Exhibit 2

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	Case No. 16-md-02741-VC MDL No. 2741
This document relates to: ALL ACTIONS	

EXPERT REPORT OF WILLIAM FLEMING, M.D., Ph.D.

July 31, 2017

I am currently a Professor of Medicine & Pediatrics at Oregon Health & Science University. I received my M.D., Ph.D. degrees from the University of Manitoba and completed my internship, medical residency and fellowship in medical oncology at Stanford University. I became board certified in Internal Medicine in 1995 and Medical Oncology in 2000. I was an Assistant Professor of Medicine at Emory University before moving to Oregon Health & Science University, where I was promoted to Associate Professor in 2001 and to Full Professor in 2008. From 2010 to 2015, I served as the Leader of the Hematologic Malignancies Program for the Cancer Center Support Grant for the Knight Cancer Institute at Oregon Health & Science University.

My research interests are focused on normal and malignant blood cells including leukemia, lymphoma and related blood cancers. In addition to clinical manuscripts, I have published more than 50 peer-reviewed scientific manuscripts in the field of experimental hematology. I have been a member of various professional organizations, including The American Society of Hematology, American Society of Blood and Bone Marrow Transplant, The International Society of Experimental Hematology, and The International Society for Stem Cell Research.

I have served as a regular member of the American Cancer Society: Leukemia, Immunology & Blood Cell Development Study Section (2001-2005). I have been a regular member of the Molecular and Cellular Hematology Study Section (2008-2009), a group that routinely reviews grant applications related to blood cancers. I also have served as an ad hoc reviewer on numerous NIH study sections and international study sections for research related to normal and leukemic blood cells. I have participated on study sections that review National Cancer Institute funded Cancer Centers and currently I am an ad hoc reviewer for National Cancer Institute subcommittee that evaluates physician-scientist trainees. During an average year, I participate on between 3 to 5 NIH study sections, special emphasis panels or related review activities. I regularly review manuscripts on hematopoietic malignancies and stem cell biology for several journals including the New England Journal of Medicine, Science, Nature, Nature Medicine, Journal of Clinical Investigation, Blood, Proceedings of the National Academy of Sciences, Stem Cells, and Experimental Hematology.

For more than 20 years, I have served as an attending physician on the Bone Marrow Transplant / Leukemia service at Emory University and Oregon Health & Science University and as an attending physician in Hematology/Oncology at the Portland Veterans Administration Hospital. During this time, my practice has been focused exclusively on the care of patients with blood cancers. Throughout my career I have participated in the diagnosis and treatment of hundreds of patients diagnosed with a non-Hodgkin's Lymphoma (NHL). This includes patients with aggressive forms of NHL that require stem cell transplants and others with more indolent subtypes that only require close observation. I have also taught classes on normal and malignant blood cell development to medical students, residents and fellows at Emory and OHSU. Details of my education, research, training and publications are provided in the attached curriculum vitae. (Attachment A)

I am being compensated at a rate of $600 per hour. During the past four years, I have not testified as an expert witness. Unless otherwise stated, all opinions expressed in this report are to a reasonable degree of scientific certainty and I reserve the right to supplement my report as new information becomes available.

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Lymphocytes: Key cellular component of the immune system

Stem cells in the bone marrow give rise to all the major cell types found in the blood throughout the life of an individual. Lymphocytes represent about one third of the total white blood cells and are a key component of the immune system. In addition to circulating in the blood, lymphocytes are the major cell type found in the spleen and in the lymph nodes (Figure1). Lymphocytes are typically long-lived cells that are responsible for protecting against infection and these immune cells are increasingly recognized to play a role in the development of cancer. These cells are functionally divided into 2 major subtypes. B-lymphocytes are the cells responsible for producing antibodies against targets (antigens) found on a variety of infections agents including bacteria and viruses. A second subset of lymphocytes referred to as T-cells function to help B-cells make these antibodies and some of these T-cells are also capable of directly attacking and killing the infectious agents.

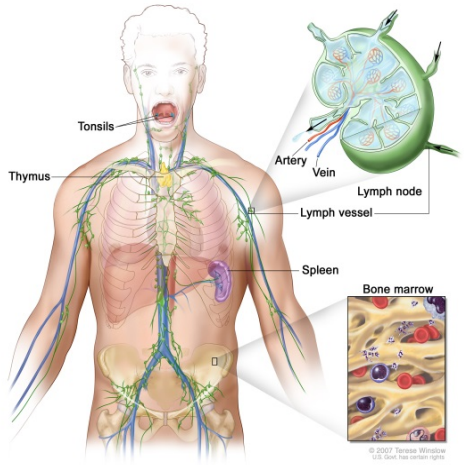

Figure1. Lymphocytes and the lymphatic system. Originating from bone marrow stem cells, lymphocytes are found in the blood (blue), lymph nodes (green) and the spleen (purple).

The coordinated response of B-cells and T-cells to antigens and other triggers is essential to provide immunity to many different types of infections. Antibodies produced by B-cells have also been engineered to kill cancer cells most notably in patients with lymphoma and breast cancer. A promising new therapy involves blocking mechanisms that inhibit T-cell function as this markedly enhances the immune response to melanoma, lung cancer and other tumors

If the immune system loses its capacity to regulate itself, instead of normally responding to the challenges of infections it may inappropriately react to normal tissues. Collectively, these disorders are referred to as autoimmune diseases. These abnormal immune cell interactions can produce significant inflammation in tissues throughout the body. Well known examples of autoimmunity include the inflammation of the joints that occurs in patients with rheumatoid arthritis and related diseases. Autoimmunity has also been associated with inflammatory bowel diseases including ulcerative colitis and Crohn's disease. Other examples of immune related diseases include systemic lupus erythematosus and Sjögren's syndrome. A history of autoimmunity is clinically relevant as several autoimmune diseases are known to be associated with a significantly increased risk for the development of cancer. Importantly, lymphocytes themselves can be come malignant then grow in an uncontrolled manner and accumulate in the blood, lymph nodes and spleen. These malignant lymphocytes are cancers of the immune system itself and are referred to as lymphoma.

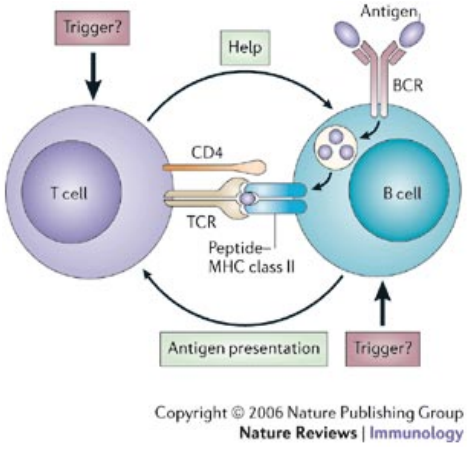

Figure 2. Lymphocyte interactions during the normal immune reponse. In response to an infectious agent (antigen or other trigger), T-cells help B-cells make antibodies and B-cells process and present antigens causing a T-cell response.

