EXHIBIT 4
UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION

This document relates to:
ALL ACTIONS

EXPERT REPORT OF ALFRED I. NEUGUT, MD, PHD

IN SUPPORT OF GENERAL CAUSATION ON BEHALF OF PLAINTIFFS
Expert Report on Glyphosate and Non-Hodgkin Lymphoma

Alfred I. Neugut, MD, PhD

I. Qualifications

I am currently the Myron M. Studner Professor of Cancer Research and Professor of Medicine and Epidemiology at Columbia University, and Associate Director for Population Sciences for the Herbert Irving Comprehensive Cancer Center at Columbia. I am also the Director of Junior Faculty Development for the Department of Epidemiology at the Mailman School of Public Health, overseeing about 30 assistant professors.

I am a medical oncologist with a particular interest in gastrointestinal tract cancers, especially colorectal and gastric cancers. Under the auspices of Columbia's Medical Scientist Training Program, I received my MD and a Ph.D. in Pathobiology in 1977. My PhD was in the laboratory of Dr. I. Bernard Weinstein, an authority in chemical carcinogenesis, and I studied growth control of cancer cells in vitro. I then trained in Internal Medicine at the Albert Einstein College of Medicine and fellowship in Medical Oncology at Memorial Sloan-Kettering Cancer Center.

I returned to Columbia for an M.P.H. in Epidemiology in 1983, and then joined the faculty at Columbia with appointments in Medicine and Epidemiology. My research has centered on cancer epidemiology and prevention. I initiated a series of important studies focused on risk factors for the occurrence and recurrence of colorectal adenomatous polyps (adenomas). These studies extended into the use and yield of colonoscopy and fecal occult blood testing for routine screening and diagnosis. An editorial I wrote in 1988 was the first to suggest the use of colonoscopy for routine screening of asymptomatic adults, a common practice now. My second major research focus was the occurrence of second malignancies, especially the impact of radiation therapy. I was also the co-PI of the Long Island Breast Cancer Study Project which investigated the high rate of breast cancer on Long Island and generated over 100 papers on environmental risk factors and breast cancer.

At the present time, a significant amount of my research is centered on studying quality of care in the use of chemotherapy and radiotherapy for cancer in the elderly and others. My group has found significant effects of age, race/ethnicity, as well as financial status and the level of co-payments in leading to lower quality care and decreased adherence to prescribed chemotherapy and hormonal therapy. I currently also have several projects ongoing in South Africa on the effect of HIV infection on cancer outcomes.

I have published over 500 peer reviewed chapters and papers. I have received over $50 million in funding from the National Cancer Institute, American Cancer Society, Department of Defense, and various foundations. I have led two NCI-funded training grants for predoctoral and postdoctoral trainees for over 25 years that have trained over 80 trainees who are now in various academic, government and industrial positions; I have also mentored over 15 K or K equivalent junior faculty award recipients. I am a recent recipient of the Distinguished Achievement Award of the American Society of Preventive Oncology. I have served on innumerable government grant review committees. My Curriculum Vitae is attached as Attachment A.
I have been asked to review the scientific literature on glyphosate and glyphosate-based formulations and to provide an opinion to a reasonable degree of medical and scientific certainty as to whether glyphosate and glyphosate-based formulations can cause non-Hodgkin lymphoma.

This review took as its takeoff the IARC report of 2015, and reviewed the studies and materials cited in that report. Further literature searches were conducted following up references in the key publications cited in the IARC report and a search conducted for any publications published subsequent to the IARC report. See References Section. We also reviewed the EPA (2016) report, the European Food Safety Authority (2015) report and the commentary by Portier (2015). In addition, I reviewed the transcripts of deposition of Aaron Blair of NIEHS, Donna Farmer of Monsanto and John Acquavella of Monsanto. With the exception of the deposition transcripts this would be the general approach utilized if one were doing a literature review for a scientific publication. More details are given in the text.

My assistant, Ayana K. April Sanders, MPH, a doctoral student in the Department of Epidemiology at Columbia University’s Mailman School of Public Health, assisted with the tasks described above, compilation of the tables, and some of the writing. I reviewed all of the studies, and all opinions, analyses and conclusions are mine and mine alone.

II. Cancer Epidemiology

Epidemiology is the study of disease in populations, including its distribution, determinants, natural history, and survival. Rather than the individual patient, its perspective is that of public health. The traditional focus and goal of cancer epidemiology has been the determination of the incidence and mortality rates of cancer in different populations and subgroups, as well as the identification of risk factors for the purpose of disease prevention and control through primary prevention and screening interventions.

Much of epidemiology involves the assessment of cancer risk. A person can be at increased risk of cancer because of extrinsic or intrinsic factors, or a mix thereof.

- Extrinsic influences are factors outside of the individual’s own body, such as environmental pollutants, cultural/lifestyle habits, medication use, infectious factors, and diet.
- Intrinsic influences are factors unique to each person, such as genetics.

From an epidemiologic perspective, an etiologic agent or risk factor is anything that increases the probability that an individual will develop the disease. These risk factors can include demographic characteristics (e.g., increasing age or race/ethnicity) or lifestyle and behavioral factors, such as smoking. They also include endogenous factors, such as genetic mutations that have been identified as predisposing a person for a disease, such as a deleterious BRCA1 or BRCA2 mutation. Most cancers undoubtedly arise from a combination of genetic and exogenous factors that interact to define certain demographic patterns.

III. Cancer characteristics

My report focuses on characteristics which are specific or idiosyncratic or more relevant to cancer as opposed to other areas of epidemiology (infectious disease, cardiovascular, psychiatric, etc).
a. Epidemiologists start with a definition of cancer which is a synonym for those diseases which involve malignancy (in contrast to being benign). While there may be various characteristics or ways in which to define this phenomenon, a good general definition would be that it is a disease in which the cell loses control of growth and proliferation. Benign cells or growths will stop growing when they reach some boundaries or limits, but malignant cells know no such limits and, in theory, will divide and proliferate forever. In many or most circumstances this is also associated with more rapid growth than in a normal cell, but this is not necessary – the defining characteristic is loss of growth control.

b. As a corollary to the above, cancers are all generally potentially fatal. This is because if you allow uncontrolled growth of a tumor (a growth) to proceed for an unlimited amount of time, it will ultimately reach a size where it will kill the host in some fashion, either because the size of the tumor (or tumors) will compete with the normal cells of the body for nutrition and oxygen, and malignant cells are always better than normal cells at this so the normal cells and tissues will starve to death (a phenomenon known as cachexia in terminal cancer patients). An alternative way in which people die from cancer is that the tumors block vital organs or passageways or replace normal functioning organs so one dies from organ failure. The tumor may be so slow growing that you would not die from it till you are very elderly and you may die from a different disease beforehand, but the point is that all malignant cancers, by definition are potentially fatal.

c. Cancer is a disease of the cell, i.e., in general, the pathophysiologic problem arises within the cell of origin as opposed to being a disease of an organ or system. All other diseases are pathologically problems of deterioration or inflammation or infection or some other disorder arising in the organ or in a system – the pancreas, the lung, the heart, the cardiovascular system, the immune system, etc. A cancer may arise in the context of an organ problem, e.g., liver cancer arising in the context of liver cirrhosis, but the cancer itself is a disorder of the liver cell.

d. Cancer cells are basically aberrant normal cells. That is, a cancer cell can retain initially many of the characteristics of the cell of origin. As it gets more aggressive or more advanced, it becomes less and less like the original normal cell.

e. From a public health and population perspective, individual cancers are uncommon, even rare. The four most common cancers in the US – breast, prostate, colorectal, lung - all occur at an age and sex-adjusted rate of about one case per 1000 population/year. From an epidemiologic perspective, this makes the use of cohort studies or intervention trials extremely difficult and expensive and indeed, such studies are uncommon. As described below, to get sufficient endpoints in such a study even with one of these “more common” cancers, one would need to follow tens of thousands of people for years. For other cancers, which are much less common, the use of cohort studies or intervention trials are
extremely uncommon and difficult to undertake and difficult to interpret unless risk ratios are very strong.

f. The latency period for a cancer can be very long, often on the order of decades. This exacerbates the problem of the use of cohort and intervention trials as described in the prior paragraph. There are, however, both tumor initiators and tumor promoters, the latter of which are short term carcinogens which can raise the risk of a cancer within very short time frames, even within a year or two. This is particularly true when looking at the hematopoietic malignancies.

g. More so than for most diseases, the diagnosis for malignant diseases is pathology-dependent, and hence highly accurate. Indeed, because it depends on histology and pathology, the subclassification of most tumors is also highly accurate. Thus to the degree that an epidemiologic study is trying to ascertain the association between a given exposure and a given disease, the width of the 95% confidence interval (i.e., the uncertainty with which one measures the association between the two variables) is increased by the uncertainty by which one estimates the presence of the exposure and the uncertainty by which one ascertains the presence of the disease. At least for studies of cancer, in most studies, more so than for most diseases, the definition and ascertainment of the disease is highly valid.

h. There are two major histologic types of cells or tissues – epithelial tissue and connective tissue. Malignancies of epithelial tissue are referred to as carcinomas, while malignancies of connective tissue are referred to as sarcomas. Both blood and lymphocytes fall under the rubric of connective tissue and hence malignancies of blood (leukemias) and malignancies of lymphocytes (either leukemias or lymphomas) are under the general category of sarcomas.

IV. Lymphoma

a. Lymphocytes are a type of white blood cell which constitute part of the immune system. There are two major types of lymphocytes. B cells are cells which respond to antigens and ultimately mature into plasma cells which make antibodies, while T cells have other functions, such as being killer cells (directly attacking foreign invaders and toxins). Lymphocytes both circulate in the blood stream, where they constitute about 15-25% of circulating white blood cells, and are concentrated in lymph nodes along the lymphatic system. These are located in contiguity with every organ and act as drainage or sewage systems for each organ in terms of disposal of toxins or invading microorganisms and are often the first sites of local metastasis.

b. Lymphocytes can become malignant in different phases and ways. Lymphocytes that are circulating in the blood stream that become malignant form lymphocytic leukemias. Lymphocytes in lymph nodes that become malignant form lymphomas, either Hodgkin lymphoma or non-Hodgkin lymphoma (NHL).
Plasma cells that become malignant (and emit antibodies) constitute the malignant cell of multiple myeloma.

c. The large majority of NHL arise from B cells as opposed to T cells but there are multiple varieties of NHL based on histology, precise cell of origin, genetic mutations or oncogenes present.

V. Basics of Causation in Epidemiology

Epidemiologic studies use a multi-step process to establish causal inferences. First, principles of causal inference are used to construct our theories, which then help us to formulate testable hypotheses. We then design studies to test causal hypotheses as rigorously as possible. The objective of an epidemiologic study is to obtain a valid and precise estimate of the frequency of a disease or of the effect of an exposure on the occurrence of a disease in the source population of the study (Rothman, 2008). Epidemiologic studies ask ‘is there a statistical association between the exposure and outcome?’

In analytic epidemiology, observational studies are carried out to ascertain whether associations exist between an exposure and an outcome. Although a statistical association may exist between the two, there is always concern that this may reflect bias in the way the study was conducted or the presence of confounding factors. Confounding factors are factors associated with both the exposure and the outcome and can lead to an observed association, which is not truly a relationship between the two. For example, a study may show that asbestos workers have an elevated risk of lung cancer compared with the general population. However, one must be concerned that asbestos workers may be heavier smokers than other individuals in the general population and cigarette smoking is associated with lung cancer risk; thus, smoking may confound the observed association. Therefore, it is important in a study that looks at this exposure and outcome to collect smoking information so that it can be statistically controlled and the individual effect of asbestos exposure can be appropriately measured.

Multicausality (aka multifactorial): Certainly it is well known and well accepted that virtually every disease or condition can and does have multiple causes and its etiology can be spoken of as a multicausal phenomenon. Some of these causes are obvious and can be thought of as almost trivial (though they are not really trivial) such as age or gender. For example, virtually all epithelial malignancies (known as carcinomas) occur in adults and are usually age-dependent. Thus age is a risk factor for most carcinomas. Being a female is a risk factor or cause for female specific cancers, like ovarian cancer, which sounds trivial, but it is also a major risk factor for breast cancer, which can occur in males.

What is important to appreciate about the multicausal nature of disease is that all the causes contribute to the probability or risk of the disease occurring and thus any or all can be important in a given individual in whom they are present. Thus if one has a 60 year old obese male who is hypertensive, has a chronic elevated cholesterol, smokes cigarettes, is sedentary, and has a family history of coronary heart disease, and he develops a myocardial infarction (heart
attack), one may ask: What caused his heart attack? The correct answer is that all of these factors did and theoretically, if one removed any one of them from his past history, he might not have developed the disease. This is not to say, they were all equally contributory – how much they each contributed may vary and would be a function of the risk ratio associated with that particular exposure.

A common example of where this multicausal phenomenon occurs is in situations that address the question of whether asbestos exposure causes lung cancer. Many people with significant asbestos exposure in asbestos mines or other occupational settings have also been cigarette smokers, obviously a well-known lung carcinogen, and the argument has been made that the tobacco was responsible for the cancer, not the asbestos exposure. The correct causal analysis of this scenario would be that certainly the cigarette smoking contributed significantly to the development of the lung cancer, but that the asbestos exposure contributed significantly as well.

VI. Types of Epidemiologic Studies

a. Cohort and Case-control Studies

Epidemiologic observational studies fall into two broad categories: cohort studies and case-control studies. Participants in cohort studies are categorized based on their exposure and then followed to determine whether the outcome develops differently in the exposed and unexposed groups. Case-control studies enroll participants who have the outcome or disease under study, in addition to a control group of healthy participants. Both groups are then assessed for exposure. Both types of studies have their advantages and disadvantages. In both types, one must try to avoid bias or directional error. For example, in a case-control study, a patient with cancer may be inclined to give a positive answer more frequently than a control participant to a question regarding smoking history—this is referred to as recall bias.

As a general rule, cohort studies are preferred when the exposure is uncommon and the outcome is common, while case-control studies are preferable with uncommon outcomes. Since the incidence of most cancers, even the most common ones, is relatively low, case-control studies usually are used in cancer research. Their disadvantage is that they are often ambiguous on the temporal relationship between the exposure and the cancer. If you compare 100 patients with colon cancer to 100 patients without colon cancer for their intake of saturated fat, it can be unclear whether a decreased intake in the cases is related to the disease or preceded the disease. In a cohort study, where the exposure is ascertained before the subjects have developed the cancer, one can be more confident that any observed association preceded the development of disease.

Advantages to a Cohort Study

• Results can be used to calculate incidence
• Results can be used to calculate prevalence
• Efficient for studying common diseases
• Can study multiple diseases/outcomes
• Ensures temporality
• Study time varying covariates
• Reduces some types of selection bias and recall bias

Disadvantages to a Cohort Study

• Expensive
• Time consuming
• Cohort studies can be ineffective for studying rare diseases, particularly when follow up time is short.
• Requires prohibitively large sample size to detect occurrence of rare diseases
• Loss to follow-up is a types of selection bias
• Information bias is detection/observer bias (as opposed to recall bias)

A case-control study is a design where two groups, known as cases and controls, are selected based on the presence and absence, respectively, of a disease/outcome of interest. The groups are then queried about various exposures that may have been a source of disease. Associations between exposures and outcomes are measured using odds ratios, which estimate the relative risk. There are several types of case-control studies that vary depending on whether the study is designed within a designated cohort or not within a designated cohort. Sampling must be independent of exposure otherwise selection bias can be a problem. As long as we sample independent of exposure for our classic case-control study, we should have a valid design to address our research question. Controls are selected as a representative sample of the population that gave rise to the cases

Advantages of classic case-control studies

• Efficient for studying rare diseases (requires smaller sample than cohort study)
• Relatively fast
• Reduces the problem of follow-up bias
• Better able to deal with long latency periods
• Relatively inexpensive

Disadvantages of classic case-control studies

• Cannot calculate prevalence
• Inefficient for rare exposures
• Can only study one outcome
• Increased susceptibility to bias
1. Sampling assumptions (selection bias)
   - It is crucial to select cases and controls before gathering any information about exposures
2. Recall/information bias (potential error in recalling exposure
   - Case-patients may recall events differently than control patients

b. Meta-Analyses

Meta-analysis is a method for summarizing epidemiologic and other scientific evidence. “Meta-analysis [that] refers to the analysis of analyses…the statistical analysis of a large collection of analysis results from individual studies for the purpose of integrating findings. It connotes a rigorous alternative to the causal, narrative discussion of research studies which typify our attempts to make sense of the rapidly expanding literature…” (Glass, 1976). A meta-analysis is a statistical analysis that combines the results of multiple scientific studies.

The basic tenet behind meta-analyses is that there is a common truth behind all conceptually similar scientific studies, but which has been measured with a certain error within individual studies. The aim then is to use approaches from statistics to derive a pooled estimate closest to the unknown common truth based on how this error is perceived. In essence, all existing methods yield a weighted average from the results of the individual studies and what differs is the manner in which these weights are allocated and also the manner in which the uncertainty is computed around the point estimate thus generated. In addition to providing an estimate of the unknown common truth, meta-analysis has the capacity to contrast results from different studies and identify patterns among study results, sources of disagreement among those results, or other interesting relationships that may come to light in the context of multiple studies (Rothman, Greenland, & Lash, 2008).

A key benefit of this approach is the aggregation of information leading to a higher statistical power and more robust point estimates than is possible from the measure derived from any individual study. However, in performing a meta-analysis, an investigator must make choices which can affect the results, including deciding how to search for studies, selecting studies based on a set of objective criteria, dealing with incomplete data, analyzing the data, and accounting for or choosing not to account for publication bias (Walker, Hernandez, & Kattan, 2008).

Meta-analyses are often, but not always, important components of a systematic review procedure. For instance, a meta-analysis may be conducted on several clinical trials of a medical treatment, in an effort to obtain a better understanding of how well the treatment works. Here it is convenient to follow the terminology used by the Cochrane Collaboration (Van Tulder, Furlan, Bombardier, Bouter, & Group, 2003), and use "meta-analysis" to refer to statistical methods of combining evidence, leaving other aspects of 'research synthesis' or 'evidence synthesis', such as combining information from qualitative studies, for the more general context of systematic reviews.
We conduct meta-analyses to summarize published literature to create a more objective summary of literature than narrative reviews and produce a quantitative statistic demonstrating the estimate average effect of all of the available data. Meta-analyses also increase statistical power of the collection of studies, which results in a more precise estimate of effect size. Finally, conducting meta-analyses of observational studies can help to identify possible heterogeneity between studies.

