller was well engrafted, afebrile, and off antibiotics. He did report severe mucositis, requiring MS Contin. On July 2, 1998, Mr. Keller saw Dr. Iacoboni in follow-up following his June 29th discharge from the University of Washington. Mr. Keller reported having a lot of difficulty swallowing and not being able to take any solids due to severe oral mucositis. He was trying to push liquids and receiving TPN ten hours every night in the meantime. He continued Oxycodone 60 mg every 8 hours sustained release and 5 mg for breakthrough pain, as well as Bactrim twice daily. Labs from June 29th showed a potassium of 4.6, creatinine of 0.9, neutrophils of 1.7, platelets of 98, and hemoglobin of 9.5. Labs obtained July 2nd showed a white count of 4.9, hemoglobin of 10.6, platelets of 182, neutrophils of 2000, potassium of 4.4, creatinine of 0.9, BUN of 36, glucose of 93, and albumin of 3.4. Dr. Iacoboni diagnosed high-grade lymphoma status post intensive therapy in the form of TBI, VePesid, and Cytoxan, followed by transplant. He also noted severe stomatitis inhibiting oral intake and uncontrolled pain. Dr. Iacoboni changed his pain regimen from Oxycodone to MS Contin 100 mg every 8 hours along with morphine sulfate immediate release (MSIR) 30 mg every one-hour PRN for pain. Leukine mouth washes were prescribed to assist with mucositis.

On July 6, 1998, Mr. Keller reported he was doing quite a bit better with decreased mouth soreness and a decreased need for pain medication. He reported taking the MS Contin every 8 hours but noted he rarely required the PRN MSIR. Labs revealed a white count of 5.9, hemoglobin of 10.8, platelets of 223, BUN of 14, creatinine of 1.0, potassium of 4.4, sodium of 139, glucose of 108, and albumin of 3.7. Dr. Iacoboni felt Mr. Keller was holding his nutritional status nicely and had excellent improvement in the stomatitis. His appetite remained diminished but was slowly improving. Dr. Iacoboni anticipated he would be off the TPN by the end of the week.

On July 13, 1998, Dr. Iacoboni noted that Mr. Keller stated his mouth was much better with only tongue pain. He reported he had stopped the morphine altogether 24 hours prior and was doing well. His TPN was being tapered with the final dose scheduled that night. He did report morning emesis after he unhooks the TPN and a need to take Ativan fairly regularly for upset stomach. He reported having dyspepsia, constipation, and nausea, and was found to be volume depleted with some weight loss present. He was advised to take Propulsid, which relieved his constipation but then caused diarrhea all night. The morning of July 14, 1998, he reported having emesis and leg cramping. Labs revealed a potassium of 4.2, BUN of 11, creatinine of 1.0, hemoglobin of 11.0, white count of 5.9, and platelets of 58. He was given three liters of D5 normal saline with 20 mEq of potassium per liter and was able to make lots of urine. He was also administered Anzemet 100 mg and Decadron 12 mg IV. He was initially also given MS 10 mg via IVP and experienced some nausea. He was switched over to Ativan and slept nicely with no further episodes of diarrhea. He was sent home with his wife with instructions to take Imodium if needed and discontinue the Propulsid.

On July 16, 1998, Mr. Keller's wife called to report that Mr. Keller had diarrhea all day and had started vomiting and complaining of lower extremity cramping. He was ordered Anzemet, Compazine, Thorazine, and Imodium. Dr. Iacoboni advised him to be liberal with the Ativan overnight and to take Morphine for the pain. He came into the office later that day and was administered two liters of D5

normal saline containing 20mEq of potassium chloride per liter. Blood cultures came back showing Staphylococcus simulans. This was felt to be a contaminant considering the rarity of the organization and the fact he had done fine without having this treated.

On July 17th, Mr. Keller came into the office stating he felt much better. He reported having a good night's sleep and eating breakfast that morning. Labs show a white count of 4.3, hemoglobin of 11.8, potassium of 2.9, and creatinine of 1.0. Because of the positive blood culture and his nausea and vomiting, Dr. Iacoboni ordered two more blood cultures. Anzemet 100 mg IV and two liters of D5 normal saline with potassium was administered, as well as 25 mg Elavil for pain and 10 mEq of Micro-K. He was prescribed potassium three times per day and advised to return on July 20th.

July 18, 1998, Dr. Iacoboni reports he was contacted being advised the blood cultures came back positive. He immediately directed Mr. Keller to the emergency room where he was seen by Dr. Newbold who pulled his line uneventfully. A progress note dated July 20, 1998, shows Mr. Keller reported feeling pretty well with return of appetite and normal bowel function. He did report some dysgeusia. Labs showed a BUN of 4, creatinine of 0.9, and potassium of 3.7. While he continued to take the Keflex that was given in the emergency room, Dr. Iacoboni elected to give him 2 grams of IV Ancef. Because his lower extremity dysesthesia was moderately severe, Dr. Iacoboni felt he would be a good candidate for Ethyol. Following Ancef administration, Dr. Iacoboni subsequently learned that the subsequent blood cultures were now growing out Staphylococcus epidermidis resistant to everything except vancomycin. Mr. Keller was advised to return the following day for Vancomycin and start Ethyol on July 22nd.

On July 28, 1998, progress note from Dr. Iacoboni reports Mr. Keller was feeling quite a bit better with improvement in his neuropathy. Mr. Keller reported taking MS Contin every 12 hours instead of every 8 hours and reported being active over the weekend. He denied nausea and stated he had not taken Thorazine for the past seven days and wanted to go to work in five days. Dr. Iacoboni opined "He seems to have had a dramatic response to the Ethyol. We can taper his morphine."

August 6, 1998, Mr. Keller was seen in follow-up with Dr. Iacoboni. Mr. Keller reported some transient benefit from the previous Ethyol treatment. He advised he had tried to go without the morphine, but his feet were burning quite a bit. He further reported being unable to tolerate any lactose-containing foods. Mr. Keller said he had returned to work on a part-time basis and was enjoying it. Dr. Iacoboni felt it was worth trying to give him more Ethyol to get a better and perhaps sustained response to his neuropathic pain. Mr. Keller was administered Anzemet, Decadron, Thorazine, Ativan, D5 normal saline, and Ethyol.

August 27, 1998, during a follow-up visit with Dr. Iacoboni, Mr. Keller reported the Ethyol he had received at his visit three weeks ago had really helped with his foot pain for 7-14 days after treatment. Over the past week, he reported having to take a lot of breakthrough morphine. Labs revealed a neutrophil count of 1800, hemoglobin of 12.5, and platelets of 174. Dr. Iacoboni noted he responded well to the Ethyol and needed to stay on it, probably every two weeks. He diagnosed adequate myeloreconstitution and significant residual neuropathy that responds to Ethyol but requires a treatment every two weeks. Dr. Iacoboni planned to call Dr. Petersdorf to see if he had any thoughts about neuropathy subsequent to the type of chemotherapy he got there, noting the Oncovin he himself had prescribed in standard dose could be playing a role in the neuropathy.

September 4, 1998, Mr. Keller was seen by Dr. Iacoboni for continued Ethyol treatment. He was premedicated with Zofran, Decadron, Thorazine, Ativan, and a liter of D5 normal saline and then

administered 1500 mg of Ethyol wide open. He was discharged ambulatory without any emesis or other adverse reaction to the treatment. At a visit on September 18, 1998, for Ethyol treatment, Mr. Keller reported his "feet feel quite a bit better." He was premedicated with Zofran, Decadron, Thorazine, Ativan, and a liter of normal saline followed by Ethyol 1500 mg wide open. The treatment was well tolerated and he was advised to return in two week for a CBC and probable Ethyol treatment.

October 1, 1998, Dr. Iacoboni saw Mr. Keller who reported being unable to cut down to one MS Contin per day per day as he was unable to go 24 hours on the one pill because of pain. He reported good pain control with 30 mg MS Contin twice a day. He reported working and having some achiness in his right collarbone after golfing. Labs showed a hemoglobin of 12.8, white count of 5.2, and platelets of 176. He was administered the Ethyol treatment which was well tolerated. An x-ray of the right clavicle was ordered and MS Contin prescription refilled. The clavicle x-ray was subsequently reported as showing a healed right clavicle fracture.

October 15, 1998, at a follow-up visit, Mr. Keller reported to Dr. Iacoboni he was feeling okay, but stated the Ethyol really wiped him out and he was not sure it was doing him any good. After discussion, it was decided to wait two weeks to see how the neuropathy was holding up and only treat him at that time if he still needed it. A prescription for 45 mg of MS Contin per day in divided doses was ordered for continued pain management.

On the November 2, 1998, follow-up visit, Mr. Keller advised Dr. Iacoboni he was felling pretty well and had been working overtime. He stated the soreness in his feet continued to slowly diminish but still required 15 mg of morphine every 12 hours. His right clavicle only ached when it was about to rain. MS Contin was continued with plans for labs and follow-up in three weeks. On December 3, 1998, Mr. Keller was once again seen by Dr. Iacoboni for reports of abdominal cramping. An x-ray of the abdomen showed quite a bit of stool throughout the abdomen, but palpation of the abdomen revealed no hepatosplenomegaly, masses, or tenderness. Dr. Iacoboni opined it could be a muscle spasm versus stool impaction and ordered Flexeril and laxatives.

On January 4, 1999, Dr. Iacoboni notes that Mr. Keller is doing great and is active at work. He stated he was trying to get off the morphine but could not go completely without it. Labs were normal. It was Dr. Iacoboni's impression Mr. Keller was doing quite well status post chemotherapy for his lymphoma with no signs of recurrent disease. Mr. Keller reported staying active. The only sequelae noted was the painful peripheral neuropathy. He was advised to follow-up in two months with labs and provided a prescription for MS Contin 15 mg.

February 17, 1999, progress note reports Mr. Keller was active, feeling well, working 70 hours per week, and traveling for work. He stated he stopped taking morphine a week ago, stating he can feel his feet, but it was not too bad. Dr. Iacoboni stated Mr. Keller was doing extremely well 12 months out from a diagnosis of a high-grade lymphoma involving the mandible, with no signs of recurrent disease and at full performance status. Prognosis remained somewhat guarded and follow-up with labs was scheduled for two months.

On February 25, 1999, Mr. Keller called Dr. Iacoboni complaining of a low-grade fever and some chest congestion, at which time a prescription for Cipro was called in. He presented to the Emergency Department on February 28, 1999, with a cough and congestion. Mr. Keller was diagnosed with bronchiolitis, felt likely secondary to viral influenza, as well as dehydration and orthostatic hypotension.

He was noted to be status post four courses of cytotoxic agents which can result in cardiac consequences. An ECG was ordered to evaluate for ejection fraction.

On June 17, 1999, Dr. Iacoboni reported Mr. Keller was 16 months out from his lymphoma diagnosis with no sign of recurrent disease. On September 30, 1999, progress note reflects Mr. Keller continued to suffer with neuropathic pain in his feet. Neurontin was prescribed. X-rays of both feet obtained November 1, 1999, were both reported negative for structural abnormality.

A December 27, 1999 progress note from Dr. Iacoboni documents Mr. Keller experienced an asthma attack while in Big Sky, Montana. He was treated locally and given Zithromax, bronchodilators, and oxygen, after his oxygen saturation levels dipped down to 84% (at 8000 feet). He opined Mr. Keller had experienced worsening of his reactive airways disease triggered by altitude, bronchitis, and a cold. Dr. Iacoboni further noted nice slow resolution of his peripheral neuropathy with no sign of recurrent lymphoma. He felt Mr. Keller's prognosis was looking excellent.

Routine follow-up visits with labs continued periodically until April 20, 2001, at which time Dr. Iacoboni noted Mr. Keller was most likely cured of his disease. Splotches on his legs were felt to be radiation skin changes. No additional follow-up was recommended, and Mr. Keller was discharged from care with instructions to return any time Dr. Berretta feels necessary. A large gap exists in the medical records for several years. Around 2006, he was reportedly diagnosed with gout. In 2009, he was diagnosed with Type 2 diabetes mellitus and iron deficient anemia. He was diagnosed with Barrett's esophagus without evidence of dysplasia in 2009. In 2010, he was diagnosed with hyperlipidemia, and in 2012, Mr. Keller was diagnosed with anemia in chronic kidney disease.

On March 11, 2014, Mr. Keller was seen in follow-up with nephrologist, Dr. Jennifer Leach, who diagnosed chronic kidney disease stage 3, "likely related to history of chemotherapy and non-Hodgkin's lymphoma, hyperglycemia/diabetes mellitus type 2, secondary hyperparathyroidism, and pseudogout. Labs showed a creatinine of 1.75, BUN of 22, sodium of 135, glucose of 223, and potassium of 3.9.

November 9, 2016, Mr. Keller was hospitalized for septic olecranon bursitis of left elbow. An x-ray of the left elbow showed a large elbow joint effusion that was felt could be related to septic arthritis. He was treated with Zosyn, Clindamycin and Vancomycin status post left elbow irrigation and debridement on November 11th, (arthroscopy of Left knee, insertion drains to left knee and left elbow). The left knee and left elbow drains were removed on November 15, 2019, and he was discharged home the following day. Mr. Keller presented to the ED on December 22, 2016, for a wound check. A wound vac was placed after his last hospital admission. Some of the wound packing was stuck inside of the wound and he was unable to remove it at home. He's had no fever, cough, or other associated symptoms. The wound vac dressing was changed, and Mr. Keller was discharged the same day.

On January 4, 2017, Mr. Keller was seen by Dr. Robert Morasch following his recent ten-day hospitalization for left olecranon bursitis. Dr. Morasch notes that Mr. Keller is now followed by rheumatology who is tapering him off the colchicine and he is scheduled for follow-up with ortho. Mr. Keller presented for orthopedic follow-up with Joshua B. Wicks, PA-C that same day. Wicks noted Mr. Keller's abscess to be decreasing in size, free of tunneling and drainage. Mr. Keller was advised to maintain his current treatment plan including the use of wound vac, continue to follow with Jean Sherman, ARNP for wound care and to return for ortho reevaluation if needed in three to four weeks. Mr. Keller was evaluated by Jean Sherman, ARNP on January 13th and January 23rd. During his January

23rd visit, Mr. Keller's left elbow wound is noted to be closed, and his wound vac and wound care orders were discontinued.

On January 31, 2017, Mr. Keller was sent to the Emergency Department at Providence St. Mary Medical Center by Dr. Morasch for abnormal labs. He presented with hypocalcemia and hypomagnesemia (calcium was low at 5.9 with ionized calcium low at 3, magnesium was low at 0.8). He had recently started a prednisone dose pack x5 days for right hand pain. Mr. Keller had an abnormal 12-lead EKG which showed: Sinus Tachycardia, Left Axis Deviation, Prolonged QTc, Non-specific T-wave abnormality, compared to EKG from November 9th, 2016, T-wave abnormality present in lateral leads, anterolateral and inferior leads. ED physician noted that the EKG was "reassuring." Since Mr. Keller was asymptomatic, he was treated with IV magnesium as well as with IV calcium. His repeat calcium was 0.7 and repeat magnesium was 1.5. Mr. Keller had an x-ray taken of his right hand which displayed no radiographic abnormality. He was discharged home with magnesium oxide and to follow up with his PCP.

On March 31, 2017, Mr. Keller was seen by rheumatologist Mandy R. Schiefelbein, ARNP for follow-up on his gout. Mr. Keller reports he continues to take his allopurinol regularly and is not aware of any gout flare ups since his last visit three months prior in December 2016. Schiefelbein notes Mr. Keller's most recent uric acid collected on January 31, 2017, was on target at less than 5.8. Current gout medications listed for visit include allopurinol 300mg daily and 0.6mg colchicine 0.6mg daily. ROS is negative for joint swelling and arthralgia. Schiefelbein ordered repeat uric acid during visit with noted plan to gradually wean Mr. Keller off colchicine if his uric acid remained on target. She further recommends Mr. Keller continue to follow with nephrology for management of his chronic kidney disease and to have him return to the rheumatology clinic in four months.