Non-Hodgkin's Lymphoma

In 1832, Thomas Hodgkin discovered that the accumulation of large numbers of abnormal lymphocytes could lead to enlargement of the lymph nodes or lymphoma. This was an important finding, as enlarged lymph nodes were previously thought to be caused by either infection or from metastatic cancer that had spread to the lymph nodes from breast cancer, colon cancer or other solid tumors. Additional research revealed a unique cell type under the microscope, characteristic contiguous patterns of lymph node involvement and high frequency of patients in their late teens and early twenties. This disorder came to be known as Hodgkin's disease and later Hodgkin's lymphoma. Over time it was recognized many patients with lymphoma did not meet the original diagnostic criteria for Hodgkin's lymphoma and this entire group of diseases became known as non-Hodgkin's lymphoma or NHL. Population based studies in the U.S. reveals that NHL occurs almost 9 times more frequently than Hodgkin's disease and today NHL is the seventh most common cancer. The American Cancer Society (ACS) estimates there will be 72,000 new cases of NHL in the United States in 2017. The most recent ACS data also estimates that adult men and women have approximately a 2% chance of developing NHL during their lifetime.

Over the past 3 decades it has been increasingly recognized that NHL is a highly diverse group of lymphoid tumors. The 2016 update of the classification of lymphoid tumors by the World Health Organization now recognizes more than 60 distinct subtypes of NHL. This classification system is of practical importance because of the remarkable differences between these NHL subtypes. The clinical behavior of NHL ranges from aggressive, high grade tumors that without treatment can lead to death in a matter of weeks to months (Burkitt's lymphoma) to indolent diseases such as follicular lymphoma and small lymphocytic lymphoma where long-term observation without therapy is standard management in the majority of patients. For these reasons the term NHL is not specific enough to either determine prognosis or to guide therapy in individual patients. Although the classification of NHL subtypes continues to evolve, the historical classification of NHL remains useful in order to study the incidence and demographics of patients with NHL over time.

NHL Trends in the U.S.

In order to better understand how NHL impacts the U.S. population, it is helpful to evaluate the annual incidence on a nationwide basis. The U.S. Surveillance, Epidemiology, and End Results (SEER) Program administered by the National Cancer Institute collects detailed information on cancer statistics as part of a national effort in an effort to reduce the cancer burden in the U.S. In addition to leadership in the science of cancer surveillance, SEER provides a number of analytical tools for interpreting and disseminating reliable population-based statistics. This robust data set is publically available along with well-established analytical tools that allow investigators to ask specific questions about the incidence, age at diagnosis and mortality of NHL (https://seer.cancer.gov/resources/). Detailed demographic data from 2008-2012 is also available and this data set can be used to evaluate the incidence of NHL at both the state-wide level and at the county level throughout the U.S. (https://gis.cancer.gov/geoviewer/).

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The SEER 9 database includes NHL cases from 1975 through 2014 and shows the changing incidence of NHL over this time interval. As shown in Figure 3A, between 1975 and 1990 the annual incidence of new cases of NHL per year in the U.S in persons over 50 years of age increased from 30 per 100 thousand to 50 per 100 thousand or about 66%. During the next 15 year interval, the incidence of NHL increased more modestly from 50 to 64 per 100 thousand and by 2004 it began to plateau before beginning to slowly decline over the next 10 years. For younger individuals, between 20 and 49 years of age, even more dramatic changes in incidence were observed. Between 1980 and 1990 the incidence doubled, increasing from 5 per 100 thousand to 10 per 100 thousand. By 1997 the incidence of NHL had declined by about 25% then it plateaued followed by a further decline that continued through 2014.

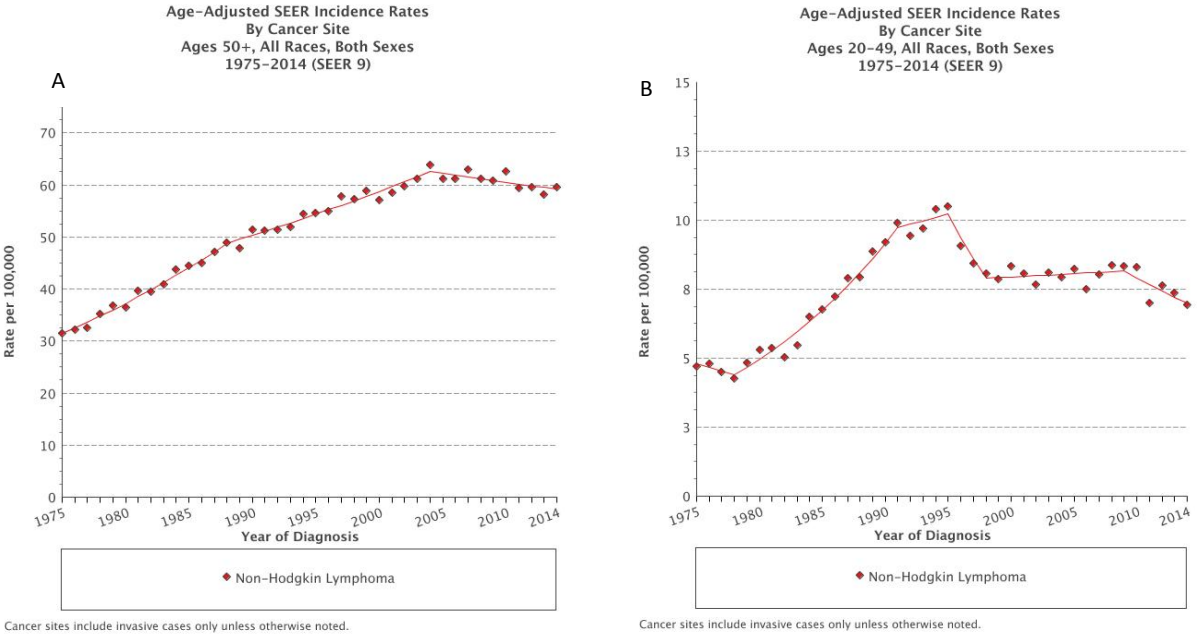


Figure 3. SEER data for the incidence of NHL in the U.S. population between 1975 and 2014. Panel A: individuals > 50 years of age. Panel B: individuals 20-49 years of age.

One theory some experts have offered to explain part of the increased in incidence in NHL from 1975 through 2003 in adult over 50 years of age is the exposure to extrinsic factor(s). The observed plateau in NHL incidence beginning in about 2004 followed by a decline over through 2014 could potentially be explained by i) a decrease in the presence of extrinsic factor(s) that previously increased the risk for NHL, ii) the introduction of new external factor(s) that in some way protect against the development of NHL or iii) a combination of both i) and ii). To date, I am not aware of any data that identifies any specific extrinsic factors that are definitively responsible for the initial rise and subsequent decline in the incidence of NHL during this almost 40 year time interval.

The Etiology of Lymphoma

It is generally accepted that most cancer results from the gradual accumulation of gene mutations over time. Consequently, the greatest risk factor for developing most types of cancer is aging. The question of what causes these mutations is of considerable interest to the cancer research community. In the case of lung cancer, the influence of cigarette smoking has been well documented by epidemiological studies. Additionally, the important role of inherited genes is apparent from many studies that have identified several different genes that predispose family members to develop cancer. Although both genetics and external agents certainly play an important role in the etiology of cancer, it has long been appreciated that they do not account for the majority of cancers. Recently, researchers have utilized international cancer databases, cancer genome sequencing and epidemiologic data to address this important question. These investigators have found that random, unavoidable gene mutations that accumulate over time are likely to be responsible for about two-thirds of the mutations in human cancer (Tomasetti et. al. Nature 2017).

For the great majority of patients, the cause of their NHL is unknown. Unlike breast cancer, ovarian cancer and colon cancer, a family history of NHL is uncommon. Although an increasing number of gene mutations have been identified as driving the development of several cancers, to date, none have been definitively identified for NHL. There are however, several well established risk factors for NHL. These are useful to review as they define the magnitude of the increased risk for certain groups of individuals. Furthermore, they help define the time interval required for the development of NHL, a time period known as latency.

