

# **Exhibit 11**

**UNITED STATES DISTRICT COURT  
NORTHERN DISTRICT OF CALIFORNIA**

IN RE: ROUNDUP PRODUCTS  
LIABILITY LITIGATION

MDL No. 2741

Case No. 16-md-02741-VC

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<sup>1</sup> 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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<sup>1</sup> 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

Meta-Analyses	Pooled Analyses
Generally no contact with original study	Investigators of each study agrees to participate
Retrieve publication and extract data of interest -Study design -Population -Exposure, confounders -Risk estimates, confidence intervals	Obtain primary data -Outcomes -Exposures -Confounders
Check data abstracted for errors	Check primary data for errors
Differences in exposure, covariates, and contrasts across studies	Calculate risk estimates from primary data
	More standardized definitions for exposures, covariates, and contrasts across studies
	Standardize formatting of data
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 Carolina 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*, 9<sup>th</sup> 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  $\times$  days/year, categorized in tertiles among users: 1-20, 21-56, 57-2,678); and (c) intensity-weighted cumulative exposure days (years of use  $\times$  days/year  $\times$  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 elevated 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 lymphohaematopoietic 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, Carlberg, & 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, while 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 sprayed 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; Mladinic, 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 (Mladinic, 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, IC<sub>50</sub>) for 18 hours increased production of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) 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- $\alpha$  and IL-1  $\beta$  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 & Shukla, 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. Temporal: 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, Mc Duffie (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

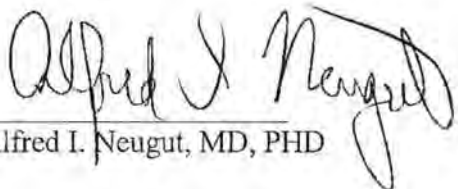
  
Alfred I. Neugut, MD, PHD



TABLE 1. OBSERVATIONAL HUMAN STUDIES OF GLYPHOSATE EXPOSURE AND NHL (Adapted from IARC Monograph 112)

Author	Year	Title	Study Design	Subjects	Location/ Enrollment Period	Exposure	Outcome	Risk Estimate (95% CI)
Cantor et al.	1992	Pesticides and other agricultural risk factors for non-Hodgkins lymphoma among men in Iowa and Minnesota	Case-Control	Cases: 622 (response rate, 89.0%); Iowa health registry records and Minnesota hospital and pathology records Controls: 1245 (response rate, 76–79%); population-based; no cancer of the lympho-haematopoietic system; frequency-matched to cases by age (5-year group), vital status, state. Random-digit dialing (aged < 65 years); Medicare records (aged ≥ 65 years); state death certificate files (deceased subjects) Exposure assessment method: questionnaire; in-person interview	Iowa and Minnesota, USA 1980–1982	Ever handled glyphosate	NHL	1.1 (0.7–1.9)
McDuffie et al.	2001	Non-Hodgkins Lymphoma and specific pesticide exposures in men cross-Canada study of pesticides and health	Case-Control	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	Canada 1991–1994	Exposed to glyphosate Unexposed >0 and ≤ 2 days >2 days	NHL	1.2 (0.83–1.74) 1 1.0 (0.63–1.57) 2.12 (1.2–3.73)

Hardell et al.	2002	Exposure to pesticides as risk factor for non-Hodgkins lymphoma and hairy cell leukemia: pooled analysis of two Swedish case-control studies	Case-Control	Cases: 515 (response rate, 91% in both studies); Swedish cancer registry Controls: 1141 (response rates, 84% and 83%); national population registry Exposure assessment method: questionnaire	Sweden; four Northern counties and three counties in mid Sweden 1987-1992	Ever glyphosate exposure (univariate) Ever glyphosate exposure (multivariate)	NHL and HCL	3.04 (1.08-8.5) 1.85 (0.55-6.2)
De Roos et al.	2003	Integrative assessment of multiple pesticides as risk factors for non-Hodgkins lymphoma among men	Pooled Case-Control	Cases: 650 (response rate, 74.7%); cancer registries and hospital records Controls: 1933 (response rate, 75.2%); random-digit dialing, Medicare, state mortality files Exposure assessment method: questionnaire; interview (direct or next-of-kin)	Nebraska, Iowa, Minnesota, Kansas, USA 1979-1986	Any glyphosate exposure	NHL	2.1 (1.1-4.0)
Lee et al.	2004	Non-Hodgkins lymphoma among asthmatics exposed to pesticides	Case-Control	Cases: 872 (response rate, NR); diagnosed with NHL from 1980 to 1986 Controls: 2381 (response rate, NR); frequency-matched controls Exposure assessment method: questionnaire; information on use of pesticides and history of asthma was based on interviews	Iowa, Minnesota and Nebraska, USA 1980-1986	Exposed to glyphosate - non-asthmatics / Exposed to glyphosate - asthmatics	NHL	1.4 (0.98-2.1) 1.2 (0.4-3.3)



De Roos et al.	2005	Cancer incidence among glyphosate-exposed pesticide applicators in the Agricultural Health Study	Prospective Cohort	54 315 (after exclusions, from a total cohort of 57 311) licensed pesticide applicators Exposure assessment method: questionnaire; semi-quantitative assessment from self-administered questionnaire	Iowa and North Carolina, USA 1993–2001	Ever use 1–20 21–56 57–2678 Trend-test P value 0.73	NHL	1.1 (0.7–1.9) 1 (ref.) 0.7 (0.4–1.4) 0.9 (0.5–1.6)
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Eriksson et al.	2008	Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis	Case-Control	Cases: 910 (response rate, 91%); incident NHL cases were enrolled from university hospitals Controls: 1016 (response rate, 92%); national population registry Exposure assessment method: questionnaire	Sweden. Four health service areas (Lund, Linköping, Örebro and Umeå) 1999–2002	Any glyphosate Any glyphosate* <=10 days per year use >10 days per year use  1–10 years >10 years B-cell lymphoma Lymphocytic lymphoma/B-CLL Diffuse large B-cell lymphoma Follicular grade I–III Other specified B-cell lymphoma Unspecified B-cell lymphoma T-cell lymphoma Unspecified NHL	NHL	2.02 (1.1–3.71) 1.51 (0.77–2.94)  1.69 (0.7–4.07) 2.36 (1.04–5.37) 1.11 (0.24–5.08) 2.26 (1.16–4.4)  1.87 (0.998–3.51)  1.63 (0.53–4.96) 1.47 (0.33–6.61)  2.29 (0.51–10.4)  5.63 (1.44–22)
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Orsi et al.	2009	Occupational exposure to pesticides and lymphoid neoplasms among men: results of a French case-control study	Case-Control	<p>Cases: 491 (response rate, 95.7%); cases (244 NHL; 87 HL; 104 LPs; 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</p> <p>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</p> <p>Exposure assessment method: questionnaire</p>	France 2000–2004	Any glyphosate exposure	NHL	1.0 (0.5-2.2)
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Cocco et al.	2013	Lymphoma risk and occupational exposure to pesticides: results of the Epilymph study	Case-Control	<p>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</p> <p>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</p> <p>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</p>	Czech Republic, France, Germany, Italy, Ireland and Spain 1998-2004	Occupational exposure to glyphosate	B-cell lymphoma	3.1 (0.6-17.1)
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Schinasi et al.

2014 Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis

Meta-Analysis 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)

NHL

1.5 ( 1.1-2.0)

Chang et al.

2016 Systematic review and meta-analysis of glyphosate exposure and risk of lymphohematopoietic cancers

Meta-Analysis

LHC incl NHL

meta-RR = 1.3  
(1.0-1.6)  
meta-RR = 1.4  
(1.0-1.9)  
meta-RR = 1.1  
(0.7-1.6)

Multiple Myeloma

HL

TABLE 2. ANIMAL EXPERIMENTS REGARDING CARCINOGENICITY OF GLYPHOSATE (Cited in IARC Monograph 112)

Author	Year(s)	Title	Species, strain (sex) Duration	Dosing regimen, Animals/groups at start	For each target organ: incidence (%) and/or multiplicity of tumors	Significance
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EPA 1985a, b, 1986, 1991a	Glyphosate; EPA Reg.#: 524-308; Mouse oncogenicity study. Document No. 004370.  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) 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%)  Renal tubule adenoma or carcinoma (combined): 1/49 (2%), 0/49, 1/50 (2%), 3/50 (6%)	P for trend = 0.016; see comments          [NS]       [P=0.037; Cochran-Armitage trend test]    [P=0.034; Cochran-Armitage trend test]
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JMPR	2006	Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004: toxicological evaluations. Report No. WHO/PCS/06.1. Geneva	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
						NS