April 30th, 2017, Mr. Keller was admitted at Providence Sant Mary Medical Center for acute hypoxemic respiratory failure secondary to asthma exacerbation related to right lower lobe pneumonia and Influenza A, sepsis, and acute kidney injury superimposed on his stage 3 chronic kidney disease. He was treated with Azithromycin, Rocephin, Tamiflu, and IV fluid hydration and discharged three days later on May 3rd, 2017. Labs performed on April 30 show Creatinine of 2.25. On May 1st, his creatine was 2.16 and then ultimately improved to 1.97 by Mr. Keller's date of discharge on May 3rd. Mr. Keller is seen by PMG SE WA Internal Medicine Physician, Dr. Robert Morasch on May 9th, 2017, where Mr. Keller's pneumonia is noted as resolved and a return to his baseline. Dr. Morasch ordered Mr. Keller to return in one week.

On May 11th, 2017, eight days following Mr. Keller's pneumonia hospitalization discharge, he presented to PMG SE WA Urgent Care Southgate for acute swelling and tenderness of the right elbow, right index finger, and left knee. Mr. Keller denied recent acute injury or fall. Labs performed at the urgent care visit show a WBC of 13.8, eGFR of 43, BUN of 21, Creatinine of 1.70, BUN/Creatinine ratio of 12.4, Uric Acid of 5.2, ESR of 104, and CRP of 26.29. Mr. Keller was evaluated by Dr. Eric Schwartzkopf, MD and subsequently diagnosed with acute gout due to renal impairment involving right hand, olecranon bursitis of right elbow, prepatellar bursitis of left knee, and unspecified leukocytosis. Dr. Schwartzkopf's plan of treatment included colchicine for the acute gout flare-up, further labs to include blood cultures, and for Mr. Keller to follow up with Dr. Morasch to review the results of his bloodwork.

On May 17th, 2017, Mr. Keller presented to for follow-up with Dr. Robert Morasch at PMG SE WA. Mr. Keller's musculoskeletal ROS was noted to be negative for myalgias and swelling and positive for "some joint pain". Dr. Morasch writes Mr. Keller looks and feels at his baseline and labs were reviewed with Mr.

Keller and "acceptable". Mr. Keller was instructed to return to the clinic in two months for follow-up and to continue his current medical regimen of allopurinol, glimepiride, Januvia, insulin glargine, omeprazole, simvastatin, and Lutein.

July 31, 2018, Dr. Robert Morasch notes that Mr. Keller's labs were reviewed with him and his renal functioning "has been worsening over the last year" and that it was time for a follow up with nephrology. Dr. Morasch notes Mr. Keller will work on diet and continue to follow with endocrine and suggests follow-up in three months. On September 2, 2018, Mr. Keller underwent renal ultrasound testing. The report impression lists the findings as consistent with chronic medical renal disease, no hydronephrosis present, and no significant post void urinary bladder residual.

September 28, 2018, Mr. Keller was seen by Rheumatologist Mandy Schiefelbeinfor continued management of his gout. Mr. Keller reported no overall health changes since his last rheumatology visit six months earlier in March of 2018. Musculoskeletal examination showed no synovitis, tenderness, soft tissue swelling or instability in any of Mr. Keller's joints. Mr. Keller reports current adherence and success with his medication regimen of allopurinol 300 mg daily and colchicine 0.6 mg 1-3 tabs PRN and denies gout flare/attack. Labs performed on April 14th, 2018, were reviewed by Dr. Scheiefelbein at this visit who noted Mr. Keller had a 4.6 uric acid level at that time. Dr. Scheiefelbein recommended Mr. Keller continue his current allopurinol regimen, present for lab testing in one week to reassess uric acid, and return to the clinic in six months.

On October 9, 2018, Mr. Keller was seen by Dr. Jennifer Leach for a nephrology consultation following a referral by Dr. Morasch for the evaluation of chronic kidney disease and noted decline in renal functioning from that year. Dr. Leach notes in the HPI that Mr. Keller was last seen four years prior on March 11, 2014, at which time Mr. Keller's serum creatinine was 1.75 and his eGFR was 42. Mr. Keller denied acute illness and current nephrotoxin use and reported the rare use of NSAIDs for headaches. Dr. Leach noted Mr. Keller's last A1C was less than 7% and he has been reportedly able to optimize glycemic control. Renal ultrasound imaging performed last month was reviewed by Dr. Leach who noted the ultrasound showed echogenic kidneys without obstructive nephropathy. Labs performed on October 2, 2018, were reviewed with Mr. Keller and showed a serum Creatinine of 2.19, uric acid of 3.9, GFR of 32, BUN of 35, BUN/Creatinine ratio of 16.0, random urine Creatinine of 84, urine protein of 105, and the presence of a small amount of blood in his urine. Dr. Leach noted greater proteinuria and decline in kidney functioning compared to Mr. Keller's last nephrology visit in 2014, and further stated "given overt proteinuria, will start ACEI therapy." Mr. Keller was instructed to start on lisinopril 2.5 mg once daily, restrict dietary intake of protein to less than 70 g per day, and return to the nephrology clinic in six months.

Mr. Keller continued to see Dr. Leach periodically in follow-up for his chronic kidney disease. On a May 21, 2019, office visit, labs were significant for an elevated creatinine of 2.33 and BUN of 42, with an estimated GFR of only 30. At a November 26, 2019, office visit, Mr. Keller was noted to be bradycardic with a heart rate of 48, which was asymptomatic. An EKG was recommended in the future if bradycardia persists.

May 26, 2020, Dr. Jennifer Leach saw Mr. Keller in follow-up for chronic kidney disease, stage III. He reported no longer requiring insulin, advising he had transitioned to a new diabetes medication, Rybelsus, which he had been taking for about 80 days. Mr. Keller reported one recent episode of a mild gout attack, which was successfully treated with colchicine. Labs revealed elevated liver enzymes and an

elevated erythrocyte sedimentation rate of 42. Mr. Keller's creatinine was 2.63, and BUN was 42. Dr. Leach diagnosed sub-nephrotic proteinuria in the setting of chronic kidney disease, stage IV. Additional diagnoses included hypomagnesemia, for which a Mg supplement at 400-500 mg daily was ordered. He was advised to follow-up in six months.

On November 17, 2020, Mr. Keller was seen in follow-up by nephrologist, Dr. Jennifer Leach. Labs showed a sodium of 133, glucose of 248, BUN of 44, creatinine of 2.35, and magnesium of 1.1. Diagnoses included proteinuric chronic kidney disease, felt to be due to diabetic kidney disease. Kidney function was found to be at baseline. He was also diagnosed with hypomagnesemia, which Dr. Leach felt was likely due to renal magnesium wasting due to diabetic kidney disease. Supplementation was ordered.

March 27, 2021, Mr. Keller was admitted to Providence St. Mary Medical Center for syncope and collapse. Upon his arrival on March 27th, Mr. Keller was found to have a critical Magnesium level of 1.0, and an elevated troponin of 0.07. He was found to be in atrial flutter with slow ventricular heart response with recurrent episodes of pauses. He underwent biphasic DC cardioversion on March 31st. Following the cardioversion, Mr. Keller underwent successful pacemaker placement on April 1, 2021, due to the presence of a Mobitz Type 2 heart block with Dr. Arthur C. Lee, MD and was discharged home on April 1, 2021. Mr. Keller received a Boston Scientific Pacemaker, model number L331. During this hospitalization, Mr. Keller's labs continued to reflect a decline in his renal functioning – initial labs drawn on the 27th show a BUN of 47, Creatinine of 2.90, eGFR of 23, BUN/Creatinine ratio of 16.2.

May 25, 2021, Dr. Jennifer Leach saw Mr. Keller in follow-up. Diagnoses included chronic kidney disease stage 4, with a GFR of 15-29 ml/min, hypomagnesemia, abnormal liver function tests with an elevated AST of 42 and an elevated ALT of 64. His albumin level was 5.0, while his alkaline phosphatase level was elevated at 120. His erythrocyte sedimentation rate was elevated at 38, uric acid was decreased at 3.2, and magnesium was decreased at 1.0.

On September 8, 2021, Mr. Keller presented to Providence St. Mary Medical Center and was diagnosed with pacemaker lead displacement with suboptimal readings of ventricular lead. Cardiology consultation note by Dr. Arthur C. Lee from September 19th notes Mr. Keller has done well since his pacemaker placement in April, however, follow-up interrogation showed lead displacement and poor readings.

On June 7, 2022, Mr. Keller presented to his nephrologist Dr. Jennifer Leach for follow-up of his chronic kidney disease. Mr. Keller reported he was experiencing an upper respiratory illness with exacerbation of asthma and that he was recently seen at an urgent care two days earlier on June 5th where he was prescribed a Medrol pack, which had improved his breathing and oxygenation levels. Dr. Leach noted Mr. Keller was scheduled for an echocardiogram that week. Mr. Keller denies presyncopal episodes or syncopal episodes at this visit and reported to Dr. Leach that he had a high resting heart rate usually at "around 90 bpm." Dr. Leach reviewed recent lab work performed on June 2nd with Mr. Keller at this visit. Labs showed a red blood cell count of 3.86, hemoglobin of 11.4, hematocrit of 36.0, BUN of 46, creatinine of 2.66, eGFR of 27, magnesium of 1.3, and uric acid of 4.6. Urinalysis showed the presence of 30 mg of protein, a small amount of blood, and epithelial cells. Dr. Leach recommend continuing lisinopril of 2.5 mg and allopurinol of 200 mg in response to his chronic kidney disease.

On June 24, 2022, Mr. Keller presented to St. Mary Medical Center's Emergency Department the, reporting persistent upper respiratory infection symptoms for which he has had three visits for over the past month, with complaints of generalized body aches, chills, and overnight elevated temperatures. Mr.

Keller's initial vitals show he was fevered with a temperature of 101.6, tachycardic with a heart rate of 109, and hypotensive with a blood pressure of 89/69. Labs performed on June 24th show an elevated WBC count of 38.6, Procalcitonin of 2.60, BUN of 76, and Creatinine of 3.39. Mr. Keller was hospitalized from June 24, 2022, to June 28, 2022, with sepsis, community-acquired pneumonia, and acute on chronic kidney injury. Pt was treated with ceftriaxone and azithromycin as inpatient and discharged on cefdinir. CT revealed a possible mass. He was scheduled for a repeat chest CT. His serum creatinine peaked at 3.84 mg/dL (baseline 2.5-2.7 mg/dL).

July 19, 2022, Dr. Leach saw Mr. Keller for follow-up for his stage IV chronic kidney disease. She diagnosed acute renal failure superimposed on stage 4 chronic kidney disease, sustained in the setting of sepsis and volume depletion. His latest creatinine was 3.09 (baseline 2.5-2.7). Electrolytes had improved but examination revealed Mr. Keller was more edematous. Dr. Leach ordered Lisinopril to be held and restarted furosemide 80 mg daily with a plan to recheck labs in one month. Additional diagnoses included diabetic neuropathy, hyponatremia, and anemia in stage 4 chronic kidney disease.

November 4, 2022, Mr. Keller was hospitalized at Providence St. Mary Medical Center until November 11, 2022, for shortness of breath, and was found to have acute diastolic heart failure. A chest x-ray was negative, but an ultrasound revealed a significant pleural effusion. Mr. Keller responded well to Lasix and Metolazone and lost 26 pounds over 5 days. Notably, Mr. Keller's creatinine increased to 5.10 during the hospitalization, only slightly improving prior to discharge. He was discharged home on PO Lasix and Metolazone with instructions to start outpatient care with cardiology and his primary care physician, and to follow-up with his nephrologist. Discharge diagnoses included acute on chronic congestive heart failure with reduced ejection fraction, acute kidney injury on chronic kidney disease stage IV, hyperphosphatemia, reactive leukocytosis, and acute hypomagnesemia.

On January 17, 2023, Mr. Keller was seen by Dr. James Otis Mudd of Providence Center for Advanced Heart Disease and Transplantation Outpatient Clinic, who noted chronic systolic heart failure, stage C, due to ischemic cardiomyopathy, coronary artery disease with stent placement on October 25, 2022, in the LAD and proximal RCA, diabetes mellitus type 2, atrial fibrillation/flutter, heart block with dual-chamber pacemaker, and chronic kidney disease. The etiology of cardiomyopathy was felt to be likely ischemic, however the possibility of it being caused by the chemotherapy for non-Hodgkin's lymphoma and treatment with unknown chemotherapeutic agents. Mr. Keller reported starting cardiac rehab this month, but reported they were unable to titrate medications due to low blood pressure. An echocardiogram obtained the previous day was interpreted as demonstration an ejection fraction of 25-30% with global hypokinesis with normal right ventricular size and function, calcified aortic valve with normal opening and trace insufficiency, calcified mitral valve with mild central regurgitation, and moderate TR with an RVSP of 55-60 mmHg. Mr. Keller reported feeling well and being able to perform all of his activities of daily living.

March 10, 2023, Mr. Keller presented to Providence St. Mary Medical Center for outpatient infusion of ferric carboxymaltose (Injectafer) as ordered by Dr. Leach for his iron deficiency anemia. Mr. Keller tolerated the infusion without complication and was discharged home.

On June 20, 2023, Mr. Keller presented to Providence St. Mary Medical Center for concern of pacemaker lead failure following the surgical placement of his Boston ICD G139 Momentum X4 on June 12, 2023. Mr. Keller's chest X-ray impression from June 20th, notes the "left chest triple lead cardiac device appears unchanged."

On August 11, 2023, Mr. Kellers was admitted to Providence St. Mary Medical Center with a diagnosis of enterocolitis and sepsis, after presenting with complaints of diarrhea after visiting Australia. He was discharged August 14, 2023, with instructions to complete the Ciprofloxacin and Metronidazole. An October 5, 2023, echocardiogram obtained at Providence St. Mary Medical Center was interpreted as showed moderately reduced systolic function in the left ventricle with an estimated left ventricle ejection fraction of 35%. No significant change was noted when compared to the prior examination of April 4, 2023.

On February 20, 2024, saw his PMD for review of lab work. At this visit, Dr. Oleksandr Selevertov notes Mr. Keller had started dialysis for his chronic kidney disease "2 weeks ago." A vascular ultrasound was performed at St. Mary Medical Center on March 1, 2024, for upper extremity vein mapping in case an arteriovenous fistula was needed. Mr. Keller underwent surgical creation by Dr. Mun J. Po, MD, of an arteriovenous anastomosis to his left arm on March 19, 2024, at St. Mary Medical Center.

On March 29, 2024, Mr. Keller presented to Dr. Oleksandr Seleverstov for postoperative follow-up after the surgical placement of his arteriovenous fistula. Normal findings from Dr. Seleverstov's assessment reveal Mr. Keller's left upper extremity surgical incision to be healing, palpable thrill, and good vascular flow to the fistula and wrist arteries and free from clot formation. However, Dr. Seleverstov notes that Mr. Keller has a concerning amount postoperative edema and cyanosis below the left elbow despite elevating the arm and writes that Mr. Keller may need vascular reevaluation and a compression sleeve. Dr. Seleverstov orders include follow-up visit.

April 10, 2024, Mr. Keller was admitted to Providence St. Mary Medical Center by Dr. Steve Mazyck, MD for AV fistula infection/cellulitis of the left antecubital area. He was initially started on Zosyn/Vancomycin. Wound cultures and staining performed on April 10th revealed the growth of MSSA and Mr. Keller was then switched to Ceftriaxone/Vancomycin on Aprill 11th. Vascular surgery and nephrology were consulted and recommended Mr. Keller discharge home on cephalexin 500mg twice daily, and for him to receive an additional dose of Vancomycin following his scheduled hemodialysis on April 13th. Mr. Keller was discharged home on April 12, 2024, with a plan for follow-up to vascular surgery clinic in one week.