Aging is one of the most important risk factors for developing NHL. In 2014, the incidence of NHL was ~7 cases per 100,000 people for individuals between 20-50 years of age while the incidence in individuals over 50 years of age was 8.6-fold higher or 60 cases per 100,000. (See Figure 3 above). Defects in the immune function are an important risk factor for developing several types of cancer including NHL. Impaired immune function can result from a number of causes including i) inherited genetic disorders; ii) autoimmune diseases such as systemic lupus erythematosus and iii) the use of immunosuppressive drugs. The risk of developing NHL in certain subsets of patients with immune dysfunction is often increased 5-fold and in certain circumstances the risk may exceed 20-fold. In some clinical settings, this increased risk can be observed as early as one year after the onset of immune dysfunction and it may persist for many years.

In addition to the factors listed above that strongly increase the risk for developing NHL, other risk factors have been identified. Viral infections are also known to play a role in NHL in certain patient populations. For example Hepatitis C infection causes inflammation of the liver and this is associated with a 1.8-fold increased risk for developing NHL. Although NHL does not run in families, if any first degree relative has any form of blood cancer, this increases the overall risk of NHL by about 2-fold. Other potential risk factors continue to be identified, particularly for specific NHL subtypes.

Latency of NHL

It is well established that some patients with non-blood cell tumors who have previously treated with chemotherapy and or radiation therapy later develop second cancers of the blood. These secondary cancers are thought to be due to direct DNA damage of blood stem cells and their progeny. DNA damage accumulates in normal blood cells and can lead to malignant transformation over time. The blood cells that typically become malignant are from the myeloid blood cell lineage, a family of blood cells that are biologically very distinct from lymphocytes. Myeloid cancer, predominately acute myeloid leukemia and a form of pre-leukemia known as myelodysplastic syndrome usually develops between 6-9 years after cancer therapy. This time delay between exposure to DNA damaging chemotherapy/radiation and the onset of a secondary blood cancer is referred to as latency.

In contrast to acute myeloid leukemia or myelodysplastic syndrome, NHL does not typically occur after treatment for non-blood cell cancers. However, it has long been recognized that patients treated for Hodgkin's lymphoma with chemotherapy and/or radiation therapy have an increased risk for developing NHL (Krikorian et. al. 1979). Analysis of multicenter, long-term survivor data from cancer registries reveals that NHL occurs in about 5% of Hodgkin's lymphoma patients and arises as soon as 5 years after treatment and the increased risk peaks at about 10 years (Krishnan et. al. 2007). Secondary NHL has also been reported in children and adolescents after cancer treatment. This treatment related NHL occurs with a latency period of about 6 years (Landmann et. al. 2008). Outside of the context of prior chemotherapy/radiation therapy and immune suppression, the latency of NHL is unknown. However, based on the available data, 6 to 10 years is certainly a reasonable time interval to begin to evaluate the role of specific exposures on the incidence of NHL.

Agricultural workers and the risk of developing NHL

Despite having a healthy lifestyle and a low overall mortality, it has been observed that agricultural workers are at an increased risk for developing several types of cancer including brain tumors, prostate cancer, multiple myeloma and NHL. Some experts have hypothesized that certain occupational exposures may explain these higher rates of these cancers in farmers and these exposures may potentially influence cancer rates in the general population. Studying this problem can be challenging as agricultural workers are often exposed to a wide range of chemicals over the course of several years. The complexity of this problem was part of the impetus for NIH to conduct a large prospective study of cancer in agricultural workers in 1993 called the Agricultural Health Study. This study has enrolled than about 89,000 farmers and their spouses and continues to provide valuable information about the incidence of specific cancers and other health outcomes. Results of this study as it pertains to the risk of developing NHL will be reviewed below.

Total glyphosate usage and the risk of developing NHL

As part of my expert opinion on NHL, I was asked to comment on whether glyphosate was potentially associated with an increased risk of developing NHL. To begin to evaluate this question, I examined data for the total usage of glyphosate in the U.S. (USGS) and compared this to the overall incidence of NHL (SEER9) from 1975 through 2014. Glyphosate was initially registered in the U.S. in 1974. Therefore, assuming a 10 year latency period, glyphosate cannot be implicated in the ~38% increase in the incidence of NHL seen between 1975 and 1985 (Figure 3.). By 1990 the annual usage of glyphosate in the U.S. had increased from 1.4 million lbs. to 15 million lbs. Annual usage increased further to 40 million lbs. by 1995 and then to 98 million lbs. in 2000. By 2014, a full 14 years after the annual usage of glyphosate reached 98 million lbs., the annual incidence of NHL continued to slowly decline.

In summary, although the annual usage of glyphosate in the U.S. increased 70-fold from 1974 to 2000, after a 10 year latency following this high rate of usage, the incidence of NHL has actually decreased. This finding is the opposite of the anticipated result if total glyphosate use was associated with an increased risk of developing NHL. It is worth noting that although this analysis evaluates the temporal relationship between total glyphosate usage and the overall incidence of NHL, it does not take into account any regional differences in either glyphosate usage or NHL incidence. These are potentially important variables that will be considered in the next section.

Regional differences in glyphosate usage and the incidence of NHL

In order to identify potential correlations between regional differences in glyphosate use and NHL incidence, it is important to examine databases that provide a nation-wide map of these variables. To address the question of regional glyphosate use, data from the U.S. Geological Survey (USGS) was evaluated. The USGS National Water-Quality Assessment Project (NAWQA) provides annual pesticide use maps of the U.S. from 1992 through 2014. Where available, estimates for the amount of glyphosate used per square mile nationwide are provided using 4 defined levels namely i) < 4.52 lbs; ii) 4.52 – 21.12 lbs. iii) 21.13 – 88.06 lbs. and iv) >88.06 lbs. Plotting these 4 levels of glyphosate usage throughout the U.S. provides a visual representation of the annual amount of glyphosate used (Figure 4). This data indicates the highest levels of usage (>88 lbs.) on the west coast of the U.S. are in eastern Washington, eastern Oregon and the central valley of California. Heavy annual glyphosate usage was also found throughout the Midwest, in Texas and along the Mississippi River Valley to the Gulf coast. A similar pattern of high usage was observed along the eastern seaboard extending from southern New York into Florida. As would be expected, high levels of glyphosate use are found throughout the major agricultural regions across the nation.