**Steps in Conducting a Meta-Analysis**

- Identify objective and hypotheses
- Define outcome, exposure, population
- Formulate study inclusion criteria
- Formulate search strategy
- Extract data
- Assess study quality
- Estimate summary effect
  - Use published estimates \(^1\) for each included study (RR-Risk Ratio/Relative Risk, OR-Odds Ratio, HR-Hazard Ratio)
  - Convert results to a common scale, if needed (z-transformation (standardization), log-transformation)
  - Combine estimates of effect using a weighted average of individual estimates to estimate summary effect (Fixed or Random Effects)

Fixed effects assume that all studies are estimating the same underlying effect size (i.e., true effect) and that the variability between studies is due to sampling of people within each study. Random effects allow the studies to have different underlying effect, which vary around a mean over all studies and allows variation between studies as well as within studies.

Selecting the correct statistical model (fixed or random effects) is critically important in a meta-analysis. If one cannot assume that all studies are sampled from the same population, then a random-effects model should be implemented for the meta-analysis. In fact, the random-effects model should be the logical starting point of a meta-analysis with the assumption that the true effect size may or may not vary from study to study and a fixed-effects model can follow as a form of sensitivity analysis.

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\(^1\) Measures of relative effect express the outcome in one group relative to that in the other. For all measures of relative effect, a value of 1 indicates that the estimated effects are the same for both comparative groups.

- Risk ratio (aka relative risk; RR): the ratio of the risk of event in the two groups
- Odds ratio (OR): the ratio of the odds of an event in two groups
- Hazard ratio (HR): the ratio of the hazard rates in the two groups
There are two types of ways to summarize scientific evidence: 1) systematic review – meta-analysis of published data - and 2) pooled analysis – meta-analysis of individual level data.

**Comparison of Meta-analyses and Pooled Analyses: Data Management/Analysis**

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<thead>
<tr>
<th>Meta-Analyses</th>
<th>Pooled Analyses</th>
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<tr>
<td>Generally no contact with original study</td>
<td>Investigators of each study agrees to participate</td>
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<tr>
<td>Retrieve publication and extract data of interest</td>
<td>Obtain primary data</td>
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<tr>
<td>- Study design</td>
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<td>- Exposure, confounders</td>
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<td>- Risk estimates, confidence intervals</td>
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<tr>
<td>Check data abstracted for errors</td>
<td>Check primary data for errors</td>
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<tr>
<td>Differences in exposure, covariates, and contrasts across studies</td>
<td>Calculate risk estimates from primary data</td>
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<tr>
<td></td>
<td>More standardized definitions for exposures, covariates, and contrasts across studies</td>
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<td>Standardize formatting of data</td>
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Check whether results are heterogeneous
Check summary estimates, if appropriate
Conduct sensitivity analyses

VII. Review of Studies

a. Cohort Study (See Table 1)

**De Roos et al. (2005)** evaluated the association between exposure to glyphosate and cancer incidence on the Agricultural Health Study (AHS) cohort (A. J. De Roos et al., 2005).

Methods & Results
Population Description: The AHS is a prospective cohort study in Iowa and North Carolina, which includes 57,311 private and commercial applicators who were licensed to apply restricted-use pesticides at the time of enrollment into the study. Recruitment of the applicators occurred between 1993 and 1997. Members of the AHS cohort were matched to cancer registry files in Iowa and North Caroline for case identification and to state death registries and the National Death Index to ascertain vital statistics.
Outcome Assessment: Incident cancers were identified for the time period from the date of enrollment (1993-1997) until December 31, 2001 and were coded according to the *International Classification of Disease, 9th Revision* (ICD-9). Cohort members who moved from the state were censored in the year they left.

The prevalence of ever-use of glyphosate was 75.5% (> 97% of users were men). In this analysis, exposure to glyphosate was defined as: (a) ever personally mixed or applied products containing glyphosate; (b) cumulative lifetime days of use, or “cumulative exposure days” (years of use × days/year, categorized in tertiles among users: 1-20, 21-56, 57-2,678); and (c) intensity-weighted cumulative exposure days (years of use × days/year × estimated intensity level, categorized in tertiles: 0.1-79.5, 79.6-337.1, 337.2-18, 241). Poisson regression was used to estimate exposure–response relations between exposure to glyphosate and incidence of all cancers combined, and incidence of 12 cancer types: lung, melanoma, multiple myeloma, and non-Hodgkin lymphoma as well as oral cavity, colon, rectum, pancreas, kidney, bladder, prostate, and leukemia (results not tabulated). Exposure to glyphosate was not associated with all cancers combined (RR, 1.0; 95% CI, 0.9–1.2; 2088 cases). For non-Hodgkin lymphoma, the relative risk was 1.2 (95% CI, 0.7–1.9; 92 cases) when adjusted for age, and was 1.1 (95% CI, 0.7–1.9) when adjusted for multiple confounders (age, smoking, other pesticides, alcohol consumption, family history of cancer, and education); in analyses by cumulative exposure-days and intensity-weighted exposure-days, the relative risks were less than 1.0 in the highest tertiles. In summary, there was no association between glyphosate exposure and all cancer incidence or most of the specific cancer subtypes that were evaluated, including NHL. The strength of this analysis was the use of a large cohort with specific assessment of glyphosate and semi-quantitative exposure assessment. The De Roos et al. (2005) report demonstrates several major limitations that hinder the inferences made by the report: (1) a short follow up period of the cohort that does not allow for a meaningful evaluation of cancer risk; (2) the inability to determine disease latency in relation to glyphosate exposure and the risk of NHL; (3) using a comparison groups that is at an elevated risk of NHL; and (4) a potential for differential exposure misclassification.

(1) Short follow-up period: Participants who were licensed restricted use pesticide applicators were only enrolled in the study cohort from 1993-1997. Participants were followed to 2001, making the follow-up period for this data to range from 4-8 years. The report showed that the median follow-up period for this group was 6.7 years. Another important factor is that participants in the cohort were generally young with 46% being <50 years of age at the time of enrollment. These statistics suggest that the cohort may be too young to adequately evaluate cancer risk. Cancer epidemiology shows us that cancer incidence does not substantially increase until the ages of 50-55 years when we see an exponential increase in cancer incidence (Cancer Research UK, 2016). Thus, the study would have needed to follow this particular cohort for a much longer period of time in order to adequately evaluate cancer, and specifically NHL, risk from glyphosate exposure.

(2) Inability to determine disease latency period for NHL in AHS cohort: To determine the latency period between exposure to glyphosate and the onset of detectable NHL, the
investigators would have had to not only collect information on exposure to glyphosate, but also the time period of the initial exposure. Determining the latency period of the outcome is important in recognizing whether there is a meaningful increased risk in disease in a population because we can use that knowledge to rule out other causes of the disease.

(3) Elevated risk of NHL in control group: In comparison to the cases, it is expected that the control group used in the analysis for “ever/never” exposure to glyphosate would have an elevate risk for NHL. Evidence for this determination includes the following: A) Farmers who were licensed to use restricted-use pesticides comprise 91% of the controls in the De Roos et al., 2005 study. Several studies have demonstrated a significant increased risk of NHL in farmers (Morton et al., 2014; Orsi et al., 2009). B) Factors considered a risk for increased likelihood of NHL in farmers was tested in the Hardell et al. (L. Hardell, Eriksson, & Nordstrom, 2002) study that ultimately found that the exposure to “all herbicides” is a risk factor for NHL, OR=1.75, 95% CI: 1.26-2.41. Theoretically, if farmers had not adopted glyphosate as an herbicide they were likely to use other herbicides and hence have an increased risk of NHL. C) Finally, and most specifically, the majority of the control group (53.3%) in De Roos et al. (2005) was exposed to 2,4-D, an herbicide with carcinogenic potential. The meta-analysis conducted by Schinasi and Leon (2014) indicated a NHL meta-risk of 1.40 (95% CI: 1.0-1.9) for 2,4-D exposure. IARC recently classified 2, 4-D as possibly carcinogenic to humans (category 2b). Therefore, the effect estimate reported by De Roos et al. (2005) would be an underestimate of the NHL risk in the “ever/never” glyphosate exposure analysis.

(4) Non-differential Exposure Misclassification

Intensity of exposure to glyphosate was collected only at enrollment from 1993 – 1997. Yet, with the movement of agriculture to genetically engineered crops in 1996, participants already using glyphosate would have a dramatic increase in their intensity of exposure. By not collecting follow-up data on exposure status the analysis of exposure to glyphosate and association with NHL would be underestimated.

b. Case-control Studies (See Table 1)

Cantor et al. (1992) conducted a case-control study of incident non-Hodgkin lymphoma (NHL) in 622 white men compared to 1245 population-based controls in Iowa and Minnesota (Cantor et al., 1992). The study measured the risk of NHL associated with farming occupation and specific agricultural exposures. Men who ever farmed had a relative increased risk of NHL than non-farmers (OR=1.2, 95% CI: 1.0-1.5) independent of crop or animal types. Men who ever handled glyphosate also showed a slight increased risk of NHL, but the association was not statistically significant (OR=1.1, 95% CI: 0.7-1.9) when adjusted for vital status, age, state, cigarette smoking status, family history of lymphohaemotapoietic cancer, high-risk occupations and high-risk exposures. A major strength of this analysis was that it used a large population-based sample in a farming community. However, the study had significant limitations. Specifically, there was low power to assess the risk of NHL with glyphosate with only 26 cases of NHL.
Interpretation of the results is also limited by lack of adjustment for other herbicides used by the cohort.

**McDuffie et al. (2001)** conducted a multisite population-based incident case-control design conducted in six Canadian provinces (McDuffie et al., 2001). The study investigated the associations between exposure to specific herbicides and NHL. A total of 517 male cases and 1506 controls were interviewed by phone. The risk of NHL was observed to be elevated but not statistically significant for men exposed to glyphosate [51 exposed cases (OR=1.26, 95% CI:0.87-1.81; adjusted for age and province) and (OR=1.20, 95% CI: 0.83-1.74; adjusted for age, province, high-risk exposure)]. In a frequency analysis of exposure to glyphosate, men with > 2 days of exposure per year had an increased risk of NHL (OR=2.12, 95% CI: 1.20-3.73; 23 exposed cases; adjusted for age and province) compared to those with ≤ 2 days of exposure. Overall, this study is strengthened by using a large population-based sample, but there was a low response rate, albeit having a non-differential effect on the reported estimates when respondents were compared to non-respondents.

**Hardell et al. (2002)** conducted a pooled analysis on two case-control studies in Sweden (Lennart Hardell, Eriksson, & Nordström, 2002), one of NHL (originally reported in (L. Hardell & Eriksson, 1999)) and another on hairy cell leukemia (HCL), a rare subtype of (originally reported in (Nordstrom, Hardell, Magnuson, Hagberg, & Rask-Andersen, 1998)). The pooled analysis of NHL and HCL was based on 515 cases and 1141 controls. In univariate analysis, glyphosate increased the risk of NHL and HCL (OR=3.04; 95% CI: 1.08-8.52; 8 exposed cases). After accounting for study, study area and vital status in multivariate analysis, the odds of disease due to exposure to glyphosate decreased to 1.85 (95% CI: 0.55-6.20). Although using the pooled analysis contributed to an overall stronger power for analysis, agent-specific exposures had minimal cases. The exposure frequency was low for glyphosate and limited the power to test the effect of the exposure.

**De Roos et al. (2003)** used pooled data from three case-control studies on NHL conducted in the 1980s in Nebraska (Zahm et al., 1990), Kansas (Hoar et al., 1986), and Iowa and Minnesota (Cantor et al., 1992) to examine pesticide exposure in farming as a risk factor for NHL among men (A. De Roos et al., 2003). The pooled sample population included 870 cases and 2,569 controls – the majority of cases (n=650) and controls (n=1933) were included for the analysis of 47 pesticides controlling for potential confounding by other pesticides. Logistic regression and hierarchical regression models (which provides more conservative estimates compared to logistic regression due to adjusting estimates based on prior evidence, from past IARC or EPA reports, that any of the 47 pesticides may cause any type of cancer) were used in data analysis and all models were adjusted for age, study site, and other pesticides. Reported use of glyphosate, as well as several individual pesticides, was associated with increased incidence of NHL. In the logistic regression model based on 36 cases, the odds ratios for association between exposure to glyphosate and NHL were 2.1 (95% CI: 1.1-4.0) and 1.6 (95% CI: 0.9-2.8) in hierarchical regression models. The pooled population used in this analysis was a considerable strength compared to single-population empirical studies limited by small cases sizes. Additionally, the study was population based. De Roos et al (2003) did
include an advanced methodological technique (hierarchical regression) for accounting for multiple exposures by adjusting for estimates based on prior distributions for the pesticide effects. However, this hierarchical regression method has limited scientific merit since the adjustments are based on prior evidence of factors that may cause any cancer and not specifically NHL, and the opinions of carcinogenicity of each pesticide can change over time. Therefore, the modeling is subject to the opinions on carcinogenicity at the time of analysis (i.e., the opinions about the carcinogenic potential of glyphosate and other herbicides in the late 1980’s and early 1990’s) and the result would likely be different from current opinions. Thus, the conservative odds ratios of the hierarchical regression may not be an accurate portrayal of the association between glyphosate and NHL and would limit how to interpret the findings of the hierarchical regression.

Lee et al. (2004) evaluated whether asthma acts as an effect modifier of the association between pesticide exposure and NHL (Lee, Cantor, Berzofsky, Zahm, & Blair, 2004). The study was conducted using a pooled analysis of population-based case-control studies in Iowa, Minnesota and Nebraska. The sample included both men and women; 872 cases with NHL from 1980 to 1986 and 2,381 frequency-matched controls. In-person interviews were conducted to collect exposure information on pesticide use and history of asthma. A total of 177 subjects (45 cases, 132 controls) reported having been told by a clinician that they had asthma. Asthmatics had a non-significantly lower risk of NHL than non-asthmatics (OR=0.5, 95% CI; 0.2-1.4), and there was no main effect of pesticide exposure (OR=1.0, 95% CI: 0.8-1.2). Overall, those with a history of asthma typically had large odds ratios associated with exposure to pesticides than subjects without a history of asthma. Among non-asthmatics, the odds ratio associated with glyphosate use was 1.4 (95% CI: 0.98-2.1; 54 exposed cases) and 1.2 (95% CI: 0.4-3.3; 6 exposed cases) for asthmatics when compared to non-asthmatic non-exposed farmers. There was no indication of effect modification, such that the main effect does not vary based on asthma status.

In a Swedish-based study, Eriksson et al (2008) reported the results of a population based case-control study of exposure to pesticides as a risk factor for non-Hodgkin lymphoma (Eriksson, Hardell, Carlb erg, & Akerman, 2008). Men and women ages 18-74 years were included during December 1, 1999 to April 30, 2002. Incident cases of NHL were recruited from the University Hospitals in Lund, Linköping, Örebro and Umeå and controls were age and sex matched from the national population registry. Exposure to different agents was assessed by questionnaire. In total, 910 (91%) cases and 1016 (92%) controls participated in the study. Latency period calculations and multivariable analyses included agents with statistically significant increased odds ratios (OR) or with an OR > 1.5 and at least 10 exposed subjects. The odds of NHL for exposure to glyphosate was 2.02 (95% CI: 1.10-3.71) in univariate analysis and 1.51 (95% CI: 0.77-2.94) in a multivariable analysis. When considering exposure for more than 10n days per year, the OR was 2.36 (95% CI: 1.04-5.37). With a latency period of > 10 years, the odds ratio for exposure to glyphosate was 2.26 (95% CI: 1.16-4.40). Exposure to glyphosate was associated with increased odds for lymphoma subtypes and elevated odds of B-cell lymphoma (OR=1.87, 95% CI: 0.998-3.51) and the subcategory of small lymphocytic
lymphoma/chronic lymphocytic leukemia (OR=3.35, 95% CI: 1.42-7.89). Strengths of this study include having a population-based case-control study investigation, the ability to study different NHL subtypes and high response rate of cases and controls. Additionally, Eriksson et al. (2008) is one of the only studies to demonstrate elevated risk for glyphosate exposure in relation to several categories of NHL and evaluate the risk of NHL related to latency period. Limitations to interpreting the results derive from self-reported exposure assessment and possible confounding from use of other pesticides including MCPA – another herbicide that is commonly used together with glyphosate – but these were controlled for in the analysis. More so, it is expected that any residual confounding would result in an underestimation of the effect of a single pesticide. Given that the results demonstrated increased risk suggests there being a causal relationship despite confounding.

*Orsi et al. (2009)* reported the results of a hospital-based case-control study conducted in six clinics in France between 2000 and 2004 (Orsi et al., 2009). The study population included men and women aged 20-75 years and controls of the same age and sex as the cases were recruited in the same hospital – most were patients in the orthopedic and rheumatologically departments during the study period. In-person interviews and expert review of cases were used to evaluate pesticide exposure. The analysis included 491 cases (95.7% response rate; 244 cases of NHL, 87 cases of Hodgkin lymphoma, 104 cases of lymphoproliferative syndrome, and 56 cases of multiple myeloma) and 56 cases 456 age- and sex-matched controls. The study had a good response rate for the participants, but it enrolled hospital-based rather than population-based cases and controls. This could induce selection bias depending on whether individuals with high exposure to herbicide/pesticides, like glyphosate, (i.e., farmers) were more or less likely be hospitalized than the average person in the population that gave rise to the cases. A key limitation is that there was a small sample of participants reporting exposure to glyphosate thus limiting the power of the analysis to test for a true effect of glyphosate on any of the outcomes.