On April 19, 2024, Mr. Keller returned for his three-week postoperative follow-up with Dr. Seleverstov. Mr. Keller's documented vitals were within normal limits and show he was afebrile. Mr. Keller's left arm edema is noted to be resolved, however, a 2 mm abscess with draining fluid formed at the surgical incision site and a faint thrill was noted at the fistula. During doppler flow testing of Mr. Keller's left arm at this visit, a 1 cm irregular fluid collection at 1-2 cm depth beneath the draining sinus was assessed. Dr. Seleverstov's writes for Mr. Keller to notify his surgeon ASAP, continue to take the antibiotic, obtain an OTC probiotic, obtain a tourniquet from the pharmacist, and monitor himself for diarrhea, fever, and chills. Dr. Seleverstov ordered follow-up in one week.

The treatment that Mr. Keller received relating to his NHL was appropriate and reasonable.

2. Past Medical History

In 1999, Mr. Keller had noted x-rays for neuropathic pain. In 2009, he was diagnosed with Diabetes Type 2, fibroepithelial skin tags of the buttock and thigh requiring surgical removal, iron deficient anemia, and Barrett's esophagus without dysplasia. The following year in 2010, he was diagnosed with

hyperlipidemia. In 2012, several diagnoses were made: anemia in chronic kidney disease, secondary hyperparathyroidism, and hyperuricemia. In 2014, Mr. Keller was diagnosed with CKD3 "likely due to hx of chemo and HL", macular degeneration (dry), peripheral neuropathy, and elevated liver function. In 2015, asthma was diagnosed. In 2016 he was diagnosed with: colon polyps, OSA with CPAP (noted after weight loss no longer using CPAP), basal cell carcinoma, CKD3, staphylococcal arthritis of the left knee, gout due to renal impairment in the left elbow, and obesity class II (BMI 35-39.9). Lastly, in 2017, hypocalcemia syndrome was diagnosed.

3. Family History

Mr. Keller's mother, a former smoker, was diagnosed with breast cancer, a malignant tumor of the lung (lung cancer), diabetes, and dementia. She also suffers from arthritis. His maternal grandmother died of breast cancer at age 92. His maternal grandfather died of congestive heart failure in his mid-60's.

Mr. Keller's father was diagnosed with colon cancer, prostate cancer, diabetes, hyperlipidemia, and obesity. He also suffers from arthritis. His paternal grandmother died after contracting pneumonia at age 94, shortly after breaking her hip. His paternal grandfather was diagnosed with prostate cancer (active at time of death) and dementia and died at age 93.

4. Social History

Mr. Keller consumed alcohol moderately, with an intake of approximately one can of beer per week. He had a period of heavy drinking when he was a younger man.

Mr. Keller has a military record from Camp Lejeune where he marked off that he was exposed to asbestos. However, he does not recall filling out the document and does not recall ever being exposed to asbestos. (Keller Dep. 99:6-99:24)

Mr. Keller's medical record indicates that he was exposed to radiofrequency (RF) radiation during his military service, specifically from working around radar systems. He reported that he was not required to wear radiation badges nor was he instructed to report to medical for radiation exposure. (Keller Dep. 102:13-104:3)

Mr. Keller has a history of woodworking as a hobby, including building cabinetry, flooring, and working on pinewood derbies with his children. He consistently used safety measures during these activities. Specifically, Mr. Keller wore a mask while woodworking and used only latex-based paints. He was not exposed to solvents or other hazardous substances during his woodworking projects. (Keller Dep. 124:3-125:1)

5. Prognosis and Future Care

Mr. Keller has a history of NHL treated with chemotherapy and SCT and now has CKD and cardiomyopathy. He is in remission from NHL. Because of that treatment, Mr. Keller has an increased risk of future hematopoietic cancers, other non-hematopoietic cancers, late recurrence of cancer, and other benzene associated cancers. He is also at risk of post-treatment chronic conditions, and in fact he likely contracted severe kidney disease as a result of his NHL treatment. He will need continued long-term follow-up and management for the future increased risk of cancer. In addition, because of his treatment

with chemotherapy and SCT, he has severe kidney disease, and significant cardiomyopathy, both of which will require long term treatment and management, overall reduce his function, increase his of morbidity and mortality. His NHL and its treatment are expected to reduce his overall life expectancy.

6. Risk Factors for NHL as Relates to Mr. Keller

In performance of a differential etiology, I considered the medical and scientific literature as well as reviewed the medical records and deposition testimony as well as other medical records to determine whether Mr. Keller had risk factors relating to his NHL and, of so, which risk factor(s).

Mr. Keller is a male. Being male in itself is a risk factor but not known to be a cause of NHL; it is not an independent cause of cancer.

Mr. Keller was only 31 years old at the time of his NHL diagnosis, well below the age at which incidences of NHL rise.

Mr. Keller has a family history that includes cancer but not hematopoietic cancer or NHL. Family history of cancer is not a cause of cancer but can be associated with the increased susceptibility to cancer and cancers caused by carcinogens. Hence, even if Mr. Keller has a familial susceptibility to cancer this would not have been a cause of his cancer but instead have increased his susceptibility to carcinogenesis.

Mr. Keller also has a history of obesity. Obesity does increase the risk of many diseases including cancer, so his obesity might have increased his risk for cancer. Obesity is a risk factor for cancer and NHL, by mechanisms including causing inflammation and impeding the immune system. In Mr. Keller, obesity at least as likely as not increased his susceptibility to cancer by influencing the ability to suppress carcinogenic processes. Obesity may increase susceptibility to environmental carcinogenesis.

Mr. Keller also used alcohol as a younger man. Alcohol can be a risk factor for many diseases including cancer but has not been generally associated with hematopoietic cancers and NHL. Mr. Keller's history of use of alcohol as a young man is not likely to have caused his NHL or not likely to have contributed to the onset of his NHL.

Mr. Keller was exposed to RF radiation. RF radiation possibly is associated with an increase in some types of cancer, but it has not been associated with NHL. In Mr. Keller, RF radiation is unlikely to be a risk factor for his NHL.

Mr. Keller was also exposed to tobacco smoke as child. Tobacco smoke exposure is known to be a cause of cancer and exposure increases the risk of cancer generally and has been associated with a risk of some types of NHL. In Mr. Keller, exposure to tobacco smoke as a child is unlikely to be risk factor for his NHL.

Asbestos is a known carcinogen and can be associated with some types of cancer risk but has not been generally associated with NHL. Further, Mr. Keller does not recall ever being exposed to asbestos. Keller Dep. 99:6-99:24.

Mr. Keller did woodworking that can be associated with exposure to carcinogens, but this is unlikely to be a contributing cause of his cancer.

Mr. Keller was diagnosed with diabetes after his diagnosis of NHL, diabetes can be associated with an increase risk of cancer, however, the timing suggests that it is unlikely to be a contributing cause.

Mr. Keller has a family history that includes cancer, but has no known familial susceptibility syndrome. Although I cannot rule out a familial susceptibility, there is no evidence for this, and familial susceptibility syndromes are not causes of cancer per se but increase the risk exposure to environmental carcinogens would be a cause of his cancer.

Finally, and of most significance, Mr. Keller was exposed to benzene at Camp Lejeune. Benzene exposure is a known cause of hematopoietic cancers including NHL, as described above. Mr. Keller's exposure to benzene was medically more likely that not a significant contributing cause of his NHL.

7. Damages

Mr. Keller has NHL that required significant treatment with chemotherapy and a SCT that involved substantial hospitalization, lost work, and loss of enjoyment of life and is a significant morbidity and an increased risk of mortality. As a consequence of his NHL treatment, he has CKD and worsened CM, both of which will further reduce his overall life expectancy and require further medical treatment.

B. Exposure History

1. Time at Camp Lejeune

Mr. Scott Keller served in the United States Marine Corps from January 1985 through December 1997, during which he reenlisted twice. He was stationed at Camp Lejeune from November 1985 to January 1988, primarily residing on base until September 1987. His work was concentrated at the communications building near the Delta Battery barracks, where he was assigned to Delta Battery, 2nd Battalion, 10th Marines of the 2nd Marine Division. These details are documented in the Keller Deposition (pages 41:1 to 60:20) and supported by deposition exhibits 2, 4, 5, and 6.

Throughout his time at Camp Lejeune, Mr. Keller was exposed to the water supply at Hadnot Point, where he lived, worked, ate, and maintained personal hygiene until his marriage in September 1987. After his marriage, he relocated off-base to Jacksonville, NC, but continued his daily base activities, including working, eating, and often showering before leaving the base as he was required to change out of his uniform before commuting (Keller Deposition 45:11-20, 136:6-17).

His recreational activities also contributed to his exposure to the water at Hadnot Point. Mr. Keller frequently used the base's fieldhouse for sports such as racquetball and soccer, and he participated in wrestling practice three to four times weekly depending on the season (Keller Dep. 160:21-161:4, 167:9-167:20).

Mr. Keller's military occupational specialties at Camp Lejeune were as a radio operator and Morse code operator. (Keller Dep. 72:23-73:1) Occasionally on the weekends, he was assigned fire watch duty. During this duty, he remained at the barracks for 24 hours, alternating between being on watch and resting every six hours, without permission to leave. He was assigned this duty once to twice a month, typically on weekends. (Keller Dep. 138:13-139:12)

During his service, Mr. Keller participated in field training on the base approximately twice a month. These training sessions varied in length, ranging from several days to two weeks, during which he did not return to the barracks. Water needs in the field were met using "water buffaloes" (large mobile water containers), and he consumed meals ready to eat (MREs), which involved rehydrating food with this same water supply.

Mr. Keller estimated he would drink 2-4 canteens of water per day, each canteen holding one quart. In hotter conditions, his consumption could reach up to two and a half gallons per day, including water and Kool-Aid (Keller Dep. 140:2-140:14).

When factoring deployments and other times off base, Mr. Keller was exposed to the water at Hadnot Point for approximately 525 days.

Exposure Start	Exposure End	Total Days	Benzene [µg/L]
11/30/1985	11/30/1985	1	3
12/1/1985	12/31/1985	31	3
1/1/1986	1/31/1986	31	3
2/1/1986	2/28/1986	28	3
3/1/1986	3/7/1986	7	3
3/19/1986	3/31/1986	13	3
4/1/1986	4/30/1986	30	4
5/1/1986	5/31/1986	31	3
6/1/1986	6/30/1986	30	3
7/1/1986	7/31/1986	31	3
8/1/1986	8/12/1986	12	3
9/20/1986	9/30/1986	11	3
10/1/1986	10/31/1986	31	3
11/1/1986	11/30/1986	30	3
12/1/1986	12/4/1986	4	3
12/17/1986	12/31/1986	15	3
1/1/1987	1/20/1987	20	2
7/16/1987	7/31/1987	16	3
8/1/1987	8/31/1987	31	3
9/1/1987	9/30/1987	30	3
10/1/1987	10/31/1987	31	3
11/1/1987	11/30/1987	30	2
12/1/1987	12/31/1987	31	2
	Total days:	**525**	

In Dr. Kelly Reynolds' report, she estimated the ingestion exposure of Mr. Keller. The contaminants at Camp Lejeune, as stated above, are volatile organic compounds and as a result Mr. Keller experienced

significant exposure through inhalation and dermal routes. To a reasonable degree of medical and scientific certainty that exposure increased both the total exposure as well as the cancer risk.

C. Camp Lejeune Water Contamination

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

> The reconstructed duration of contamination of finished water exceeding the current MCL for benzene was 63 months (January 1979–November 1984) (Table 7.16); the maximum reconstructed finished water concentration of benzene was about 12 µg/L during April 1984 (Table 7.16). Measured benzene concentrations at the HPWTP ranged from below detection limits (10 µg/L) to 38 µg/L during the period December 1984– December 1985. An unexplained value of 2,500 µg/L of benzene was measured on November 11, 1985 (Table 7.16).

Additionally, I have examined Appendix J of the same report, which contains the ATSDR water modeling tables. These tables provide detailed monthly mean contaminant concentrations for Hadnot Point, expressed in micrograms per liter in finished water. The concentration levels from the ATSDR and Maslia report for the dates Mr. Keller was on base is reflected in the table above.

VII. CONCLUSION

I conclude with a reasonable degree of medical certainly, after review of Mr. Keller's medical records, other expert reports and documents and performing a differential etiology, using well established methods that are generally accepted, and after consideration and review of the medical and scientific literature that more likely than not his exposure to benzene from contaminated drinking water at Camp Lejeune was a significant contributing cause of his NHL.

Mr. Keller was exposed to drinking water at Camp Lejeune between 1985 and 1987 exposing him to 2-4 PPB of benzene and he was diagnosed at age 31 of NHL. Benzene has been shown to be a cause of NHL, exposure of benzene during this time at this exposure are medically more likely than not a significant contributing cause of his NHL.

Mr. Keller is young for this disease and this is consistent with an environmental exposure being a cause of his cancer. He has some family history of cancer, but no family history of hematopoietic cancers so there is no evidence for a familial susceptibility. He has as ~~a~~n environmental exposure his being exposed to benzene in the contaminated drinking while based in Camp Lejeune that, more likely than not can be a cause of hematopoietic, lympho-hematopoietic cancers including NHL.

He does not have a known history of smoking, but was exposed as a child, does have a prior history of use of alcohol, does not have a history of exposure to ionizing radiation exposure but was exposed to RF radiation. Exposure to tobacco smoke may be associated with some types of NHL, but his childhood exposure is unlikely to be a contributing cause. His exposure to RF radiation is not known to be a cause of NHL. Alcohol use is a risk for some types of cancer, but unlikely to be a cause of NHL in Mr. Keller.

He is over-weight and this may generally increase his susceptibility to cancer and would have increased his risk upon a carcinogenic exposure.

His cancer does have evidence for multiple genetic abnormalities that can be caused by exposure to carcinogens, including the VOCs such as benzene in the contaminated drinking water ~~of~~ at Camp Lejeune.

Mr. Keller has NHL treated with chemotherapy and SCT and now has CKD and cardiomyopathy. Although he is now in remission from NHL, because of history of NHL and the treatment of his disease, Mr. Keller has an increased risk of future hematopoietic cancers, other non-hematopoietic cancers, late recurrence of cancer, as well as other benzene associated cancers. He is also at risk of post-treatment chronic conditions, including progression of his severe kidney disease that was a result of his NHL treatment and significant cardiomyopathy, both of which will require long term treatment and management, overall reduce his function, increase his of morbidity and mortality.

His NHL and its treatment are expected to reduce his overall life expectancy.