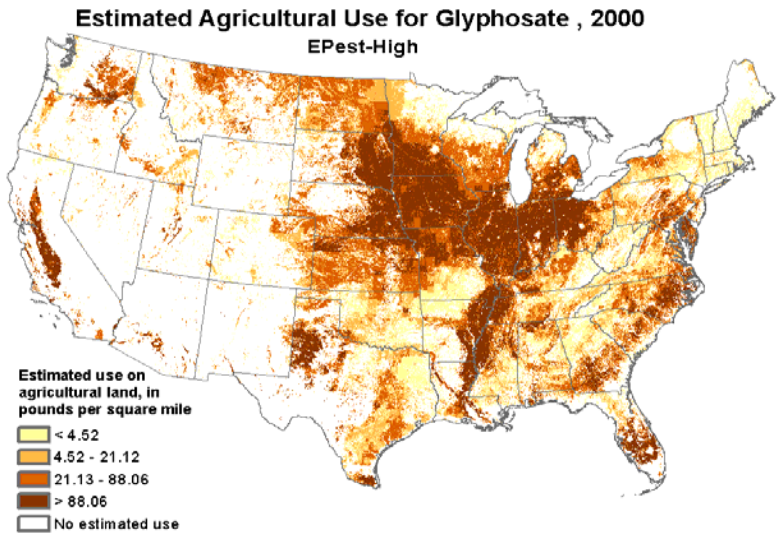


Figure 4: Glyphosate Use in the U.S. (2000)

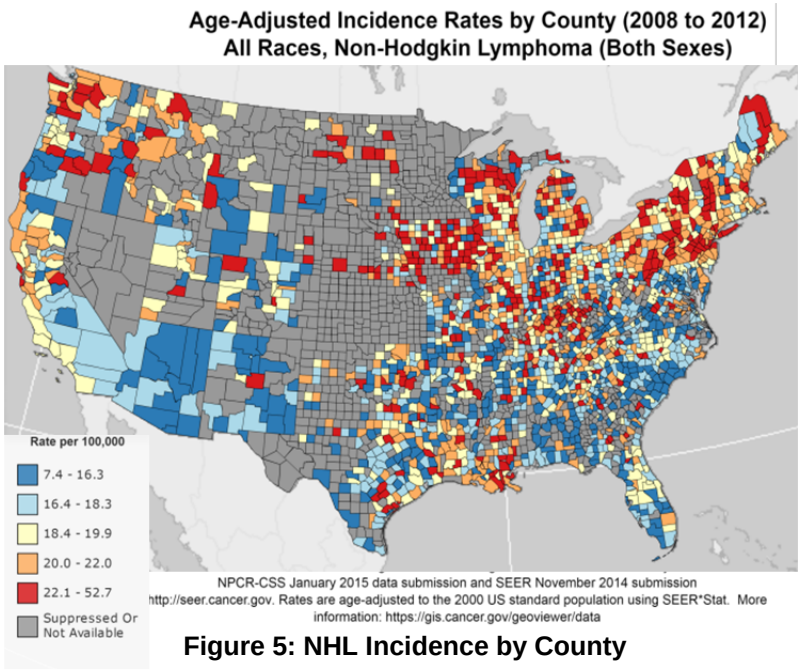


Figure 5: NHL Incidence by County

A useful feature of the SEER database is that it can provide age-adjusted incidence rates and mortality rates for various cancers by county. Using the SEER Geoviewer tool, the incidence of NHL in the U.S. can be visualized on a county by county basis from 2008 through 2012. The incidence rates for NHL are indicated by 4 color coded groups ranging from a low of 7.4 cases per 100,000 person per year to up to 52.7 cases per 100,000 persons per year. (Figure 5; Counties with no evaluable data are shown in grey). Comparing the patterns seen in Figure 4 and Figure 5 provides a detailed visual representation of glyphosate use in 2000 that can be compared to the incidence of NHL up to 12 years later. On the west coast of the U.S., the most prominent region of high glyphosate use is the central valley of California, one of the largest agricultural areas in the nation. This area includes the counties of Sacramento, San Joaquin and Fresno, all of which have relatively high glyphosate usage but also have relatively low incidences of NHL (blue and yellow). By contrast, Marin and San Mateo County counties include several major urban areas in the San Francisco Bay area that have a high incidence of NHL (red) but relatively low usage of glyphosate.

In Washington State, glyphosate use is concentrated in agricultural areas of eastern Washington while conversely, the highest incidences of NHL are found mostly clustered in western Washington, particularly King, Snohomish and surrounding counties. This region encompasses several large urban centers including Tacoma, Seattle and Everett with low

glyphosate usage. Additional inverse correlations are found in the north eastern U.S. with modest usage of glyphosate in northern Maine but high incidences of NHL in these counties. Significant agricultural activity occurs in the southeastern U.S. and this extends deep into Florida. These areas typically show a high use of glyphosate along with a modest incidence of NHL. Similar patterns are seen in the lower portion of the Mississippi river valley. Parts of the midwestern U.S. are challenging to evaluate as there is incomplete county level data for the incidence of NHL in the SEER data base. Overall, the data shown above indicates that incidence of NHL generally appears to be highest in urban and suburban areas with modest glyphosate usage while major agricultural regions, that often have with a ≥ 20-fold usage of glyphosate have relatively low incidences of NHL. A similar nation-wide distribution of glyphosate usage in the USGS, NAWQA database is observed going back as far as 1992. In summary, these findings are essentially the opposite of what would be expected if glyphosate exposure was positively associated with the risk of developing NHL in a dose dependent manner.

Studies of Glyphosate and the Risk of NHL

To date, there has been only one prospective study that has investigated the risk of NHL associated with exposure to glyphosate. The Agricultural Health Study (AHS) is a prospective cohort study that enrolled 57,311 licensed pesticide applicators from 1993 through 1997 (De Roos et. al. 2005) with follow up through the end of 2001. The investigators studied the relationship between glyphosate exposure and the incidence of all cancers combined as well as 12 common cancer subtypes. The important issue of exposure dose was evaluated using both cumulative lifetime days of use and intensity-weighted cumulative exposures. Glyphosate exposure was not associated with an increased risk of developing cancer overall. Specifically for NHL, the RR was 1.1 (95% CI 0.7-1.9) and this risk did not increase with increasing levels of glyphosate exposure.

Some have criticized this study claiming that the median follow-up time was only 6.7 years and that a longer follow up period may be necessary to observe an increased incidence of NHL following glyphosate exposure. It is important to note that the median exposure time of subjects prior to enrolling in this study was an average of 15 years. In other words, these study subjects had ~20 years of total occupational exposure when this data was analyzed. This time period is more than adequate to allow for the expected latency for NHL.

A review of the medical literature revealed additional follow up data from the AHS study that focused on the risk of developing NHL. Prompted by prior studies suggesting an inverse correlation between allergies and NHL, the AHS investigators confirmed a significant reduction in NHL in farmers and spouses with allergic rhinitis (HR=0.63, 95% CI=0.51–0.79) and following exposures to either soybeans or stored grain (Hofmann et. al. 2015). This study also showed that growing up on a farm through 18 years of age was associated with an increased risk of NHL (HR=1.51, 95% CI=1.15–1.98). As approximately half of AHS study participants were less than 50 years old in 1993, study participants, on average would have reached 18 years of age

by 1961. This is more than a decade before glyphosate was registered in the U.S. in 1974. Consequently, glyphosate exposure cannot be implicated with increased risk of NHL observed in individuals who grew up on a farm. A second manuscript from the AHS study focused on NHL after exposure to 26 pesticides (Alavanja et. al. 2014). This study found an increased risk of certain NHL subtypes associated with exposure to DDT, lindane, permethrin, diazinon and terbufos. The authors concluded that while pesticides from different chemical and functional classes were associated with an increased risk of NHL, not all members of a class were associated with an elevated risk of total NHL or its subtypes.