George et al.	2010	Studies on glyphosate-induced carcinogenicity in mouse skin: a proteomic approach.	Mouse, Swiss (M) 32 wk	Initiation-promotion study Skin application of glyphosate-based formulation (glyphosate, 41%; POEA, ~15%) (referred to as "glyphosate") dissolved in 50% ethanol; DMBA dissolved in 50% ethanol, and TPA dissolved in 50% acetone, used in the groups described below 20 M/group	Skin tumors [called "papillomas" by the authors, following gross examination only]	*P < 0.05 vs groups VI and VII
				Group I: untreated control (no treatment)	Group I: 0/20	
				Group II: glyphosate only: 25 mg/kg bw topically, 3 × /wk, for 32 wk	Group II: 0/20	
				Group III: single topical application of DMBA, 52 µg/mouse, followed 1 wk later by TPA, 5 µg/mouse, 3 × /wk, for 32 wk	Group III: 20/20*, 7.8 ± 1.1	
				Group IV: single topical application of glyphosate, 25 mg/kg bw, followed 1 wk later by TPA, 5 µg/mouse, 3 × /wk, for 32 wk	Group IV: 0/20	
				Group V: 3 × /wk topical application of glyphosate, 25 mg/kg bw, for 3 wk, followed 1 wk later by TPA, 5 µg/mouse, 3 × /wk, for 32 wk	Group V: 0/20	*P < 0.05 vs group VI
				Group VI: single topical application of DMBA, 52 µg/mouse	Group VI: 0/20	
				Group VII: topical application of TPA, 5 µg/mouse, 3 × /wk, for 32 wk	Group VII: 0/20	
				Group VIII: topical application of TPA, 5 µg/mouse, 3 × /wk, for 32 wk	Group VIII: 8/20*, 2.8 ± 0.9	



Group VIII: single topical  
application of DMBA, 52  
 $\mu\text{g}/\text{mouse}$ , followed 1 wk later  
by topical treatment with  
glyphosate, 25 mg/kg bw, 3  $\times$   
/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 × 10 <sup>-8</sup> % (glyphosate, 5.0 × 10 <sup>-5</sup> mg/L), 0.09% (glyphosate, 400 mg/L) or 0.5% (glyphosate, 2.25 × 103 mg/L), ad libitum, for 24 mo 10 M and 10 F/group (age, 5 wk)	<p>Males</p> <p>No significant increase in tumor incidence observed in any of the treated groups</p> <p>NS</p> <p>*[P&lt;0.05]</p> <p>Females</p> <p>Mammary tumors (mainly fibroadenomas and adenocarcinomas): 5/10 (50%), 9/10 (90%), 10/10 (100%)*, 9/10 (90%)</p> <p>NS</p> <p>Pituitary lesions (hypertrophy, hyperplasia, and adenoma): 6/10 (60%), 8/10 (80%), 7/10 (70%), 7/10 (70%)</p>
Chrucielska et al.	2000	Glyphosate - Evaluation of chronic activity and possible far-reaching effects. Part I. Studies on chronic toxicity	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)	<p>No significant increase in tumor incidence observed in any of the treated groups</p> <p>NS</p>
JMPR	2006	Glyphosate. In: Joint FAO/WHO Meeting on Pesticide Residues. Pesticide residues in food – 2004; toxicological evaluations. Report No. WHO/PCS/06.1. Geneva	Rat, Wistar-Alpk:APSD (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]	<p>No significant increase in tumor incidence observed in any groups of treated animals</p> <p>NS</p>

JMPR	2006	Glyphosate, In: Joint FAO/WHO Meeting on Pesticide Residues, Pesticide residues in food – 2004; toxicological evaluations. Report No. WHO/PCS/06.1. Geneva	Rat, Sprague-Dawley (M, F) 104 wk	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]	No significant increase in tumor incidence observed in any groups of treated animals	NS
JMPR	2006	Glyphosate, In: Joint FAO/WHO Meeting on Pesticide Residues, Pesticide residues in food – 2004; toxicological evaluations. Report No. WHO/PCS/06.1. Geneva	Rat, Wistar-Alpk:API/D (M, F) 24 mo	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]	No significant increase in tumor incidence observed in any groups of treated animals	NS



EPA	1991 A,B,C,D	Rat Sprague-Dawley (M, F) 24 mo	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	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%)	Adenoma, * P ≤ 0.05 (Fisher exact test with Bonferroni inequality); see comments
				Liver: Hepatocellular adenoma: 2/60 (3%), 2/60 (3%), 3/60 (6%), 7/60 (12%) Hepatocellular carcinoma: 3/60 (5%), 2/60 (3%), 1/60 (2%), 2/60 (3%) 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	Adenoma, P for trend = 0.016; see comments  NS
				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	Adenoma, P for trend = 0.031; see comments

EPA	1991 a,b,c,d	Second peer review of glyphosate	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]
		Glyphosate; 2-year combined chronic toxicity/carcinogenicity study in Sprague-Dawley rats - List A pesticide for reregistration. Document No. 008390.				
		Peer review on glyphosate. Document No. 008527.				
		Glyphosate – EPA registration No. 524–308 – 2- year chronic feeding/oncogenicity study in rats with technical glyphosate. Document No. 008897.				NS

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

Author	Year(s)	Title	Cell type	End-point	Test	Description of exposure and controls	Test results/Significance
Paz-y-Miño et al.	2007	Evaluation of DNA damage in an Ecuadorian population exposed to glyphosate	NA	DNA damage	DNA strand breaks, comet assay	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	+ / $P < 0.001$



Paz-y- Miño et al.	2011	Baseline determination in social, health, and genetic areas in communities affected by glyphosate aerial spraying on the northeastern Ecuadorian border	N/A	Chromosomal damage	Chromosomal aberrations	92 individuals in 10 communities, 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)	-
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Bolognesi et al.	2009	Biomonitoring of genotoxic risk in agricultural workers from five Colombian regions: association to occupational exposure to glyphosate	Lymphocytes	Chromosomal damage	Micronucleus formation	<p>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> <p>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> <p>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>	<p>+ / P&lt;0.001</p> <p>*p-value for after spraying vs. before spraying in the same individuals</p> <p>+ / P=0.01</p> <p>*p-value for after spraying vs before spraying in the same individuals</p> <p>+ / P&lt;0.001</p> <p>*p-value for after spraying vs before spraying in the same individuals</p>
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**TABLE 3B. STUDIES OF GLYPHOSATE, AMPA, AND GLYPHOSATE--BASED FORMULATIONS EXPOSURE TO HUMAN CELLS IN VITRO (Adapted from IARC Monograph 112)**

Author	Year(s)	Title	Tissue, cell line	End-point	Test	Results		Dose (LED or HID)	Significance
						Without metabolic activation	With metabolic activation		
GLYPHOSATE									
Mañas et al.	2009a	Genotoxicity of glyphosate assessed by the comet assay and cytogenetic tests	Liver Hep-2	DNA damage	DNA strand breaks, comet assay	+	NT	3mM [507.2 µg/mL]	P <0.01
Mladinic et al.	2009b	Evaluation of genome damage and its relation to oxidative stress induced by glyphosate in human lymphocytes in vitro.	Lymphocytes	DNA damage	DNA strand breaks, standard and hOGGI modified comet assay	+	+	3.5 µg/mL	P <0.01 (with hOGGI modified comet assay, + S9 at highest dose tested (580 µg/mL)
Alvarez-Moya et al.	2014	Comparison of the in vivo and in vitro genotoxicity of glyphosate isopropylamine salt in three different organisms.	Lymphocytes	DNA damage	DNA strand breaks, comet assay	+	NT	0.00007 mM [0.12 µg/mL]	P≤0.01
Monroy et al.	2005	Cytotoxicity and genotoxicity of human cells exposed in vitro to glyphosate	Fibroblast GM 38	DNA damage	DNA strand breaks, comet assay	+	NT	4mM [676 µg/mL]	P <0.01



Lueken et al.	2004	Synergistic DNA damage by oxidative stress (induced by H2O2) and nongenotoxic environmental chemicals in human fibroblasts	Fibroblast GM 5757	DNA damage	DNA strand breaks, comet assay	+	NT	75 mM [12.680 µg/mL]	Not Reported
Monroy et al.	2005	Cytotoxicity and genotoxicity of human cells exposed in vitro to glyphosate	Fibrosarcoma HT1080	DNA damage	DNA strand breaks, comet assay	+	NT	4.75 mM [803 µg/mL]	P<0.001
Koller et al.	2012	Cytotoxic and DNA-damaging properties of glyphosate and Roundup in human-derived buccal epithelial cells	Buccal carcinoma TR146	DNA damage	DNA strand breaks, SCGE assay	+	NT	20 µg/mL	P<0.05, dose-dependent increase
Mañas et al.	2009a	Genotoxicity of glyphosate assessed by the comet assay and cytogenetic tests	Lymphocytes	Chromosomal damage	Chromosomal aberrations	-	NT	6 mM [1015 µg/mL]	Not Significant
Mladinic et al.	2009a	Characterization of chromatin instabilities induced by glyphosate, terbuthylazine and carbofuran using cytome FISH assay	Lymphocytes	Chromosomal damage	Micronucleus formation	-	+	580 µg/mL	P < 0.01 at highest exposure + S9
Bolognesi et al.	1997	Genotoxic activity of glyphosate and its technical formulation Roundup	Lymphocytes	Chromosomal damage	Sister-chromatid exchange	+	NT	1000 µg/mL	P < 0.05
<b>AMPA</b>									