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

VIII. REFERENCES

1. Bryder D, Rossi D, Weissman I. Hematopoietic stem cells: The paradigmatic tissuespecific stem cell. *Am J Pathol*. 2006;169:338-346. doi:10.2353/ajpath.2006.060312

2. Filipek-Gorzała J, Kwiecińska P, Szade A, Szade K. The dark side of stemness – the role of hematopoietic stem cells in development of blood malignancies. *Front Oncol*. 2024;14. doi:10.3389/fonc.2024.1308709

3. Greaves MF, Wiemels J. Origins of chromosome translocations in childhood leukaemia. *Nat Rev Cancer*. 2003;3(9):639-649. doi:10.1038/nrc1164

4. Küppers R. Mechanisms of B-cell lymphoma pathogenesis. *Nat Rev Cancer*. 2005;5(4):251-262. doi:10.1038/nrc1589

5. Weigert O, Weinstock DM. The evolving contribution of hematopoietic progenitor cells to lymphomagenesis. *Blood*. 2012;120(13):2553-2561. doi:10.1182/blood-2012-05-414995

6. Weniger MA, Seifert M, Küppers R. B Cell Differentiation and the Origin and Pathogenesis of Human B Cell Lymphomas. In: Küppers R, ed. *Lymphoma: Methods and Protocols*. Springer US; 2025:1-30. doi:10.1007/978-1-0716-4188-0_1

7. Weissman IL, Shizuru JA. The origins of the identification and isolation of hematopoietic stem cells, and their capability to induce donor-specific transplantation tolerance and treat autoimmune diseases. *Blood*. 2008;112(9):3543-3553. doi:10.1182/blood-2008-08-078220

8. de Leval L, Jaffe ES. Lymphoma Classification. *Cancer J*. 2020;26(3):176. doi:10.1097/PPO.0000000000000451

9. Singh R, Shaik S, Negi BS, et al. Non-Hodgkin's lymphoma: A review. *J Fam Med Prim Care*. 2020;9(4):1834-1840. doi:10.4103/jfmpc.jfmpc_1037_19

10. Pasqualucci L, De Simone P, Dalla-Favera R. Chapter 14 - Molecular Pathogenesis of B-Cell Lymphomas. In: Honjo T, Reth M, Radbruch A, Alt F, Martin A, eds. *Molecular Biology of B Cells (Third Edition)*. Academic Press; 2024:309-333. doi:10.1016/B978-0-323-95895-0.00020-9

11. Cerhan JR, Slager SL. Familial predisposition and genetic risk factors for lymphoma. *Blood*. 2015;126(20):2265-2273. doi:10.1182/blood-2015-04-537498

12. Kurz KS, Ott M, Kalmbach S, et al. Large B-Cell Lymphomas in the 5th Edition of the WHO-Classification of Haematolymphoid Neoplasms—Updated Classification and New Concepts. *Cancers*. 2023;15(8):2285. doi:10.3390/cancers15082285

13. Koff JL, Chihara D, Phan A, Nastoupil LJ, Williams JN, Flowers CR. To Each Its Own: Linking the Biology and Epidemiology of NHL Subtypes. *Curr Hematol Malig Rep*. 2015;10(3):244-255. doi:10.1007/s11899-015-0267-0

14. Szmyd B, Mlynarski W, Pastorczak A. Genetic predisposition to lymphomas: Overview of rare syndromes and inherited familial variants. *Mutat Res Mutat Res*. 2021;788:108386. doi:10.1016/j.mrrev.2021.108386

15. Ekström Smedby K, Vajdic CM, Falster M, et al. Autoimmune disorders and risk of non-Hodgkin lymphoma subtypes: a pooled analysis within the InterLymph Consortium. *Blood*. 2008;111(8):4029-4038. doi:10.1182/blood-2007-10-119974

16. Grulich AE, Vajdic CM, Cozen W. Altered immunity as a risk factor for non-Hodgkin lymphoma. *Cancer Epidemiol Biomark Prev Publ Am Assoc Cancer Res Cosponsored Am Soc Prev Oncol*. 2007;16(3):405-408. doi:10.1158/1055-9965.EPI-06-1070

17. Engels EA. Infectious Agents as Causes of Non-Hodgkin Lymphoma. *Cancer Epidemiol Biomarkers Prev*. 2007;16(3):401-404. doi:10.1158/1055-9965.EPI-06-1056

18. Harbron RW, Pasqual E. Ionising radiation as a risk factor for lymphoma: a review. *J Radiol Prot*. 2020;40(4):R151. doi:10.1088/1361-6498/abbe37

19. Smith MT, Jones RM, Smith AH. Benzene exposure and risk of non-Hodgkin lymphoma. *Cancer Epidemiol Biomark Prev Publ Am Assoc Cancer Res Cosponsored Am Soc Prev Oncol*. 2007;16(3):385-391. doi:10.1158/1055-9965.EPI-06-1057

20. Vlaanderen J, Lan Q, Kromhout H, Rothman N, Vermeulen R. Occupational benzene exposure and the risk of lymphoma subtypes: a meta-analysis of cohort studies incorporating three study quality dimensions. *Environ Health Perspect*. 2011;119(2):159-167. doi:10.1289/ehp.1002318

21. Steinmaus C, Castriota F, Ferreccio C, et al. Obesity and excess weight in early adulthood and high risks of arsenic-related cancer in later life. *Environ Res*. 2015;142:594-601. doi:10.1016/j.envres.2015.07.021

22. Rana I, Dahlberg S, Steinmaus C, Zhang L. Benzene exposure and non-Hodgkin lymphoma: a systematic review and meta-analysis of human studies. *Lancet Planet Health*. 2021;5(9):e633-e643. doi:10.1016/S2542-5196(21)00149-2

23. Ge C, Spoerri A, Egger M, et al. Occupational exposure to benzene and mortality risk of lymphohaematopoietic cancers in the Swiss National Cohort. *Scand J Work Environ Health*. 50(5):351-358. doi:10.5271/sjweh.4164

24. Bassig BA, Shu XO, Friesen MC, et al. Occupational exposure to benzene and risk of non-Hodgkin lymphoma in an extended follow-up of two population-based prospective cohorts of Chinese men and women. *Int J Cancer*. 2024;155(12):2159-2168. doi:10.1002/ijc.35095

25. Program NT. NTP Listing Recommendation. In: *Report on Carcinogens Monograph on Trichloroethylene: RoC Monograph 05 [Internet]*. National Toxicology Program; 2015. Accessed February 3, 2025. https://www.ncbi.nlm.nih.gov/books/NBK575908/

26. Catalani S, Donato F, Madeo E, et al. Occupational exposure to formaldehyde and risk of non hodgkin lymphoma: a meta-analysis. *BMC Cancer*. 2019;19:1245. doi:10.1186/s12885-019-6445-z

27. Zhang L, Rana I, Shaffer RM, Taioli E, Sheppard L. Exposure to glyphosate-based herbicides and risk for non-Hodgkin lymphoma: A meta-analysis and supporting evidence. *Mutat Res Mutat Res*. 2019;781:186-206. doi:10.1016/j.mrrev.2019.02.001

28. Davoren MJ, Schiestl RH. Glyphosate-based herbicides and cancer risk: a post-IARC decision review of potential mechanisms, policy and avenues of research. *Carcinogenesis*. 2018;39(10):1207-1215. doi:10.1093/carcin/bgy105

29. Assessment UENC for E. Evaluation of the Inhalation Carcinogenicity of Ethylene Oxide (2016 Interagency Science Discussion Draft) | IRIS | US EPA. Accessed February 3, 2025. https://iris.epa.gov/Document/&deid=306671

30. Cavalier H, Trasande L, Porta M. Exposures to pesticides and risk of cancer: Evaluation of recent epidemiological evidence in humans and paths forward. *Int J Cancer*. 2023;152(5):879-912. doi:10.1002/ijc.34300

31. Francisco LFV, da Silva RN, Oliveira MA, et al. Occupational Exposures and Risks of Non-Hodgkin Lymphoma: A Meta-Analysis. *Cancers*. 2023;15(9):2600. doi:10.3390/cancers15092600

32. Kricheldorff J, Maria Fallenberg E, Solbach C, Gerber-Schäfer C, Rancsó C, von Fritschen U. Breast Implant-Associated Lymphoma. *Dtsch Ärztebl Int*. 2018;115(38):628-635. doi:10.3238/arztebl.2018.0628

33. Lichtman MA. Obesity and the Risk for a Hematological Malignancy: Leukemia, Lymphoma, or Myeloma. *The Oncologist*. 2010;15(10):1083-1101. doi:10.1634/theoncologist.2010-0206

34. Skibola CF. Obesity, Diet and Risk of Non-Hodgkin Lymphoma. *Cancer Epidemiol Biomark Prev Publ Am Assoc Cancer Res Cosponsored Am Soc Prev Oncol*. 2007;16(3):392. doi:10.1158/1055-9965.EPI-06-1081

35. Willett EV, Morton LM, Hartge P, et al. Non-Hodgkin lymphoma and obesity: A pooled analysis from the InterLymph Consortium. *Int J Cancer*. 2008;122(9):2062-2070. doi:10.1002/ijc.23344

36. Reagan JL, Ingham RR II, Dalia S, et al. Association Between Obesity/Overweight and Leukemia: A Meta-Analysis of Prospective Cohort Studies,. *Blood*. 2011;118(21):3588. doi:10.1182/blood.V118.21.3588.3588

37. Xu J, Wang T. Association of diabetes mellitus with non-Hodgkin lymphoma risk: a meta-analysis of cohort studies. *Hematology*. 2019;24(1):527-532. doi:10.1080/16078454.2019.1636485

38. Chang ET, Boffetta P, Adami HO, Mandel JS. A critical review of the epidemiology of Agent Orange or 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and lymphoid malignancies. *Ann Epidemiol*. 2015;25(4):275-292.e30. doi:10.1016/j.annepidem.2015.01.002

39. Radkiewicz C, Bruchfeld JB, Weibull CE, et al. Sex differences in lymphoma incidence and mortality by subtype: A population-based study. *Am J Hematol*. 2023;98(1):23-30. doi:10.1002/ajh.26744

40. Carbone M, Arron ST, Beutler B, et al. Tumor predisposition and cancer syndromes as models to study gene X environment interactions. *Nat Rev Cancer*. 2020;20(9):533-549. doi:10.1038/s41568-020-0265-y

41. Full article: Unraveling the interactions between environmental factors and genetic polymorphisms in non-Hodgkin lymphoma risk. Accessed February 6, 2025. https://www.tandfonline.com/doi/10.1586/era.09.194?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed

42. CP W, E W, BW S. *World Cancer Report: Cancer Research for Cancer Prevention*. Accessed February 6, 2025. https://publications.iarc.fr/Non-Series-Publications/World-Cancer-Reports/World-Cancer-Report-Cancer-Research-For-Cancer-Prevention-2020

43. Sarpa M, Costa VÍ do B da, Ferreira SN, et al. Investigation of occupational risk factors for the development of non-Hodgkin's lymphoma in adults: A hospital-based case-control study. *PLOS ONE*. 2024;19(2):e0297140. doi:10.1371/journal.pone.0297140

44. Boyle J, Ward MH, Cerhan JR, Rothman N, Wheeler DC. Modeling historic environmental pollutant exposures and non-Hodgkin lymphoma risk. *Environ Res*. 2023;224:115506. doi:10.1016/j.envres.2023.115506

45. Cohn P, Klotz J, Bove F, Berkowitz M, Fagliano J. Drinking water contamination and the incidence of leukemia and Non-Hodgkin's lymphoma. *Environmental Health Perspectives.* 1994;102(6-7):556-561. doi:10.1289/ehp.94102556

46. Weisel CP, Jo WK. Ingestion, inhalation, and dermal exposures to chloroform and trichloroethene from tap water. *Environ Health Perspect*. 1996;104(1):48-51. doi:10.1289/ehp.9610448

47. Lutz WK. Susceptibility differences in chemical carcinogenesis linearize the dose–response relationship: threshold doses can be defined only for individuals. Mutat Res Mol Mech Mutagen. 2001;482(1):71-76. doi:10.1016/S0027-5107(01)00211-1

IX. ADDITIONAL MATERIALS CONSIDERED

Deposition Transcript & Exhibits

1. Deposition Transcript - Scott Keller (Plaintiff) - April 15, 2024, and exhibits

2. Deposition Transcript - Chad Keller (Plaintiff's Son) - July 29, 2024, and exhibits

3. Deposition Transcript - Kimberly Keller (Plaintiff's Wife) - May 13, 2024, and exhibits

4. Deposition Transcript - James Mudd, M.D. (Treating Physician) - July 30, 2024, and exhibits

5. Deposition Transcript - Stephen Iacoboni, M.D. (Treating Physician) - August 8, 2024, and exhibits

6. Deposition Transcript - Jennifer W. Leach, M.D. (Treating Physician) - May 24, 2024, and exhibits

7. Deposition Transcript - Oleksandr Seleverstov, M.D. (Treating Physician) - July 25, 2024, and exhibits

Expert Reports

1. Expert Report of Morris L. Maslia, P.E., D.WRE, DEE, Fellow EWRI - October 25, 2024
2. General Causation Expert Report of Dean W. Felsher, MD, PHD. [Leukemia, and Non-Hodgkin's Lymphoma] - December 9, 2024
3. General Causation Expert Report of Howard Hu, MD, MPH, SC. D. - December 9, 2024
4. General Causation Expert Report of Kathleen M. Gilbert, PHD. [TCE, Non-Hodgkin Lymphoma, and Leukemia] - December 9, 2024
5. General Causation Expert Report of Lukasz Gondek, MD, PhD. [Leukemia] - December 9, 2024
6. General Causation Expert Report of Steven B. Bird, MD. [Hematopoietic Cancers: Leukemia & Non-Hodgkin's Lymphoma] - December 9, 2024
7. Specific Causation Expert Report of Kelly Reynolds, MSPH, PhD – February 7, 2025

Medical

1. St. Mary Medical Center/ Univ. of WA Medical Center/ PROVIDENCE MEDICAL GROUP WALLA WALLA CARDIOLOGY, 01501_KELLER_0000000001-01501_KELLER_0000001816
2. St. Mary Medical Center, 01501_KELLER_PSMMC_0000000001-01501_KELLER_PSMMC_0000000258
3. Kadlec Regional Medical Center, 01501_KELLER_VBA_0000000058-01501_KELLER_VBA_0000000089
4. VA Puget Sound Health Care System, 01501_KELLER_VHA_0000000002-01501_KELLER_VHA_0000000014
5. Walla Walla Clinic, 01501_KELLER_WWC_0000000001-01501_KELLER_WWC_0000000566
6. Walla Walla VAMC, 01501_KELLER_VHA_0000000055-01501_KELLER_VHA_0000000675
7. Providence Medical Center, 01501_KELLER_VBA_0000000268-01501_KELLER_VBA_0000000416
8. Medical Records - Jonathan M. Wainwright VAMC, 01501_KELLER_VHA_0000000676-01501_KELLER_VHA_0000000847
9. Providence Saint Mary Medical Center-01501_KELLER_VBA_0000000869-01501_KELLER_VBA_0000000946

10. Morasch Medical Pc, 01501_KELLER_HCOS_0000000001-01501_KELLER_HCOS_0000000024

11. University of Washington Medical Center, 01501_KELLER_VBA_0000001588-01501_KELLER_VBA_0000001664

12. Walla Walla Clinic, 01501_KELLER_VBA_0000001673-01501_KELLER_VBA_0000001688

13. Walla Walla Clinic, 01501_KELLER_VBA_0000001703-01501_KELLER_VBA_0000001782

14. Providence Medical Center, 01501_KELLER_VBA_0000002038-01501_KELLER_VBA_0000002186

15. Jonathan M. Wainwright VAMC, 01501_KELLER_VBA_0000002383-01501_KELLER_VBA_0000002536

16. Providence Saint Mary Medical Center, 01501_KELLER_VBA_0000002583-01501_KELLER_VBA_0000002660

17. Providence Saint Mary Medical Center, 01501_KELLER_VBA_0000002707-01501_KELLER_VBA_0000002798

18. Kadlec Regional Medical Center, 01501_KELLER_VBA_0000003000-01501_KELLER_VBA_0000003031

19. Providence Heart Institute, 01501_KELLER_PHI_0000000001-01501_KELLER_PHI_0000000616

Short-Form Complaint & Profile Form

1. Scott Keller Discovery Pool Profile Form, 01501_KELLER_DPPF_0000000001-01501_KELLER_DPPF_0000000030

2. Scott Keller Short-Form Complaint

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

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

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

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

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

Exhibit 1

Biographical and Bibliographic Information

Identifying Information:

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

Academic History:

Colleges and University

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

Scholarships and Honors

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

Residency and Post-Doctoral Training

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

Board Certification

1996	Internal Medicine
1998	Medical Oncology

Employment History:

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

Public and Professional Service:

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

Graduate Programs

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

Research and Professional Experience

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

Clinical Experience

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

University Services

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

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

Clinical Teaching

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

Community Service

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

Teaching Activities /Courses

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

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

Trainees

High School Students

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

College Students

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

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

Graduate Students/Medical Students

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

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

Post-Doctoral Fellows

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

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

Graduate Student Committees

Orals Committees

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

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

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

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

Invited Journal Reviews
American Journal of Pathology

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

NIH Study Sections

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

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

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

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

Program Reviews
2009 Review Panel: UCSF BMS Graduate Program

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

Search Committees

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

Honors, Awards and Memberships:

Honors

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

Awards

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

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

Memberships

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

Major Invited Addresses

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

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

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

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

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

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

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

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

Research Support:

Ongoing

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Completed

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

This project investigators a novel CD inhibitor for cancer.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

"Lipogenesis inhibition in cancer"

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

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

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

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

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

Patents:

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

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

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

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

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

Publications:

Chapters (total of 3)

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

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

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

Peer-reviewed articles (total of 134)

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

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

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

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

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.