Recently, I was provided with the copy of a draft manuscript entitled "Lymphoma risk and pesticide assessment in the Agricultural Health Study" by Alavanja et. al. dated March 15, 2013. This was marked at the deposition of Dr. Aaron Blair, which I have also reviewed. At his deposition, Dr. Blair noted that this manuscript analyzed all primary NHL cases through December 31, 2008 adding an additional 7 years to the original follow up period. Dr. Blair also noted that this research captured another five years of exposure data. As expected, the incidence of NHL increased significantly with increasing age in the AHS cohort by 2008. The total number of cases of NHL more than tripled from 92 to 333 between the 2001 and 2008 analysis. When other occupational factors were evaluated, the number of livestock on the farm and the use of farm equipment with diesel engines showed a significantly increased risk for developing NHL. However, when the lifetime days of exposure or intensity-weight exposure of glyphosate was examined, the RR for developing NHL was between 0.8 and 1.0 and the trend was not significant (Table 2 in March 2013 draft). When subtypes of NHL were evaluated, most major subtypes showed a trend towards a decreased risk of NHL at the highest doses of glyphosate (RR= 0.6 to 0.7); this did not reach statistical significance with the exception of diffuse large B-cell lymphoma (DLBCL; P trend= 0.05). It is important to note that this this draft manuscript evaluated a total of 50 compounds and in contrast to glyphosate, 8 chemically diverse compounds were significantly associated with an increased risk of specific subtypes of NHL. A subset of the results in this draft manuscript evaluating the increased risk of NHL after exposure to 5 pesticides was published in 2014 (Alavanja et. al. 2014; see above). In summary, with a minimum of follow up of 13.7 years in the AHS, there was a borderline significant inverse association with glyphosate exposure in the diffuse large B-cell lymphoma subtype of NHL. No positive associations were observed.

Summary

Some experts have used the Bradford-Hill criteria to make the argument that glyphosate exposure increases the risk of NHL. However, applying the Bradford-Hill elements of specificity, temporality, and the presence of a biological gradient to the available data on glyphosate does not support this conclusion.

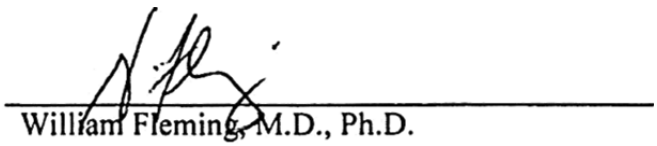
- There was a steep rise in the incidence of NHL incidence between 1975 and 1985. No etiologic agent has been identified, however assuming a latency of $\geq$10years, any related exposures would have occurred many years before glyphosate was introduced in 1974. Consequently there is no temporal relationship between glyphosate and NHL during this time period and other unknown factors must be responsible.

- From the time of its registration in 1974 through the year 2000, the total usage of glyphosate increased more than 70-fold while the incidence of NHL nationwide actually peaked then declined. Allowing for a 10 year latency period, this is the opposite of the expected result if glyphosate increased the risk of developing NHL.

- Regional differences in the usage of glyphosate in the U.S. at the county level can vary by more than 20-fold, yet many of the counties with the highest usage of glyphosate have a relatively low incidence of NHL. This data further argues against a relationship between glyphosate exposure and an increased risk of developing NHL.

- The AHS, the only prospective cohort study of glyphosate and NHL, examined more than 57,000 subjects and did not show an increased risk of NHL following glyphosate exposure. Analysis of the data does not support an increased risk for NHL overall, while a subset analysis of diffuse large B-cell lymphoma reveals a trend towards a decreased risk with increasing glyphosate exposure.

Based on my expertise, and after reviewing the documents I have been provided in addition to my review of the medical literature, I conclude with a reasonable degree of medical certainty that the available evidence does not support the conclusion that glyphosate exposure increases the risk of developing NHL.



William Fleming, M.D., Ph.D.

DATED: July 31, 2017

CURRICULUM VITAE

June 2017

William Harvard Fleming M.D., Ph.D.

PRESENT POSITION AND ADDRESS

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EDUCATION

Undergraduate and graduate:

B.A. Philosophy, University of Winnipeg, Winnipeg, Manitoba, Canada 1979
M.D. Ph.D., Faculty of Medicine, Joint M.D./Ph.D. Program, 1987
University of Manitoba, Winnipeg, Manitoba. Thesis Title: *Human Growth Hormone Mediated Oncogene Expression in Nb2 Lymphoma Cells.* Research Supervisors: Drs. Henry Friesen and Robert J. Matusik.

Postgraduate:

Intern and Medical Resident, Stanford University Hospital 1987-1989
Oncology Fellow, Stanford University Medical Center 1989-1993
Postdoctoral Fellow, Stanford University, Stanford, CA 1990-1993
(Stem Cell Biology: Mentor Irving L. Weissman)

Licenses: Oregon Medical License (MD20761); California Medical License (A45492)

Current ABIM Board Certification: Medical Oncology

PROFESSIONAL EXPERIENCE

Faculty Appointments:

Assistant Professor of Medicine, Emory University 1993-1997
Assistant Professor, Winship Cancer Center, Emory University
Adjunct Assistant Professor, Department of Microbiology and Immunology
Adjunct Assistant Professor, Yerkes Regional Primate Center

Assistant Professor of Medicine, Oregon Health & Sciences University 1997-2001

Adjunct Assistant Professor, Department of Cell and Developmental Biology
Adjunct Assistant Professor, Department of Microbiology and Immunology
Oregon Health & Sciences University
Member Oregon Cancer Institute

Associate Professor of Medicine 2001-2008
Adjunct Associate Professor, Department of Cell and Developmental Biology
Adjunct Associate Professor, Department of Microbiology and Immunology
Oregon Health & Sciences University
Member, Oregon Cancer Institute

Professor of Medicine (Tenured) 2008- Present
Professor of Pediatrics 2010- Present

Adjunct Professor, Department of Cell and Developmental Biology 2008- Present
Adjunct Professor, Department of Microbiology and Immunology
Oregon Health & Science University
Member, OHSU Knight Cancer Institute

Member, Oregon Stem Cell Center 2004- Present
Hematologic Malignancies Program
OHSU Knight Cancer Institute 2004- Present

Administrative and Hospital Appointments:

Attending Physician BMT Program, Emory University Hospital 1993-1997
Attending Physician, Grady Memorial Hospital, Atlanta GA
Attending Physician Leukemia/BMT Program, OHSU 1997-2013
Staff Physician (WOC), Hematology/Oncology Portland VA 2015-present
Director, Molecular Hematology Training Grant (T-32) 2006-2010
Leader, Hematologic Malignancies Program, Knight Cancer Institute 2010-2015
Director, Pediatric Clinical Trials Office, OHSU 2013-2017

SCHOLARSHIP

Area(s) of Research/Scholarly Interest:

Hematopoietic stem cells, leukemia stem cells, endothelial cells, hematopoietic microenvironment

Ongoing Research Support:

R01DK109694-01A1 Fleming (PI) 07/01/16-05/31/21

Rcor1 Regulates Myelomonocytic Progenitor Cell Fate. The goal of this proposal is to identify Rcor1 targets that can be used to restore the normal differentiation program of myelomonocytic progenitor cells.

2P01HL048546-21A1 Fleming (Core Director) 09/01/16-05/31/21

Pathophysiology and Treatment of Fanconi Anemia. The goal of this proposal is to identify the therapeutic potential of small molecule therapy for the treatment of bone marrow failure in FA.

No number Fleming (Co-PI) 09/15/15-09/14/17

Fanconi Anemia Research Foundation (FARF)

A Porcine Model of Fanconi Anemia. The goal of this project is to create a large animal model to study the pathophysiology of Fanconi anemia

No number Fleming (Co-PI)

V Foundation 09/01/14-09/01/17

Combination Therapy for KIT-mutant Cancer: an Integrated Systems Biology Approach

Using a systems biology approach, we will identify and validate novel molecular pathways that can be targeted in combination with KIT TKIs to synergistically inhibit the growth/survival of KIT-mutant cancer cells. Promising combination treatments will be advanced to human clinical studies. Role: Co-Investigator for xenograft studies.