Mañas et al.	2009b	Genotoxicity of AMPA, the environmental metabolite of glyphosate, assessed by the Comet assay and cytogenetic tests.	Liver Hep-2	DNA damage	DNA strand breaks, comet assay	+	NT	4.5 mM [500µg/mL]	P < 0.05 at 4.5 mM; P < 0.01 at up to 7.5 mM Dose-response relationship (P<0.05)
Mañas et al.	2009b	Genotoxicity of AMPA, the environmental metabolite of glyphosate, assessed by the Comet assay and cytogenetic tests.	Lymphocytes	Chromosomal damage	Chromosomal aberrations	+	NT	1.8 mM [200 µg/mL]	P < 0.05
<b>GLYPHOSATE-BASED FORMULATIONS</b>									
Gasnier et al.	2009	Glyphosate-based herbicides are toxic and endocrine disruptors in human cell lines	Liver Hep-2	DNA damage	DNA strand breaks, comet assay	+	NT	5ppm	Not Reported
Koller et al.	2012	Cytotoxic and DNA-damaging properties of glyphosate and Roundup in human-derived buccal epithelial cells	Buccal carcinoma TR146	DNA damage	DNA strand breaks, SCGE assay	+	NT	20 µg/mL	Dose-dependent increase (P≤0.05)



Vigfusson & Vyse	1980	The effect of the pesticides, Dexon, Captan and Roundup, on sister-chromatid exchanges in human lymphocytes in vitro	Lymphocytes	Chromosomal damage	Sister-chromatid exchange	+	NT	250 µg/mL	P < 0.001
Bolognesi et al.	1997	Genotoxic activity of glyphosate and its technical formulation Roundup	Lymphocytes	Chromosomal damage	Sister-chromatid exchange	+	NT	100 µg/mL	P < 0.05

AMPA, aminomethyl phosphonic acid; HIID, highest ineffective dose; hOGG1, human 8-hydroxyguanine DNA-glycosylase; LED, lowest effective dose; NR, not reported; NT, not tested; S9, 9000 × g supernatant; SCGE, single cell gel electrophoresis; vs, versus

FIGURE 1. FOREST PLOT FOR GLYPHOSATE/NHL – SHINASI &amp; LEON, 2014

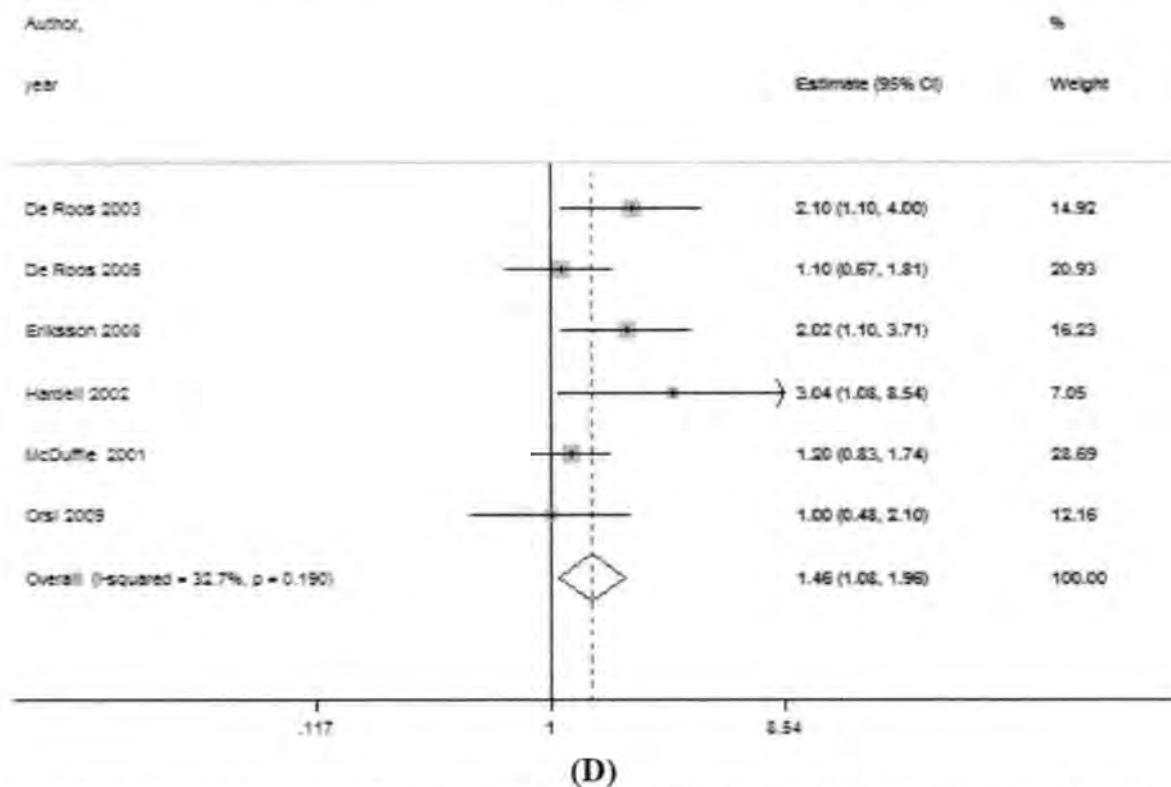


Figure D. (Schinasi &amp; Leon, 2014) Forest plots showing estimates of association between non-Hodgkins Lymphoma and occupational, agricultural exposure to (D) glyphosate.

FIGURE 2. FOREST PLOT – CHANG &amp; DELZELL, 2016

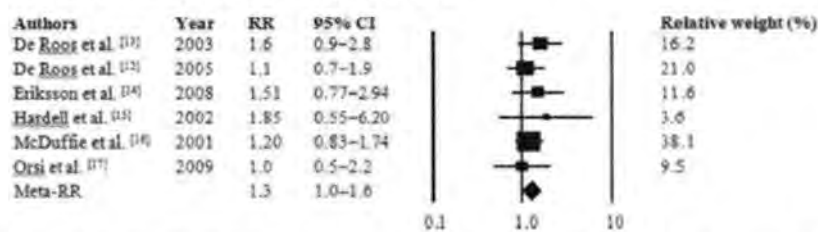


Figure 1. Forest plots of relative risk (RR) estimates and 95% confidence intervals (CIs) for the association between glyphosate exposure and risk of non-Hodgkin lymphoma. Meta-RRs were identical in random-effects and fixed-effects models.



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# Attachment A



**Alfred I. Neugut**  
**Curriculum Vitae**  
April 1, 2017

**HOME ADDRESS:**

[REDACTED]  
[REDACTED]

**OFFICE ADDRESS:**

[REDACTED] [REDACTED]  
[REDACTED] [REDACTED]  
[REDACTED] [REDACTED]  
[REDACTED] [REDACTED]  
[REDACTED] [REDACTED]

**TELEPHONE:**

[REDACTED]

**FAX:**

[REDACTED]

**DATE OF BIRTH:**

[REDACTED]

**E-Mail:**

[REDACTED]

**PLACE OF BIRTH:**

[REDACTED]

**CITIZENSHIP:**

[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

Sloan-Kettering Institute for Cancer Research, New York. Research Fellow.

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:**

1980 - 1981	Cornell University Medical College, New York. Clinical Instructor in Medicine.
1991 - 1983	Columbia University School of Public Health, New York. Staff Associate in Epidemiology.
1983 - 1985	Columbia University College of Physicians & Surgeons, New York, N.Y., Assistant Professor of Medicine.
1985 - 1991	Columbia University College of Physicians & Surgeons, New York, N.Y., Assistant Professor of Medicine and Public Health (Epidemiology).
1991 - 1998	Columbia University College of Physicians & Surgeons, New York, N.Y. Associate Professor of Clinical Medicine and Public Health (Epidemiology).
1998 - 1999	Columbia University College of Physicians & Surgeons, New York, N.Y. Associate Professor of Medicine and Public Health (Epidemiology).
1999 -	Strang Cancer Prevention Center, New York, N.Y. Consultant in Epidemiology.
1999 - 2001	Columbia University College of Physicians & Surgeons, New York, N.Y. Professor of Medicine and Public Health (Epidemiology).
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.
2005 -	Columbia University College of Physicians & Surgeons, New York, N.Y. Myron M. Studner Professor of Cancer Research in 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:**

1983 - 1991	Presbyterian Hospital, New York, N.Y. Assistant Attending Physician.
1989 - 1998	Co-Director, Oncology Outpatient Unit, Presbyterian Hospital, New York, N.Y.
1991 - 1999	Presbyterian Hospital, New York, N.Y. Associate Attending Physician.
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.
2002 - 2010	New York Presbyterian Hospital, New York, N.Y. Member, New York Presbyterian Oncology Editorial Board.