*Cocco et al. (2013)* reported on a pooled analysis of case-control studies conducted in six European countries between 1998-2004 (EPILYMPH, Czech Republic, France, Germany, Ireland, Italy, and Spain) investigating the role of occupational exposure to specific groups of chemicals in the etiology of lymphoma overall, B-cell lymphoma, and its most prevalent subtypes (Cocco et al., 2013). There was an approximately 1:1 ratio of cases (n=2,348) to controls (n=2,462) recruited by the six studies. Controls from Germany and Italy were randomly selected by sampling from the general population, whiles the other countries used matched hospital controls. Participation was adequate, 88% of cases participated and 81% of hospital controls and 52% of population controls participated. In-person interviews were conducted to collect detailed information on occupational history on farm-specific work related to type of crop, farm size, pest being treated, type of schedule of pesticide use. Industrial hygienists and occupational experts at each study center was used to assess exposure to specific groups of pesticides and individual compounds with assistance from agronomists. This method was used to reduce differential misclassification of exposure. Regression models were adjusted for age, sex, education, and study center. Lymphoma overall, and B-cell lymphoma were not
associated with any class of the investigated pesticides, while the risk of chronic lymphocytic leukemia was elevated among those ever exposed to inorganic and organic pesticides. The odds ratio for exposure to glyphosate and B-cell lymphoma was 3.1 (95% CI: 0.6-17.1; 4 exposed cases and 2 exposed control). The study was significantly limited in its power to assess the effects of glyphosate on risk of NHL due to substantially small sample of exposed cases.

c. Meta-analyses

In summary, the two published meta-analyses demonstrated statistically significant elevated risk of NHL in relation to glyphosate exposure. Estimates varied slightly based on the inclusion/exclusion of certain articles and the specific data points used in the meta-analyses.

Schinasi & Leon (2014) conducted a systematic review and a series of meta-analyses of approximately three decades of epidemiologic research on the relationship between NHL and occupational exposure to agricultural pesticide active ingredients and chemical groups, including glyphosate (Schinasi & Leon, 2014). The meta-analysis included six studies (A. De Roos et al., 2003; Eriksson et al., 2008; L. Hardell et al., 2002; McDuffie et al., 2001; Orsi et al., 2009) and yielded a meta risk-ratio of 1.5 (95% CI: 1.1-2.0) (See Fig. 1). Of note, the meta risk-ratio did not use the most fully adjusted estimates were from Hardell et al. (2002) and Eriksson et al. (2008) studies. The IARC Working Group re-assessed the meta-analysis by including the more adjusted estimates and generated similar but slightly diminished estimate (meta-RR=1.3, 95% CI: 1.03-1.65), $I^2=0\%$, P for heterogeneity = 0.589].

Chang and Delzell (2016) used the same six studies as Schinasi and Leon (2014) to conduct a systematic review and meta-analysis examining the relationship between glyphosate exposure and risk of lymphohematopoietic cancer including NHL, Hodgkin lymphoma, multiple myeloma and leukemia (Chang & Delzell, 2016). The meta-analysis yielded a meta-risk ratio of 1.3 (95% CI: 1.0-1.6) based on the six studies (Chang and Delzell, 2016 Figure 1). The investigators also conducted a meta-analysis substituting the logistic regression results of the De Roos et al. (2003) study for the hierarchical regression results and used the update data from McDuffie et al. (2001) and yielded a meta-risk ratio of 1.4 (95% CI: 1.0-1.8) (See Fig. 2).

VIII. Toxicity Studies

Animal Evidence (See Table 2)

Several rodent studies were conducted (EPA, 1985a, 1985b, 1986, 1991a, as cited in IARC Monograph 112) evaluating the effect of pure glyphosate exposure at varying concentrations. A significant positive trend for renal tumors in male CD-1 mice (EPA, 1985a), typically rare in mice, although there were no comparisons of any individual exposure group were statistically significant. In the Joint FAO/WHO Meeting on Pesticide Residues (JMPR, 2006) where CD-1 male and female mice were given diets containing glyphosate (purity,
98.6%), a significant positive trend for hemangiosarcoma in male CD-1 mice was reported. Again no individual exposure group was found to be statistically significant different from the control group. Finally, EPA’s (EPA, 1991a, 1991b, 1991c, 1991d) reports saw a significant increase in the incidence of pancreatic islet cell adenomas in two studies in male Sprague-Dawley male and female rats that were exposed to increasing concentrations of 96.5% purity glyphosate diets. These reports also demonstrated increased thyroid gland adenoma in females and liver adenoma in males.

The IARC working group reached the conclusion of sufficient evidence of glyphosate carcinogenicity in animals based on the significance of trend tests. The European Food and Safety Authority (EFSA) concluded that based on lack of individual significant differences and consistency between historical control ranges that there is no evidence of carcinogenicity of glyphosate in animal studies. Guidelines for evaluating toxicity in animal studies and relevant scientific reports and publications recommended that the key data points are the use of concurrent controls and trend tests (OECD, 2012; European Chemicals Agency, 2015). Trend tests are more powerful than pairwise comparisons, particularly for rare tumors where data are sparse. Likewise, historical control data should be garnered from the studies in the same time frame, animal strain and preferably the same laboratory and reviewed by the same pathologist.

**Carcinogenic Mechanisms in Humans (See Tables 3a & 3b)**

The genotoxic potential for glyphosate has been studied in a variety of assays including human, non-human mammal and non-mammalian systems. In the following, we summarize the findings of studies carried out in exposed humans and in human cells in vitro (as cited in IARC Monograph 112).

Studies in exposed humans (see Table 3a)

Available publications assessing the effect of a glyphosate-based formulation have focused on communities where the agent was aerially-sprayed in areas of northern Ecuador (Paz-y-Miño et al., 2007)) and five regions in Colombia (C. Bolognesi, Carrasquilla, Volpi, Solomon, & Marshall, 2009). In 24 exposed individuals in Ecuador, a statistically significant increase in DNA damage (DNA strand breaks) were observed in blood cells collected 2 weeks to 2 months after glyphosate was spayed in the area. Paz-y-Miño et al. (2011) studies continued by evaluating blood cells from 92 residents in 10 communities of northern Ecuador, sampled 2 years after the aerial spraying with an herbicide mix containing glyphosate (Paz-y-Miño et al., 2011). The results assessing chromosomal damage showed that the subject’s karyotypes were similar to levels reported in the control group. In Colombia, 137 married couples (137 women of reproductive age and their 137 spouses) were recruited from five regions. In three regions with exposed to glyphosate-based formulations from aerial spraying, blood samples were taken from the same individuals at three time points – (1) before spraying (baseline), (2) 5 days after spraying, and (3) 4 months after spraying – to determine the frequency of micronucleus formation in lymphocytes. Compared to a reference region without use of pesticides, subjects residing in the three regions where there had been aerial spraying of glyphosate-based formulations and in a fourth region with pesticide exposure (but not administered aerially) had significantly higher baseline frequency of binucleated cells with micronuclei. Increased
frequency of micronucleus formation in peripheral blood lymphocytes compared to baseline frequencies was also reported in subjects from the three regions. Directly after aerial sprays with the glyphosate-based formulation, subjects showed higher frequency of binucleated cells with micronuclei. However, the observed increases in micronuclei formation was reported to be inconsistent with the rates of application used in the regions; there was no association between self-reported direct contact with pesticide sprays and frequency of binucleated cells with micronuclei. In one of the 3 regions, subjects’ frequency of binucleated cells with micronuclei was significantly decreased 4 months after spraying compared to their frequencies immediately after spraying.

Studies in human cells in vitro (See Table 3b)

In studies using human cells in vitro, glyphosate induced DNA strand breaks (measured using the comet assay) in liver HEP-2 cells (F Mañas et al., 2009), lymphocytes (Alvarez-Moya et al., 2014; Mladenic, Berend, et al., 2009), GM38 fibroblasts, the HT1080 fibrosarcoma cell line (Monroy et al., 2005), and the TR146 buccal carcinoma line (Koller et al., 2012). DNA strand breaks were induced by AMPA in Hep-2 cells (Fernando Mañas et al., 2009), and by a glyphosate-based formulation in the TR146 buccal carcinoma cell line (Koller et al., 2012). AMPA, the degradation product of glyphosate and main metabolite of glyphosate, and glyphosate-based formulation also induces DNA strand breaks in Hep-2 cells and in the TR146 buccal carcinoma cells line, respectively. AMPA (F Mañas et al., 2009), but not glyphosate (Fernando Mañas et al., 2009), was found to produce chromosomal aberrations in human lymphocytes. Sister-chromatid exchange was induced by glyphosate (Bolognesi et al., 1997) and by a glyphosate-based formulation (Claudia Bolognesi et al., 1997; Vigfusson & Vyse, 1980) in human lymphocytes exposed in vitro. Glyphosate did not induce concentration-related increases in micronucleus formation in human lymphocytes at levels estimated to correspond to occupational and residential exposure (Mladenic, Perkovic, & Zeljezic, 2009).

Several studies have been conducted assessing the effect of glyphosate and its variations on oxidative stress, inflammation and immunosuppression. In studies examining the effects of glyphosate on oxidative stress parameters in the human keratinocyte cell line (HaCaT), a glyphosate-based formulation was found to be cytotoxic to HaCaT cell – addition of antioxidants reduced cytotoxicity (Gehin, Guillaume, Millet, Guyon, & Nicod, 2005). Another study showed that incubation of HaCaT cells with glyphosate at 21 mM (the half maximal inhibitory concentration for cytotoxicity, IC₅₀) for 18 hours increased production of hydrogen peroxide (H₂O₂) as shown by dichlorodihydrofluorescein diacetate assay (Elie-Caille, Heu, Guyon, & Nicod, 2010). Similar findings were reported by George & Shukla (2013) using 41% pure glyphosate up to 0.1 mM (George & Shukla, 2013). Dichlorodihydrofluorescein diacetate was used as a marker of oxidative stress limiting the validity of the findings (Bonini, Rota, Tomasi, & Mason, 2006; Kalyanaraman et al., 2012). The oxidative effects of glyphosate, AMPA, and a glyphosate-based formulation on oxidative stress in HepG2 cells was evaluated by Chaufan et al. (2014) and showed only the formulation had an adverse effects by increasing levels of reactive oxygen species, nitrotyrosine formation, superoxide dismutase activity, and glutathione, but did not have an effect on catalase or glutathione-S-transferase activities (Chaufan, Coalova, & Molina, 2014). Coalova et al (2014) found that exposing Hep2 cells to a formulation resulted in
various elevated parameters of oxidative stress. Exposure to the glyphosate-based formulation for 24 hours increased catalase activity and glutathione levels, with no effect on superoxide dismutase or glutathione-S-transferase activity (Coalova, de Molina, & Chaufan, 2014). Mladinic et al. (2009b) used blood samples from non-smoking male donors to examine the effects of in-vitro exposure to glyphosate on oxidative DNA damage in primary lymphocyte cultures and on lipid peroxidation in plasma. In both parameters glyphosate exposure significantly elevated the DNA damage when concentrations were 580 µg/mL or higher. Examining the effects of glyphosate, AMPA, and other related compounds in human erythrocytes collected from healthy donors, Kwiatkowska et al. (2014) found that exposed erythrocytes had increased production of reactive oxygen species (Kwiatkowska, Huras, & Bukowska, 2014). One study was available investigating the effects of glyphosate on cytokine production in human peripheral blood mononuclear cells (Nakashima et al., 2002). At 1mM glyphosate had a slight inhibitory effect on cell proliferation and modestly inhibited the production of IFN-gamma and IL-2. The production of TNF-α and IL-1 β was not affected by glyphosate at concentrations that significantly inhibited proliferative activity and T-cell-derived cytokine production.

Several studies have been developed to assess the effect of glyphosate exposure on cell proliferation and death. George & Shulka (2013) found that a glyphosate-based formulation increased the number of viable cells in HaCaT keratinocytes (George & Shulka, 2013). Eight human cancer cell lines was inhibited cell growth when exposed to glyphosate and AMPA – the greatest loss of viability were in ovarian and prostate cell lines (Li et al., 2013). Immortalized prostate cell lines were not affected. Using t47D breast cancer cells, Thongprakaisang et al. (2013) saw an increased growth in the cancer cells when exposed to glyphosate only when endogenous estrogen was minimized in the culture medium (Thongprakaisang et al., 2013). The growth of hormone-independent cultured breast cancer cells was not affected by glyphosate. The effect on apoptotic cell death given glyphosate exposed has been studied in HepG2 human hepatoma cell line. Glyphosate-based formulations induced apoptosis, while glyphosate alone generally was ineffective or showed effects at considerably high concentrations (Chaufan et al., 2014; Coalova et al., 2014; Gasnier et al., 2009; Mesnage, Bernay, & Séralini, 2013). Formulations showed to be more cytotoxic than glyphosate alone in studies with glyphosate and nine different glyphosate-based formulations in three cell lines (Mesnage et al., 2013). In HUVEC primary neonate umbilical cord vein cells, and 293 embryonic kidney and JEG3 placental cell lines, Benachour & Séralini (2008) found that glyphosate at relatively high concentrations induced apoptosis (Benachour & Séralini, 2008). The umbilical cord HUVEC cells were the most sensitive (by about 100-fold) to the apoptotic effects of glyphosate. Heu et al. (2012) evaluated apoptosis in immortalized human keratinocytes (HaCaT) exposed to glyphosate (5–70 mM) (Heu, Berquand, Elie-Caille, & Nicod, 2012). Based on annexin V, propidium iodide and mitochondrial staining, exposures leading to 15% cytotoxicity gave evidence of early apoptosis, while increases in late apoptosis and necrosis were observed at higher levels of cytotoxicity.

IX. Bradford Hill Criteria for Causation

While studies may assess associations, the decision regarding whether causality, as opposed to reverse causality, confounding, or some other relationship exists between
a putative exposure and outcome reflects a judgment call on the part of an educated experienced observer. The issue of causation in science can be appreciated by how extensively it is discussed and expounded upon within the writings of various philosophers, thinkers, and scientists going back to Plato and Aristotle, but the discussion of this topic profoundly accelerated during the time frame of the Empiricists in the sixteenth to eighteenth centuries, reflecting the growth of true experimental science and observation and an effort to be able to systematize and understand it.

Prior to the twentieth century, in medicine, the scientific endeavors, such as they were, focused almost exclusively on infectious diseases, and even there causality was a major concern. One solution to this problem were the so-called Koch’s Postulates, an algorithm by which to establish the etiologic agent for an infectious disease. It had a few instances of spectacular success, but in truth, it could not be often applied in human disease as it required that the infectious agent be introduced into a naïve host and cause the disease, something which was usually unacceptable, and today would almost always be unethical.

The advent of chronic diseases as the major health problems of the latter half of the twentieth century revived the causation issue, as again one could not apply any form of Koch’s Postulates. Indeed, most scientists were skeptical of whether lifestyle or behavioral factors could even be responsible for disease, in contrast to infectious or toxic agents. Tobacco and lung cancer became the salient testing ground for this issue, and it proved difficult to convince both the scientific and lay public of the etiologic relationship between the two, especially in the face of fierce tobacco company opposition but with growing observational data in support of the hypothesis. For obvious reasons, it was impossible to undertake a study along the lines of Koch’s Postulates.

In response to this problem there arose a set of criteria known as the Bradford Hill Criteria, published in 1965, named after their author, which became a checklist of sorts against which to weigh the collected evidence for a putative association in chronic disease epidemiology. They have remained to this day as the centerpiece of most circumstances in which a causal decision has to be made. Of particular relevance to this case is that they are also central to the methodology by which IARC reaches its judgments regarding carcinogenesis. We list them below and address each one in regard to the glyphosate/NHL question.

a. Temporality: This is always a key criterion for causality as it is an absolutely necessary condition, i.e., the cause must precede the effect. Certainly in this case, by the nature of the studies conducted, there is no doubt that this criterion was met. Exposure to glyphosate and its formulations preceded the development of NHL in all the human and all the animal studies.
b. Consistency: This criterion assesses whether the various studies essentially found similar results. Figures 1 and 2 summarize the findings of the case-control studies in Forest plots. If there were no association between glyphosate and NHL, i.e., if the two phenomena were truly random, then the measured associations in the studies should have randomly distributed themselves around 1. If one looks in the literature at exposures that have been shown not to be associated with certain outcomes, that is what one finds in the Forest plots. But that is not what one finds here. Here one finds that all the studies show a positive estimate of association between the exposure and the outcome. It is true that they are not all statistically significant. Many things attenuate the measurement of a statistical association ranging from any degree of misclassification in the measurement of the exposure or outcome to biases. But what is telling in the Forest plots is the consistency – they are primarily positive and to the right of 1. This consistency is amplified by the finding that when the data are meta-analyzed, they do indeed come out to be statistically significant.

c. Dose-Response: Two of the studies do suggest that there is a dose-response relationship, and that there is both a stronger association with increased exposure, as well as a statistically significant relationship. (See McDuffie (2002), Eriksson (2008)).

d. Biological Plausibility: Glyphosate has been shown to cause tumors in animal studies and there are at least two biological mechanisms (i.e., genotoxicity and oxidative stress) adduced for its mode of action. IARC considered this a strong rationale for carcinogenicity.

e. Strength: Meta-analyses suggest that the strength of association between ever use of glyphosate and NHL is in the range of 1.3-1.5. Of course, as mentioned in section c above, there is a dose-response so those exposed with high levels or for long durations have higher levels of risk. For example, McDuffie (2002) shows an OR=2.12 for people who used glyphosate greater than 2 days per year, and Eriksson (2008) showed an OR=2.36 for people who used glyphosate longer than 10 years.

f. Analogy: Not applicable.

g. Specificity: This is a criterion that is often not applicable in assessing a causal relationship and is ignored. However, this is one instance in which specificity does appear to apply. Glyphosate has not been associated with a broad range of malignancies, like epithelial cancers or even Hodgkin lymphoma, which would have suggested that methodological issues or biases in the studies could be the reasons rather than a true causal relationship. The fact that glyphosate has been linked specifically to NHL provides further reassurance that the association is causal.
X. Conclusions

My general view is that the approach and conclusions reflected in the IARC report of 2015 were reasonable and within the bounds of scientific and epidemiological normative practice and, with those practices in mind, reach the correct conclusion. While decisions regarding causal effects are, and usually will remain, judgement calls the epidemiologic and scientific evidence currently available leads to the conclusion to a reasonable degree of scientific certainty for most expert, objective, and reasonable viewers, myself included, that the use of glyphosate in its various combinations can cause non-Hodgkin lymphoma.