Recently Completed Research Support:

U19 AI091175-01 Fleming (Project Leader) 08/01/10-7/30/16

Stem Cell Based Therapies for Mitigation of Acute Radiation Syndrome. The major goal of this project is to identify factors that mitigate acute irradiation syndrome.

Publications:

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Friesen HG, Elsholtz HE, **Fleming WH**, Shiu RPC: The Nb2 node lymphoma cells, PRL receptors and mechanism of action of PRL. In: Shizume K, Imura H, Shimizu N, Endocrinology. Amsterdam, Exerpta Medica, p. 244-250, 1983

Fleming WH, Murphy PR, Murphy LJ, Hatton TW, Matusik RJ, Friesen HG: Human growth hormone induces and maintains c-myc expression in Nb2 lymphoma cells. Endocrinology 117: 2547-1549, 1985.

Fleming WH, Hamel A, MacDonald R, Ramsey E, Pettigrew NM, Johnston B, Dodd JG, Matusik RJ: Expression of the c-myc proto-oncogene in human prostatic carcinoma and benign prostatic hyperplasia. Cancer Res 46: 1535-1538, 1986.

Matusik RJ, Kries C, McNichol P, Sweetland R, Mullin C, **Fleming WH**, Dodd JG: Regulation of prostatic genes: Role of androgens and zinc in gene expression. Biochem Cell Biol 64: 601-607, 1986

Matusik RJ, **Fleming WH**, Hamel A, Westenbrink TG, Hrabarchuk B, Pettigrew NM, Johnston B, Gartner J, Ramsey E, MacDonald R, Dodd JG: Expression of the c-myc proto-oncogene in prostatic tissue. Prog Clin Biol Res 239: 91-112, 1987.

Fleming WH, Alpern EJ, Uchida N, Ikuta K, Weissman IL: Steel factor influences the distribution and activity of murine hematopoietic stem cell in vivo. Proc Natl Acad Sci USA 90: 3760-3764, 1993.

Fleming WH, Alpern EJ, Uchida N, Ikuta K, Spangrude GJ, Weissman IL: Functional heterogeneity is associated with the cell cycle status of hematopoietic stem cells. J Cell Biol 122: 897-902, 1993.

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Uchida N, **Fleming WH**, Alpern EJ, Weissman IL: Heterogeneity of hematopoietic stem cells. Curr Opin Immunol 5: 177-184, 1993

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Chen G, Jaffrezou JP, **Fleming WH**, Duran GE, Sikic BI: Prevalence of the multidrug resistance related to the activation of the mdr-1 gene in human sarcoma mutants derived by single step selection doxorubicinand selection. Cancer Research 54:4980-4987, 1994.

Fleming WH, Weissman IL: Hematopoietic stem cells. In: Abeloff M, Armitage J, Lichter A, Neiderhuber J (eds), Clinical Oncology. New York, Churchill Livingstone. p. 127-135, 1995.

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Bomberger C, Jairam M, Rodey G, Guerriero A, Yeager AM, **Fleming WH**, Holland HK, Waller EK: Lymphoid reconstitution following autologous PBSC transplantation with CD34^{+} progenitor cells. Blood 91:2588-2600,1998.

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Fleming WH, Lankford-Turner P, Turner, CW, Wong J, Strobert E, McKearn JP: Administration of daniplestim and granulocyte colony-stimulating factor for the mobilization of hematopoietic stem cells in nonhuman primates. Biology of Blood and Marrow Transplantation. 5: 8-14, 1999.

Battaile KP, Bateman RL, Mortimer D, Mulcahy JM, Rathbun RK, Bagby G, **Fleming WH**, Grompe M: In vivo selection of wild-type hematopoietic cells in a murine model of Fanconi Anemia. Blood 94:2151-2158, 1999

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Hayes-Lattin BM, Curtin PT, **Fleming WH**, et al. Toxic megacolon: a life-threatening complication of high-dose therapy and autologous stem cell transplantation among patients with AL amyloidosis. Bone Marrow Transplant 30:279-285, 2002

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Anderson DA, Wu Y, Shuguang J, Zhang X, Streeter PR, Spangrude GD, Archer DR, **Fleming,WH**: Donor marker infidelity in transgenic hematopoietic stem cells. Stem Cells 23:638-643, 2005

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Bailey AS, Willenbring H, Jiang S, Anderson DA, Schroeder DA, Wong MH, Grompe M, **Fleming WH**: Myeloid lineage progenitors give rise to vascular endothelium. Proc Natl Acad Sci USA 103:13156-13161, 2006

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Zhang, Q. Marquez-Loza L, Eaton L, Duncan AW, Goldman, DC, Anur P, Smith K, Rathbun RK, **Fleming, WH** Bagby, GC Grompe, M: Resveratrol alleviates the defects in hematopoietic stem cells and microenvironment of Fancd2 deficient mice. Fancd2-/- mice have hematopoietic defects that can be partially corrected by resveratrol. Blood 116:5140-8, 2010.

Umashankar M, Petrucelli A, Cicchini L, Caposio P, Kreklywich CN, Rak M, Bughio F, Goldman DC, Hamlin KL, Nelson JA**, Fleming WH**, Streblow DN, Goodrum F: A novel human cytomegalovirus locus modulates cell type-specific outcomes of infection. PLoS Pathog. 2011 Dec;7(12):e1002444. PubMed PMID: 22241980; PubMed Central PMCID: PMC3248471.

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Hakki M, Goldman DC, Streblow DN, Hamlin KL, Krekylwich CN, **Fleming WH**, Nelson JA. HCMV infection of humanized mice after transplantation of G-CSF mobilized peripheral blood stem cells from HCMV-seropositive donors. Biol Blood Marrow Transplant. 2014 Jan 20(1):132-5. PMID: 24161922

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Yao H, Goldman DC, Nechiporuk T, Kawane S, McWeeney SK, Tyner JW, Fan G, Kerenyi MA, Orkin SH, **Fleming WH**, Mandel G. Corepressor Rcor1 is essential for murine erythropoiesis. Blood. 2014 May 15;123(20):3175-84

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Yao H, Goldman DC, Fan G, Mandel G, **Fleming WH**.The co-repressor Rcor1 is essential for normal myeloerythroid lineage differentiation. Stem Cells 2015; 33: 3304-3314. PMID:26119982

Patents:

METHOD FOR ENHANCING HEMATOPOIESIS
U.S. Patent No. 6,060,643

MOUSE MODEL WITH HUMAN IMMUNE SYSTEM
U.S. Patent No. 6,821,513

ENHANCEMENT OF HEMATOPOIETIC STEM CELL SURVIVAL
Patent Pending

Invited Lectures, Conference Presentations or Professorships (Selected)

International and National:

Fondation des Treilles, Paris, France
Department of Biochemistry Melbourne, Australia
Institute for Stem Cell Research, Edinburgh, Scotland
International Society of Experimental Hematology, Presidential Symposium, Glasgow Scotland
International Symposia on In Utero Stem Cell Transplantation, Venice Italy
University of New Mexico Cancer Center, Albuquerque, NM
Fred Hutchinson Cancer Research Center, Seattle, WA
Stem Cell Biology Program, University of Southern California, L.A., CA
Stem Cell Biology Program, Case Western University, Cleveland, OH