**HONORS AND AWARDS:**

1972 - 1973	B.A. awarded cum laude, Columbia College.
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.
1982 - 1983	Participant, Student Workshop of Society for Epidemiologic Research.
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 Mentor of the Year, Office of Academic Affairs and Irving Institute for Clinical and Translational Research, Columbia University Medical Center.
2016	Joseph R. Fraumeni Distinguished Achievement Award, American Society of Preventive Oncology

**GRANTS:**

1981 - 1990	Fellowship Awardee, Andrew Mellon Foundation Program for Epidemiology in Medicine. Principal Investigator, M. Susser - \$40,000 per year.
1984 - 1986	Junior Faculty Fellowship, American Cancer Society - \$10,000 per year.
1986 - 1989	Principal Investigator, NCI grant #1-R01-CA37196. A Case-Control Study of Colorectal Polyps - \$450,383.
1986 - 1990	Principal Investigator, American Institute for Cancer Research grant #86A77. Obesity-Related Risk Factors for Prostate Cancer - \$96,596.
1986 - 1991	Principal Investigator, NCI grant #1-KO7-CA01211. Preventive Oncology Academic Award \$270,000.
1987 - 1992	Project Principal Investigator on Epidemiology component, Aaron Diamond Foundation. Colon Cancer Program at Columbia University. Principal Investigator, IB Weinstein - \$750,000.
1988 - 1989	Principal Investigator, Cancer Research Society. Implications of Chemotherapy and Radiation Therapy for Second Primary Cancers in the United States Population - \$54,000.
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.
1990 - 1991	Principal Investigator, ACS Institutional Seed Grant. A Preliminary Study of Cancer Incidence among Dominican Immigrants in Washington Heights - \$15,000.
1991 - 1992	Principal Investigator, ACS - NYC Division. Increasing Pap Smear Compliance Among Young Women in Northern Manhattan - \$13,000.
1991 - 1996	Principal Investigator, NCI grant #5-T32-CA09529. Cancer Epidemiology, Biostatistics, Environmental Sciences Training Program - \$2,538,595.
1992 - 1993	Co-Investigator, ACS - NYC Division. Dissemination of Cancer Screening and Prevention Guidelines Among Urban Community Physicians Through Educational Outreach. Principal Investigator, AR Ashford - \$29,000.
1992 - 1995	Principal Investigator, on Columbia University subcontract, NCI grant #2-PO1-CA32617-09A1. Smoking, Diet and Other Risk Factors for Tobacco-Related Cancers. Principal Investigator, G. Kabat, American Health Foundation - \$202,285.
1992 - 1995	Principal Investigator, NCI grant #R25-CA57905. Cancer Prevention Education Outreach in North Manhattan - \$298,011.
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	Principal Investigator, NCI grant #P20-CA66224. Timing of Breast Cancer Surgery and Biopsy within the Menstrual Cycle: Effects on Recurrence and Survival - \$19,839.
1995 - 2005	Co-Principal Investigator, NCI grant #U01-CA66572. Breast Cancer and the Environment on Long Island. Principal Investigator, M. Gammon \$6,318,789.
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	Principal Investigator, Bristol-Myers/Squibb grant. Second Malignancies and Ovarian Cancer: Incidence, Therapy Effects, Clinical Characteristics, and Survival - \$46,000.
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	Principal Investigator, NCI grant #1-R25-CA66882. Breast Cancer Education in North Manhattan - \$770,273.
1996 - 2001	Principal Investigator, NCI grant T32-CA09529. Training Program in Cancer Epidemiology, Biostatistics, Environmental Health Sciences -
1997 - 1998	Principal Investigator, Bristol-Myers/Squibb grant. Population-Based Study of Health Outcomes in Colorectal Cancer - \$54,000.
1997 - 2001	Principal Investigator, supplemental award to NCI grant #3-P30-CA13696-2551 (K. Antman, PI). Studying Complications in Breast Cancer Survivors - Lung Cancer After Breast Cancer Radiotherapy - \$235,980.
1998 - 1999	Co-Investigator, USAMRMC MP980014. Race/Ethnic Based Genetic Variations in Human Genes: Defining the Genetic Evidence for Disparity of Prostate Cancer Risk and Mortality Between Different Populations. Principal Investigator, J. Franklin - \$49,840.
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	Co-Investigator, USAMRMC BC972005. Cytochrome p450-17cx Polymorphism and Risk of Breast Cancer. Principal Investigator, H. Ahsan - \$232,732.
1998 - 2001	Co-Principal Investigator, Robert Wood Johnson Foundation. Addressing Tobacco in Managed Care Program. Principal Investigator, D. Sadowsky - \$500,000.
1999 - 2000	Co-Investigator, USAMRMC PC 991287. Assessment of Genetic Variations Among Different Ethnic/Racial Groups: An Explanation of Ethnic/Racial Disparities in Prostate Cancer Risk and Mortality. Principal Investigator, J. Franklin - \$74,999.



**Alfred I. Neugut**  
**Curriculum Vitae**

1999 - 2002	Principal Investigator, Gustavus and Louise Pfeiffer Research Foundation. The Patterns and Effectiveness of Advanced Ovarian Cancer Treatment in Elderly Women - \$223,398.
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	Co-Principal Investigator, NIH grant #SO6-GM54650. Psychosocial Deterrents to Breast Cancer Screening in Three Ethnic Groups of Older Black Women. Principal Investigator, C. Magai, Long Island University - \$1,551,631.
1999 - 2007	Principal Investigator, NYSDOH. Colorectal Cancer Screening and Prostate Cancer Education - \$177,161.
2000 - 2001	Principal Investigator, Avon Products Foundation. Breast Cancer Research Program. Follow-up of breast cancer survivors in the elderly - \$50,000.
2001 - 2003	Principal Investigator, American Cancer Society RSGHP-01-024-01-CCE. Effectiveness of Cancer Care in the Elderly - \$622,000.
2001 - 2003	Co-Investigator, NCI grant R03-CA92748. Barrett's Esophagus Ablation with Celecoxib Trial. Principal Investigator, C. Lightdale - \$100,000.
2001 - 2003	Co-Investigator, NCI grant R03-CA96422. Cognitive effects of breast cancer treatment. Principal Investigator, Felice Tager - \$100,000.
2001 - 2004	Principal Investigator, NCI grant P20-CA91372. Cancer in Older Minority Populations - \$375,000.
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 - 2003	Principal Investigator, Avon Products Foundation/ACS grant. Brooklyn Physicians Against Cancer: An Academic Detailing Project - \$395,551.
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.
2002 - 2007	Principal Investigator, NCI grant R25 CA94061. Training Program in Cancer Related Populations Sciences - \$2,052,258.
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.
2003 - 2005	Co-Investigator, American Cancer Society grant RSGHP PBP-105710. Decision analysis of population screening for BRCA1/2 mutations. Principal Investigator, Victor Grann - \$181,000.
2003 - 2006	Principal Investigator, Columbia University subcontract, NCI grant U10-CA101178. Fox Chase Cancer Center CCOP-Research Base Program. Principal Investigator, Paul Engstrom - \$2,805,791, subcontract \$132,393.



**Alfred I. Neugut**  
**Curriculum Vitae**

2003 – 2008	Principal Investigator, NCI U54 CA101598, Cancers in Older Minority Populations: Caribbean American Emphasis - \$3,173,271.
2003 – 2008	Co-Principal Investigator, NCI U54 CA101388, Cancers in Older Minority Populations: Caribbean American Emphasis. Principal Investigator, Carol Magai - \$4,330,399.
2003 - 2006	Co-Investigator, NCI R01 CA80197. The Jerusalem Perinatal Study. Principal Investigator, Susan Harlap - \$1,452,622.
2003 - 2006	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.
2003 - 2004	Principal Investigator, Columbia University subcontract, NCI N01-CN-17103 Early therapeutics development with phase 2 emphasis. Principal Investigator, E. Lesser, Montefiore Medical Center
2005- 2007	Principal Investigator, American Cancer Society grant RSGT-01-02404-CPHPS. Effectiveness of Cancer Care in the Elderly - \$623,000.
2005- 2017	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.
2006-2008	Co-Investigator, NCI R21 CA114064. Columbia-GHI Consortium for Claims-based Cancer. Principal Investigator, S. Stellman - \$275,150.
2007-2008	Principal Investigator, NYSDOH Colorectal Cancer Screening and Prostate Cancer Education Initiative for Bronx County - \$50,000.
2007-2009	Co-Investigator, Komen Foundation. Post-Treatment Care of Latina Breast Cancer Survivors. Principal Investigator, D. Hershman - \$300,000.
2007-2011	Co-Investigator, Columbia University subcontract, NCI R01 CA124924, Using Physiologic Age to Predict Chemotherapy Toxicity. Principal Investigator, J. Mandelblatt - \$304,638 (Columbia University Subcontract).
2007-2012	Principal Investigator, NCI grant R25 CA94061. Training Program in Cancer Related Populations Sciences - \$2,445,223.
2008-2010	Co-Principal Investigator, American Cancer Society grant RSG 08-009-01-CPHPS. Adjuvant Breast Cancer Therapies - Racial Variations in Care and Survival. Principal Investigator, D. Hershman - \$715,327.
2008-2013	Principal Investigator, NCI grant T32-CA09529. Training Program in Cancer Epidemiology, Biostatistics, and Environmental Health Sciences – \$2,235,920.
2008-2013	Principal Investigator, NYSDOH. Breast Cancer Screening, Colorectal Cancer Screening and Prostate Cancer Education - \$250,000/yr.
2008-2012	Co-Investigator, NCI grant R01 CA134964. Determinants and risks of use and overuse of expensive drugs. Principal Investigator, D. Hershman - \$639,600.
2009-2010	Principal Investigator, Brain Tumor Foundation. MRI Early Detection Program – \$48,210.
2009-2010	Principal Investigator, Herbert Irving Comprehensive Cancer Center Pilot Grant. Adherence to hormonal therapy in breast cancer: an intervention trial - \$75,000.
2009-2010	Principal Investigator, NCI grant U54 CA1015978-0551. ARRA Supplement to Cancer in Older Minority Populations: Caribbean American - \$94,270.
2009-2014	Co-Investigator, American Cancer Society grant RSGT-09-012-01-CPPB. Promoting CRC screening in a hard-to-reach, low-income minority population. Principal Investigator, CE Basch - \$1,975,355.
2010-2011	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.