XI. Statement of Compensation and Previous Testimony

I am being compensated for my review and testimony at the rate of $450.00 per hour. The cases where I have testified at deposition or trial in the last four years are listed in Attachment B.

Dated: April 28, 2017

[Signature]
Alfred I. Neugut, MD, PHD
# TABLE 1. OBSERVATIONAL HUMAN STUDIES OF GLYPHOSATE EXPOSURE AND NHL (Adapted from IARC Monograph 112)

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Title</th>
<th>Study Design</th>
<th>Subjects</th>
<th>Location/Enrollment Period</th>
<th>Exposure</th>
<th>Outcome</th>
<th>Risk Estimate (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cantor et al.</td>
<td>1992</td>
<td>Pesticides and other agricultural risk factors for non-Hodgkin lymphoma among men in Iowa and Minnesota</td>
<td>Case-Control</td>
<td>Cases: 622 (response rate, 89.6%); Iowa health registry records and Minnesota hospital and pathology records Controls: 1245 (response rate, 76–79%); population-based; no cancer of the lymphohematopoietic system; frequency-matched to cases by age (5-year group), vital status, state. Random-digit dialing (aged &lt; 65 years); Medicare records (aged ≥ 65 years); state death certificate files (deceased subjects) Exposure assessment method: questionnaire; in-person interview</td>
<td>Iowa and Minnesota, USA 1980–1982</td>
<td>Ever handled glyphosate</td>
<td>NHL</td>
<td>1.1 (0.7-1.9)</td>
</tr>
<tr>
<td>McDuffie et al.</td>
<td>2001</td>
<td>Non-Hodgkin Lymphoma and specific pesticide exposures in men cross-Canada study of pesticides and health</td>
<td>Case-Control</td>
<td>Cases: 517 (response rate, 67.1%); from cancer registries and hospitals Controls: 1506 (response rate, 48%); random sample from health insurance and voting records Exposure assessment method: questionnaire, some administered by telephone, some by post</td>
<td>Canada 1991–1994</td>
<td>Exposed to glyphosate Unexposed &gt;0 and &lt;= 2 days &gt;2 days</td>
<td>NHL</td>
<td>1.2 (0.83-1.74) 1.0 (0.63-1.57) 2.12 (1.2-3.73)</td>
</tr>
<tr>
<td>Authors</td>
<td>Year</td>
<td>Study Description</td>
<td>Cases</td>
<td>Controls</td>
<td>Exposure Assessment Method</td>
<td>Location</td>
<td>NHL and HCL</td>
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<tr>
<td>Hardell et al.</td>
<td>2002</td>
<td>Exposure to pesticides as risk factor for non-Hodgkins lymphoma and hairy cell leukemia: pooled analysis of two Swedish case-control studies</td>
<td>515</td>
<td>1141</td>
<td>questionnaire</td>
<td>Sweden; four Northern counties and three counties in mid Sweden 1987-1992</td>
<td>Ever glyphosate exposure (univariate) Ever glyphosate exposure (multivariate)</td>
<td>3.04 (1.08-8.5) 1.85 (0.55-6.2)</td>
</tr>
<tr>
<td>De Roos et al.</td>
<td>2003</td>
<td>Integrative assessment of multiple pesticides as risk factors for non-Hodgkins lymphoma among men</td>
<td>650</td>
<td>1933</td>
<td>questionnaire; interview (direct or next-of-kin)</td>
<td>Nebraska, Iowa, Minnesota, Kansas, USA 1979-1986</td>
<td>Any glyphosate exposure</td>
<td>NHL 2.1 (1.1-4.0)</td>
</tr>
<tr>
<td>Lee et al.</td>
<td>2004</td>
<td>Non-Hodgkins lymphoma among asthmatics exposed to pesticides</td>
<td>872</td>
<td>2381</td>
<td>questionnaire; information on use of pesticides and history of asthma was based on interviews</td>
<td>Iowa, Minnesota and Nebraska, USA 1980-1986</td>
<td>Exposed to glyphosate - non-asthmatics Exposed to glyphosate - asthmatics</td>
<td>NHL 1.4 (0.98-2.1) 1.2 (0.4-3.3)</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Study Design</td>
<td>Number (after exclusions)</td>
<td>Location/Cohort</td>
<td>NHL Use</td>
<td>NHL Risk</td>
<td></td>
<td></td>
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<td>------------------</td>
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</tr>
<tr>
<td>De Roos et al.</td>
<td>2005</td>
<td>Prospective Cohort</td>
<td>54,315 (after exclusions, from a total cohort of 57,311 licensed pesticide applicators)</td>
<td>Iowa and North Carolina, USA 1993-2001</td>
<td>Ever use</td>
<td>1.1 (0.7-1.9)</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-20</td>
<td>1 (ref.)</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>21-56</td>
<td>0.7 (0.4-1.4)</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>57-2678</td>
<td>0.9 (0.5-1.6)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Trend-test P value 0.73</td>
<td></td>
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<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Study Design</th>
<th>Number (response rate, 91%); incident NHL cases were enrolled from university hospitals</th>
<th>Location/Cohort</th>
<th>NHL Use</th>
<th>NHL Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erikkson et al.</td>
<td>2008</td>
<td>Case-Control</td>
<td>910</td>
<td>Sweden; Four health service areas (Lund, Linkoping, Orebro, and Umea) 1999-2002</td>
<td>Any glyphosate Any glyphosate*</td>
<td>2.02 (1.1-3.71)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>&lt;=10 days per year use</td>
<td></td>
<td>1-10 years</td>
<td>1.69 (0.7-4.07)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>&gt;10 days per year use</td>
<td></td>
<td>&gt;10 years</td>
<td>2.36 (1.04-5.37)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>B-cell lymphoma</td>
<td></td>
<td>B-cell lymphoma</td>
<td>1.11 (0.24-5.08)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Lymphocytic lymphoma/B-CLL</td>
<td></td>
<td>Lymphocytic lymphoma/B-CLL</td>
<td>2.26 (1.16-4.4)</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Diffuse large B-cell lymphoma</td>
<td></td>
<td>Diffuse large B-cell lymphoma</td>
<td>1.87 (0.998-3.51)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Follicular grade I-II</td>
<td></td>
<td>Follicular grade I-II</td>
<td>1.63 (0.53-4.96)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other specified B-cell lymphoma</td>
<td></td>
<td>Other specified B-cell lymphoma</td>
<td>1.47 (0.33-6.61)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Unspecified B-cell lymphoma</td>
<td></td>
<td>Unspecified B-cell lymphoma</td>
<td>2.29 (0.51-10.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>T-cell lymphoma</td>
<td></td>
<td>T-cell lymphoma</td>
<td>5.63 (1.44-22)</td>
</tr>
<tr>
<td>Orsi et al. 2009</td>
<td>Occupational exposure to pesticides and lymphoid neoplasms among men: results of a French case-control study</td>
<td>Case-Control</td>
<td>Cases: 491 (response rate, 95.7%); cases (244 NHL; 87 HL; 104 LPSs; 56 MM) were recruited from main hospitals of the French cities of Brest, Caen, Nantes, Lille, Toulouse and Bordeaux, aged 20–75 years; ALL cases excluded Controls: 456 (response rate, 91.2%); matched on age and sex, recruited in the same hospitals as the cases, mainly in orthopedic and rheumatological departments and residing in the hospital’s catchment area Exposure assessment method: questionnaire</td>
<td>France 2000–2004</td>
<td>Any glyphosate exposure</td>
<td>NHL</td>
</tr>
<tr>
<td>Cocco et al. 2013</td>
<td>Lymphoma risk and occupational exposure to pesticides: results of the Epilymph study</td>
<td>Case-Control</td>
<td>Cases: 2348 (response rate, 88%); cases were all consecutive adult patients first diagnosed with lymphoma during the study period, resident in the referral area of the participating centers Controls: 2462 (response rate, 81% hospital; 52% population); controls from Germany and Italy were randomly selected by sampling from the general population and matched to cases on sex, 5-year age-group, and residence area. The rest of the centers used matched hospital controls, excluding diagnoses of cancer, infectious diseases and immunodeficiency diseases Exposure assessment method: questionnaire; support of a crop exposure matrix to supplement the available information, industrial hygienists and occupational experts in each participating center reviewed the general questionnaires and job modules to assess exposure to pesticides</td>
<td>Czech Republic, France, Germany, Italy, Ireland and Spain 1998–2004</td>
<td>Occupational exposure to glyphosate</td>
<td>B-cell lymphoma</td>
</tr>
<tr>
<td>Author</td>
<td>Year(s)</td>
<td>Title</td>
<td>Species, strain (sex) Duration</td>
<td>Dosing regimen, Animals/groups at start</td>
<td>For each target organ: incidence (%) and/or multiplicity of tumors</td>
<td>Significance</td>
</tr>
<tr>
<td>-----------------</td>
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<td>-----------------------------------------------------------------------</td>
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</tr>
<tr>
<td>Schinasi et al.</td>
<td>2014</td>
<td>Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis</td>
<td>Meta-Analysis</td>
<td>The meta-analysis for glyphosate included six studies (McDuffie et al., 2001; Hardell et al., 2002; De Roos et al., 2003; 2005a; Eriksson et al., 2008; Orsi et al., 2009)</td>
<td>NHL 1.5 (1.1–2.0)</td>
<td></td>
</tr>
<tr>
<td>Chang et al.</td>
<td>2016</td>
<td>Systematic review and meta-analysis of glyphosate exposure and risk of lymphohematopoietic cancers</td>
<td>Meta-Analysis</td>
<td>LHC incl NHL</td>
<td>meta-RR = 1.3 (1.0–1.6) meta-RR = 1.4 (1.0–1.9) meta-RR = 1.1 (0.7–1.6)</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 2. ANIMAL EXPERIMENTS REGARDING CARCINOGENICITY OF GLYPHOSATE (Cited in IARC Monograph 112)**
| EPA | 1985a, b, 1986, 1991a | Glyphosate; EPA Reg.#: 524–308; Mouse oncogenicity study. Document No. 004370. | Mouse, CD-1 (M, F) 24 mo Diet containing glyphosate (technical grade; purity, 99.7%) at concentrations of 0, 1000, 5000, or 30 000 ppm, ad libitum, for 24 mo 50 M and 50 F/group [age, NR] | Males Renal tubule adenoma: 0/49, 0/49, 1/50 (2%), 3/50 (6%) Females No data provided on the kidney Report from the PWG of the EPA (1986): Males Renal tubule adenoma: 1/49 (2%), 0/49, 0/50, 1/50 (2%) Renal tubule carcinoma: 0/49, 0/49, 1/50 (2%), 2/50 (4%) [NS] Renal tubule adenoma or carcinoma (combined): 1/49 (2%), 0/49, 1/50 (2%), 3/50 (6%) [P=0.037; Cochran-Armitage trend test] [P=0.034; Cochran-Armitage trend test] |

EPA Reg.#: 524–308; Roundup; glyphosate; pathology report on additional kidney sections. Glyphosate; EPA Registration No. 524–308; Roundup; additional histopathological evaluations of kidneys in the chronic feeding study of glyphosate in mice. Document No. 005590. Second peer review of glyphosate.

Mouse, CD-1 (M, F) 104 wk

Diet containing glyphosate (purity, 98.6%) at doses of 0, 100, 300, 1000 mg/kg bw, ad libitum, for 104 wk

50 M and 50 F/group [age, NR]

Males

Haemangiosarcoma: 0/50, 0/50, 0/50, 4/50 (8%)

Histiocytic sarcoma in the lymphoreticular/haemopoietic tissue: 0/50, 2/50 (4%), 0/50, 2/50 (4%)

Females

Haemangiosarcoma: 0/50, 2/50 (4%), 0/50, 1/50 (2%)

Histiocytic sarcoma in the lymphoreticular/haemopoietic tissue: 0/50, 3/50 (6%), 3/50 (6%), 1/50 (2%)

[P < 0.001; Cochran–Armitage trend test]

NS

NS
<table>
<thead>
<tr>
<th>Group</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Untreated control (no treatment)</td>
</tr>
<tr>
<td>II</td>
<td>Glyphosate only: 25 mg/kg bw topically, 3 x/wk, for 32 wk</td>
</tr>
<tr>
<td>III</td>
<td>Single topical application of DMBA, 52 µg/mouse, followed 1 wk later by TPA, 5 µg/mouse, 3 x/wk, for 32 wk</td>
</tr>
<tr>
<td>IV</td>
<td>Single topical application of glyphosate, 25 mg/kg bw, followed 1 wk later by TPA, 5 µg/mouse, 3 x/wk, for 32 wk</td>
</tr>
<tr>
<td>V</td>
<td>3 x/wk topical application of glyphosate, 25 mg/kg bw, for 3 wk, followed 1 wk later by TPA, 5 µg/mouse, 3 x/wk, for 32 wk</td>
</tr>
<tr>
<td>VI</td>
<td>Single topical application of DMBA, 52 µg/mouse</td>
</tr>
<tr>
<td>VII</td>
<td>Topical application of TPA, 5 µg/mouse, 3 x/wk, for 32 wk</td>
</tr>
<tr>
<td>VIII</td>
<td>8/20*, 2.8 ± 0.9</td>
</tr>
</tbody>
</table>

Skin tumors [called “papillomas” by the authors, following gross examination only] *P < 0.05 vs groups VI and VII
Group VIII: single topical application of DMBA, 52 μg/mouse, followed 1 wk later by topical treatment with glyphosate, 25 mg/kg bw, 3 × /wk, for 32 wk
Séralini et al. 2014 Republished study: long-term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize.

Rat, Sprague-Dawley (M, F) 24 mo

Drinking-water containing a glyphosate-based formulation at a concentration of 0 (control), 1.1 x 10^{-8} \text{mg/L} \text{ (glyphosate, 5.0 x 10^{-5} mg/L)}, 0.09\% \text{ (glyphosate, 400 mg/L) or 0.5\% (glyphosate, 2.25 x 103 mg/L), ad libitum, for 24 mo}}

10 M and 10 F/group (age, 5 wk)

Males
No significant increase in tumor incidence observed in any of the treated groups

Females
Mammary tumors (mainly fibroadenomas and adenocarcinomas): 5/10 (50\%), 9/10 (90\%), 10/10 (100\%){*}, 9/10 (90\%)

Pituitary lesions (hypertrophy, hyperplasia, and adenoma): 6/10 (60\%), 8/10 (80\%), 7/10 (70\%), 7/10 (70\%)


Rat, Wistar (M, F) 24 mo

Drinking-water containing ammonium salt of glyphosate (13.85\% solution) [purity of glyphosate, NR] was used to make aqueous solutions of 0, 300, 900, and 2700 mg/L [Details on dosing regimen, NR]

55 M and 55 F/group (age, 6–7 wk)

No significant increase in tumor incidence observed in any of the treated groups

NS


Rat, Wistar-Alpk:APiSD (M, F) 1 yr

Diet containing glyphosate (purity, 95.6\%) at concentrations of 0, 2000, 8000, or 20,000 ppm, ad libitum, for 1 yr

24 M and 24 F/group [age, NR]

No significant increase in tumor incidence observed in any groups of treated animals

NS
<table>
<thead>
<tr>
<th>JMPR</th>
<th>Year</th>
<th>Study Description</th>
<th>Species</th>
<th>Treatment Details</th>
<th>Tumor Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2006</td>
<td>Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva</td>
<td>Rat, Sprague-Dawley (M, F)</td>
<td>Diet containing glyphosate (purity, 98.7–98.9%) at doses of 0, 10, 100, 300, or 1000 mg/kg bw, ad libitum, for 104 wk 50 M and 50 F/group [age, NR]</td>
<td>No significant increase in tumor incidence observed in any groups of treated animals</td>
</tr>
<tr>
<td></td>
<td>2006</td>
<td>Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva</td>
<td>Rat, Wistar-Alpk:APiSD (M, F)</td>
<td>Diet containing glyphosate (purity, 97.6%) at concentrations of 0, 2000, 6000, or 20 000 ppm, ad libitum, for 2 yr 52 M and 52 F/group [age, NR]</td>
<td>No significant increase in tumor incidence observed in any groups of treated animals</td>
</tr>
<tr>
<td>EPA</td>
<td>1991 A.B.C.D</td>
<td>Rat Sprague-Dawley (M, F)</td>
<td>Diet containing glyphosate (technical grade; purity, 96.5%) at concentrations of 0, 2000, 8000, or 20 000 ppm, ad libitum, for 24 mo 60 M and 60 F/group (age, 8 wk) 10 rats/group killed after 12 mo</td>
<td>Males Pancreas (islet cell): Adenoma: 1/58 (2%), 8/57 (14%)*, 5/60 (8%), 7/59 (12%) Carcinoma: 1/58 (2%), 0/57, 0/60, 0/59 Adenoma or carcinoma (combined): 2/58 (3%), 8/57 (14%), 5/60 (8%), 7/59 (12%)</td>
<td>Adenoma, * P ≤ 0.05 (Fisher exact test with Bonferroni inequality); see comments</td>
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<td></td>
<td>Liver: Hepatocellular adenoma: 2/60 (3%), 2/60 (3%), 3/60 (5%), 7/60 (12%) Hepatocellular carcinoma: 3/60 (5%), 2/60 (3%), 1/60 (2%), 2/60 (3%)</td>
<td>Females Pancreas (islet cell): Adenoma: 5/60 (8%), 1/60 (2%), 4/60 (7%), 0/59 Carcinoma: 0/60, 0/60, 0/60, 0/59 Adenoma or carcinoma (combined): 5/60 (8%), 1/60 (2%), 4/60 (7%), 0/59</td>
<td>Adenoma, P for trend = 0.016; see comments</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Thyroid: C-cell adenoma: 2/60 (3%), 2/60 (3%), 6/60 (10%), 6/60 (10%) C-cell carcinoma: 0/60, 0/60, 1/60, 0/60</td>
<td></td>
<td>Adenoma, P for trend = 0.031; see comments</td>
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</table>
EPA 1991
a,b,c,d Second peer review of glyphosate


Peer review on glyphosate. Document No. 008527.