21. Deb-Basu, D., Karlsson, A., Li, Q., Dang, C. V., and Felsher, D. W. MYC can enforce cell cycle transit from G1 to S and G2 to S, but not mitotic cellular division, independent of P27 inhibition of cyclin E/CDK2. Cell Cycle, 5(12): 1348-55, 2006.

22. Ray, S., Atkuri, K. R., Deb-Basu, D., Adler, A. S., Chang, H. Y., Herzenberg, L. A., and Felsher, D. W. MYC can induce DNA breaks in vivo and in vitro independent of ROS. Cancer Research, 66(13): 6598-605, 2006.

23. Weng, A. P., Millholland, J. M., Yashiro-Ohtani, Y., Arcangeli, M. L., Lau, A., Wai, C., del Bianco, C., Rodriguez, C. G., Sai, H., Tobias, J., Li, Y., Wolfe, M. D., Shachaf, C., Felsher, D. W., Blacklow, S. C., Pear, W. S., and Aster, J. C. cMyc is an important direct

target of Notch1 in T cell acute lymphoblastic leukemia/lymphoma. Genes and Development, 20(15): 2096-109, 2006.

24. Giuriato, S., Ryeom, S., Fan, A. C., Bachireddy, P., Lynch, R. C., Rioth, M. J., van Riggelen, J., Kopelman, A. M., Passegue, E., Tang, F., Folkman, J., and Felsher, D. W. Sustained regression of tumors upon MYC inactivation requires p53 or TSP-1 to reverse the angiogenic switch. Proceedings of the National Academy of Sciences, 103(44): 16266-71, 2006.

25. Pan, M., Winslow, M. M., Chen, L., Kuo, A., Felsher, D. W., and Crabtree, R. Enhanced NFATc1 nuclear occupancy causes T cell activation independent of CD28 costimulation. Journal of Immunology, 178(7): 4315-21, 2007.

26. Arvanitis, C., Bendapudi, P. K., Bachireddy, P., and Felsher, D. W. Identifying critical signaling molecules for the treatment of cancer. Cancer Research, 172: 5-24, 2007.

27. Shachaf, C., Perez, O. D., Youssef, S., Fan, A. C., Elchuri, S., Goldstein, M. J., Shirer, A. E., Sharpe, O., Chen, J., Mitchell, D. J., Chang, M., Nolan, G. P., Steinman, L., and Felsher, D. W. Inhibition of HMGcoA reductase by atorvastatin prevents and reverses MYC-induced lymphomagenesis. Blood DOI 110(7): 2674-84, 2007.

28. Giuriato, S., Faumont, N., Bousquet, E., Foisseau, M., Bibonne, A., Moreau, M., Al Saati, T., Felsher, D. W., Delsol, G., Meggetto, F. Development of a conditional bioluminescent transplant model for TPM3-ALK-induced tumorigenesis as a tool to validate ALK-dependent cancer targeted therapy. Cancer Biology Therapy, (8): 1318-23, 2007.

29. Wu, C., van Riggelen, J., Yetil, A., Fan, A. C., Bachireddy, P., and Felsher, D. W. Cellular senescence is an important mechanism of tumor regression upon c-Myc inactivation. Proceedings of the National Academy of Sciences, 104(32): 13028-33, 2007.

30. Gao, P., Zhang, H., Dinavahi, R., Li, F., Xiang, Y., Raman, V., Bhujwalla, Z. M., Felsher, D. W., Cheng, L., Pevsnr, J., Lee, L. A., Semenza, G. L., and Dang, C. V. HIF-dependant antitumorigenic effect of antioxidants in vivo. Cancer Cell, 12(3): 230-8, 2007.

31. Opavsky, R., Tsai, S., Guimond, M., Arora, A., Opavska, J., Becknell, B., Kaufmann, M., Walton, N. A., Stephens, J. A., Fernandez, S. A., Muthusamy, N., Felsher, D. W., Porcu, P., Caligiuri, M. A., and Leone, G. Specific tumor suppressor function for E2F2 in Myc-induced T cell lymphomagenesis. Proceedings of the National Academy of Sciences, 104(39): 15400-15405, 2007.

32. Opavsky, R., Wang, S., Trikha, P., Raval, A., Huang, Y., Wu, Y., Rodriguez, B., Keller, B., Liyanarachi, S., Wei, G., Davuluri, R. V., Weinstein, M., Felsher, D. W., Ostrowski, M., Leone, G., and Plass, C. CpG island methylation in a mouse model of lymphoma is driven by the genetic configuration of tumor cells. PLOS Genetics, 3(9): e167, 2007.

33. Omidvar, N., Kogan, S., Beurlet, S., le Pogam, C., Janin, A., West, R., Noguera, M. E., Reboul, M., Soulie, A., Leboeuf, C., Setterblad, N., Felsher, D. W., Lagasse, E., Mohamedali, A., Thomas, N. S., Fenaux, P., Fontenay, M., Pla, M., Mufti G. J., Weissman, I., Chomienne, C., and Padua, R. A. BCL-2 and mutant NRAS interact physically and functionally in a mouse model of progressive myelodysplasia. Cancer Research, 67(24): 11657-6, 2007.

34. Tran, P. T., Fan, A. C., Bendapudi, P. K., Koh, S., Komatsubara, K., Chen, J., Horng, G., Bellovin, D. I., Giuriato, S., Wang, C. S., Whitsett, J. A., and Felsher, D. W. Combined inactivation of MYC and k-ras oncogenes reverses tumorigenesis in lung adenocarcinomas and lymphomas. PLoS One, 3(5): e2125, 2008.

35. Wu, C., Sahoo, D., Arvanitis, C., Bradon, N., Dill, D., and Felsher, D. W. Combined analysis of murine and human microarrays and ChIP analysis reveals genes associated with ability of MYC to maintain tumorigenesis. PLoS Genetics, 4(6): e1000090, 2008.

36. Beer, S., Komatsubara, K., Bellovin, D., Kurobe, M., Sylvester, K., and Felsher, D. W. Hepatotoxin-induced changes in the adult murine liver promote MYC-induced tumorigenesis. PLoS One, 3(6): e2493, 2008.

37. Shachaf, C., Gentles, A., Elchuri, S., Sahoo, D., Soen, Y., Sharpe, O., Perez, O., Chang, M., Mitchel, D., Robinson, W., Nolan, G., Dill, D., Plevritis, S., and Felsher, D. W. Genomic and proteomic analysis reveals a threshold level of MYC required for tumor maintenance. Cancer Research, 68(13): 5132-42, 2008.

38. Traykova-Brauch, M., Schönig, K., Greiner, O., Miloud, T., Jauch, A., Bode, M., Felsher, D. W., Glick, A. B., Kwiatkowski, D. J., Bujard, H., Horst, J., von Knebel Doeberitz, M., Niggli, F. K., Kriz, W., Gröne, H. J., and Koesters, R. An efficient and versatile system for acute and chronic modulation of renal tubular function in transgenic mice. Nature Medicine, 14(9): 979-84, 2008.

39. Fan, A. C., Goldrick, M. M., Ho, J., Liang, Y., Bachireddy, P., and Felsher, D. W. A quantitative PCR method to detect blood microRNAs associated with tumorigenesis in transgenic mice. Molecular Cancer, 7(1): 74, 2008.

40. Arvanitis, C., Bendapudi, P. K., Tseng, J. R., Gambhir, S. S., and Felsher, D. W. (18)F and (18)FDG PET imaging of osteosarcoma to non-invasively monitor in situ changes in cellular proliferation and bone differentiation upon myc inactivation. Cancer Biology and Therapy, 7(12): 1947-51, 2008.

41. Felsher, D. W. Reversing cancer from inside and out: Oncogene addiction, cellular senescence, and the angiogenic switch. Lymphatic Research and Biology, 6(3-4): 149-54, 2008.

42. Ney, J. T., Schmidt, T., Kurts, C., Zhou, Q., Eckert, D., Felsher, D. W., Schorle, H., Knolle, P., Tuting, T., Barchet, W., Buttner, R., Limmer, A., and Gutgemann, I. Autochthonous liver tumors induce systemic T cell tolerance associated with T cell receptor down-modulation. Hepatology, 49(2): 471-81, 2009.

43. Tran, P. T., and Felsher, D. W. The current STATe of biomarkers to predict the response to anti-angiogenic therapies. Cancer Biology & Therapy, 7(12): 2004-6, 2008.

44. Lee, H. G., Casadesus, G., Nunomura, A., Zhu, X., Castellani, R. J., Richardson, S. L., Perry, G., Felsher, D. W., Petersen, R. B., and Smith, M.A. The neuronal expression of MYC causes a neurodegenerative phenotype in a novel transgenic mouse. American Journal of Pathology, 174(3): 891-7, 2009.

45. Kampa, K. M., Acoba, J. D., Chen, D., Gay, J., Lee, H., Beemer, K., Padiernos, E., Boonmark, N., Zhu, Z., Fan, A. C., Bailey, A. S., Fleming, W. H., Corless, C., Felsher, D. W., Naumovski, L., and Lopez, C. D. Apoptosis-stimulating protein of p53 (ASPP2) heterozygous mice are tumor-prone and have attenuated cellular damage-response thresholds. Proceedings of the National Academy of Science USA, 106(11): 4390-5, 2009.

46. Levashova, Z., Backer, M. V., Horng, G., Felsher, D. W., Backer, J. M., and Blankenberg, F. G. SPECT and PET imaging of EGF receptors with site-specifically labeled EGF and Dimeric EGF. Bioconjugate Chemistry, 20(4): 742-9, 2009.

47. Fan, A. C., Deb-Basu, D., Orban, M. W., Gotlib, J. R., Natkunam, Y., O'Neil, R., Padua, R. A., Xu, L., Taketa, D., Shirer, A. E., Beer, S., Yee, A. X., Voehringer, D. W., and Felsher, D. W. Nano-fluidic proteomic assay for serial analysis of oncoprotein activation in clinical specimens. Nature Medicine, 15(15): 566-71, 2009. Also see: G. Alderton, Proteomics: Biomarkers on a nanoscale, Nature Reviews Cancer, 9:382, 2009

48. Liu, Z, Fan, A. C., Rakhra, K., Sherlock, S., Goodwin, A., Chen, X., Yang, Q., Felsher, D. W., and Dai, H. Supramolecular stacking of doxorubicin on carbon nanotubes for in vivo cancer therapy. Angewandte Chemie, 48(41): 7668-72, 2009.

49. Lee, H.G., Chen, Q., Wolfram, J. A., Richardson, S. L., Liner, A., Siedlak, S. L., Zhu, Z., Ziats, N. P., Fujioka, F., Felsher, D. W., Castellani, R. J., Valencik, M. L., McDonald, J. A., Hoit, B. D., Lesnefsky, E. J., and Smith, M. A. Cell cycle re-entry and mitochondria defects in Myc-mediated hypertrophic cardiomyopathy and heart failure. PLoS One, 4(9): e7172, 2009.

50. Beer, S., Bellovin, D. I., Lee, J. S., Komatsubara, K., Wang, L. S., Koh, H., Borner, K., Storm, T. A., Davis, C. R., Kay, A. K., Felsher, D. W., and Grimm, D. Low-level shRNA cytotoxicity can contribute to MYC-induced hepatocellular carcinoma in adult mice (the contribution). Molecular Therapy, 18(1): 161-70, 2009.

51. Giuriato, S., Foisseau, M., Dejean, E., Felsher, D. W., Al Saati, T., Demur, C., Ragab, A., Kruczynski, A., Schiff, C., Delsol, G., and Meggetto, F. Conditional TPM3-ALK and NPM-ALK transgenic micedevelop reversible ALK-positive early B cell lymphoma/leukemia. Blood, 115(20): 4061-70, 2010.

52. Zhou, H., Rodriguez, M., van den Haak, F., Nelson, G., Jogani, R., Xu, J., Zhu, X., Xian, Y., Tran, P. T., Felsher, D. W., Keall, P. J., and Graves, E. E. Development of a MicroCT-Based Image-Guided Conformal Radiotherapy System for Small Animals. International Journal of Radiation Oncology, Biology, Physics, 78(1): 297-305, 2010.

53. van Riggelen J., Müller J, Otto T., Beuger V., Yetil A., Choi P. S., Kosan C., Möröy T, *Felsher D. W., and *Eilers M. The interaction between Myc and Miz1 is required to antagonize TGFbeta-dependent autocrine signaling during lymphoma formation and maintenance. Genes Dev., 24(12): 1281-94, 2010. *co-senior and corresponding authors

54. Woodard, L. E., Keravala, A., Jung, W. E., Wapinski, O. L., Yang, Q., Felsher, D. W., and Calos, M. P. Impact of hydrodynamic injection and phiC31 integrase on tumor latency in a mouse model of MYC-induced hepatocellular carcinoma. PLoS One, 5(6): e11367, 2010.

55. Fan-Minogue H., Cao, Z., Paulmurugan R., Chan C. T., Massoud, T. F., Felsher, D. W., and Gambhir, S. S. From the cover: Noninvasive molecular imaging of c-Myc activation in living mice. Proceedings of the National Academy of Sciences, 107(36): 15892-7, 2010.

56. Graves, E. E., Vilalta, M., Cecic, I. K., Erler, J. T., Tran, P. T., Felsher, D. W., Sayles, L., Sweet-Cordero, A., Thu Le, Q., and Giaccia, A., J. Hypoxia in models of lung cancer: Implications for targeted therapeutics. Clinical Cancer Research, 16(19): 4843-52, 2010.

57. Contag, C. H., Sikorski, R., Negrin, R., Schmidt, T., Fan, A., Bachireddy, P., Felsher, D. W., and Thorne S. H. Definition of an enhanced immune cell therapy in mice that can target stem-like lymphoma cells. Cancer Research, 70(23): 9837-45, 2010.

58. Rakhra, K., Bachireddy, P., Zabuawala, T., Zeiser, R., Xu, L., Kopelman, A., Fan, A. C., Yang, Q., Braunstein, L., Crosby, E., Ryeom, S., and Felsher, D. W. CD4+ T-cells contribute to the remodeling of the microenvironment required for sustained tumor regression upon oncogene inactivation. Cancer Cell, 18(5): 485-98, 2010.

59. Nielsen, C. H., Kimura, R. H., Withofs, N., Tran P. T., Miao, Z., Cochran, J. R., Cheng, Z., Felsher, D. W., Kjaer, A., Willmann, J. K., and Gambhir, S. S. PET Imaging of tumor neovascularization in a transgenic mouse model with a novel 65Cu-DOTA-knottin peptide. Cancer Research, 70(22): 9022-30, 2010.