**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.
2010-2013	Principal Investigator, NCI grant D43 CA153715. Columbia-South Africa Training Program for Research on HIV-Associated Malignancies - \$1,465,971.
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.
2012-2015	Principal Investigator, American Cancer Society grant RSGT-11-012-01-CPHPS. The relationship between insurance and cancer-related prescription drug use. - \$589,812.
2012-2017	Principal Investigator, NCI grant R25 CA94061. Training Program in Cancer Related Populations Sciences - \$2,437,354.
2013-2017	Co-Investigator, NCI grant R01 CA169121. The Influence of Hospital Variability on the Management of Cancer-Associated Complications. Principal Investigator, JD Wright - \$1,660,000.
2013-2016	Co-Investigator, NCI grant R01 CA186084. Using SWOG-Medicare database to evaluate long-term toxicities of cancer survivors. Principal Investigator, DL Hershman - \$1,230,000.
2013-2014	Principal Investigator, NCI grant D43 CA153715. Columbia-South Africa Training Program for Research on HIV-Associated Malignancies (supplement) - \$250,000.
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.
2014-2017	Principal Investigator, Brain Tumor Foundation. Brain Tumor Early Detection Study - \$746,476.
2014-2015	Co-Investigator, Herbert Irving Comprehensive Cancer Center Pilot Grant. Gut Microbiota, Butyrate, Inflammation and Physical Activity: A Pilot Study among Individuals at High Risk for Colorectal Cancer. Principal Investigators, H Greenlee, B Williams - \$75,000.
2015-2019	Co-Investigator, American Cancer Society Mentored Research Scholar Grant. Dietary Intake and Obesity in Children with Acute Lymphoblastic Leukemia. Principal Investigator, EJ Ladas - \$729,000.
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), Brisol-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 - 1998	Columbia-Presbyterian Cancer Center, Head of Working Group on Cancer Prevention and Control.
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.  
 Abnormal Human Biology, Oncology Preceptor, 20-25 students, 1986-1996, 2000-2004.  
 Pathophysiology for Occupational and Physical Therapy, Lecturer, 50-75 students, 1985-  
 P6729 - Preventive Medicine and Public Health, Lecturer, 25-35 students, 1987-2007.  
 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 - Lydía 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 Kubo, 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				
Name	Years	Institution/Degree/Year	Research Project Title	Current Position/Support
Garrison, Susan	'89-'91	Columbia U., MSPH MPH '91 College of Physicians &	Study of cancer among Dominican	Associate Professor of Medicine, Albert



**Alfred I. Neugut**  
**Curriculum Vitae**

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

**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 Stanford, BA '94	Hormone Receptor Status and Breast Cancer	Pharmaceutical industry



**Alfred I. Neugut**  
**Curriculum Vitae**

Abrams, Julian	'05-07	Mt Sinai MD, '00 University of Penn., BA, '94	Inflammatory liver disease and liver mets.	Assistant Professor of Medicine and Epidemiology, Columbia
Hall, Michael	'05-06	Columbia BA '93; MD '99 U of Chicago, MHS, '05	Analysis of BRCA and DNA repair data	Associate Professor of Medicine and Clinical Genetics, Fox Chase Cancer Center
Strauss, Joshua	'08-09 '14-15	Einstein MD '09; Penn BA '05	GI cancer treatments and outcomes	Private practice
Lebwohl, Benjamin	'08-10	Columbia MD '03, MS '10 Harvard BA '99	Quality of colonoscopy performance and prep	Assistant Professor of Medicine and Epidemiology, Columbia
MacDonald, Alicia	'09-'12	Pittsburgh PhD '09	Viruses and cancer in HIV patients	Assistant Professor of Epidemiology, Penn State University
Lasheen, Wael	'09-'12	Cairo University MBBS '95	Palliative care and racial disparities among cancer patients	Case Western Reserve, Research Scientist
Brouse, Corey	'09-'11	Teachers College EdD '03; University of New Hampshire BS '99	Promotion of colonoscopy among the underserved	Associate Professor, Montclair State
Becker, Daniel	'09-'10	NYU MD '04; Yale BA '00	Health outcomes studies in the treatment of colorectal cancer	Assistant Professor of Clinical Medicine, NYU
Sharaiha, Reem	'10-'11	University of London MBBS '03	Studies on GI tract tumors	Assistant Professor of Gastroenterology, Cornell
Winner, Megan	'10-'12	Washington University in St. Louis MD '07; BA '02	Studies on benign and malignant tumors of the pancreas	Instructor in Surgery, SUNY Stony Brook
Vin-Raviv, Neomi	'11-'14	University of Haifa PhD '11	Studies on PTSD and cancer	

**Alfred I. Neugut**  
**Curriculum Vitae**

Khanna, Lauren	'12-'14	Harvard MD '07; Princeton BA '02	Disparities in pancreatic cancer	Assistant Professor of Gastroenterology, NYU
Oberstein, Paul	'12-'13	Ohio State MD '06; University of Maryland BA '02	Translational studies in pancreatic cancer	Assistant Professor of Medicine, Columbia
Tsui, Jennifer	'12-'14	UCLA PhD '12; Columbia MPH '05; UC Berkeley BA '03	Insurance and health outcomes studies	Assistant Professor of Public Health, Robert Wood Johnson
Sardo Molmenti, Christine	'13-'15	Arizona PhD '13; Minnesota MPH '94; Ohio State BA '92	Diet, obesity, chemoprevention, colorectal cancer	Assistant Professor of Public Health, Hofstra
Kahn, Justine	'15-	Mt. Sinai MD '10; Barnard BA '05	Pediatric cancer and adherence, SES	Instructor in Pediatric Oncology
Leng, Siyang	'16-	SUNY Downstate MD '09; Cornell BS '05	Health outcomes for myeloma and plasma cell dyscrasias	Instructor in Medical Oncology
Joffe, Lenat	'16-	Sackler MD '10; NYU BA '06	Outcomes research in pediatric oncology	Pediatric oncology fellow
Kwon, Sung (Steve)	'16-	Washington MPH '11; Illinois MD '07; Illinois BS '03	Outcomes research in surgical oncology	Postdoctoral fellow Epidemiology
O'Neil, Daniel	'17-	Harvard MPH '16; Einstein MD '12; Cornell BS '06	Outcomes and global research	Medical Oncology Fellow

Mentor For Career Development

Victor R. Grann, M.D., M.P.H. - ACS Career Development Award - 1999-2004

Mary Beth Terry, Ph.D., K07 - 2001-2006

Dawn L. Hershman, M.D., M.S. - K07 - 2002-2007

Andrew Rundle, Ph.D. - K07 - 2003-2008

Donna Shelley, M.D., M.P.H. - K01 CDC 2005-2008

Benjamin Spencer, M.D., M.P.H. - DoD Physician Research Training Award - 2006-2011

Michael A.J. Hall, M.D., M.H.S. - ACS Career Development Award - 2007-2012

Abby Siegel, M.D. - K12 2008-2011, K23- 2010-2015

Heather Greenlee, N.D., Ph.D. - K12 - 2008-2011, K23 - 2009 - 2014 (Co-Mentor)

Katherine Crew, M.D., M.S. - ACS Career Development Award - 2008-2013 (Co-Mentor)

Julian A. Abrams, M.D., M.S. - FAMRI Career Development Award - 2008-2011; K07-2008-2013

Rose Lai, M.D., M.Sc. - K07 - 2008-2013



**Alfred I. Neugut**  
**Curriculum Vitae**

Daniel Becker, M.D. – ASCO Young Investigator Award – 2010-2011  
 Fay Kastrinos, M.D., M.P.H. –K07 – 2010-2015  
 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, Journal of Clinical Oncology, 2004-2006.  
 Editorial Board, Section Chief for Epidemiology and Prevention, Cancer Investigation, 2005-.