Rat Sprague-Dawley (M, F) Lifetime (up to 26 mo) Diet containing glyphosate (purity, 98.7%) at concentrations of 0 ppm, 30 ppm (3 mg/kg bw per day), 100 ppm (10 mg/kg bw per day), 300 ppm (31 mg/kg bw per day), ad libitum, up to 26 mo 50 M and 50 F/group [age, NR]

Males
Pancreas (islet cell): Adenoma: 0/50 (0%), 5/49* (10%), 2/50 (4%), 2/50 (4%) Carcinoma: 0/50 (0%), 0/49 (0%), 0/50 (0%), 1/50 (2%) Adenoma or carcinoma (combined): 0/50 (0%), 5/49 (10%), 2/50 (4%), 3/50 (6%) Females
Pancreas (islet cell): Adenoma: 2/50 (4%), 1/50 (2%), 1/50 (2%), 0/50 (0%) Carcinoma: 0/50 (0%), 1/50 (2%), 1/50 (2%), 1/50 (2%) Adenoma or carcinoma (combined): 2/50 (10%), 2/50 (2%), 2/50 (74%), 1/50 (2%) Adenoma, *P < 0.05; Fisher exact test

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**TABLE 3A. STUDIES OF DIRECT EXPOSURE TO GLYPHOSATE-BASED FORMULATION IN HUMANS (Cited in IARC Monograph 112)**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year(s)</th>
<th>Title</th>
<th>Cell type</th>
<th>End-point</th>
<th>Test</th>
<th>Description of exposure and controls</th>
<th>Test results/Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paz-y-Miño et al.</td>
<td>2007</td>
<td>Evaluation of DNA damage in an Ecuadorian population exposed to glyphosate</td>
<td>NA</td>
<td>DNA damage</td>
<td>DNA strand breaks, comet assay</td>
<td>24 exposed individuals in northern Ecuador; areas sprayed with glyphosate-based formulation (sampling 2 weeks to 2 months after spraying); control group was 21 non-exposed individuals</td>
<td>+ / P&lt;0.001</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Study Design</td>
<td>Sample Description</td>
<td>Key Findings</td>
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<td>-------------------------------------------------------------------------------------------------------------</td>
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<tr>
<td>Paz-y-Miño et al.</td>
<td>2011</td>
<td>Baseline determination in social, health, and genetic areas in communities affected by glyphosate aerial spraying on the northeastern Ecuadorian border</td>
<td>Chromosomal damage and aberrations 92 individuals in 10 communities in northern border of Ecuador; sampling 2 years after last aerial spraying with herbicide mix containing glyphosate; control group was 90 healthy individuals from several provinces without background of smoking or exposure to genotoxic substances (hydrocarbons, X-rays, or pesticides)</td>
<td></td>
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<td></td>
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</tbody>
</table>
| Bolognesi et al.               | 2009 | Biomonitoring of genotoxic risk in agricultural workers from five Colombian regions: association to occupational exposure to glyphosate | Lymphocytes, Chromosomal damage and Micronucleus formation 55 community residents, Nariño, Colombia; area with aerial glyphosate-based formulation spraying for coca and poppy eradication (glyphosate was tank-mixed with an adjuvant) | + / P<0.001 *p-value for after spraying vs. before spraying in the same individuals  
53 community residents, Putumayo, Colombia; area with aerial glyphosate-based formulation spraying for coca and poppy eradication (glyphosate was tank-mixed with an adjuvant) | + / P=0.01 *p-value for after spraying vs. before spraying in the same individuals  
27 community residents, Valle del Cauca, Colombia; area where glyphosate-based formulation was applied through aerial spraying for sugar-cane maturation (glyphosate was applied without adjuvant) | + / P<0.001 *p-value for after spraying vs. before spraying in the same individuals |
<table>
<thead>
<tr>
<th>Author</th>
<th>Year(s)</th>
<th>Title</th>
<th>Tissue, cell line</th>
<th>Endpoint</th>
<th>Test</th>
<th>Results</th>
<th>Dose (LED or HID)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mañas et al.</td>
<td>2009a</td>
<td>Genotoxicity of glyphosate assessed by the comet assay and cytogenetic tests</td>
<td>Liver Hep-2</td>
<td>DNA damage</td>
<td>DNA strand breaks, comet assay</td>
<td>+</td>
<td>3mM [507.2 μg/mL]</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Mladinic et al.</td>
<td>2009b</td>
<td>Evaluation of genome damage and its relation to oxidative stress induced by glyphosate in human lymphocytes in vitro.</td>
<td>Lymphocytes</td>
<td>DNA damage</td>
<td>DNA strand breaks, standard and hOGGI modified comet assay</td>
<td>+</td>
<td>3.5 μg/mL</td>
<td>P &lt; 0.01 (with hOGGI modified comet assay, + S9 at highest dose tested 580 μg/mL)</td>
</tr>
<tr>
<td>Alvarez-Moya et al.</td>
<td>2014</td>
<td>Comparison of the in vivo and in vitro genotoxicity of glyphosate isopropylamine salt in three different organisms.</td>
<td>Lymphocytes</td>
<td>DNA damage</td>
<td>DNA strand breaks, comet assay</td>
<td>+</td>
<td>0.00007 mM [0.12 μg/mL]</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Monroy et al.</td>
<td>2005</td>
<td>Cytotoxicity and genotoxicity of human cells exposed in vitro to glyphosate</td>
<td>Fibroblast GM 38</td>
<td>DNA damage</td>
<td>DNA strand breaks, comet assay</td>
<td>+</td>
<td>4mM [676 μg/mL]</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Study</td>
<td>Year</td>
<td>Description</td>
<td>Cell Type</td>
<td>Endpoint</td>
<td>Result</td>
<td>Concentration [pM]</td>
<td>p-value</td>
<td>Notes</td>
</tr>
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</tr>
<tr>
<td>Lucken et al.</td>
<td>2004</td>
<td>Synergistic DNA damage by oxidative stress (induced by H2O2) and nongenotoxic environmental chemicals in human fibroblasts</td>
<td>Fibroblast GM 5757</td>
<td>DNA damage</td>
<td>+</td>
<td>75 mM [12.680 μg/mL]</td>
<td></td>
<td>Not Reported</td>
</tr>
<tr>
<td>Monroy et al.</td>
<td>2005</td>
<td>Cytotoxicity and genotoxicity of human cells exposed in vitro to glyphosate</td>
<td>Fibrosarcoma HT1080</td>
<td>DNA damage</td>
<td>+</td>
<td>4.75 mM [803 μg/mL]</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Koller et al.</td>
<td>2012</td>
<td>Cytotoxic and DNA-damaging properties of glyphosate and Roundup in human-derived buccal epithelial cells</td>
<td>Buccal carcinoma TR146</td>
<td>DNA damage</td>
<td>+</td>
<td>20 μg/mL</td>
<td>&lt;0.05 dose-dependent increase</td>
<td></td>
</tr>
<tr>
<td>Mañas et al.</td>
<td>2009</td>
<td>Genotoxicity of glyphosate assessed by the comet assay and cytogenetic tests</td>
<td>Lymphocytes</td>
<td>Chromosomal damage</td>
<td>-</td>
<td>6 mM [1015 μg/mL]</td>
<td></td>
<td>Not Significant</td>
</tr>
<tr>
<td>Mladinic et al.</td>
<td>2009</td>
<td>Characterization of chromatin instabilities induced by glyphosate, terbuthylazine and carbofuran using cytome FISH assay</td>
<td>Lymphocytes</td>
<td>Chromosomal damage</td>
<td>-</td>
<td>580 μg/mL</td>
<td>&lt;0.01 at highest exposure + S0</td>
<td></td>
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<tr>
<td>Bolognesi et al.</td>
<td>1997</td>
<td>Genotoxic activity of glyphosate and its technical formulation Roundup</td>
<td>Lymphocytes</td>
<td>Chromosomal damage</td>
<td>+</td>
<td>1000 μg/mL</td>
<td>&lt;0.05</td>
<td></td>
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**AMPA**
<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Description</th>
<th>Tissues</th>
<th>End Points</th>
<th>Result</th>
<th>Concentration</th>
<th>P Value</th>
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</thead>
<tbody>
<tr>
<td>Mañas et al.</td>
<td>2009b</td>
<td>Genotoxicity of AMPA, the environmental metabolite of glyphosate, assessed by the Comet assay and cytogenetic tests.</td>
<td>Liver Hep-2 DNA damage, DNA strand breaks, comet assay</td>
<td>+ NT</td>
<td>4.5 mM [500μg/mL]</td>
<td>P &lt; 0.05 at 4.5 mM; P &lt; 0.01 at up to 7.5 mM Dose-response relationship (p&lt;0.05)</td>
<td></td>
</tr>
<tr>
<td>Mañas et al.</td>
<td>2009b</td>
<td>Genotoxicity of AMPA, the environmental metabolite of glyphosate, assessed by the Comet assay and cytogenetic tests.</td>
<td>Lymphocytes Chromosomal damage, Chromosomal aberrations</td>
<td>+ NT</td>
<td>1.8 mM [200 μg/mL]</td>
<td>P &lt; 0.05</td>
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</table>

**GLYPHOSATE-BASED FORMULATIONS**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Description</th>
<th>Tissues</th>
<th>End Points</th>
<th>Result</th>
<th>Concentration</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guenier et al.</td>
<td>2009</td>
<td>Glyphosate-based herbicides are toxic and endocrine disruptors in human cell lines</td>
<td>Liver Hep-2 DNA damage, DNA strand breaks, comet assay</td>
<td>+ NT</td>
<td>5ppm</td>
<td>Not Reported</td>
<td></td>
</tr>
<tr>
<td>Koller et al.</td>
<td>2012</td>
<td>Cytotoxic and DNA-damaging properties of glyphosate and Roundup in human-derived buccal epithelial cells</td>
<td>Buccal carcinoma TR146 DNA damage, DNA strand breaks, SCGE assay</td>
<td>+ NT</td>
<td>20 μg/mL</td>
<td>Dose-dependent increase (P≤0.05)</td>
<td></td>
</tr>
<tr>
<td>Author(s)</td>
<td>Year</td>
<td>Title</td>
<td>Cell Type</td>
<td>Chromosomal Damage</td>
<td>Sister-chromatid Exchange</td>
<td>Conc. (µg/mL)</td>
<td>P Value</td>
</tr>
<tr>
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<td>-----------------------------------------------------------------------</td>
<td>----------------------</td>
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<td>--------------------------</td>
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</tr>
<tr>
<td>Vigfussion &amp; Vyse</td>
<td>1980</td>
<td>The effect of the pesticides, Dexon, Capitan and Roundup, on sister-chromatid exchanges in human lymphocytes in vitro</td>
<td>Lymphocytes</td>
<td>Chromosomal damage</td>
<td>Sister-chromatid exchange</td>
<td>NT</td>
<td>250</td>
</tr>
<tr>
<td>Bolognesi et al.</td>
<td>1997</td>
<td>Genotoxic activity of glyphosate and its technical formulation Roundup</td>
<td>Lymphocytes</td>
<td>Chromosomal damage</td>
<td>Sister-chromatid exchange</td>
<td>NT</td>
<td>100</td>
</tr>
</tbody>
</table>

AMPA, aminomethyl phosphonic acid; HID, highest ineffective dose; hOGG1, human 8-hydroxyguanosine DNA-glycosylase; LED, lowest effective dose; NR, not reported; NT, not tested; S9, 9000 x g supernatant; SCGE, single cell gel electrophoresis; vs, versus
FIGURE 1. FOREST PLOT FOR GLYPHOSATE/NHL – SHINASI & LEON, 2014

Figure D. (Schinasi & Leon, 2014) Forest plots showing estimates of association between non-Hodgkins Lymphoma and occupational, agricultural exposure to (D) glyphosate.

FIGURE 2. FOREST PLOT – CHANG & DELZELL, 2016

Figure 1. Forest plots of relative risk (RR) estimates and 95% confidence intervals (CIs) for the association between glyphosate exposure and risk of non-Hodgkin lymphoma. Meta-RRs were identical in random-effects and fixed-effects models.
REFERENCES:


European Food Safety Authority. Conclusion on the peer review of the pesticide risk assessment of the active substance glyphosate. EFSA J 2015;13:4302.


George, J., & Shukla, Y. (2013). Emptying of intracellular calcium pool and oxidative stress imbalance are associated with the glyphosate-induced proliferation in human skin keratinocytes HaCaT cells. *ISRN dermatology, 2013*.


Attachment A
Alfred I. Neugut  
Curriculum Vitae  
April 1, 2017

HOME ADDRESS: [Redacted]

OFFICE ADDRESS:  
Division of Oncology  
Department of Medicine  
New York Presbyterian Hospital  
722 West 168th Street, Room 725  
New York, NY 10032  

Department of Epidemiology  
Mailman School of Public Health  
Columbia University  
722 W. 168th St., Room 725  
New York, NY 10032

TELEPHONE: [Redacted]  
FAX: [Redacted]  
E-Mail: [Redacted]

PLACE OF BIRTH: [Redacted]

EDUCATION:  
1972 B.A.  
Columbia College, New York, NY; Chemistry, cum laude.

1977 M.D.  
Columbia University College of Physicians and Surgeons, New York, NY.

1977 Ph.D.  
Columbia University Graduate School of Arts and Sciences, New York, NY;  
Pathobiology. Ph.D. Thesis: Studies on the genetic and molecular controls of the  
transformed phenotype, especially anchorage-independent growth and protease  
production. Under supervision of Dr. I. Bernard Weinstein.

1983 M.P.H.  
Columbia University School of Public Health, New York, NY; Epidemiology.

POSTDOCTORAL TRAINING:  
1977 - 1978  
Bronx Municipal Hospital Center, Bronx, N.Y., Intern in Medicine.

1978 - 1980  
Bronx Municipal Hospital Center, Bronx, N.Y., Resident in Medicine.

1980 - 1981  

1980 - 1981  
Memorial Hospital, New York, N.Y., Medical Oncology, Clinical Fellow.

1981 - 1983  
Presbyterian Hospital, New York, N.Y., Hematology-Oncology, Clinical Fellow.

LICENSURE AND CERTIFICATION:  
1978 New York license, Number 136912  
1981 New Jersey license, Number 39887  
1980 American Board of Internal Medicine  
1983 American Board of Medical Oncology

MEMBERSHIP IN SOCIETIES:  
American Association for Cancer Research.  
American College of Epidemiology, Fellow.  
American College of Physicians, Fellow.  
American Epidemiological Society.  
American Public Health Association.  
American Society of Clinical Oncology.  
American Society of Preventive Oncology.  
International Society of Cancer Chemoprevention.  
International Society for Pharmacoeconomics and Outcomes Research  
International Society for Pharmacoepidemiology.  
Society for Epidemiologic Research.
Alfred I. Neugut
Curriculum Vitae

ACADEMIC APPOINTMENTS:
1983 - 1985 Columbia University College of Physicians & Surgeons, New York, N.Y., Assistant Professor of Medicine.
2001 - Columbia University College of Physicians & Surgeons, New York, N.Y. Professor of Medicine and Epidemiology.
2003 - 2006 Columbia University College of Physicians & Surgeons, New York, N.Y. Acting Head, Division of Medical Oncology, Department of Medicine.
2010 - 2013 Head, Chronic Disease Epidemiology, Department of Epidemiology, Mailman School of Public Health.
2014 - Director of Faculty Development, Department of Epidemiology, Mailman School of Public Health.

HOSPITAL APPOINTMENTS:
1989 - 1998 Co-Director, Oncology Outpatient Unit, Presbyterian Hospital, New York, N.Y.
1993 - 1999 Harlem Hospital Center, New York, N.Y. Associate Attending Physician.
1999 - Present New York Presbyterian Hospital, New York, N.Y. Director, Cancer Prevention Center, Columbia-Presbyterian Campus.
1999 - Present New York Presbyterian Hospital, New York, N.Y. Member, NYPH Oncology Service Line Executive Council.
1999 - Present New York Presbyterian Hospital, New York, N.Y., Attending Physician.
1999 - 2008 Harlem Hospital Center, New York, N.Y., Attending Physician.
2001 - Present New York Presbyterian Hospital, New York, N.Y. Member, NYPH Preventive Service Line Executive Council.
2002 – 2007 New York Presbyterian Hospital, New York, N.Y. Member, NYPH Digestive Disease Service Line Executive Council.

HONORS AND AWARDS:
1972 - 1977 Medical Scientist Training Program, Columbia P & S.
1980 - 1981 Clinical Fellow of the American Cancer Society, Memorial Sloan-Kettering Cancer Center.
Alfred I. Neugut
Curriculum Vitae

1980 - 1981  Clinical Associate of the Clinical Cancer Education Grant, Memorial Sloan-Kettering Cancer Center.
1981 - 1990  Mellon Fellow in Epidemiology in Medicine, School of Public Health and Department of Medicine, Columbia University.
1984 - 1986  Junior Faculty Fellow of the American Cancer Society.
1996 - 1997  Distinguished Service Award, American Society of Preventive Oncology.
2005 - 2015  Myron M. Studner Professorship in Cancer Research in the Department of Medicine
2015 - 2016  Mentor of the Year, Office of Academic Affairs and Irving Institute for Clinical and Translational Research, Columbia University Medical Center.
2016 - 2016  Joseph R. Fraumeni Distinguished Achievement Award, American Society of Preventive Oncology

GRANTS:

1984 - 1986  Junior Faculty Fellowship, American Cancer Society - $10,000 per year.
1986 - 1991  Principal Investigator, NCI grant #1-K07-CA01211. Preventive Oncology Academic Award $270,000.
1989 - 1990  Principal Investigator, Rudin Foundation. The Effect of Calcium Supplements on Oncogene Expression - $85,000.
1989 - 1990  Principal Investigator, IARC. Feasibility Phase of International Study of Cancer Risk in Biology Research Laboratory Workers - $5,000.
1993 - 1994  Principal Investigator, subcontract from Cancer Prevention Research Institute. Colorectal Cancer and DNA Repair Markers - $10,000.
Alfred I. Neugut
Curriculum Vitae

1993 - 2001
Co-Investigator, CDC U64/CCU206822. A Prospective Study of Cervical Disease in HIV-Infected Women 5% effort on supplemental management project A. Management of Cervical intra-epithelial neoplasia in HIV-infected and non-infected women: A comparative study. Principal Investigator, T. Wright - $440,000 per year.