60. Muller, J., Samans, B., van Riggelen J., Faga, G., Peh, K. N. R., Wei, C. L., Muller, H., Amati, B., Felsher, D. W., and Eilers, M. TGFbeta-dependent gene expression shows that senescence correlates with abortive differentiation along several lineages in Myc-induced lymphomas. Cell Cycle, 9 (23): 4622-4626, 2010.

61. Huang, M., Whang, P., Chodaparambil, J. V., Pollyea, D. A., Kusler, B., Xu, L., Felsher, D. W., and Mitchell, B. S. Reactive oxygen species regulate nucleostemin oligomerization and protein degradation. Journal of Biological Chemistry, 286(13): 11035-46, 2011.

62. Cao, Z., Fan-Minogue, H., Bellovin, D., Arzeno, J., Yang, Q., Gambhir, S. S., and Felsher, D. W. Inhibition of HMG-CoA reductase by statins reverses hepatocellular tumorigenesis by blocking phosphorylation and activation of c-MYC. Cancer Research, 71(6): 2286-97, 2011.

63. Saddic, L. A., Wirt, S., Vogel, H., Felsher, D. W., and Sage, J. Functional Interactions between Retinoblastoma and c-MYC in a Mouse Model of Hepatocellular Carcinoma. PLoS One, 6(5): e19758, 2011. [PubMed - in process] Free PMC Article

64. Dejean, E., Renalier, M. H., Foisseau, M., Agirre, X., Joseph. N., de Paiva, G. R., Al Saati, T., Soulier, J., Desjobert, C., Lamant, L., Prósper, F., Felsher, D. W., Cavaillé, J. Prats, H., Delsol, G., Giuriato, S., and Meggetto, F. Hypoxia-microRNA-16 downregulation induces VEGF expression in anaplastic lymphoma kinase (ALK)-positive anaplastic large-cell lymphomas. Leukemia, doi: 10.1038/leu.2011.168. [Epub ahead of print]

65. Seetharam, M., Fan, A. C., Tran, M., Xu, L., Renschler, J. P., Felsher, D. W., Sridhar, K., Wilhelm, F., and Greenberg, P. L. Treatment of higher risk myelodysplastic syndrome patients unresponsive to hypomethylating agents with ON 01910.Na. Leukemia Research 2011 Sep 14. [Epub ahead of print]

66. Choi, P. S., van Riggelen, J., Gentles, A., J., Bachireddy, P., Rakhra, K., Adam, S. J., Plevritis, S. K., and Felsher, W. D. Lymphomas that recur after MYC suppression continue to exhibit oncogene addition. Proceedings of the National Academy of Sciences, 108(42): 17432-7, 2011.

67. Tran, P.T., Bendapudi, P. K., Lin H. J., Choi, P., Koh, S., Chen, J., Horng, G., Hughes, N. P. Schwartz, L. H., Miller, V. A., Kawashima, T., Kitamura, T., Paik, D., and Felsher, D. W. Survival and death signals can predict tumor response to therapy after oncogene inactivation. Science Translational Medicine, 3(103): 103ra99, 2011.

68. Hlady, R. A., Novakova, S., Opavska, J., Klinkebiel, D., Peters, S. L., Bies, J., Hannah, J., Iqbal, J., Anderson, K. M., Siebler, H. M., Smith, L. M., Greiner, T. C., Bastola, D., Joshi, S., Lockridge, O., Simpson, M. A., Felsher, D. W., Wagner, K. U., Chan, W. C., Christman, J. K., and Opavsky, R. Loss of Dnmt3b function upregulates the tumor modifier Ment and accelerates mouse lymphomagenesis. Journal of Clinical Investigation, 122(1): 163-77, 2011.

69. Bachireddy, P., Rakhra, K., and Felsher, D. W. Immunology in the clinic review series: focus on cancer; multiple roles for the immune system in oncogene addiction. Clinical and Experimental Immunology, 167(2): 188-94, 2012.

70. DeChristopher, B. A., Fan, A. C., *Felsher, D. W., and Wender, P. A. "Picolog, " a synthetically-available bryostatin analog, inhibits growth of MYC-induced lymphoma in vivo. Oncotarget, vol. 3, no. 1, 2012. *co-corresponding senior author

71. Tran, P., Shroff, E., Burns, T. F., Thiyagarajan, S., Das, S. T., Zabuawala, T., Chen, J., Cho, Y., Luong, R., Tamayo, P., Salih, T., Aziz, K., Adam, S. J., Vicent, S., Nielsen, C. H., Withofs, N., Sweet-Cordero, A., Gambhir, S. S., Rudin, C. M. and Felsher, D. W. Twist1 suppresses senescence programs and thereby accelerates and maintains mutant Kras-induced lung tumorigenesis. PLoS Genetics, 8(5): e1002650, 2012.

72. Wu, A. R., Kawahara T. L., Rapicavoli, N. A., van Riggelen, J., Shroff, E. H., Xu, L., Felsher, D. W., Chang, H. Y., and Quake, S. R. High throughput automated chromatin immunoprecipitation as a platform for drug screening and antibody validation. Lab Chip, 12(12): 2190-8, 2012. Epub 2012 May 8.

73. Das, B., Bayat-Mokhtari, R., tsui, M., Lotfi, S., Tsuchida, R., Felsher, D. W., and Yeger, H. HIF-2a suppresses p53 to enhance the stemness and regenerative potential of human embryonic stem cells. Stem Cells, 30(8): 1685-1695, 2012. Doi: 10.1002/stem. 1142.

74. To, C. A., Hsieh, R. W., McClellan, J. S., Howard, W., Fischbein, N. J., Brown, J. M., Felsher, D. W., and Fan, A. C. Cryptococcal osteomyelitis and meningitis in a patient with non-hodgkin's lymphoma treated with PEP-C. BMJ Case Rep, 2012. pii:bcr0820114578. doi: 10.1136/bcr.08.2011.4578.

75. Bisikirska, B. C., Adam, S. J., Alvarez, M. J., Rajbhandari, P. Cox, R., Lefebvre, C., Wang, K., Rieckhof, G. E., *Felsher, D. W., and Califano, A. STK38 is a critical upstream regulator of MYC's oncogenic activity in human B-cell lymphoma. Oncogene, doi: 10.1038/onc.2012.543. [Epub ahead of print] *co-corresponding senior author

76. Das, B., Kashino, S. S., Pulu, I., Kalita, D., Swami, V., Yeger, H., *Felsher, D. W., and Neto-Campos, A. CD271 bone marrow mesenchymal stem cells may provide a niche for dormant mycobacterium tuberculosis. Science Translational Medicine. 5(170): 170ra13. Doi: 10.1126/scitranslmed.3004912. *corresponding senior author

77. Lin, W., Rajbhandari, N., Liu, C., Sakamoto, K., Zhang, Q., Triplett, A. A., Batra, S. K., Opavsky, R., Felsher, D. W., DiMaio, D. J., Hollingsworth, M. A., Morris 4th, J. P., Hebrok, M., Witkiewicz, A. K., Brody, J. R., Rui H., and Wagner, K. Dormant cancer cells contribute to residual disease in a model of reversible pancreatic cancer. Cancer Research. 2013 Mar 15;73(6):1821-30. doi: 10.1158/0008-5472.CAN-12-2067. Epub 2013 Mar 6.

78. Perry, R. H., Bellovin, D. I., Shroff, E. H., Ismail, A. I., Zabuawala, T., Felsher, D. W., and Zare, R. N. Characterization of MYC-induced tumorigenesis by in situ lipid profiling. Anal Chem. 2013 May 7;85(9):4259-62. doi: 10.1021/ac400479j. Epub 2013 Apr 18.

79. Nwabugwu, C., Rakhra, K., Felsher, D. W., Paik, D. In vivo imaging-based mathematical modeling techniques that enhance the understanding of oncogene addiction in relation to tumor growth. Comput Math Methods Med. 2013;2013:802512. doi: 10.1155/2013/802512. Epub 2013.

80. Kapanadze, T., Gamrekelashvili, J., Ma, C., Chan, C., Zhao, F., Hewitt, S., Zender, L., Kapoor, V., Felsher, DW, Manns, M. P., Korangy, F., Greten T. F. Regulation of accumulation and function of myeloid derived suppressor cells in different murine models of hepatocellular carcinoma. J Hepatol. 2013 Nov;59(5):1007-13. doi: 10.1016/j.jhep.2013.06.010. Epub 2013 Jun 22.

81. Fan-Minogue H, Bodapati S, Solow-Cordero D, Fan A, Paulmurugan R, Massoud TF, Felsher DW, Gambhir SS. A c-Myc activation sensor-based high-throughput drug screening identifies an antineoplastic effect of nitazoxanide. Mol Cancer Ther. 2013 Sep;12(9):1896-905. doi: 10.1158/1535-7163.MCT-12-1243. Epub 2013 Jul 3.

82. Beurlet S, Omidvar N, Gorombei P, Krief P, Le Pogam C, Setterblad N, de la Grange P, Leboeuf C, Janin A, Noguera ME, Hervatin F, Sarda-Mantel L, Konopleva M, Andreeff M, Tu AW, Fan AC, Felsher DW, Whetton A, Pla M, West R, Fenaux P, Chomienne C, Padua RA. BCL-2 inhibition with ABT-737 prolongs survival in an NRAS/BCL-2 mouse model of AML by targeting primitive LSK and progenitor cells. Blood. 2013 Oct 17;122(16):2864-76. doi: 10.1182/blood-2012-07-445635. Epub 2013 Aug 13.

83. Ansari C, Tikhomirov GA, Hong SH, Falconer RA, Loadman PM, Gill JH, Castaneda R, Hazard FK, Tong L, Lenkov OD, Felsher DW, Rao J, Daldrup-Link HE. Development of novel tumor-targeted theranostic nanoparticles activated by membrane-type matrix metalloproteinases for combined cancer magnetic resonance imaging and therapy. Small. 2013 Aug 27. doi: 10.1002/smll.201301456. [Epub ahead of print].

84. Nwabugwu CI, Rakhra K, Felsher, DW, Paik DS. A tumor-immune mathematical model of CD4+ T helper cell dependent tumor regression by oncogene inactivation. Conf Proc IEEE Eng Med Biol Soc. 2013 Jul;2013:4529-32. doi: 10.1109/EMBC.2013.6610554.

85. Anso, E., Mullen, A. R., Felsher, DW, Mates, J. M., DeBeradinis, R. J., and Chandel, N. S. Metabolic changes in cancer cells upon suppression of MYC. Cancer & Metabolism. 2013.

86. Cao, Z., Ding, B., Guo, P., Lee, S. B., Butler, J. M., Casey, S. C., Simons, M., Tam, W., Felsher, DW, Shido, K., Rafii, A., Scandura, J. M., and Rafii, S. Angiocrine factors deployed by tumor vascular niche induce b-cell lymphoma invasiveness and chemoresistance. Running title: Tumor vascular niche induces aggressive lymphoma. Cancer Cell 2014 Mar 17;25(3):350-65. doi: 10.1016/j.ccr.2014.02.005.

87. Ye D, Shuhendler AJ, Cui L, Tong L, Tee SS, Tikhomirov G, Felsher, DW, Rao J. Bioorthogonal cyclization-mediated in situ self-assembly of small-molecule probes for imaging caspase activity in vivo. Nat Chem. 2014 Jun;6(6):519-26. doi: 10.1038/nchem.1920. Epub 2014 Apr 28.

88. Gabay M, Li Y, Felsher DW. MYC activation is a hallmark of cancer initiation and maintenance Cold Spring Harb Perspect Med. 2014 Jun 2;4(6):a014241. doi: 10.1101/cshperspect.a014241.

89. Choi PS, Li Y, Felsher, DW. Addiction to multiple oncogenes can be exploited to prevent the emergence of therapeutic resistance. Proc Natl Acad Sci U S A. 2014 Aug 12;111(32):E3316-24. doi: 10.1073/pnas.1406123111. Epub 2014 Jul 28.

90. Eberlin LS, Gabay M, Fan AC, Gouw AM, Tibshirani RJ, Felsher, DW, Zare RN. Alteration of the lipid profile in lymphomas induced by MYC overexpression. Proc Natl

Acad Sci U S A. 2014 Jul 22;111(29):10450-5. doi: 10.1073/pnas.1409778111. Epub 2014 Jul 3.

91. Li, Y., Choi, P., Casey, S. C., Dill, D. L., and Felsher, DW. MYC through miR-17-92 suppresses specific target genes to maintain survival, autonomous proliferation and a neoplastic state. Cancer Cell. 2014 Aug 11;26(2):262-72. doi: 10.1016/j.ccr.2014.06.014.

92. Casey, S.C, Li, Y, Fan, A.C, and Felsher, DW. Oncogene withdrawal engages the immune system to induce sustained cancer regression. J Immunother Cancer. 2014 Jul 15;2:24. doi: 10.1186/2051-1426-2-24. eCollection 2014.

93. Li Y, Choi PS, Casey SC, and Felsher DW. Activation of cre recombinase alone can induce complete tumor regression. PLOS One. 2014 Sep 10;9(9):e107589. doi: 10.1371/journal.pone.0107589. eCollection 2014.

94. Li Y, Choi PS, Felsher, DW. Oncogene addiction: resetting the safety switch? Oncotarget. 2014 Sep 30;5(18):7986-7. No abstract available. Free PMC Article

95. Li Y, Casey SC, and Felsher, DW. Inactivation of MYC reverses tumorigenesis. J Intern Med. 2014 Jul;276(1):52-60. doi: 10.1111/joim.12237.

96. Yetil A, Anchang B, Gouw AM, Adam SJ, Zabuawala T, Parameswaran R, van Riggelen J, Plevritis S, and Felsher DW. p19ARF is a critical mediator of both cellular senescence and an innate immune response associated with MYC inactivation in mouse model of acute leukemia. Oncotarget. 2015 Feb 28; 6(6):3563-77.

97. Xiang Y, Stine ZE, Xia J, Lu Y, O'Connor RS, Altman BJ, Hsieh AL, Gouw AM, Thomas AG, Gao P, Sun L, Song L, Yan B, Slusher BS, Zhuo J, Ooi LL, Lee CG, Mancuso A, McCallion AS, Le A, Milone MC, Rayport S, and Felsher, DW, Dang CV. Targeted inhibition of tumor-specific glutaminase diminishes cell-autonomous tumorigenesis. J Clin Invest. 2015 Apr 27. pii: 75836. doi: 10.1172/JCI75836. Epub 2015 Apr 27.

98. Shroff EH, Eberlin LS, Dang VM, Gouw AM, Gabay M, Adam SJ, Bellovin DI, Tran PT, Philbrick WM, Garcia-Ocana A, Casey SC, Li Y, Dang CV, Zare RN, and Felsher, DW. MYC oncogene overexpression drives renal cell carcinoma in a mouse model through glutamine metabolism. Proc Natl Acad Sci U S A. 2015 May 26;112(21):6539-44. doi: 10.1073/pnas.1507228112. Epub 2015 May 11.

99. Altman BJ, Hsieh AL, Sengupta A, Krishnanaiah SY, Stine ZE, Walton ZE, Gouw AM, Venkataraman A, Li B, Goraksha-Hicks P, Diskin SJ, Bellovin DI, Simon MC, Rathmell JC, Lazar MA, Maris JM, Felsher, DW, Hogenesch JB, Weljie AM, Dang CV. MYC Disrupts the Circadian Clock and Metabolism in Cancer Cells. Cell Metab. 2015 Dec 1;22(6):1009-19. doi: 10.1016/j.cmet.2015.09.003. Epub 2015 Sep 17.