Manuscript Reviewer

American Journal of Epidemiology, American Journal of Gastroenterology, American Journal of Medicine, American Journal of Public Health, Anti-Cancer Drugs, BMJ, Breast Cancer Research, Breast Cancer Research and Treatment, British Journal of Cancer, Cancer, Cancer Causes and Control, Cancer Control, Cancer Epidemiology Biomarkers and Prevention, Cancer Investigation, Cancer Medicine, Cancer Research, Chronic Diseases in Canada, Clinical Cancer Research, Clinical Gastroenterology and Hepatology, Consultant, Digestion, Digestive Diseases and Sciences, Diseases of the Colon and Rectum, Epidemiologic Reviews, Epidemiology, European Journal of Cancer, European Journal of Epidemiology, Gastroenterology, Gastrointestinal Endoscopy, Gut, Integrative Cancer Therapies, International Journal of Cancer, International Journal of Epidemiology, International Journal of Radiation Oncology Biology and Physics, Italian Journal of Gastroenterology and Hepatology, Journal of the American Geriatrics Society, Journal of the American Medical Association, JAMA Oncology, Journal of Cancer Epidemiology, Journal of Cancer Survivorship, Journal of Chronic Disease, Journal of Clinical Oncology, Journal of Global Oncology, Journal of Laboratory and Clinical Medicine, Journal of the National Cancer Institute, Journal of Occupational Medicine, Journal of Oncology Practice, Journal of Urban Health, Lancet Oncology, Nature Reviews Clinical Oncology, New England Journal of Medicine, New York State Journal of Medicine, Nutrition and Cancer, Oncology (Switzerland), Oncotarget, PLOS One, Seminars in Oncology, Southern Medical Journal, Supportive Care in Cancer, Surgical Endoscopy, The Oncologist, Therapeutic Advances in Gastroenterology

Committees

Federal

NCI, National Occupational Cancer Control Clinical Network, 1984-1985.  
 Office of Technology Assessment, U.S. Congress, paper reviewer, 1993-1994.  
 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, adhoc 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 City Department of Environmental Protection, Citizens Advisory Committee of the Greenpoint/Williamsburg Environmental Benefits Program, advisor to Health Subcommittee, 1994-1995.  
 New York State Department of Health, Commissioner's Expert Advisory Panel on

**Alfred I. Neugut**  
**Curriculum Vitae**

Cancer Screening Guidelines, 1994-1995.  
New York State Comprehensive Cancer Control Data and Surveillance Goal  
Development team, 2002-2003.  
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, Ad Hoc Contracts Technical Review Group for SEER, June, 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  
Cancer Surveillance, July 1995.  
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  
Networks, January 1998.  
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,  
January - February 1999.  
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,  
July 2002-June 2006.  
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

Cancer and Leukemia Group B, Cancer Control Science Committee, 1987-1991.  
 American Society of Preventive Oncology, Long-range Planning Committee, 1988.  
 U.S.- Israel Binational Science Foundation, grant reviewer, 1989-1990.  
 American Society of Preventive Oncology, Chairman, Publications Committee, 1989-1990.  
 Cancer and Leukemia Group B, Epidemiology Working Group, 1989-1991.  
 American Association of Cancer Institutes, Policy Advisory Committee, Centralized Cancer Patient Data System, 1989-1991.  
 CHEMOcare, Board of Trustees, 1991-Present.  
 American Association of Cancer Research representative to the LaSalle D. Leffall, Jr. Award Committee, 1991.  
 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 Clinical Oncology, Cancer Prevention and Control Committee, 1994-1996.  
 American Society of Preventive Oncology, Secretary-treasurer, 1994-1999.  
 Member, Southwest Oncology Group, 1994-Present.  
 American Gastroenterology Association, Abstract Evaluation Committee for 1995 Annual Meeting.  
 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.

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American Gastroenterology Association, Abstract Evaluation Committee for 1996 Annual Meeting.  
 Member, Review Committee of American Health Foundation Activities, 1995.  
 American Society of Clinical Oncology, 1997 Annual Meeting Program Planning Committee.  
 Center for Health Outcomes and Economics, Bristol-Myers/Squibb, Business Development Task Force, 1996-1997.  
 American Gastroenterology Association, Abstract Evaluation Committee, GI Oncology Section, 1997 Annual Meeting.  
 Member, Southwest Oncology Group Gastrointestinal Committee, 1997-Present.  
 American Society of Preventive Oncology, President-Elect, 1998-1999.  
 Member, Southwest Oncology Group Cancer Control Committee, 1998-Present.  
 President, American Society of Preventive Oncology, 1999-2001.  
 Reviewer, Doris Duke Charitable Foundation, Clinical Scientist Development Awards, 2000, 2001, 2002.  
 Past President, American Society of Preventive Oncology, 2001-2003.  
 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.  
 External Reviewer, Institute of Medicine National Academy of Sciences, Draft report on Asbestos: Selected Health Effects, 2006.  
 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.  
 Reviewer, U.S. – Israel Binational Science Foundation, 2010.  
 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

International Agency for Research on Cancer, Working Group on the International Study of Potential Cancer Risk in Cancer Research Laboratory Workers, 1987-1990.  
 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



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Guest lecturer, Cancer Epidemiology. French School of Public Health (Ecole Hautes d'Etudes Sante Publique), Paris France, November 2009, 2010, 2011, 2012, 2013  
Member, African Organization for Research and Training in Cancer, 2010 – present.  
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.  
Consultant to the United Nations Compensation Commission on the Persian Gulf War, Industrial Economics Inc, 2003-2004.  
Consultant, Safety and Development of ETC-216, Pfizer, 2004.  
Member, Program Steering Committee, U56 Partnership between University of Massachusetts-Boston and Dana-Farber/Harvard Cancer Center, 2006-.  
External Advisor, R25T Cancer Copntrol Research Postdoctoral Training Program, Cancer Research Center of Hawaii, University of Hawaii, 2006-2009.  
Member, Advisory Group on Bisphosphonates-Associated Osteonecrosis of the Jaw, American Association of Oral and Maxillofacial Surgeons, 2006-2008.  
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.

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**PUBLICATIONS:**

Peer-Reviewed

1. Neugut AI, Eisenberg D, Silverstein M, Pulkrabek P, Weinstein IB. Effects of asbestos on epithelioid cell lines. *Env Res* 17:256-265, 1978.
2. Neugut RH, Neugut AI, Kahana E, Stein Z, Alter M. Creutzfeldt-Jakob Disease: Familial clustering among Libyan-born Israelis. *Neurology* 29:225-231, 1979.
3. Neugut AI, Weinstein IB. The use of agarose in the determination of anchorage-independent growth. *In Vitro* 15:351-355, 1979.
4. Neugut AI, Weinstein IB. Growth limitation of BHK-21 cells and its relation to folate metabolism. *In Vitro* 15:363-367, 1979.
5. Neugut AI, Neugut RH. How accurate are patient histories? *J Comm Health* 9:294-301, 1984.
6. Neugut AI, Johnsen C, Forde KA, Treat MR. Recurrence rates for colorectal polyps. *Cancer* 55:1586-1589, 1985.
7. Neugut AI, Johnsen C, Fink D. Serum cholesterol levels in adenomatous polyps and cancer of the colon: A case-control study. *JAMA* 255:365-367, 1986.
8. Liskow AS, Neugut AI, Benson M, Olsson CA, Birkhoff J, Chang CH. Multiple primary neoplasms in association with prostate cancer in black and white patients. *Cancer* 59:380-384, 1987.
9. Linskey ME, Neugut AI, Hall E, Cox JD. A course to teach medical research study design and analysis to medical students. *J Med Educ* 62:143-145, 1987.
10. Guillem JG, Forde KA, Treat MR, Neugut AI, Bodian CA. The impact of colonoscopy on the early detection of colonic neoplasms in patients with rectal bleeding. *Ann Surg* 206:606-611, 1987.
11. Neugut AI, Johnsen CM, Forde KA, Treat MR, Nims NC, Murray D. Cholecystectomy among women with adenomatous polyps and cancer of the colon. *Cancer* 61:618-621, 1988.
12. Robinson E, Neugut AI, Wylie P. Clinical aspects of post-irradiation sarcomas. *J Natl Cancer Inst* 80:233-240, 1988.
13. Neugut AI, Pita A. The role of sigmoidoscopy in screening for colorectal cancer: A critical review. *Gastroenterology* 95:492-499, 1988.
14. Neugut AI. Squamous cell cancers and cigarette smoking: a matter of exposure. *Med Hyp* 26:9-10, 1988.
15. Neugut AI, Johnsen CM, Forde KA, Treat MR, Nims NC. Vitamin supplements among women with adenomatous polyps and cancer of the colon: Preliminary findings. *Dis Col Rect* 31:430-432, 1988.
16. Guillem JJ, Neugut AI, Forde KA, Waye JD, Treat MR. Colonic neoplasms in asymptomatic first-degree relatives of colon cancer patients. *Am J Gastroenterol* 83:271-273, 1988.
17. Neugut AI, Sordillo PP. Leiomyosarcomas of the extremities. *J Surg Oncol* 40:65-67, 1989.