1994 - 1999
Co-Investigator, NCI grant P30-CA13696. Cancer Center Support Grant for Columbia Presbyterian Cancer Center, 10% effort. Principal Investigator, KH Antman $1.8 million per year.

1995 - 1996

1995 - 2005

1995 - 1996
Principal Investigator, NCI grant #P20-CA66224. Preliminary Studies for Columbia-HIP Minority Cohort Study (CHIPS) - $20,000.

1995 - 1998
Principal Investigator, on Columbia University subcontract, NCI grant #2-PO1-CA32617-09A2. Smoking, Diet and Other Risk Factors for Tobacco-Related Cancers. Principal Investigator, S. Stellman, American Health Foundation - $118,259.

1996 - 1997

1996 - 1998
Principal Investigator, on Columbia University subcontract, Wireless Technology Research grant. Cellular Telephone Use and Risk of Brain Tumors. Principal Investigator, J. Muscat, American Health Foundation - $72,000.

1996 - 2001

1996 - 2001
Principal Investigator, NCI grant T32-CA09529. Training Program in Cancer Epidemiology, Biostatistics, Environmental Health Sciences - $54,000.

1997 - 1998
Principal Investigator, Bristol-Myers/Squibb grant. Population-Based Study of Health Outcomes in Colorectal Cancer - $54,000.

1997 - 2001

1998 - 1999

1998 - 1999
Co-Investigator, Columbia Clinical Trials Office. PTEN Mutations in Prostate Cancer; Pathologic Correlations and Clinical Significance. Principal Investigator, M.A. Rubin - $49,900.

1998 - 1999
Principal Investigator, Columbia University subcontract, New York City contract to Academic Medicine Development Corporation, New York Cancer Project. Principal Investigator, Maria Mitchell - $153,082.

1998 - 2001

1998 - 2001
Co-Principal Investigator, Robert Wood Johnson Foundation. Addressing Tobacco in Managed Care Program. Principal Investigator, D. Sadowsky - $500,000.

1999 - 2000
Alfred I. Neugut  
Curriculum Vitae


1999 - 2002  Co-Director, Columbia Presbyterian Campus. NYPH Cancer Prevention Center - $675,000.

1999 - 2002  Principal Investigator, American Cancer Society TIOG-99-363-01-CPC. Dissemination of Colorectal Cancer Screening to Primary Care Physicians - $976,000.

1999 - 2003  Principal Investigator, Columbia University subcontract, NCI grant #RO1-CA81932. Tailored Communications for Colorectal Cancer Screening. Principal Investigator, C. Basch, Teachers College - $170,970.


1999 - 2003  Principal Investigator, NYSDOH. Colorectal Cancer Screening and Prostate Cancer Education - $177,161.


2001 - 2003  Principal Investigator, American Cancer Society RSghp-01-024-01-CCE. Effectiveness of Care in the Elderly - $622,000.


2001 - 2006  Principal Investigator, NCI grant K05-CA89155. Established Investigator Award in Cancer Prevention, Control, Behavioral and Population Research: Colorectal Cancer and Other Cancers of the GI Tract. $660,000.

2001 - 2006  Co-Principal Investigator, CDC grant #U57/CCU220685. Dissemination of Cervical Cancer Screening to Primary Care Physicians. Principal Investigator, Sherri Sheinfeld Gorin - $1,107,735.


2002 - 2004  Principal Investigator, Pfizer/Pharmacia-NYPH National Newsletter on Cancer Prevention - $200,000.

2002 - 2005  Co-Director, Columbia Presbyterian Campus. NYPH Cancer Prevention Center - $675,000.

2002 - 2007  Principal Investigator, NCI grant T32-CA09529. Training Program in Cancer Epidemiology, Biostatistics, Environmental Health Sciences – $1,936,098.


2003 - 2014  Associate Director and Program Leader, NCI grant P30-CA13696 (Dalla-Favera). Herbert Irving Comprehensive Cancer Center Support Grant - $1,300,000.

2003 - 2004  Principal Investigator, Cellular Telecommunications and Internet Association (CTIA) grant. The Mobile Phone Study: National Brain Cancer Rates and Mobile Phone Use - $48,000.


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<th>Year</th>
<th>Role and Funders</th>
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<tr>
<td>2003 - 2006</td>
<td>Principal Investigator, Columbia University subcontract, NCI N01-CN-35159 Phase 1 and 2 Clinical Trials of Chemopreventive Agents. Principal Investigator, Scott Lippman, M.D. Anderson Cancer Center.</td>
</tr>
<tr>
<td>2003 - 2004</td>
<td>Principal Investigator, Columbia University subcontract, NCI N01-CN-17103 Early therapeutics development with phase 2 emphasis. Principal Investigator, E. Lesser, Montefiore Medical Center</td>
</tr>
<tr>
<td>2005- 2007</td>
<td>Principal Investigator, American Cancer Society grant RSGT-01-02404-CPHPS. Effectiveness of Cancer Care in the Elderly - $623,000.</td>
</tr>
<tr>
<td>2005- 2017</td>
<td>Principal Investigator, Department of Defense Breast Cancer Center of Excellence Award BC043120. Racial disparities in the initiation and intensity of adjuvant therapy for breast cancer - $9,966,608.</td>
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<tr>
<td>2007-2008</td>
<td>Principal Investigator, NYSDOH Colorectal Cancer Screening and Prostate Cancer Education Initiative for Bronx County - $50,000.</td>
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<tr>
<td>2008-2013</td>
<td>Principal Investigator, NCI grant T32-CA09529. Training Program in Cancer Epidemiology, Biostatistics, and Environmental Health Sciences – $2,235,920.</td>
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<tr>
<td>2008-2013</td>
<td>Principal Investigator, NYSDOH. Breast Cancer Screening, Colorectal Cancer Screening and Prostate Cancer Education - $250,000/yr.</td>
</tr>
<tr>
<td>2008-2012</td>
<td>Co-Investigator, NCI grant R01 CA134964. Determinants and risks of use and overuse of expensive drugs. Principal Investigator, D. Hershman - $639,600.</td>
</tr>
<tr>
<td>2009-2010</td>
<td>Principal Investigator, Herbert Irving Comprehensive Cancer Center Pilot Grant. Adherence to hormonal therapy in breast cancer: an intervention trial - $75,000.</td>
</tr>
<tr>
<td>2010-2011</td>
<td>Principal Investigator, Women at Risk. Use of urinary biomarkers for detecting drug adherence with aromatase inhibitors in women with early stage breast cancer. - $20,000.</td>
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</table>
Alfred I. Neugut
Curriculum Vitae

2010-2013  Principal Investigator, Department of Defense Prostate Cancer Research Program Health Disparity Research Award – Established Investigator PC094372. Racial Disparities in Palliative Care for Prostate Cancer. $450,000.


2010-2014  Subcontract Principal Investigator, R01 HS0197670. Comparative Effectiveness of Surgical Treatments for Lung Cancer in Elderly. Principal Investigator, J Wisinevsky, Mt. Sinai Medical Center - $255,457.

2011-2015  Principal Investigator, Conquer Cancer Foundation of ASCO-Komen Foundation Improving Cancer Care Grant. Text Messaging to Reduce Early Discontinuation of Adjuvant Hormonal Therapy in Breast Cancer: A Randomized Trial - $1,350,000.


2014-2019  Principal Investigator (Multi-PI Schwartz (Contact), Lassman), NCI grant UM1 CA189960. Columbia University Minority/Underserved Site NCI Community Oncology Research Program - $3,275,000.

2014-2019  Associate Director, NCI grant P30-CA13696 (Stephen Emerson).Herbert Irving Comprehensive Cancer Center Support Grant - $2,370,450.


2015-2020  Principal Investigator (Multi-PI Joffe (Contact), Jacobson, Ruff), NCI grant R01 CA192627. HIV’s Effects on Breast Cancer Treatment and Outcomes in South Africa - $1,450,359.

2015-2017  Principal Investigator, supplemental award to promote cancer prevention and control research in LMIC countries to NCI grant #P30-CA13696 (S. Emerson, PI). Palliative care and end-of-life issues among cancer patients in Soweto, South Africa - $260,000.

2015-2017  Principal Investigator, New York State Department of Health Prostate Cancer Hypothesis Development Grant, Impact of HIV on the Burden of Prostate Cancer in South Africa - $89,584

2016-2018  Principal Investigator, supplemental award for Pilot Program on Aging, HIV, and Outcomes in Non-AIDS Defining Cancers in Sub-Saharan Africa to NCI grant #P30-CA13696 (S. Emerson, PI) - $399,082

2017-2022  Principal Investigator (Multi-PI Terry), NCI grant T32 CA094061-16. Training program in cancer-related population sciences - $1,349,804.
Alfred I. Neugut  
Curriculum Vitae

2017-2018  Mentoring PI (Multi-PI Madiba, Sartorius), pilot grant from Pilot Program on HIV, Aging, and Outcomes in Non-AIDS Defining Cancer in Sub-Saharan African (P30 CA13696). HIV, aging and colorectal cancer among black patients in a hyperendemic HIV setting, KwaZuluNatal, South Africa - $30,000.

2017-2020  Co-investigator (Multi-PI Graham, Ruff, Black), Bristol-Myers Squibb Foundation Secure the Future Lung Cancer Programme. Centre of Respiratory Excellence (CORE), Gauteng - $1,500,000.

ADMINISTRATIVE RESPONSIBILITIES

1985 - 1992  Division of Cancer Control, Columbia University Comprehensive Cancer Center, Medical Director of Columbia Tumor Registry.

1986 - 1992  Presbyterian Hospital Cancer Committee, Member.

1988 - 1989  Division of Cancer Control, Columbia University Comprehensive Cancer Center, Director of "Test for Life" Colorectal Cancer Screening Program.

1988 - 1989  Columbia University Comprehensive Cancer Center, Co-Deputy Director for Cancer Control and Regional Activities.

1989 - 1991  Columbia University Comprehensive Cancer Center, Deputy Director for Cancer Epidemiology and Prevention.

1990 - 1991  Environmental Sciences Search Committee, Member.

1990 - 2005  Director, Columbia University Seminar on Cancer.

1990 - 2003  Columbia University Comprehensive Cancer Center, member of ACS Institutional Research Grant Committee.

1991 - 1992  Columbia University Comprehensive Cancer Center, Associate Director for Cancer Etiology, Prevention, and Control.

1992 - 1993  Presbyterian Hospital Autopsy Committee, member.


1993 - 1999  Columbia-Presbyterian Cancer Center, Head of Working Group on Gastrointestinal Tract Cancers.

1993 - 1995  School of Public Health Steering Committee, member.

1994 - 1995  Faculty Council of the Faculty of Medicine, member.

1995 - 1999  Member, Columbia-Presbyterian Cancer Center Protocol Review Committee.

1996 - 2011  Head, Epidemiology Faculty Appointments and Promotions Committees.

1999 - Present  Head, Prevention, Control and Disparities Program, Herbert Irving Comprehensive Cancer Center.

1999 - Present  Member, Executive Committee of the Department of Medicine.

2001 - 2002  Member, Avon Products Foundation Professorship Search Committee.

2002 - 2011  Chair, Committee on Appointments and Promotions, Department of Epidemiology.

2002 - Present  New York Presbyterian Hospital, Member of Advisory Committee for Celiac Disease Center.

2004 - Present  Associate Director for Population Sciences, Herbert Irving Comprehensive Cancer Center.

2004 - 2005  Member, Urology Chair Search Committee, Herbert Irving Comprehensive Cancer Center.

2005 - 2007  Member, Search Committee for Mieczyslaw Finster Professor of Anesthesiology and Epidemiology.

2007 - Present  Member, Department of Medicine, Standing Committee on Recruitment and Retention for Clinical and Epidemiology Research.

2008 - 2009  Chair, Environmental Health Sciences Chair Search Committee.

2010 - 2011  Member, Search Committee for Director of Bone Marrow Transplant Unit.

2012 -  Member, Committee on Appointments and Promotions, Department of Epidemiology.
Alfred I. Neugut
Curriculum Vitae

2016- Member, Cancer Scientist Search Committee, P&S
2017- Member, Internal Advisory Committee, Brain Tumor SPORE

TEACHING EXPERIENCE AND RESPONSIBILITIES:

Courses Taught, Primary Instructor
P8414 - Cancer Epidemiology, 15-40 students, 1982-2016
P8401 - Pharmacoepidemiology, 25 students, 1995-1998
P9480 - Epidemiology Colloquium, 50 students, 1992
G4500 – Cancer Biology II, Department of Pathology, 7-15 students, 2010-2013
EPIC, Cancer Epidemiology, Department of Epidemiology, 8-12 students, 2011-2017

Courses Taught, Preceptor or Lecturer
First Year Medical School Epidemiology, Preceptor, 20-25 students, 1983-1990.
Pathophysiology for Occupational and Physical Therapy, Lecturer, 50-75 students, 1985-.
Institute of Human Nutrition, Nutrition and Chronic Disease, Lecturer, 50 students, 2008-
The Body in Health and Disease, Hematology/Oncology Lecture on Cancer Screening, 2011

Clinical Teaching
Ward Attending, Medical Service, 1-2 months/year, 1983-.
Oncology Consult Attending, 1-2 months/year, 1983-.
Oncology Consult Attending, Harlem Hospital Center, 4-6 months/year, 1993-2006
Oncology Fellow Course, Study Design and Epidemiology, 10-12 lectures, 2015, 2017.

Graduate Student Supervision

Doctoral Advisor
1995 - Judith S. Jacobson, Dr.P.H., Epidemiology. Associate Professor of Epidemiology, Columbia.
1996 - Jeanne Mandelblatt, Ph.D., Epidemiology, pending. Professor of Oncology and Medicine,
Director of the Division of Health Outcomes and Health Behavior, and Associate Director for
Population Sciences for the Lombardi Cancer Center at Georgetown University Medical School
1996 - Ilene Prokup, Dr.P.H., Epidemiology, pending, Associate Professor of Nursing, Kutztown
University, Pennsylvania
1999 - Mary Beth Terry, Ph.D., Epidemiology. Professor of Epidemiology, Columbia.
2005 - John Doyle, Dr.P.H., Epidemiology, Vice President of Quintiles Consulting

M.S./M.P.H. Advisor (selected)
1983 - Christine Johnsen, M.P.H., Epidemiology
1988 - Salvador Pita, M.P.H., Epidemiology
1990 - Sarah Garrison, M.P.H., Epidemiology
1991 - Jose Guillem, M.P.H., Epidemiology
1992 - Clark Chen, M.S., Epidemiology
1993 - Jason Santos, M.P.H., Epidemiology
1994 - Ghada Sherif, M.S., Epidemiology
1995 - Sungmin Suh, M.S., Epidemiology
1996 - Zareen Khan, M.P.H., Epidemiology
1996 - Greg Hocking, M.P.H. Epidemiology
1997 - David J. Rosenberg, M.P.H., Epidemiology
1997 - John Doyle, M.P.H., Epidemiology
1998 - Beverly Insel, M.P.H., Epidemiology
Alfred I. Neugut
Curriculum Vitae

2001 - Pierre Krakiewicz, M.P.H. Epidemiology
2001 - Susan Sweeney, M.P.H. Epidemiology
2001 - Melissa Carlson, M.P.H. Epidemiology
2009 - Yin Cao, M.P.H. Epidemiology
2009 - Sophie Rousseau, M.P.H., Epidemiology (Ecole Hautes d’Etude Sante Publique, Paris)
2012 –Stephen J. Mooney, M.P.H., Epidemiology

Doctoral Committees
1989 - Robert Macklin, Dr.P.H., Environmental Science
1990 - Wendy Huebner, Ph.D., Epidemiology
1993 - Dale Glasser, Ph.D., Epidemiology
1993 - Immaculata DeVivo, Dr.P.H., Environmental Sciences
1994 - John Luo, Dr.P.H., Environmental Sciences
1996 - Lori Mosca, Dr.P.H., Epidemiology
1997 - Peter Kanetsky, Ph.D. Epidemiology
1998 - Bu Tian Ji, Dr.P.H, Epidemiology
1999 - Emanuela Taioli, Dr.P.H., Epidemiology
2000 - Andrew Rundle, Dr.P.H., Environmental Health Sciences
2000 - Susan Teitelbaum, Ph.D., Epidemiology
2001 - Tamara Do, Ph.D., Environmental Health Sciences
2001 - Joshua Fogel, Ph.D., Clinical Psychology (Yeshiva University)
2001 - Nandita Mitra, Ph.D., Biostatistics
2001 - Joshua Muscat, Ph.D., Epidemiology (New York University)
2002 - Sybil Eng, Ph.D., Epidemiology
2002 - Paul Terry, Ph.D., Epidemiology
2002 - Regina Zimmerman, Ph.D., Epidemiology
2002 - Lydia Zablotska, Ph.D., Epidemiology 2002
2003 - Sandro Galea, Ph.D., Epidemiology
2004 - Julie Kranick, Epidemiology
2005 - Elizabeth Kaufman, Ph.D., Epidemiology
2007 - Sylvia Taylor, Ph.D., Epidemiology,
2007 - Ai Kudo, Ph.D., Epidemiology
2007 - Heather Greenlee, Ph.D., Epidemiology
2010 - Heidi Mochari, Ph.D., Epidemiology
2012 - Russell McBride, Ph.D., Epidemiology
2011 – Meghan Work, Ph.D., Epidemiology, pending
2011 - Joseph Jaeger, Dr.P.H., Epidemiology
2012 - George Friedman-Jimenez. Dr.P.H., Epidemiology
2013 - Dariush Nasrollahzadeh Nesheli, Ph.D., Epidemiology, Karolinska Institute, Sweden
2015 - Gene Pesola, Ph.D., Epidemiology
2015 - Laura Reimers Iadeluca, Ph.D., Epidemiology
2016 - Stephen J. Mooney, Ph.D., Epidemiology
2016 - Nathalie Mehler-Horowicz, Ph.D., Epidemiology