100. Casey SC, Tong L, Li Y, Do R, Walz S, Kelly N, Fitzergerald N, Gouw, A, Baylot V Guetegemann I, Eilers, M. Felsher DW. MYC Regulates the Anti-Tumor Immune

Response through CD47 and PD-L1. Science. 2016 Mar 10. pii: aac9935. [Epub ahead of print]

101. Ma C, Kesarwala AH, Eggert T, Medina-Echeverz J, Kleiner DE, Jin P, Stroncek DF, Terabe M, Kapoor V, ElGindi M, Han M, Thornton AM, Zhang H, Egger M, Luo J, Felsher DW, McVicar DW, Weber A, Heikenwalder M, Greten TF. NAFLD causes selective CD4(+) T lymphocyte loss and promotes hepatocarcinogenesis. Nature. 2016 Mar 10;531(7593):253-7. doi: 10.1038/nature16969. Epub 2016 Mar 2.

102. Swords RT, Greenberg PL, Wei AH, Durrant S, Advani AS, Hertzberg MS, Lewis ID, Rivera G, Gratzinger D, Fan AC, Felsher DW, Cortes JE, Watts JM, Yarranton GT, Walling JM, Lancet JE. KB004, a first in class monoclonal antibody targeting the receptor tyrosine kinase EphA3, in patients with advanced hematologic malignancies: Results from a phase 1 study. Leuk Res. 2016 Nov; 50:123-131. doi: 10.1016/j.leukres.2016.09.012.

103. Li Y, Deutzmann A, Choi PS, Fan AC, Felsher DW. BIM mediates oncogene-inactivation induced apoptosis in multiple transgenic mouse models of acute lymphoblastic leukemia. Oncotarget. 2016 May 10;7(19):26926-34. doi: 10.18632/oncotarget.8731.

104. Gouw AM, Eberlin, LS, Margulis K, Sullivan DK, Toal GG, Tong L, Zare RN, and Felsher DW. Oncogene KRAS activates fatty acid synthase, resulting in specific ERK and lipid signatures associated with lung adenocarcinoma. PNAS 2017; published ahead of print April 11, 2017, doi:10.1073/pnas.1617709114.

105. Bailey ST, Smith AM, Kardos J, Wobker SE, Wilson HL, Krishnan B, Saito R, Lee HJ, Zhang J, Eaton SC, Williams LA, Manocha U, Peters DJ, Pan X, Carroll TJ, Felsher DW, Walter V, Zhang Q, Parker JS, Yeh JJ, Moffitt RA, Leung JY, and Kim WY. MYC activation cooperates with Vhl and Ink4a/Arf loss to induce clear cell renal cell carcinoma. Nature Communications; April 2017, doi:10.1038/ncomms15770.

106. Felsher DW, Dhanasekaran R, Gabay-Ryan M, Baylot V, Lai I, Mosley A, Huang X, Zabludoff S, Li J, Kaimal V, Karmali P. Anti-miR-17 Therapy Delays Tumorigenesis in MYC-driven Hepatocellular Carcinoma (HCC). Oncotarget, 2017-08-18 10:57:29. In press

107. Hebb J, Felsher DW, Mosley AR, Vences-Catalan F, Rajasekaran N, Rosen A, Ellmark P. Administration of low dose combination anti-CTLA4, anti-CD137, and anti-OX40 into murine tumor or proximal to the tumor draining lymph node induces systemic tumor regression. Cancer Immunology, Immunotherapy, 2018 Jan;67(1):47-60. doi: 10.1007/s00262-017-2059-y. Epub 2017 Sep 13.

108. Miess H, Dankworth B, Gouw AM, Rosenfeldt M, Schmitz W, Jiang M, Saunders B, Howell M, Downward J, Felsher DW, Peck B, Schulze A. The glutathione redox system is essential to prevent ferroptosis caused by impaired lipid metabolism in clear cell renal cell carcinoma. Oncogene. 2018 Jun 5. doi: 10.1038/s41388-018-0315-z.

109. Taparra K, Wang H, Malek R, Lafargue A, Barbhuiya MA, Wang X, Simons BW, Ballew M, Nugent K, Groves J, Williams RD, Shiraishi T, Verdone J, Yildirir G, Henry R, Zhang B, Wong J, Wang KK, Nelkin BD, Pienta KJ, Felsher D, Zachara NE, Tran PT. O-GlcNAcylation is required for mutant KRAS-induced lung tumorigenesis. J Clin Invest. 2018 Nov 1;128(11):4924-4937. doi: 10.1172/JCI94844. Epub 2018 Sep 24.

110. Lai, I, Swaminathan, S, Baylot, V, Adriane Mosley, A, Dhanasekaran, R, Ryan, M, Felsher DW. Lipid Nanoparticles that Deliver IL-12 Messenger RNA Suppress Tumorigenesis in MYC Oncogene-Driven Hepatocellular Carcinoma. Journal for Immune Therapy of Cancer, 2018 Nov 20;6(1):125. doi: 10.1186/s40425-018-0431-x.

111. Yang P, Jiang Y, Pan Y, Ding X, Rhea P, Ding J, Hawke DH, Felsher D, Narla G, Lu Z, Lee RT. Mistletoe extract Fraxini inhibits the proliferation of liver cancer by down-regulating c-Myc expression. Sci Rep. 2019 Apr 23;9(1):6428. doi: 10.1038/s41598-019-41444-2.

112. Struntz NB, Chen A, Deutzmann A, Wilson RM, Stefan E, Evans HL, Ramirez MA, Liang T, Caballero F, Wildschut MHE, Neel DV, Freeman DB, Pop MS, McConkey M, Muller S, Curtin BH, Tseng H, Frombach KR, Butty VL, Levine SS, Feau C, Elmiligy S, Hong JA, Lewis TA, Vetere A, Clemons PA, Malstrom SE, Ebert BL, Lin CY, Felsher DW, Koehler AN. Stabilization of the Max Homodimer with a Small Molecule Attenuates Myc-Driven Transcription. Cell Chem Biol. 2019 May 16;26(5):711-723.e14. doi: 10.1016/j.chembiol.2019.02.009. Epub 2019 Mar 14.

113. Das B, Pal B, Bhuyan R, Li H, Sarma A, Talukdar J, Sandhya S, Gayan S, Bhuyan S, Gogoi G, Gouw AM, Baishya D, Gotlib JR, Kataki AC, and Felsher DW. MYC regulates the HIF-2α stemness pathway via Nanog and Sox2 to maintain self-renewal in cancer stem cells versus non-stem cancer cells. Cancer Research 2019 Aug 15;79(16):4015-4025. doi: 10.1158/0008-5472.CAN-18-2847. Epub 2019 Jul 2.

114. Gouw AM, Margulis K, Liu NS, Raman SJ, Mancuso A, Toal GG, Tong L, Mosley A, Hsieh AL, Sullivan DK, Stine ZE, Altman BJ, Schulze A, Dang CV, Zare RN, and Felsher DW. The MYC oncogene cooperates with sterol regulated element-binding protein to regulate lipogenesis essential for neoplastic growth. Cell Metabolism, 2019 Aug 19. pii: S1550-4131(19)30385-7. doi: 10.1016/j.cmet.2019.07.012. [Epub ahead of print]

115. Dhanasekaran R, Baylot V, Kim M, Kuruvilla S, Bellovin D, Adeniji N, Rajan A, Lai I, Gabay M, Tong L, Krishnan M, Park J, Hu T, Barbhuiya M, Gentles A, Kannan K, Tran P, Felsher DW. MYC and Twist1 cooperate to drive metastasis by eliciting crosstalk between cancer and innate immunity. eLife Sciences, 2020 Jan 14;9. pii: e50731. doi: 10.7554/eLife.50731.

116. Smith MT, Guyton KZ, Kleinstreuer N, Borrel A, Cardenas A, Chiu WA, Felsher DW, Gibbons CF, Goodson WH, Houck KA, Kane A, La Merrill MA, Lebrec H, Lowe L, McHale CM, Minocherhomji S, Rieswijk L, Sandy MS, Sone H, Wang A, Zhang L, Zeise L, Fielden M. The key characteristics of carcinogens: Relationship to the hallmarks of cancer, relevant biomarkers, and assays to measure them. Cancer

Epidemiology Biomarkers Prevention. 2020 Mar 9. pii: cebp.1346.2019. doi: 10.1158/1055-9965.EPI-19-1346. [Epub ahead of print].

117. Swaminathan S, Hansen AS, Heftdal LD, Dhanasekaran R, Deutzmann A, Fernandez WDM, Liefwalker DF, Horton C, Mosley A, Liebersbach M, Maecker HT, Felsher DW. MYC functions as a switch for natural killer cell-mediated immune surveillance of lymphoid malignancies. Nat Commun. 2020 Jun 5;11(1):2860. doi: 10.1038/s41467-020-16447-7.

118. Dhanasekaran R, Park J, Yevtodiyenko A, Bellovin DI, Adams SJ, Rajan AKD, Gabay M, Fernando H, Arzeno J, Arjunan V, Gryanzov S, Felsher DW. MYC ASO impedes tumorigenesis and elicits oncogene addiction in autochthonous transgenic mouse models of HCC and RCC. Molecular Therapy Nucleic Acids. 2020 Sep 4; 21: 850–859. Published online 2020 Jul 10 doi: 10.1016/j.omtn.2020.07.08

119. Mahauad-Fernandez WD, Felsher DW. The Myc and Ras partnership in cancer: Indistinguishable alliance or contextual relationship? Cancer Research | Perspective 2020, doi: 10.1158/0008-5472.CAN-20-0787. Published September 2020.

120. Cui L, Gouw AM, LaGory EL, Guo S, Attarwala N, Tang Y, Qi J, Chen YS, Gao Z, Casey KM, Bazhin AA, Chen M, Hu L, Xie J, Fang M, Zhang C, Zhu Q, Wang Z, Giaccia AJ, Gambhir SS, Zhu W, Felsher DW, Pegram MD, Goun EA, Le A, Rao J. Mitochondrial copper depletion suppresses triple-negative breast cancer in mice. Nat Biotechnol. 2020 Oct 19. doi: 10.1038/s41587-020-0707-9. Online ahead of print.

121. Krishnan MS, Rajan Kd A, Park J, Arjunan V, Garcia Marques FJ, Bermudez A, Girvan OA, Hoang NS, Yin J, Nguyen MH, Kothary N, Pitteri S, Felsher DW, Dhanasekaran R. Genomic analysis of vascular invasion in hepatocellular carcinoma (HCC) reveals molecular drivers and predictive biomarkers. Hepatology. 2020 Nov 3. doi: 10.1002/hep.31614. Online ahead of print.

122. Hori SS, Tong L, Swaminathan S, Liebersbach M, Wang J, Gambhir SS, Felsher DW. A mathematical model of tumor regression and recurrence after therapeutic oncogene inactivation. Science Reports. 2021; 11: 1341. Published online 2021 Jan 14. doi: 10.1038/s41598-020-78947-2.

123. Eguiarte-Solomon F, Blazanin N, Rho O, Carbajal S, Felsher DW, Tran PT, DiGiovanni J. Twist1 is required for development of UVB-Induced squamous cell carcinoma. Molecular Carcinogenesis. Published online 13 March 2021. doi: 10.1002/mc.23296.

124. Dhanasekaran R, Deutzmann A, Mahauad-Fernandez WD, Hansen AS, Gouw, Felsher DW. The MYC oncogene — the grand orchestrator of cancer growth and immune evasion. Nature Reviews Clinical Oncology (2021) Published 10 September 2021. doi.org/10.1038/s41571-021-00549-2.

125. Lee CK, Atibalentja DF, Yao LE, Park J, Kuruvilla S, Felsher DW. Anti-PD-L1 F(ab) conjugated PEG-PLGA nanoparticle enhances immune checkpoint therapy. Nanotheranostics 2022; 6(3):243-255. doi:10.7150/ntno.65544.

126. Gouw A, Kumar V, Resendez A, Alvina F, Liu N, Margulis K, Tong L, Zare R, Malhotra S, and Felsher DW. Azapodophyllotoxin causes lymphoma and kidney cancer regression by disrupting tubulin and monoglycerols. ACS Medicinal Chemistry Letters, ml-2021-00673x.R1. doi:10.1021/acsmedchemlett.1c00673.

127. Tong L, Shan M, Zou W, Liu X, Felsher DW, and Wang J. Cyclic adenosine monophosphate/phosphodiesterase 4 pathway associated with immune infiltration and PD-L1 expression in lung adenocarcinoma cells. Frontiers in Oncology, 12:904969. Doi: 10.3389/fonc. 2022.904969.

128. Lowe L, LaValley JW, and Felsher DW. Tackling heterogeneity in treatment resistant breast cancer with personalized combination therapeutics. Cancer Drug Resistance, Manuscript ID: CDR-2022-40, dx.doi.org/10.20517/cdr.xxx.

129. Gong YY, Shao H, Li Y, Brafford P, Stine ZE, Sun J, Felsher DW, Orange JS, Albelda SM, and Dang CV. Na/H -exchanger 1 enhances antitumor activity of engineered NK-92 natural killer cells. AACR Cancer Research Communications, doi:org/10.1158/2767-9764.CRC-22-0270. Aug 2022

130. Sullivan DK, Deutzmann A, Yarbrough J, Krishnan MS, Gouw AM, Bellovin DI, Adam SJ, Liefwalker DF, Dhanasekaran R, and Felsher DW. MYC oncogene elicits tumorigenesis associated with embryonic, ribosomal biogenesis, and tissue-lineage dedifferentiation gene expression changes. Springer Nature, Oncogene October 2022, https://doi.org/10.1038/s41388-022-02458-9.

131. Dhanasekaran R, Hansen AS, Park J, Lemaitre L, Lai I, Adeniji N, Kuruvilla S, Suresh A, Zhang J, Swamy V, and Felsher DW. MYC overexpression drives immune evasion in hepatocellular carcinoma that is reversible through restoration of pro-inflammatory macrophages. Cancer Research, February 2023, DOI 10.1158/0008-5472 CAN-22-0232.

132. Smith BAH, Deutzmann A, Correa KM, Delaveris CS, Dhanasekaran R, Dove CG, Sullivan DK, Wisnovsky S, Stark JC, Pluvinage JV, Swaminathan S, Riley NM, Rajan A, Majeti R, Felsher DW and Bertozzi CR. MYC driven synthesis of Siglec ligands is a novel glyco-immune checkpoint. Proc Natl Acad Sci, 2023 March 14;120(11):e2215376120. doi: 10.1073/pnas.2215376120

133. Deutzmann A, Sullivan DK, Dhanasekaran R, Li W, Chen X, Tong L, Mahauad-Fernandez WD, Bell J, Mosley A, Koehler AN, Li Y, Felsher DW. Nuclear to cytoplasmic transport is a druggable dependency in MYC-driven hepatocellular carcinoma. Nature Communications 15, 963 (2024). https://doi.org/10.1038/s41467-024-45128-y.

134. Lemaitre L, Adeniji N, Suresh A, Reguram R, Zhang J, Park J, Reddy A, Trevino AE, Mayer AT, Deutzmann A, Hansen A, Tong L, Arjunan V, Kambham N, Visser B, Dua M, Bonham A, Kothary N, D'Angio HB, Preska R, Rosen Y, Zou J, Charu V, Felsher DW, Dhanasekaran R. Multi-omic Spatial Analysis Reveals Targetable Macrophage-Mediated Mechanisms of Immune Evasion in Hepatocellular Carcinoma Minimal Residual Disease. Nat Cancer. 2024;(in press).