Stem Cell Biology Program, City of Hope, Duarte, CA
American Society of Hematology (Session Chair, Stem Cell Plasticity)
Gordon Research Conference (Vascular Biology), Santa Barbara, CA
Egelston Children's Hospital Emory University, Atlanta, GA
Montagna Symposium, Stem Cells in Skin, Snowmass, CO
Cancer Research Institute, University of Indiana, Indianapolis, ID
Stem Cell Biology Program, University of Florida, Gainesville, FL
Stem Cell Biology Program, Washington University School of Medicine, St. Louis MO
NIH NHLBI Stem Cell Investigators Meeting, Bethesda MD
Institute for Genetic Medicine, University of Southern California, L.A., CA
Department of Medicine, Santa Clara Valley Medical Center, San Jose CA
Division of Hematology, Mount Sinai School of Medicine, New York NY
Division of Hematology & Oncology, MUSC, Charleston SC
Department of Medical Microbiology, Immunology & Cell Biology, SIU, Springfield IL
Hematologic Malignancies Program, University of Florida Shands, Gainesville FL
9th International Myeloid Conference, Cincinnati Children's Hospital

Regional and Local:

Oregon Graduate Institute (Biomedical Engineering), Beaverton, OR
Cell and Developmental Biology Seminar Series, OHSU
Hematology and Medical Oncology Fellows Research Retreat
Pulmonary Division Research Conference OHSU
Pediatric Hematology/Oncology Research Conference OHSU
Heart Research Center OHSU
Clinical Transplant Conference OHSU
Department of Medicine, Willamette Valley Medical Center, Oregon City, OR
Markham Hill Lecture Series, Portland, OR

SERVICE

Membership in Professional Societies:

American Society of Hematology
American Society for Blood and Marrow Transplantation
International Society for Stem Cell Research
International Society for Experimental Hematology

Peer Review Activities:

2001-2005	American Cancer Society: Leukemia, Immunology & Blood Cell Development Study Section (Regular Member)
2002	BioScience Foundation (Israel)
2003	Austrian Science Fund
2004	NIH Special Emphasis Panel Stem Cell Biology (NIA)
2004-2008	NIH Hematopoiesis Study Section (HP) (ad hoc member)
2008	NHLBI T-32 Review Group (ad hoc member)
2006-2010	NIH NCI P-30 Initial Review Group (ad hoc member)
2009-2013	NIH Molecular and Cellular Hematology Study Section (Regular member)
2005-present	NIH NHLBI Special Emphasis Panels
2014-present	NIH NIDDK Special Emphasis Panels

Journal Editorial and Ad Hoc Review Activities:

Editorial Board, Stem Cell Research 2010- present.
Science
Nature
Nature Medicine
PNAS
New England Journal of Medicine
Journal of Clinical Investigation
Blood
Cancer Research
Stem Cells
Experimental Hematology
Journal of Physiology
Cytometry

Committees:

Institutional and Regional: (OHSU)

2001-2007	IRB (Institutional Review Board)
2004-2005	Oregon Stem Cell Center Steering Committee
2006-Present	Oregon Stem Cell Center Search Committee
2006	BMT/Leukemia Discharge Committee
2006-Present	TALENT Program
2006-Present	Oregon Stem Cell Center Faculty Search Committee
2006-2012	M.D. Ph.D Program Committee
2006-2011	Hematology & Medical Oncology Fellowship Committee

Community Service:

2001	Markham Hill Lecture Series, Portland, OR
2004	American Cancer Society, (Oregon)
2004-Present	Oregon Cancer Institute, CURE Program for Minority Students
2005-Present	OHSU High School Science Class Program (PSI)
2005-Present	Murdock Scholars Program
2006	Grand Rounds, Willamette Valley Medical Center, Oregon City, OR
2007-Present	Leukemia & Lymphoma Society /ONS

Clinical Service:

1997-2010	Attending Physician: Inpatient BMT/Leukemia Service
1997-2013	Attending Physician: BMT/Leukemia Outpatient Clinic
2015-present	Attending Physician (WOC): Hematology/Oncology Portland VA

TEACHING:

Medical Student and Undergraduate Teaching:

1993-1997	Emory University
Member	Department of Graduate Studies
Member	Immunology and Molecular Pathogenesis Program
Member	Executive Committee, Immunology and Molecular Pathogenesis Program
Courses	Research Topics in Immunology, Immunology Colloquium

1997-Present Undergraduate Medicine (Second Year Blood Course)
2004-Present Cure Program (OCI)
2006-Present Partnership for Scientific Inquiry (Casey Eye Institute)
2006-2012 Bench to Bedside (M.D. Ph.D. Program)

Clinical Teaching:

1993-1997 Attending Physician BMT Program, Emory University Hospital
1993-1997 Attending Physician, General Medicine, Grady Memorial Hospital, Atlanta GA
1997-2000 Medicine House Staff Lecture Series (OSHU)
1997-2010 Attending Physician: Inpatient BMT/Leukemia Service (OHSU)
1997-2013 Attending Physician: BMT/Leukemia Outpatient Clinic (OHSU)

Graduate Student Teaching:

1993-1997 Courses, Research Topics In Immunology Colloquium (Emory University)
1993-1997 Member, Immunology and Molecular Pathogenesis Program (Emory University)
1993-1997 Member, Executive Committee, Immunology and Molecular Pathogenesis Program
1997-Present PMCB Graduate Courses (CON 665, CELL 605, CELL 616 all at OHSU)

Former Pre and Postdoctoral Research Trainees:

Eytan Alpern, M.D.
Thomas Neal M.D.
David Archer Ph.D.
Curtis Turner M.D.
Megan Montfort Ph.D
Jean Mulcahy M.D.
Christopher Olivares M.D.
David Schroeder M.D.
Bei Li M.D.
Shugang Jiang M.D. Ph.D
Luke Walker M.D.
Christina Baumann M.D.
Holger Willenbring M.D.
Devorah Goldman Ph.D.
Azzah Masri Ph.D
Ronn Leon Ph.D
Derek Zachman M.D., Ph.D

Current Pre and Postdoctoral Research Trainees:

2011- 2016 Derek Zachman MD/PhD student

Service and Membership on Educational Committees:

Past Ph.D. Thesis Advisory Committees:

(Jack Lee, Gokhan Dalgin Ph.D., Maclaine Pahl, Patrice Held Ph.D.)

Current Ph.D. Thesis Advisory Committees:

(Alison Macleod, Branden Tarlow, Ashley Kamimae-Lanning, Hulian Yao, Derek Zachman)

2006-2012 M.D. Ph.D. Program Admissions Committee

2006-2012 Hematology & Medical Oncology Fellowship Committee

2006-2010 Molecular Hematology Training Program Advisory Committee

Materials Considered List

1. Acquavella, J. et al., *Glyphosate Epidemiology Expert Panel Review: A Weight of Evidence Systematic Review of the Relationship between Glyphosate Exposure and Non-Hodgkin 's Lymphoma or Multiple Myeloma*, 46 Critical Rev. Toxicology 28 (2016).

2. Alavanja, M. et al., *Non-Hodgkin Lymphoma Risk and Insecticide, Fungicide and Fumigant Use in the Agricultural Health Study*, 9 PLoS One (2014).

3. Alavanja, M. et al., *DRAFT-Lymphoma risk and pesticide use in the Agricultural Health Study* (Mar. 15, 2013) (unpublished study) (on file with Authors).