Post-doctoral trainees

<table>
<thead>
<tr>
<th>Name</th>
<th>Years</th>
<th>Institution/Degree/Year</th>
<th>Research Project Title</th>
<th>Current Position/Support</th>
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<tr>
<td>Garrison, Susan</td>
<td>'89-'91</td>
<td>Columbia U., MSPH MPH ‘91 College of Physicians &amp;</td>
<td>Study of cancer among Dominican</td>
<td>Associate Professor of Medicine, Albert</td>
</tr>
</tbody>
</table>
### Alfred I. Neugut

**Curriculum Vitae**

<table>
<thead>
<tr>
<th>Name</th>
<th>Years</th>
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<tbody>
<tr>
<td>Lee, Won Chul</td>
<td>'89-'91</td>
<td>Catholic U., Korea, M, PhD</td>
<td>Risk factors for colorectal adenomas and cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Professor of Preventive Medicine, Catholic U., Korea</td>
</tr>
<tr>
<td>Mosca, Lori</td>
<td>'91-'92</td>
<td>Columbia U., MSPH MPH '92 SUNY at Syracuse, MD '84</td>
<td>Physical activity and colorectal neoplasia</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Professor of Medicine, Columbia U.</td>
</tr>
<tr>
<td>Ahsan, Habibul</td>
<td>'93-'95</td>
<td>U. of Western Australia, M Med Sc ‘92 U of Dhakar, MBBS ‘88</td>
<td>Studies on second malignancies/brain tumors</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Professor of Health Studies, Human Genetics, U. of Chicago</td>
</tr>
<tr>
<td>Sherif, Ghada</td>
<td>'93-'94</td>
<td>Columbia U., MS ‘94 Cairo U., Egypt ’90 M.B.B.Ch.</td>
<td>Coronary artery disease and colorectal cancer</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Epidemiologist, NCI of Egypt</td>
</tr>
<tr>
<td>Grann, Victor R.</td>
<td>'95-'97</td>
<td>Columbia MSPH, MPH ‘97 New York Medical, MD ‘62 Yale, BA</td>
<td>Decision analysis for women at high risk for breast cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Professor of Medicine and Epidemiology emeritus, Columbia U.</td>
</tr>
<tr>
<td>Davidow, Amy</td>
<td>'94-'95</td>
<td>NYU, PhD ‘89 Tufts U, BA ‘80</td>
<td>Development and application of biostatistical methods</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Associate Professor of Preventive Medicine (Biostatistics), UMDNJ</td>
</tr>
<tr>
<td>Rosenberg, David</td>
<td>'94-'97</td>
<td>Columbia U., MSPH MPH ‘97 SUNY Downstate, MD ‘91 CUNY, BA ‘87</td>
<td>Coronary heart disease and diabetes mellitus and the risk of prostate cancer</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Director of Evidence-based Medicine, Associate Professor of Medicine, Hofstra Northwell</td>
</tr>
<tr>
<td>Sharir, Sharon</td>
<td>'97-'99</td>
<td>Columbia U., MSPH MPH ‘99 U. of Toronto, MD’95, BS ‘91</td>
<td>Prognostic significance of p27 and Ki-67 in prostate cancer after prostatectomy</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Associate Professor of Urology, University of Toronto</td>
</tr>
<tr>
<td>Sheinfeld Gorin, Sherri</td>
<td>'97-'99</td>
<td>U. of Michigan, PhD ‘96 U. of Pennsylvania, MS ‘82</td>
<td>Uptake of screening recommendations among physicians</td>
</tr>
</tbody>
</table>
| Alfred I. Neugut  
| Curriculum Vitae  |

| Sundararajan Vijaya | 98–’00 | U. North Carolina, MPH ’96  
| | | U. of Oklahoma, MD ‘89  
| | | U. of Oklahoma, BA ‘85 | Preference of high risk women for prophylactic surgery | Professor of Medicine, University of Melbourne School of Medicine, Melbourne |
| Hershman, Dawn | ’97–’01 | Columbia U., MSPH, MS ‘01  
| | | Albert Einstein , MD ’94  
| | | UCLA, BA ‘87 | Cancer survivorship, racial disparities in chemotherapy for cancer | Professor of Medicine and Epidemiology, Columbia U. |
| Chen, Allen | ’01–’03 | Columbia U., MSPH, MS ‘03  
| | | Columbia U., MD ‘97  
| | | Harvard College, BA ‘93 | PSA levels among Afro-Caribbean immigrant populations | Assistant Professor of Medicine and Assistant Attending Physician, NYU |
| Honda, Keiko | ’02–’05 | NYU., PhD. ‘02  
| | | NYU., MPH ‘99 | Identifying psychosocial pathways to cancer screening behaviors |  |
| Matasar, Matthew | ’03–’05 | Columbia U., MSPH MS ‘05  
| | | Harvard U., MD ‘01  
| | | Harvard U., AB ‘96 | Impact of dose density of 5-FU therapy on survival in colon cancer. | Assistant Professor on Lymphoma Service at MSKCC |
| Crew, Katherine | ’03–’05 | Columbia U., MD , MS ‘05  
| | | Brown University, BS ‘94 | Polyphenon E and breast cancer prevention | Associate Prof of Medicine and Epidemiology, Columbia U. |
| Greenlee, Heather | ’04–07 | Columbia U, MSPH, Ph.D. (P)  
| | | Univ of Washington, MPH ‘03  
| | | Bastyr U., ND ‘99  
| | | University of Washington, BA | Complementary and Alternative Medicine Use among Long Island Breast Cancer Study Cases | Assistant Professor of Epidemiology MSPH, Columbia |
| Link, Lilli B. | ’02– 05 | Cornell U., MS ‘02  
| | | Univ of Chicago, MD.,  
| | | ’94  
| | | Wesleyan U. BA 89 | Hippocrates Follow-up Study - feasibility and effects of a raw food diet | Private practice |
| Zojwalla, Naseem | ’01–’03 | Temple U., MD, ‘98  
<p>| | | Stanford, BA ‘94 | Hormone Receptor Status and Breast Cancer | Pharmaceutical industry |</p>
<table>
<thead>
<tr>
<th>Name</th>
<th>Years</th>
<th>Institution and Degree</th>
<th>Research Focus</th>
<th>Position and Institution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abrams, Julian</td>
<td>'05-07</td>
<td>Mt Sinai MD, '00</td>
<td>Inflamatory liver disease and liver mets.</td>
<td>Assistant Professor of Medicine and Epidemiology, Columbia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>University of Penn., BA, '94</td>
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<tr>
<td>Hall, Michael</td>
<td>'05-06</td>
<td>Columbia BA ’93; MD ’99</td>
<td>Analysis of BRCA and DNA repair data</td>
<td>Associate Professor of Medicine and Clinical Genetics, Fox Chase Cancer Center</td>
</tr>
<tr>
<td></td>
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<td>U of Chicago, MHS, ‘05</td>
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<tr>
<td>Strauss, Joshua</td>
<td>'08-09 ’14-15</td>
<td>Einstein MD ’09; Penn BA ’05</td>
<td>GI cancer treatments and outcomes</td>
<td>Private practice</td>
</tr>
<tr>
<td>Lebwohl, Benjamin</td>
<td>'08-10</td>
<td>Columbia MD ’03, MS ’10</td>
<td>Quality of colonoscopy performance and prep</td>
<td>Assistant Professor of Medicine and Epidemiology, Columbia</td>
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<tr>
<td></td>
<td></td>
<td>Harvard BA ‘99</td>
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<tr>
<td>MacDonald, Alicia</td>
<td>'09-’12</td>
<td>Pittsburgh PhD ’09</td>
<td>Viruses and cancer in HIV patients</td>
<td>Assistant Professor of Medicine and Epidemiology, Columbia</td>
</tr>
<tr>
<td>Lasheen, Wael</td>
<td>'09-’12</td>
<td>Cairo University MBBS ‘95</td>
<td>Palliative care and racial disparities among cancer patients</td>
<td>Case Western Reserve, Research Scientist</td>
</tr>
<tr>
<td>Brouse, Corey</td>
<td>'09-’11</td>
<td>Teachers College EdD ’03; University of New Hampshire BS ’99</td>
<td>Promotion of colonoscopy among the underserved</td>
<td>Associate Professor, Montclair State</td>
</tr>
<tr>
<td>Becker, Daniel</td>
<td>'09-’10</td>
<td>NYU MD ’04; Yale BA ‘00</td>
<td>Health outcomes studies in the treatment of colorectal cancer</td>
<td>Assistant Professor of Clinical Medicine, NYU</td>
</tr>
<tr>
<td>Sharaiha, Reem</td>
<td>’10-’11</td>
<td>University of London MBBS ‘03</td>
<td>Studies on GI tract tumors</td>
<td>Assistant Professor of Gastroenterology, Cornell</td>
</tr>
<tr>
<td>Winner, Megan</td>
<td>’10-’12</td>
<td>Washington University in St. Louis MD ’07; BA ’02</td>
<td>Studies on benign and malignant tumors of the pancreas</td>
<td>Instructor in Surgery, SUNY Stony Brook</td>
</tr>
<tr>
<td>Vin-Raviv, Neomi</td>
<td>’11-’14</td>
<td>University of Haifa PhD ‘11</td>
<td>Studies on PTSD and cancer</td>
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<tr>
<td>Mentor For Career Development</td>
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<td>Mary Beth Terry, Ph.D., K07 - 2001-2006</td>
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<tr>
<td>Dawn L. Hershman, M.D., M.S. - K07 - 2002-2007</td>
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<tr>
<td>Andrew Rundle, Ph.D. - K07 - 2003-2008</td>
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<td>Donna Shelley, M.D., M.P.H. - K01 CDC 2005-2008</td>
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<tr>
<td>Benjamin Spencer, M.D., M.P.H. - DoD Physician Research Training Award - 2006-2011</td>
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<tr>
<td>Michael AJ. Hall, M.D., M.H.S. - ACS Career Development Award - 2007-2012</td>
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<tr>
<td>Katherine Crew, M.D., M.S. - ACS Career Development Award - 2008-2013 (Co-Mentor)</td>
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<tr>
<td>Julian A. Abrams, M.D., M.S. - FAMRI Career Development Award - 2008-2011;K07-2008-2013</td>
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<tr>
<td>Rose Lai, M.D., M.Sc. – K07 – 2008-2013</td>
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<tr>
<td>Mary Beth Terry, Ph.D., K07 - 2001-2006</td>
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<tr>
<td>Dawn L. Hershman, M.D., M.S. - K07 - 2002-2007</td>
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<tr>
<td>Andrew Rundle, Ph.D. - K07 - 2003-2008</td>
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<tr>
<td>Donna Shelley, M.D., M.P.H. - K01 CDC 2005-2008</td>
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<tr>
<td>Benjamin Spencer, M.D., M.P.H. - DoD Physician Research Training Award - 2006-2011</td>
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<td>Michael AJ. Hall, M.D., M.H.S. - ACS Career Development Award - 2007-2012</td>
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<tr>
<td>Katherine Crew, M.D., M.S. - ACS Career Development Award - 2008-2013 (Co-Mentor)</td>
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<tr>
<td>Julian A. Abrams, M.D., M.S. - FAMRI Career Development Award - 2008-2011;K07-2008-2013</td>
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<td>Rose Lai, M.D., M.Sc. – K07 – 2008-2013</td>
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<tr>
<th>Name</th>
<th>Time</th>
<th>Institution(s)</th>
<th>Research Interest(s)</th>
<th>Position</th>
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<tr>
<td>Khanna, Lauren</td>
<td>‘12-‘14</td>
<td>Harvard MD ‘07; Princeton BA ‘02</td>
<td>Disparities in pancreatic cancer</td>
<td>Assistant Professor of Gastroenterology, NYU</td>
</tr>
<tr>
<td>Oberstein, Paul</td>
<td>‘12-‘13</td>
<td>Ohio State MD ‘06; University of Maryland BA ‘02</td>
<td>Translational studies in pancreatic cancer</td>
<td>Assistant Professor of Medicine, Columbia</td>
</tr>
<tr>
<td>Tsui, Jennifer</td>
<td>‘12-‘14</td>
<td>UCLA PhD ’12; Columbia MPH ’05; UC Berkeley BA ’03</td>
<td>Insurance and health outcomes studies</td>
<td>Assistant Professor of Public Health, Robert Wood Johnson</td>
</tr>
<tr>
<td>Sardo Molmenti, Christine</td>
<td>‘13-‘15</td>
<td>Arizona PhD ’13; Minnesota MPH ’94; Ohio State BA ’92</td>
<td>Diet, obesity, chemoprevention, colorectal cancer</td>
<td>Assistant Professor of Public Health, Hofstra</td>
</tr>
<tr>
<td>Kahn, Justine</td>
<td>‘15-</td>
<td>Mt. Sinai MD ’10; Barnard BA ’05</td>
<td>Pediatric cancer and adherence, SES</td>
<td>Instructor in Pediatric Oncology</td>
</tr>
<tr>
<td>Leng, Siyang</td>
<td>‘16-</td>
<td>SUNY Downstate MD ’09; Cornell BS ’05</td>
<td>Health outcomes for myeloma and plasma cell dyscrasias</td>
<td>Instructor in Medical Oncology</td>
</tr>
<tr>
<td>Joffe, Lenat</td>
<td>‘16-</td>
<td>Sackler MD ’10; NYU BA ’06</td>
<td>Outcomes research in pediatric oncology</td>
<td>Pediatric oncology fellow</td>
</tr>
<tr>
<td>Kwon, Sung (Steve)</td>
<td>‘16-</td>
<td>Washington MPH ’11; Illinois MD ’07; Illinois BS ’03</td>
<td>Outcomes research in surgical oncology</td>
<td>Postdoctoral fellow Epidemiology</td>
</tr>
<tr>
<td>O’Neil, Daniel</td>
<td>‘17-</td>
<td>Harvard MPH ’16; Einstein MD ’12; Cornell BS ’06</td>
<td>Outcomes and global research</td>
<td>Medical Oncology Fellow</td>
</tr>
</tbody>
</table>
Alfred I. Neugut
Curriculum Vitae

Daniel Becker, M.D. – ASCO Young Investigator Award – 2010-2011
Rachel Shelton, Ph.D. – KL2 – 2012-2014; ACS Career Development Award – 2013-2018
Benjamin Lebwohl, M.D., M.S.-KL2 - 2011-2013; AGA Research Scholar Award 2014-2017
Paul E. Oberstein, M.D. – ASCO Young Investigator Award, 2013-2014
Elena Ladas, Ph.D.- ACS Career Development Award 2015-2019
Pamela Valera, Ph.D. – K22 – 2015-2018
Daniel Freedberg, MD – KL2 – 2015-2017 (Co-mentor)

OTHER PROFESSIONAL ACTIVITIES:

Editorial Boards

Editorial Advisory Board, Cancer Epidemiology, Biomarkers, and Prevention, 1990-1998; Associate Editor, 1998-2006.
Editorial Board, Integrative Cancer Therapies, 2002-Present.
Editorial Board, Section Chief for Epidemiology and Prevention, Cancer Investigation, 2005-.

Manuscript Reviewer


Committees

Federal
NCI, Board of Scientific Advisors, ad hoc Cancer Control Program Review Group, 1996-1997.
FDA, Center for Devices and Radiological Health, Orthopedic and Rehabilitation Devices Panel, ad hoc Orthopedics Spinal Device Panel, July 2010.
NCORP Minority/Underserved site representative to the NCI Cancer Care Delivery Research Steering Committee. 2015-

State/Local
New York State Department of Health, Commissioner’s Expert Advisory Panel on
Alfred I. Neugut
Curriculum Vitae

New York State Comprehensive Cancer Control Data and Surveillance Goal
New York City Department of Health and Mental Hygiene, Citywide Colorectal Cancer
Control Coalition, Co-Chair of Research Committee, 2003-.

Study Sections
NCI, Special Review Committee for Yale CPRU, August 1988.
NCI, Special Review Committee for Chemoprevention Trials RFA, March 1989.
NCI, Special Review Committee for Program Project Grant, February 1990.
ACS, Scientific Advisory Committee on Clinical and Cancer Control Investigations I - Epidemiology,
Diagnosis and Therapy, 1992.
National Cancer Institute, Site Visit Team for New York University Program Project Grant, May 1994.
National Cancer Institute, Site Visit Team for Wake Forest Cancer Center, October 1994.
National Cancer Institute, Special Emphasis Panel, Nutrition Study Section, October 1994.
National Cancer Institute, Special Review Committee for RFA 95-CA-004 on Breast
National Cancer Institute, Special Review Committee for RFA 95-CA-18 on Cancer Prevention
Research Units, March 1996.
National Cancer Institute, Site Visit Team for UCLA Program Project Grant,
February 1997.
National Cancer Institute, Ad Hoc Member of Epidemiology and Disease Control-2
Study Section, June 1997.
National Cancer Institute, Special Review Committee for RFA 97-CA-004 on Cancer G Genetics
National Cancer Institute, Ad hoc member of Scientific Review Committee E, July 1998.
California Cancer Research Program, member of Biomedical Study Section,
October 1998.
Middle East Cancer Consortium Small Grants Program, reviewer,
National Cancer Institute, Ad hoc member of Scientific Review Group E, April 1999,
December 1999.
National Cancer Institute, Cancer Genetics Network Pilot Projects Review
Team, Chair, May 1999.
California Cancer Research Program, member of Epidemiology Study Section,
February 2000.
National Cancer Institute, Site Visit Team for Norris Comprehensive Cancer Center, University of
Southern California, June 2000.
National Cancer Institute, Ad hoc member of Scientific Review Group SNEM-3, August 2000.
National Cancer Institute, Ad hoc member of Scientific Review Group SNEM-2, November 2000.
National Cancer Institute, Special Emphasis Panel for SNEM-1, Chair, April 2001.
California Cancer Research Program, member of Epidemiology Study Section, January 2002.
National Cancer Institute, Special Emphasis Panel for SNEM-5, Member, March 2002.
National Cancer Institute, Special Emphasis Panel for SNEM-4 Member, April 2002.
National Cancer Institute, Member of Epidemiology and Disease Control-2 Study Section,
National Cancer Institute, Site Visit Team for Norris Comprehensive Cancer Center, University of
Southern California, May 2005.
Alfred I. Neugut
Curriculum Vitae

American Cancer Society, Member of Cancer Control and Prevention Peer Review Committee in Psychosocial and Behavioral Research, January 2006-December 2010.
National Cancer Institute, Site Visit Team for Yale University Cancer Center, October 2006.
National Cancer Institute, Member of Special Emphasis Panel for PAR 07-230, Minority Institutions/ Cancer Center Initiatives, June 2007.
National Cancer Institute, Member of Special Emphasis Panel/Scientific Review Group 2009/10 ZRG1 PSE-J for RFA OD 09-003, Challenge grants Panel, June 2009.
Department of Defense, Congressionally Directed Medical Research Programs, Prostate Cancer Research Program Peer Review, 2009-2013.
National Cancer Institute, Member of Special Emphasis Panel for PAR Cancer Health Disparities ZRG1 OBT-Z, March 2010.
National Institutes of Health, Member of Review Panel for Director’s Opportunity 5 Theme Hematology and Cardiovascular-Respiratory Sciences, (OD-10-005), June 2010.
National Institutes of Health, Site Visit Team for Norris Comprehensive Cancer Center, University of Southern California, June 2010.
National Institutes of Health, Site Visit Team for Helen Diller Family Comprehensive Cancer Center, University of California San Francisco, January 2012.
National Cancer Institute, Member of Special Emphasis Panel for PAR 11-156 Cancer Health Disparities and Diversity in Basic Cancer Research ZRG1 OBT-A (55) R, March 2012.
National Institutes of Health, Member of Special Emphasis Panel for PAR-10-278 Global Research Initiative program, AIDS and AIDS-Related Research ZRG1 AARR-K (95) S, August 2012.
National Cancer Institute, Member of Subcommittee I – Transition to Independence, June 2014.
National Institutes of Health, Member of Special Emphasis Panel/Scientific Review Group Neurological, Aging and Musculoskeletal Epidemiology Committee, October 2014.
National Cancer Institute, Member of Special Emphasis Panel for PAR-13-081 2015/05 ZCA1 SRB(M3)-S Bridging the Gap between Cancer Mechanism and Population Science, January 2015
National Institutes of Health, Site Visit Team for Norris Comprehensive Cancer Center, University of Southern California, June 2015.
National Institutes of Health, Site Visit Team for Moffitt Cancer Center, Tampa FL, May 2016.