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18. Robinson E, Neugut AI. Clinical aspects of multiple primary neoplasms. *Cancer Det Prev* 13:287-292, 1989.
19. Neugut AI, Fink DJ, Radin D. Serum cholesterol and primary brain tumors: A case-control study. *Int J Epidemiol* 18:798-801, 1989.
20. Robinson E, Neugut AI. The clinical behavior of radiation-induced thyroid cancer in patients with prior Hodgkin's disease. *Radiother Oncol* 17:109-113, 1990.
21. Neugut AI, Nieves J. The use of clinical data in studies of cancer etiology. *Cancer Prev* 1:8-12, 1990.
22. Neugut AI, Leighton J, Habif DV, Markowitz AM. Conjugal breast cancer. *Breast Dis* 3:107-110, 1990.
23. Neugut AI, Robinson E, Nieves J, Murray T, Tsai W-Y. Poor survival of treatment-related acute non-lymphocytic leukemia. *JAMA* 264:1006-1008, 1990.
24. Neugut AI, Timony D, Murray T. Colorectal cancer: Differences between community and geographically distant patients seen at an urban medical center. *Dis Col Rect* 34:64-68, 1991.
25. Neugut AI, Lee WC, Garbowski GC, Waye JD, Forde KA, Treat MR, Fenoglio-Preiser C. Obesity and colorectal adenomatous polyps. *J Natl Cancer Inst* 83:359-361, 1991.
26. Robinson E, Neugut AI, Murray T, Rennert G. A comparison of the clinical characteristics of first and second primary head and neck cancers: A population-based study. *Cancer* 68:189-192, 1991.
27. Garfield RM, Neugut AI. Epidemiologic aspects of warfare. *JAMA* 266:688-692, 1991.
28. Neugut AI, Murray TI, Garbowski GC, Forde KA, Treat MR, Waye JD, Fenoglio-Preiser C. Cholecystectomy as a risk factor for colorectal adenomatous polyps and carcinoma. *Cancer* 68:1644-1647, 1991.
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30. Neugut AI, Murray TI, Garbowski GC, Treat MR, Forde KA, Waye JD, Fenoglio-Preiser C. The association of asbestos exposure with colorectal adenomatous polyps and cancer. *J Natl Cancer Inst* 83:1827-1828, 1991.
31. Luo J-CJ, Neugut AI, Nieves J, Benson M, Niman H, Brandt-Rauf P. Presence of int-1 related protein in serum of prostate cancer patients and controls. *Med Sci Res* 19:453-454, 1991.
32. Neugut AI, Sherr D, Robinson E, Murray T, Nieves J. Differences in histology between first and second primary lung cancer. *Cancer Epidemiol Biomarkers Prev* 1:109-112, 1992.
33. Murray TI, Neugut AI, Garbowski GC, Waye JD, Forde KA, and Treat MR. Relationship between breast cancer and colorectal adenomatous polyps: A case-control study. *Cancer* 69:2232-2234, 1992.
34. Robinson E, Nasrallah S, Adler Z, Rennert G, Neugut AI. The clinical, demographic, and follow-up characteristics of patients with two primary metachronous tumors (one of the tumors in the colon). *Dis Col Rect* 35:457-461, 1992.

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35. Guillem JG, Forde KA, Treat MR, Neugut AI, O'Toole K, Diamond BE. Colonoscopic screening for neoplasms in asymptomatic first degree relatives of colon cancer patients: a controlled, prospective study. *Dis Col Rect* 35:523-529, 1992.
36. Robinson E, Bar-Deroma R, Rennert G, Neugut AI. A comparison of the clinical characteristics of second primary and single primary sarcoma: A population-based study. *J Surg Oncol* 50:263-266, 1992.
37. Neugut AI, Garbowski GC, Lee WC, Murray T, Nieves JW, Forde KA, Treat MR, Waye JD, Fenoglio-Preiser C. Dietary risk factors for the incidence and recurrence of colorectal adenomatous polyps: a case-control study. *Ann Int Med* 118:91-95, 1993.
38. Lee WC, Neugut AI, Garbowski GC, Forde KA, Treat MR, Waye JD, Fenoglio-Preiser C. Cigarettes, alcohol, coffee and caffeine as risk factors for colorectal adenomatous polyps. *Ann Epidemiol* 3:239-244, 1993.
39. Robinson E, Rennert G, Rennert HS, Neugut AI. Survival of first and second primary breast cancer. *Cancer* 71:172-176, 1993.
40. Neugut AI, Robinson E, Lee WC, Murray T, Karwoski K, Kutcher GJ. Lung cancer after radiation therapy for breast cancer. *Cancer* 71:3054-3057, 1993.
41. Neugut AI, Garbowski GC, Waye JD, Forde KA, Treat MR, Tsai JL, Lee WC. Diagnostic yield of colorectal neoplasia with the use of colonoscopy for abdominal pain, change in bowel habits, and rectal bleeding. *Am J Gastroenterol* 88:1179-1183, 1993.
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44. Neugut AI and Santos J. The association between cancers of the small and large bowel. *Cancer Epidemiol Biomarkers Prev* 2:551-553, 1993.
45. Brandt-Rauf PW, Luo J-C, Carney WP, Smith S, DeVivo I, Milling C, Hemminki K, Koskinen H, Vaino H, Nieves J, Neugut AI. Detection of increased amounts of extracellular domain of the c-erbB-2 oncoprotein in serum during pulmonary carcinogenesis in humans. *Int J Cancer* 56:383-386, 1994.
46. Neugut AI, Murray T, Santos J, Amols H, Hayes MK, Flannery JT, Robinson E. Increased risk of lung cancer after breast cancer radiation therapy in cigarette smokers. *Cancer* 73:1615-1620, 1994.
47. Chen CC, Neugut AI, Rotterdam H. Risk factors for adenocarcinomas and malignant carcinoids of the small intestine: Preliminary findings. *Cancer Epidemiol Biomarkers Prev* 3:205-207, 1994.
48. Jacobson JS, Neugut AI, Murray T, Garbowski GC, Forde KA, Treat MR, Waye JD, Santos J, Ahsan H. Cigarette smoking, and other risk factors for recurrent colorectal adenomatous polyps. *Cancer Causes Control* 5:215-220, 1994.



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49. Lautenbach E, Forde KA, Neugut AI. Colonoscopic surveillance following curative resection of colorectal cancer. *Ann Surg* 220:206-211, 1994.
50. Neugut AI, Kizelnik S, Ackerman C. Black-white differences in risk for cutaneous, ocular, and visceral melanoma. *Am J Publ Health* 84:1828-1829, 1994.
51. Jacobson JS, Kono S, Todoroki I, Honjo S, Shinchu K, Imanishi K, Nishikawa H, Ogawa S, Katsurada M, Neugut AI. Fatherhood and risk for colorectal adenomas: a study of male self-defense officials in Japan. *Cancer Epidemiol Biomarkers Prev* 3:655-659, 1994.
52. Neugut AI, Jacobson JS, Ahsan H, Santos J, Garbowski GC, Forde KA, Treat MR, Waye JD. Incidence and recurrence rates of colorectal adenomas: a prospective study. *Gastroenterology* 108:402-408, 1995.
53. Rennert G, Robinson E, Rennert HS, Neugut AI. Clinical characteristics of metachronous colorectal tumors. *Int J Cancer* 60:743-747, 1995.
54. Moss SF, Neugut AI, Wang S, Garbowski G, Forde K, Treat M. *Helicobacter pylori* and colorectal neoplasia: evidence against an association. *J Natl Cancer Inst* 87:762-763, 1995.
55. Neugut AI, Jacobson JS. Breast cancer screening in first-degree relatives of breast cancer patients. *Am J Publ Hlth* 85:832-834, 1995.
56. Robinson E, Adler Z, Nasrallah S, Rennert G, Neugut AI. Clinical characteristics of second primary tumors following breast cancer. *Isr J Med Sci* 31:169-171, 1995.
57. Luo J-C, Neugut AI, Garbowski GC, Forde KA, Treat MR, Smith S, Carney WP, Brandt-Rauf PW. Levels of p53 antigen in the plasma of patients with colon adenomas and carcinomas. *Cancer Lett* 91:235-240, 1995.
58. Neugut AI, Jacobson JS, Sherif G, Ahsan H, Garbowski GC, Waye J, Forde KA, Treat MR. Coronary artery disease and colorectal neoplasia. *Dis Colon Rectum* 38:873-877, 1995.
59. Xing X, Burr JA, Brasure JR, Neugut AI, Marshall JR. Reproducibility of food intake in food frequency questionnaire used in a general population. *Nutr Cancer* 24:85-95, 1995.
60. Robinson E, Adler Z, Nasrallah S, Kuten A, Steiner M, Rennert G, Neugut AI. Clinical characteristics of patients with a second primary tumor in the endometrium or ovary. *Eur J Gynaecol Oncol* 16:195-198, 1995.
61. Neugut AI, Ahsan H, Robinson E. Pancreatic cancer as a second primary malignancy: a population-based study. *Cancer* 76:589-592, 1995.
62. Ahsan H, Neugut AI, Bruce JN. Trends in incidence of primary malignant brain tumors in U.S.A., 1981-90. *Int J Epidemiol* 24:1078-1085, 1995.
63. Ahsan H, Neugut AI, Bruce JN. Association of malignant brain tumors and cancers of other sites. *J Clin Oncol* 13:2931-2935, 1995.
64. Jacobson JS, Neugut AI, Garbowski GC, Ahsan H, Waye JD, Treat MR, Forde KA. Reproductive risk factors for colorectal adenomatous polyps. *Cancer Causes Control* 6:513-518, 1995.