4. American Cancer Society, *Cancer Facts & Figures* (2017).

5. Andreadis, C. et al., *Members of the glutathione and ABC-transporter families are associated with clinical outcome in patients with diffuse large B-cell lymphoma*, 109 Blood J. 3409 (2007).

6. Bangham, C. and L. Ratner, *How does HTLV-1 cause adult T-cell leukemia/lymphoma (ATL)?*, 14 Curr Opin Virol 93 (2015).

7. BfR, *Assessment of IARC Monographies Volume 112 (2015); Glyphosate*, Renewal Assessment Report: Glyphosate Addendum I to RAR (2015).

8. Brusick, D. et al., *Genotoxicity Expert Panel Review: Weight of Evidence Evaluation of the Genotoxicity of Glyphosate, Glyphosate-Based Formulations, and Aminomethy/phosphonic Acid*, 46 Critical Rev. Toxicology 56 (2016).

9. Cancer Research UK (2016) Cancer incidence by age. https://www.cancerresearchuk.org/health-professional/cancer-statistics/incidence/age#heading-zero

10. Cantor, K. et al., *Pesticides and Other Agricultural Risk Factors for Non-Hodgkin's Lymphoma among Men in Iowa and Minnesota*, 52 Cancer Res. 2447 (1992).

11. Carbone, A. and A. Gloghini, *HHV-8-Associated Lymphoma: State-of-the-Art Review*, 117 Acta Haematol 129 (2007).

12. Carroll, V. and A. Garzino-Demo, *HIV-associated lymphoma in the era of combination antiretroviral therapy: shifting the immunological landscape*, 73 Pathogens and Disease 1 (2015).

13. Chang, E. and E. Delzell, *Systematic Review and Meta-Analysis of Glyphosate Exposure and Risk of Lymphohematopoietic Cancers*, 51 J Envtl. Science Health 402 (2016).

14. Chihara, D. et al., *New insights into the epidemiology of non-Hodgkin lymphoma and implications for therapy*, 15 Expert Rev. Anticancer Therapy 531 (2015).

15. Cozen, W. et al., *Fecal microbiota diversity in survivors of adolescent/young adult Hodgkin lymphoma: a study of twins*, 108 British J. of Cancer 1163 (2013).

16. De Roos, A. et al., *Cancer Incidence among Glyphosate-Exposed Pesticide Applicators in the Agricultural Health Study*, 113 Envtl. Health Persp. 49 (2005).

17. De Roos, A. et al., *Integrative assessment of multiple pesticides as risk factors for non-Hodgkin's lymphoma among men*, 60 Occupational Envtl. Medicine 1 (2003).

18. EPA, Cancer Assessment Review Committee, Health Effects Division, Office of Pesticide Programs, U.S. Environmental Protection Agency, *Cancer Assessment Document – Evaluation of the Carcinogenic Potential of Glyphosate* (Oct. 1, 2015).

19. EPA, Office of Pesticide Programs, *Glyphosate Issue Paper: Evaluation of Carcinogenic Potential*, Regulations.gov (September 12, 2016).

20. Eriksson, M. et al., *Pesticide Exposure as Risk Factor for Non-Hodgkin Lymphoma Including Histopathological Subgroup Analysis*, 123 Int'l J Cancer 1657 (2008).

21. European Food Safety Authority (EFSA), *Conclusion on the Peer Review of the Pesticide Risk Assessment of the Active Substance Glyphosate*, 13(11) EFSA Journal 1-107 (2015).

22. Expert Report of Alfred I. Neugut, MD, PHD In Support of General Causation on Behalf of Plaintiffs, In Re: Roundup Products Liability Litigation, No. 16-md-02741-VC (N.D. Cal. May 1, 2017).

23. Expert Report of Dr. Dennis Weisenburger, M.D. In Support of General Causation on Behalf of Plaintiffs, In Re: Roundup Products Liability Litigation, No. 16-md-02741-VC (N.D. Cal. May 1, 2017).

24. Expert Report of Dr. Nabhan In Support of General Causation on Behalf of Plaintiffs, In Re: Roundup Products Liability Litigation, No. 16-md-02741-VC (N.D. Cal. May 1, 2017).

25. FIFRA SAP, *Meeting Minutes and Final Report No. 2017-01: EPA's Evaluation of the Carcinogenic Potential of Glyphosate,* (Mar. 16, 2017).

26. Hardell, L. et al., *Exposure to Pesticides as Risk Factor for Non-Hodgkin's Lymphoma and Hairy Cell Leukemia: Pooled Analysis of Two Swedish Case-control Studies*, 43 Leukemia and Lymphoma 1043 (2002).

27. Healy, J. and S. Dave, *The Role of EBV in the Pathogenesis of Diffuse Large Cell Lymphoma*, 390 Curr Top Microbiol Immunol 315 (2015).

28. Hietanen, E. et al., *Effects of Phenoxyherbicides and Glyphosate on the Hepatic and Intestinal Biotransformation Activities in the Rat*, 53 Acta Pharmacologica et Toxicologica 103 (1983).

29. Hofmann, J. et al., *Farm characteristics, allergy symptoms, and risk of non-Hodgkin lymphoid neoplasms in the Agricultural Health Study*, 24 Cancer Epidemiology Biomarkers Prevention 587 (2015).

30. Hohenadel, K. et al., *Exposure to Multiple Pesticides and Risk of Non-Hodgkin Lymphoma in Men from Six Canadian Provinces*, 8 Int. J. Environ. Res. Public Health 20320 (2011).

31. Howlader, N. et al., *Contributions of Subtypes of Non-Hodgkin Lymphoma to Mortality Trends*, 25 Cancer Epidemiology Biomarkers Prevention 174 (2016).

32. International Agency for Research on Cancer (IARC), Monograph Vol. 112 on the Evaluation of Carcinogenic Risks to Humans, *Some Organophosphate Insecticides and Herbicides: Diazinon, Glyphosate, Malathion, Parathion, and Tetrachlorvinphos* (2015).

33. JMPR, *Pesticide residues in food – 2004: Toxicological evaluations – Toxicological Monographs and Monograph Addenda,* Joint FAO/WHO Meeting on Pesticide Residues (2006).

34. Krikorian, J. et al., *Occurrence of Non-Hodgkin's Lymphoma after Therapy for Hodgkin's Disease*, 300 New England J Med. 452 (1979).

35. Krishnan, B. & G. Morgan, *Non-Hodgkin Lymphoma Secondary to Cancer Chemotherapy*, 16 Cancer Epidemiol Biomarkers Prevention 377 (2007).

36. Lan, Q. et al., *Genetic polymorphisms in the oxidative stress pathway and susceptibility to non-Hodgkin lymphoma*, 121 Hum. Genetics 161 (2007).

37. Landmann, E. et al., *Secondary non-Hodgkin lymphoma (NHL) in children and adolescents after childhood cancer other than NHL*, 143 British J Haematology 387 (2008).

38. Lee, W. et al., *Non-Hodgkin's Lymphoma Among Asthmatics Exposed to Pesticides*, 111 Intl. J. Cancer 298 (2004).

39. Lichtman, M., *Obesity and the Risk for a Hematological Malignancy: Leukemia, Lymphoma, or Myeloma*, 15 The Oncologist 1083 (2010).

40. Lightfoot, T. et al., *Polymorphisms in the oxidative stress genes, superoxide dismutase, glutathione peroxidase and catalase and risk of non-Hodgkin's lymphoma*, 91 Haematologica 1222 (2006).

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