Private/Foundation
CHEMOcare, Board of Trustees, 1991-Present.
Institute of Medicine, National Academy of Sciences, Committee for Review of the Health Effects on Vietnam Veterans of Exposure to Herbicides; Chairman, Cancer Subcommittee, 1992-1993.
American Society of Preventive Oncology, Secretary-treasurer, 1994-1999.
Member, Southwest Oncology Group, 1994-Present.
American Society of Preventive Oncology, Program Planning Committee for 1996 Meeting.
Member, Expert Panel for Prostate Cancer as a Cause of Death Project. Harlem Hospital Center, 1995.
Alfred I. Neugut
Curriculum Vitae

Member, Review Committee of American Health Foundation Activities, 1995.
American Society of Clinical Oncology, 1997 Annual Meeting Program Planning Committee.

Member, Southwest Oncology Group Gastrointestinal Committee, 1997-Present.
Member, Southwest Oncology Group Cancer Control Committee, 1998-Present.

Israel Science Foundation, grant reviewer, 2001.
Member, Medical Advisory Board, Executive Health Group, New York, 2001-Present.
American Society of Clinical Oncology, Cancer Education Committee, Cancer Prevention/Epidemiology Track Team, 2003-2006.
American Association of Cancer Research, member of 2004 Cancer Epidemiology and Prevention Awards Committee.
American Association of Cancer Research, member of 2004 Clinical Research/Prevention Abstract Review Committee.
Member, Southwest Oncology Group Health Disparities & Outcomes Committee, 2009-Present.
Member, Southwest Oncology Group Cancer Survivorship Committee, 2009-Present.
Member, India Spreading Wellness and Prevention (SWAP) Advisory Board, Ortho-Clinical Diagnostics, 2009.
American Society of Clinical Oncology, Test Materials Development Committee for National Medical Oncology In-Training Examination, 2009-2011.
Member, Advisory Board, Mouse Models of Human Cancers Consortium, HICCC, 2009-2014.
Member, Medullary Thyroid Carcinoma Registry Consortium-Registry Data Monitoring Committee, 2014-
Member, Otsuka Pharmaceuticals Global Pharmacovigilance Advisory Committee, 2015-
Member, Pfizer Data Generation Advisory Board, 2015-
Grant reviewer, Dutch Cancer Society, 2016.
American Society of Preventive Oncology, 2017 Annual Meeting Program Planning Committee Member
American Society of Preventive Oncology, 2018 Annual Meeting Program Planning Committee Member

International

Cancer Research Campaign, Clinical Trials Committee, grant reviewer, May 2000.
Dutch Cancer Society, grant reviewer, August 2004.
Cancer Research UK, Program Grant Reviewer, September 2009.
Prostate Cancer Foundation of Australia, grant reviewer, September 2009
Alfred I. Neugut
Curriculum Vitae


Chair, Track Committee for Cancer Care and Survivorship, UICC World Cancer Congress, Montreal Canada, August 2012

French National Cancer Institute, Hospital Clinical Research program 2012, grant reviewer, February 2012

Karolinska Institutet, Department of Medical Epidemiology and Biostatistics, external reviewer for faculty selection committee, November 2013.

Workshop on Cancer Epidemiology, Nelson R. Mandela School of Medicine, University of KwaZuluNatal, Durban, South Africa, November 2013

Other

External Consultant, Ohio State University Comprehensive Cancer Center, Columbus, Ohio, October 1999.

Member, Data Safety Monitoring Board: Randomized Double-Blind, Placebo-Controlled Study of the Efficacy and Safety of Celecoxib in the Prevention of Colorectal Sporadic Adenomatous Polyps (PreSAP), Pfizer, 2001-2006.


Member, Program Steering Committee, U56 Partnership between University of Massachusetts-Boston and Dana-Farber/Harvard Cancer Center, 2006-.


Member, Program Steering Committee, U56 Partnership between University of the District of Columbia and Lombardi Comprehensive Cancer Center, 2007-.

Member, External Scientific Advisory Committee, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, 2008-.

Member, Scientific Advisory Committee, Population Science Research Program, NYU Langone Medical Center, New York, July 2010.

Member, Mock Site Visit Team, UAB Comprehensive Cancer Center, Birmingham AL, September 2010.

Member, External Advisory Board, Center of Excellence in Disparities Research and Community Engagement (CEDREC), Weill Cornell Medical College, 2010-.

Member, External Advisory Committee, University of Kansas Cancer Center, University of Kansas, 2011-.

Consultant, Population Sciences Programs, Moores Cancer Center, University of California San Diego, February 2013.

Member, External Review Committee, Graduate Programs in Epidemiology and Biostatistics, Perelman School of Medicine, University of Pennsylvania, Philadelphia PA, April 2013.

Consultant, Population Sciences Programs, University of Maryland Greenbaum Cancer Center, MD, 2014-

Honorary Police Surgeon, New York Police Department, New York NY, 2015-

Consultant, Cancer Prevention and Control Program, Wake Forest Baptist Comprehensive Cancer Center, November 2015, February 2016 mock site visit.
Alfred I. Neugut
Curriculum Vitae

PUBLICATIONS:
Peer-Reviewed


Alfred I. Neugut  
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Curriculum Vitae


Zacharia BE, Goldstein H, Bruce SS, Malone HR, Neugut AI, Bruce JN. Incidence, treatment and survival of patients with craniopharyngioma in the Surveillance, Epidemiology and End Results Program. Neuro Oncol 14:1070-1078, 2012.


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Curriculum Vitae


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fogger trucks and breast cancer incidence in the Long Island Breast Cancer Study Project (LIBCSP). Env
Health 12-24, 2013.

372. Oberstein PE, Hershman DL, Khanna L, Chabot JA, Insel BJ, Neugut AI. Uptake and patterns of use of

373. Wright JD, Deutsch I, Wilde ET, Ananth CV, Neugut AI, Lewin SN, Siddiq Z, Herzog TJ, Hershman DL.
Uptake and outcomes of intensity-modulated radiation therapy for uterine cancers. Gynecol Oncol 130:43-
48, 2013.

374. Wright JD, Neugut AI, Lewin SN, Lu YS, Herzog TJ, Hershman DL. Trends in hospital volume and

375. Cubasch H, Joffe M, McCormack V, Hanisch R, Neugut AI, Karstaedt A, Browse N, van den Berg E,
Schuz J, Jacobson JS. Breast cancer characteristics and HIV among 1092 women in Soweto, South Africa.

376. Clarke Hillyer G, Hershman DL, Kushi LH, Lamerato L, Ambrosone CB, Bobjerg DH, Fu OS, Rana S,
Mandelblatt JS, Neugut AI. A survey of breast cancer physicians regarding patient involvement in breast

Prognostic significance of mucinous differentiation of endometrioid adenocarcinoma of the endometrium.

378. Wright JD, Herzog TJ, Tsui J, Ananth CV, Lewin SN, Lu YS, Neugut AI, Hershman DL. Nationwide

379. Lebwohl B, Granath F, Ekbom A, Smedby KE, Murray JA, Neugut AI, Green PHR, Ludvigsson JF.
Mucosal healing and risk of lymphoproliferative malignancy in celiac disease. Ann Int Med 159:169-175,
2013.

380. Link AR, Gammon MD, Jacobson JS, Abrahamson P, Bradshaw P, Terry MB, Teitelbaum S, Neugut AI,
Greenlee H. Use of self-care and practitioner-based forms of complementary and alternative medicine

381. Bashir S, Ananth CV, Lewin SN, Burke WM, Lu YS, Neugut AI, Herzog TJ, Hershman DL, Wright JD.
Utilization and safety of sodium hyaluronate-carboxymethylcellulose (HA-CMC) adhesion barrier. Dis Col

382. Winner M, Mooney SJ, Hershman DL, Feingold DL, Allendorf JD, Wright JD, Neugut AI. Incidence and
predictors of bowel obstruction in stage IV colon cancer patients: a population-based cohort study. JAMA

383. Hershman DL, Wright JD, Lim E, Buono DL, Tsai WY, Neugut AI. Contraindicated use of bevacizumab
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Curriculum Vitae


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410. Neugut AI, Hillyer GC, Kushi LH, Lamerato L, Leoce N, Ambrosone CB, Bovbjerg DH, Mandelblatt JS, Hershman DL. Non-initiation and early discontinuation of adjuvant trastuzumab in women with localized
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515. Wright JD, Herzog TJ, Neugut AI, Burke WM, Lu YS, Lewin SN, Hershman DL. Comparative effectiveness of minimally invasive radical hysterectomy for cervical cancer. Submitted for publication.


518. Wright JD, Neugut AI, Tergas A, Lewin SN, Burke WM, Lu YS, Herzog TJ, Hershman DL. Surveillance testing in women following a diagnosis of early-stage endometrial cancer. Submitted for publication.


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525. Wright JD, Chen L, Burke WM, Hou JY, Tergas AI, Hu JC, Neugut AI, Ananth CV, Hershman DL. Trends in use and outcomes of women undergoing hysterectomy with electric power morcellation. Submitted for publication.


532. Onishi M, Vasan S, Accordino M, Hillyer GC, Neugut AI, Wright JD, Hershman DL. Factors associated with neurokinin-1 receptor antagonist use among commercially insured cancer patients receiving highly emetogenic chemotherapy. Submitted for publication.

533. Youngerman BE, Neugut AI, Yang J, Hershman DL, Wright JD, Bruce JN. The modified frailty index and 30-day adverse events in oncologic neurosurgical oncology. Submitted for publication.


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540. Wright JD, Chen L, Hou JY, Burke WM, Tergas AI, Ananth CV, Neugut AI, Hershman DL. Impact of hospital volume and quality of care on survival for ovarian cancer. Submitted for publication.

541. Wright JD, Chen L, Gabor L, Burke WM, Tergas AI, Hou JY, Ananth CV, Neugut AI, Hershman DL. Trends in specialty-based referral and outcomes for women with endometrial cancer undergoing hysterectomy. Submitted for publication.


545. Accordino MK, Wright JD, Vasan S, Neugut AI, Gross T, Hillyer GC, Hershman DL. Association between time alive with metastatic breast cancer and aggressive end-of-life care. Submitted for publication.


547. Neugut AI, MacLean SA, Dai WF, Jacobson JS. Impact of physician characteristics on decisions regarding cancer screening: a systematic review. Submitted for publication.

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549. Parada H, Jr., Gammon MD, Chen J, Calafat AM, Neugut AI, Santella RM, Wolff MS, Teitelbaum SL. Urinary phthalate concentrations and breast cancer incidence and survival following breast cancer. Submitted for publication.


Case Reports


Invited Reviews


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Book Chapters


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Editorials.


624. Neugut AI, Abi-Rached B. Lessons from a follow-up study of large colorectal adenomas: BE or not BE, that is the question. Am J Gastroenterol 91:420-422, 1996.
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636. Hall MJ, Neugut AI. Only women with specific family histories should be referred for counseling or evaluation for BRCA breast and ovarian cancer susceptibility testing. ACP J Club 144 (Mar-April):3; 2006.


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Books

Letters:


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INVITED PRESENTATIONS:


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10. Cancer screening, stage, and impact on survival. Third Annual Cancer Symposium for Physicians, Crozer Regional Cancer Center, Upland, PA, October 1993.
27. Recurrence of adenomatous polyps. Medical Grand Rounds, Ichilov Hospital, Tel Aviv, Israel, June 1998.
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35. Use of large administrative databases for answering questions in cancer etiology and treatment. Faculty Research Seminar, Department of Medicine, Columbia-Presbyterian Medical Center, New York NY, February, 2001.
39. Use of large data sets in cancer research. Team Rounds, Department of Medicine, Columbia Presbyterian Medical Center, New York, May 2001.
43. Using large-scale databases for policy making in cancer prevention. Ruttenberg Cancer Center, Mt. Sinai School of Medicine, New York, NY, December 2001.
44. Risk factors for colorectal neoplasia. International Meeting: GI Maligancies can be Presented and Treated from the Bench to the Bedside. Dead Sea, Israel, January 2002.
50. Screening for breast and prostate cancer. First Annual Dr. Paul A. Marks Oncology Symposium, New Milford, CT, May 2003.
51. Epidemiology and Screening for colorectal cancer. Northeast Regional Caner Institute, University of Scranton, Scranton, PA, June 2003.
54. Use of large datasets in cancer research. Department of Epidemiology and Social Medicine, Albert Einstein College of Medicine, Bronx, NY, December 2003.
55. Update on Long Island Breast Cancer Study. Grand Rounds, Department of Medicine, Winthrop University Hospital, Mineola, NY, February 2004.
56. Second malignancies in the etiology of GI tract cancers. Frontiers in Gastroenterology and Hepatology, Division of Gastroenterology, Case Western Reserve University, School of Medicine, Cleveland, Ohio, April 2004.
64. Racial disparities in adjuvant therapy for breast cancer. Division of Oncology, University of Chicago, Chicago IL, April 2005
65. Use of epidemiologic methods to study issues in oncology, NYU Cancer Institute Seminar, NYU Medical Center, May 2005.
68. Racial disparities in cancer treatment. Department of Medicine Grand Rounds, UMDNJ-New Jersey Medical School, Newark, NJ, March 2006.
70. NSAIDS and breast and ovarian cancer. The Emerging Role of Screening and Prevention in Women’s Cancers, NYU School of Medicine, New York, NY, May 2006.
75. Quality of Care in Cancer Treatment. Department of Medicine Grand Rounds, Mary Imogene Bassette Hospital, Cooperstown NY, December 2009.
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76. Preparation of a K award. Workshop on Career development for Junior Faculty, Junior Researchers, and Trainees. 33rd Annual Meeting of the American Society of Preventive Oncology, Tampa FL, March 2009.
80. Quality of Care in Cancer Treatment. Oncology Grand Rounds, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, Washington DC, July 2009.
85. Getting ahead of the curve through early detection. 8th Annual Brain Tumor Awareness Day Conference, Brain Tumor Foundation, New York NY, November 2009.
92. Controversies in cancer screening. Department of Pathology, Faculty of Health Sciences, Wits Medical School, University of Witswatersrand, Johannesburg, South Africa, February 2011.
93. Workshop in cancer epidemiology and methodology. Department of Medicine, Faculty of Health Sciences, Tygerberg Hospital, University of Stellenbosch Medical School, Cape Town, South Africa, February 2011.
94. Cancer Epidemiology Workshop, Medical Education Partnership Initiative, Nelson R. Mandela School of Medicine, University of KwaZuluNatal, Durban, South Africa, March 2011.
96. Utility of observational and health outcomes studies for pharmaceutical issues. Real World and Non-Interventional Study Preceptor Meeting, Pfizer, La Jolla CA, November 2011.
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110. Selected cases in malpractice and lessons to be learned. GI Grand Rounds, Columbia University Medical Center, New York NY, September 2013.
111. Risk Factors for Esophageal Cancer. Department of Medical Epidemiology and Bistatistics, Karolinska Institute, Stockholm, Sweden, October 2013.
115. Lesotho and Swaziland. HICCC Cancer Center Retreat, New York NY October 2014.
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120. Creating successful population science programs in a cancer center. Annual Meeting of the American Society of Preventive Oncology, Columbus Ohio, March 2016.


Attachment B
Attachment B:

Page v. Crombie M.D., No. CV2013105090 (Ohio Com.Pl.)

Booker v. Macneal Hospital, No. 11 L 10545 (Ill.Cir.Ct.)

Collar v. R.j. Reynolds, No. 31-2011-CA000115 (Fla.Cir.Ct.)

Skolnik v. R. J. Reynolds, No. 09-4045 (Fla. Cir.Ct.)

Pijuan, et al. v. R.J. Reynolds, et al., Case No. 10-8359 (Fla.Cir.Ct.)


Kristufek v. Takeda Pharmaceuticals America, Inc. Court of Common Pleas of Pennsylvania, Philadelphia County, No. 1207002275


Myers v. Takeda Pharmaceuticals America, Inc., Circuit Court of West Virginia, Berkeley County, No. 13-C-315.