Non-Peer-Reviewed articles (total of 2)

1. Braun, J., Ando, D., Denis, K., Felsher, DW., Govan, H., and Krall, W. Generation and characterization of murine Ly-1 (CD5) B-cell lines. In: O. N. Witte, N. Klinman, M. C. Howard (Eds), B- cell development: UCLA symposia in Molecular and Cellular Biology, New Series, pp. 55-67, New York: Alan R. Liss, Inc., 1988.

2. Braun, J., Felsher, D. W., and Goodglick, L. A. C-MYC, MHCI, and NK resistance in immunodeficiency lymphomas. Annals NY Academy of Sciences, 651:467, 1992.

Reviews (total of 37)

1. Felsher, DW. Cancer Revoked: Oncogenes as therapeutic targets. Nature Reviews Cancer, 3(5) 375, 2003.

2. Giuriato, S., Felsher, DW. How cancers escape their oncogene habit. Cell Cycle, 2(4): 329, 2003.

3. Felsher, DW., and Bradon, N. Pharmacological inactivation of MYC for the treatment of cancer. Drug News Perspect, 16(6): 370-4 2003.

4. Felsher, DW. Oncogenes as therapeutic targets. Seminars in Cancer Biology, (1): 1, 2004.

5. Giuriato, S., Rabin, K, Fan, A. C., Shachaf, C. M., and Felsher, DW. Conditional animal models: a strategy to define when oncogenes will be effective targets to treat cancer. Seminars in Cancer Biology, 14(1): 3-11, 2004.

6. Felsher, DW. Reversibility of oncogene induced cancer. Current Opinion Genetics and Development, 14(1): 37-42, 2004.

7. Felsher, DW. Putting oncogenes into a developmental context. Cancer Biology & Therapy, 3(10): 942-4 EPUB 2004.

8. Shachaf, C. M., and Felsher, DW. Tumor dormancy and MYC inactivation: Pushing cancer to the brink of normalcy. Cancer Research, 65:(11) 4471-4, 2005.

9. Shachaf, C. M., and Felsher, DW. Rehabilitation of cancer through oncogene inactivation. Trends in Molecular Medicine, 11(7): 316-21, 2005.

10. Bachireddy, P. Bendapudi, P., and Felsher, DW. Getting at MYC through RAS. Clinical Cancer Research, 11(12): 4278-81, 2005.

11. Arvanitis, C., and Felsher, DW. Conditionally MYC: insights from novel transgenic models. Elsevier, Cancer Letters, 226(2): 95-9, 2005.

12. Felsher, DW. Tumor Dormancy-Death and resurrection of cancer as seen through transgenic mouse models. Cell Cycle, 5(16): 1808-11, 2006.

13. Arvanitis, C., and Felsher, DW. Conditional transgenic models define how MYC initiates and maintains tumorigenesis. Seminars in Cancer Biology, 16(4): 313-7, 2006.

14. Felsher, DW. Oncogene addiction versus oncogene amnesia: Perhaps more than just a bad habit? Cancer Research, 68(9): 3081-6, 2008.

15. Felsher, DW. Tumor dormancy and oncogene addiction. Acta Pathologica, Microbiologica et Immunologica Scandinavica, 116(7-8): 629-37, 2008.

16. van Riggelen, J. and Felsher, DW. Myc and a Cdk2 senescence switch. Nature Cell Biology, 12(1): 7-9, 2010.

17. van Riggelen, J., Yetil, A., and Felsher, DW. MYC as a regulator of ribosome biogenesis and protein synthesis. Nature Reviews Cancer, 10(4): 301-9, 2010.

18. Felsher, D. W. MYC inactivation elicits oncogene addiction through both tumor cell-intrinsic and host-dependent mechanisms. Genes Cancer, 1(6): 597-604, 2010.

19. Bellovin, D.I., Das, B., Felsher, DW. Tumor dormancy, oncogene addiction, cellular senescence, and self-renewal programs. Adv Exp Med Biol. 2013;734:91-107. doi: 10.1007/978-1-4614-1445-2_6.

20. Felsher, D. W. Role of mycn in retinoblastoma. Lancet Oncol. 2013 Apr;14(4):270-1. doi: 10.1016/S1470-2045(13)70070-6. Epub 2013 Mar 13.

21. Casey, S. C., Bellovin, D. I., and Felsher, DW. Noncanonical roles of the immune system in eliciting oncogene addiction. Current Opinion Immunology, 2013 doi:pii: S0952-7915(13)00023-X. 10.1016/j.coi.2013.02.003 [Epub ahead of print].

22. Fan, A.C., O'Rourke, J.J., Praharaj, D.R., Felsher DW. Real-time nanoscale proteomic analysis of the novel multi-kinase pathway inhibitor rigosertib to measure the response to treatment of cancer. Expert Opin Investig Drugs. 2013 Nov;22(11):1495-509. doi: 10.1517/13543784.2013.829453. Epub 2013 Aug 12.

23. Rakhra, K., Felsher, DW. Generation of a tetracycline regulated mouse model of MYC-induced T-cell acute lymphoblastic leukemia. Methods Mol Biol. 2013;1012:221-35. doi: 10.1007/978-1-62703-429-6_15.

24. Casey, C. C., Li, Y., Felsher, DW. An essential role for the immune system in the mechanism of tumor regression following targeted oncogene inactivation. Immunologic Research, May 2014 58(2-3):282-91. doi: 10.1007/s12026-014-8503-6.

25. Gabay M, Li Y, Felsher DW. MYC activation is a hallmark of cancer initiation and maintenance Cold Spring Harb Perspect Med. 2014 Jun 2;4(6):a014241. doi: 10.1101/cshperspect.a014241.

26. Casey SC, Amedei A, Aquilano K, Azmi AS, Benencia F, Bhakta D, Bilsland AE, Boosani CS, Chen S, Ciriolo MR, Crawford S, Fujii H, Georgakilas AG, Guha G, Halicka D, Helferich WG, Heneberg P, Honoki K, Keith WN, Kerkar SP, Mohammed SI, Niccolai E, Nowsheen S, Vasantha Rupasinghe HP, Samadi A, Singh N, Talib WH, Venkateswaran V, Whelan RL, Yang X, Felsher DW. Cancer prevention and therapy through the modulation of the tumor microenvironment. Semin Cancer Biol. 2015 Dec;35 Suppl:S199-223. doi: 10.1016/j.semcancer.2015.02.007. Epub 2015 Apr 10. Review.

27. Casey SC, Vaccari M, Al-Mulla F, Al-Temaimi R, Amedei A, Barcellos-Hoff MH, Brown DG, Chapellier M, Christopher J, Curran CS, Forte S, Hamid RA, Heneberg P, Koch DC, Krishnakumar PK, Laconi E, Maguer-Satta V, Marongiu F, Memeo L, Mondello C, Raju J, Roman J, Roy R, Ryan EP, Ryeom S, Salem HK, Scovassi AI, Singh N, Soucek L, Vermeulen L, Whitfield JR, Woodrick J, Colacci A, Bisson WH, Felsher DW. The effect of environmental chemicals on the tumor microenvironment. Carcinogenesis. 2015 Jun;36 Suppl 1:S160-83. doi: 10.1093/carcin/bgv035. Review.

28. Goodson WH 3rd, Lowe L, Carpenter DO, Gilbertson M, Manaf Ali A, Lopez de Cerain Salsamendi A, Lasfar A, Carnero A, Azqueta A, Amedei A, Charles AK, Collins AR, Ward A, Salzberg AC, Colacci A, Olsen AK, Berg A, Barclay BJ, Zhou BP, Blanco-Aparicio C, Baglole CJ, Dong C, Mondello C, Hsu CW, Naus CC, Yedjou C, Curran CS, Laird DW, Koch DC, Carlin DJ, Felsher DW, Roy D, Brown DG, Ratovitski E, Ryan EP, Corsini E, Rojas E, Moon EY, Laconi E, Marongiu F, Al-Mulla F, Chiaradonna F, Darroudi F, Martin FL, Van Schooten FJ, Goldberg GS, Wagemaker G, Nangami GN, Calaf GM, Williams G, Wolf GT, Koppen G, Brunborg G, Lyerly HK, Krishnan H, Ab Hamid H, Yasaei H, Sone H, Kondoh H, Salem HK, Hsu HY, Park HH, Koturbash I, Miousse IR, Scovassi AI, Klaunig JE, Vondráček J, Raju J, Roman J, Wise JP Sr, Whitfield JR, Woodrick J, Christopher JA, Ochieng J, Martinez-Leal JF, Weisz J, Kravchenko J, Sun J, Prudhomme KR, Narayanan KB, Cohen-Solal KA, Moorwood K, Gonzalez L, Soucek L, Jian L, D'Abronzo LS, Lin LT, Li L, Gulliver L, McCawley LJ, Memeo L, Vermeulen L, Leyns L, Zhang L, Valverde M, Khatami M, Romano MF, Chapellier M, Williams MA, Wade M, Manjili MH, Lleonart ME, Xia M, Gonzalez MJ, Karamouzis MV, Kirsch-Volders M, Vaccari M, Kuemmerle NB, Singh N, Cruickshanks N, Kleinstreuer N, van Larebeke N, Ahmed N, Ogunkua O, Krishnakumar PK, Vadgama P, Marignani PA, Ghosh PM, Ostrosky-Wegman P, Thompson PA, Dent P, Heneberg P, Darbre P, Sing Leung P, Nangia-Makker P, Cheng QS, Robey RB, Al-Temaimi R, Roy R, Andrade-Vieira R, Sinha RK, Mehta R, Vento R, Di Fiore R, Ponce-Cusi R, Dornetshuber-Fleiss R, Nahta R, Castellino RC, Palorini R, Abd Hamid R, Langie SA, Eltom SE, Brooks SA, Ryeom S, Wise SS, Bay SN, Harris SA, Papagerakis S, Romano S, Pavanello S, Eriksson S, Forte S, Casey SC, Luanpitpong S, Lee TJ, Otsuki T, Chen T, Massfelder T, Sanderson T, Guarnieri T, Hultman T, Dormoy V, Odero-Marah V, Sabbisetti V, Maguer-Satta V, Rathmell WK, Engström W, Decker WK, Bisson WH, Rojanasakul Y, Luqmani Y, Chen Z, Hu Z. Assessing the carcinogenic potential of low-dose exposures to chemical mixtures in the environment: the challenge ahead. Carcinogenesis. 2015 Jun;36 Suppl 1:S254-96. doi: 10.1093/carcin/bgv039. Review.

29. Kearney AY, Anchang B, Plevritis S, Felsher DW. ARF: connecting senescence and innate immunity for clearance. Aging (Albany NY). 2015 Sep;7(9):613-5.

30. Block KI, Gyllenhaal C, Lowe L, Amedei A, Amin AR, Amin A, Aquilano K, Arbiser J, Arreola A, Arzumanyan A, Ashraf SS, Azmi AS, Benencia F, Bhakta D, Bilsland A, Bishayee A, Blain SW, Block PB, Boosani CS, Carey TE, Carnero A, Carotenuto M, Casey SC, Chakrabarti M, Chaturvedi R, Chen GZ, Chen H, Chen S, Chen YC, Choi BK, Ciriolo MR, Coley HM, Collins AR, Connell M, Crawford S, Curran CS, Dabrosin C, Damia G, Dasgupta S, DeBerardinis RJ, Decker WK, Dhawan P, Diehl AM, Dong JT, Dou QP, Drew JE, Elkord E, El-Rayes B, Feitelson MA, Felsher DW, Ferguson LR, Fimognari C, Firestone GL, Frezza C, Fujii H, Fuster MM, Generali D, Georgakilas AG, Gieseler F, Gilbertson M, Green MF, Grue B, Guha G, Halicka D, Helferich WG, Heneberg P, Hentosh P, Hirschey MD, Hofseth LJ, Holcombe RF, Honoki K, Hsu HY, Huang GS, Jensen LD, Jiang WG, Jones LW, Karpowicz PA, Keith WN, Kerkar SP, Khan GN, Khatami M, Ko YH, Kucuk O, Kulathinal RJ, Kumar NB, Kwon BS, Le A, Lea MA, Lee HY, Lichtor T, Lin LT, Locasale JW, Lokeshwar BL, Longo VD, Lyssiotis CA, MacKenzie KL, Malhotra M, Marino M, Martinez-Chantar ML, Matheu A, Maxwell C, McDonnell E, Meeker AK, Mehrmohamadi M, Mehta K, Michelotti GA, Mohammad RM, Mohammed SI, Morre DJ, Muralidhar V, Muqbil I, Murphy MP, Nagaraju GP, Nahta R, Niccolai E, Nowsheen S, Panis C, Pantano F, Parslow VR, Pawelec G, Pedersen PL, Poore B, Poudyal D, Prakash S, Prince M, Raffaghello L, Rathmell JC, Rathmell WK, Ray SK, Reichrath J, Rezazadeh S, Ribatti D, Ricciardiello L, Robey RB, Rodier F, Rupasinghe HP, Russo GL, Ryan EP, Samadi AK, Sanchez-Garcia I, Sanders AJ, Santini D, Sarkar M, Sasada T, Saxena NK, Shackelford RE, Shantha Kumara HM, Sharma D, Shin DM, Sidransky D, Siegelin MD, Signori E, Singh N, Sivanand S, Sliva D, Smythe C, Spagnuolo C, Stafforini DM, Stagg J, Subbarayan PR, Sundin T, Talib WH, Thompson SK, Tran PT, Ungefroren H, Vander Heiden MG, Venkateswaran V, Vinay DS, Vlachostergios PJ, Wang Z, Wellen KE, Whelan RL, Yang ES, Yang H, Yang X, Yaswen P, Yedjou C, Yin X, Zhu J, Zollo M. Designing a broad-spectrum integrative approach for cancer prevention and treatment. Semin Cancer Biol. 2015 Dec;35 Suppl:S276-304. doi: 10.1016/j.semcancer.2015.09.007. Review.

31. Gouw AM, Toal GG, Felsher DW. Metabolic vulnerabilities of MYC-induced cancer. Oncotarget. 2016 Feb 6. doi: 10.18632/oncotarget.7223. [Epub ahead of print] Review.

32. Li Y, Deutzmann A, Felsher DW.BIM-mediated apoptosis and oncogene addiction. Aging (Albany NY). 2016 Sep 29;8(9):1834-1835. doi: 10.18632/aging.101072. No abstract available. PMID: 27688082 Free PMC Article

33. Felsher DW, Lowe L. Affordable Cancer Medications are within reach but we need a different approach. J Clin Oncol. 2016 Jun 20;34(18):2194-5. doi: 10.1200/JCO.2016.67.2436. No abstract available. PMID: 27161965

34. Casey SC, Baylot V, Felsher DW. The MYC oncogene is a global regulator of the immune response. Blood. 2018 May 3;131(18):2007-2015. doi: 10.1182/blood-2017-11-742577. Epub 2018 Mar 7. Review. PMID: 29514782

35. Felsher DW. A Tale of Two Complications of Obesity: Nonalcoholic steatohepatitis (NASH) and hepatocellular carcinoma (HCC). Hepatology, 2019 Apr 8. doi: 10.1002/hep.30649. [Epub ahead of print] No abstract available. PMID: 30958566

36. Dhanasekaran R, Deutzmann A, Mahauad-Fernandez WD, Hansen AS, Gouw, Felsher DW. The MYC oncogene — the grand orchestrator of cancer growth and immune evasion. Nature Reviews Clinical Oncology (2021) Published 10 September 2021. doi.org/10.1038/s41571-021-00549-2.
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.

14. Sundberg, C. D., Tang, F., and Felsher, D. W. The loss of p53 function prevents MYC inactivation from causing sustained tumor regression. SALK/EMBL, San Diego, CA, August 2001.
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.

Exhibit 2

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

Dear Lori

Here is my CV.

My rates:

1000/hour review and preparation

1500/hour trial and depo, patient interview

Dean

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

Exhibit 3

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

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

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