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65. Robinson E, Bar-Deroma R, Epelbaum R, Rennert G, Neugut AI. Clinical characteristics of malignant non-Hodgkins' lymphoma as a second primary tumor: a population-based survey. *Leukemia & Lymphoma* 20:297-301, 1996.
66. Sutter T, Arber N, Findling RI, Moss SF, Neugut AI, Weinstein IB, Holt PR. Frequent K-ras mutations in small bowel adenocarcinomas. *Dig Dis Sci* 41:115-118, 1996.
67. Ahsan H, Neugut AI. High risk of Kaposi's sarcoma and central nervous system lymphoma in the same individuals: a finding related to acquired immunodeficiency syndrome. *Int J Cancer* 66:176-178, 1996.
68. Davidow AL, Neugut AI, Jacobson JS, Ahsan H, Garbowski GC, Forde KA, Treat MR, Waye JD. Recurrent adenomatous polyps and body mass index. *Cancer Epidemiol Biomarkers Prev* 5:313-315, 1996.
69. Luo JC, Neugut AI, Garbowski GC, Forde KA, Treat M, Smith S, Niman H, Brandt-Rauf P. Expression of p21 ras-related protein in the tissue of patients with adenomas and carcinomas of the colon. *Biomarkers* 1:29-33, 1996.
70. Gammon MD, Wolff MW, Neugut AI, Terry MB, Britton JA, Greenebaum E, Hibshoosh H, Levin B, Wang Q, Santella R. Treatment for breast cancer and blood levels of chlorinated hydrocarbons. *Cancer Epidemiol Biomarkers Prev* 5:467-471, 1996.
71. Arber N, Lightdale C, Rotterdam H, Han KH, Sgambato A, Neugut AI, Finegold J, Weinstein IB. Increased expression of cyclin D1 in Barrett's esophagus. *Cancer Epidemiol Biomarkers Prev* 5:457-459, 1996.
72. Xing X, Burr JA, Brasure JR, Neugut AI, Marshall JR. Reproducibility of nutrient intake in a food frequency questionnaire used in a general population. *Nutr Cancer* 25:259-268, 1996.
73. Weissman LB, Corson JM, Neugut AI, Antman KH. Malignant mesothelioma following radiation therapy for Hodgkin's disease. *J Clin Oncol* 14:2098-2100, 1996.
74. Neugut AI, Horvath K, Whelan RL, Terry MB, Garbowski GC, Bertram A, Forde KA, Treat MR, Waye J. The effect of calcium and vitamin supplements on incidence and recurrence rates of colorectal adenomatous polyps. *Cancer* 78:723-728, 1996.
75. Chow JS, Chen CC, Ahsan H, Neugut AI. A population-based study of the incidence of malignant small bowel tumors: SEER, 1973-90. *Int J Epidemiol* 25:722-728, 1996.
76. Marchetti A, Tak Apiech C, McGhan WF, Neugut AI, Smith BT. Pharmacoeconomic analysis of oral therapies for onychomycosis: a U.S. model. *Clinical Therapeutics* 18:757-777, 1996.
77. Neugut AI, Lautenbach E, Abi-Rached B, Forde KA. Incidence of adenomas following curative resection for colorectal cancer. *Am J Gastroenterol* 91:2096-2098, 1996.
78. Neugut AI, Terry MB, Hocking G, Mosca L, Garbowski GC, Forde KA, Treat MR, Waye J. Leisure and occupational physical activity and risk of colorectal adenomatous polyps. *Int J Cancer* 68:744-748, 1996.
79. McGuire W, Neugut AI, Arikian S, Doyle J, Dezii CM. Analysis of the cost-effectiveness of paclitaxel as alternative combination therapy for advanced ovarian cancer. *J Clin Oncol* 15:640-645, 1997.



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81. Gammon MD, Wolff MS, Neugut AI, Terry MB, Papadopoulos K, Levin B, Wang Q, Santella RM. Temporal variation in chlorinated hydrocarbons in healthy women. *Cancer Epidemiol Biomarkers Prev* 6:327-332, 1997.
82. Neugut AI, Ahsan H, Antman KH. Incidence of malignant pleural mesothelioma following thoracic radiotherapy. *Cancer* 80:948-950, 1997.
83. Muscat JE, Stellman SD, Zhang Z-F, Neugut AI, Wynder EL. Cigarette smoking and large cell carcinoma of the lung. *Cancer Epidemiol Biomarkers Prev* 6:477-480, 1997.
84. Ahsan H, Neugut AI, Gammon MD. Association of adenocarcinoma and squamous cell carcinoma of the esophagus with tobacco-related and other malignancies. *Cancer Epidemiol Biomarkers Prevention* 6:779-782, 1997.
85. Arber N, Neugut AI, Weinstein IB, Holt PR. Molecular genetics of small bowel cancer. *Cancer Epidemiol Biomarkers Prev* 6:745-748, 1997.
86. Ahsan H, Neugut AI. Radiation therapy for breast cancer and increased risk of esophageal carcinoma. *Ann Int Med* 128:114-117, 1998.
87. Neugut AI, Jacobson JS, Suh S, Mukherjee R, Arber N. Epidemiology of cancer of the small intestine. *Cancer Epidemiol Biomarkers Prev* 7:243-251, 1998.
88. Grann VR, Panageas RS, Whang W, Antman KH, Neugut AI. A decision analysis of prophylactic mastectomy and oophorectomy in BRCA1 positive patients. *J Clin Oncol* 16:979-985, 1998.
89. Terry MB, Neugut AI. Smoking and the colorectal adenoma-carcinoma sequence: explaining the paradox. *Am J Epidemiol* 147:903-910, 1998.
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93. Khan ZR, Neugut AI, Ahsan H, Chabot JA. Risk factors for biliary tract cancer. *Am J Gastroenterol* 94:149-152, 1999.
94. Grann VR, Whang W, Jacobson JS, Heitjan DF, Antman KH, Neugut AI. Benefits and costs of screening Ashkenazi Jewish women for BRCA1 and BRCA2. *J Clin Oncol* 17:494-500, 1999.

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96. Su Y, Ahsan H, Neugut AI. Association between biliary tract cancers and cancers of other sites. *Am J Gastroenterol* 94:2256-2262, 1999.
97. Grann VR, Jacobson JS, Sundararajan V, Albert SM, Troxel AB, Neugut AI. Quality of life associated with prophylactic treatments for women with BRCA1/2 mutations. *Cancer J Sci Am* 5:283-292, 1999.
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22. Terry MB, Neugut AI. Re: "Cigarette smoking and the colorectal adenoma-carcinoma sequence: a hypothesis to explore the paradox: The authors reply." *Am J Epidemiol* 149:788, 1999.
23. Terry MB, Neugut AI. RE: Cigarette smoking and the colorectal adenoma-carcinoma sequence: A hypothesis to explain the paradox: The authors reply. *Am J Epidemiol* 150:542-543, 1999.
24. Grann VR, Whang W, Jacobson JS, Heitjan DF, Antman KH, Neugut AI. Re-evaluation of benefits of BRCA1/2 testing. *J Clin Oncol* 17:1962, 1999.



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25. Muscat JE, Stellman SD, Malkin MG, Thompson S, Shore RE, Neugut AI. Handheld cellular telephones and brain cancer risk. *JAMA* 285:1838-1839, 2001.
26. Fogel J, Albert SM, Schnabel F, Ditkoff BA, Neugut AI. Quality of health information on the internet. *JAMA* 286:2093-2094, 2001.
27. Gatto NM, Frucht H, Neugut AI. Response: Re: Risk of perforation after colonoscopy and sigmoidoscopy: a population-based study. *J Natl Cancer Inst* 95:831, 2003.
28. Terry MB, Gammon M, Neugut AI. Association of aspirin use and hormone receptor status with breast cancer risk-Reply. *JAMA* 292:1427, 2004.
29. Hershman D, Neugut AI. Author reply. *Cancer* 109:2384, 2007.
30. Rundle A, Richards C, Neugut AI. Hemodilution of prostate specific antigen levels among obese men. Author reply. *Cancer Epidemiol Biomarkers Prev* 18:2343-2344, 2009.
31. Neugut AI, Lebwohl B. Screening colonoscopy vs flexible sigmoidoscopy – reply. *JAMA* 304P:2017-2018, 2010.
32. Sonabend AM, Zacharia BE, Golstein H, Bruce SS, Hershman D, Neugut AI, Bruce JN. Response. *J Neurosurg* 120:298-299, 2014.
33. Prigerson HG, Neugut AI, Maciejewski PK. Evaluating chemotherapy at the end of life – reply. *JAMA Oncol* 2:144, 2016.

**INVITED PRESENTATIONS:**

1. Discussant, Symposium on Biological Markers in Colorectal Cancer. Twelfth Meeting of the American Society of Preventive Oncology, Bethesda, MD, March 1988.
2. Discussant, plenary paper: Kritchevsky SB, and Wilcosky TC. Lipid, lipoprotein cholesterol, and weight change preceding the diagnosis of cancer in the Lipid Research Clinics Coronary Primary Prevention Trial. 22nd Annual Meeting of the Society for Epidemiologic Research, Birmingham, AL, June 1989.
3. Use of endoscopic screening for individuals at average risk for colorectal cancer. Workshop on Colon Cancer Screening for Average-Risk Populations. Second International Conference on Gastrointestinal Cancer, Jerusalem, Israel, August 1989.
4. Natural history and management of colonic polyps. Update in Gastroenterology and Hepatology, Columbia University College of Physicians & Surgeons, New York, NY, December 1991.
5. Epidemiologic perspective on war related casualties. 5th Annual Acute Combat Trauma Symposium, Norfolk, VA, June 1992.
6. Colorectal cancer. NCI Cancer Prevention and Control Academic Course, Rockville, MD, July 1992.
7. Basic epidemiology. Maxwell Abramson Otolaryngology Basic Science Course, New York Academy of Medicine, New York, NY, October 1992.
8. Association of fat with the incidence and recurrence of colorectal adenomas and a proposed mechanism for its role. Ole Moller Jensen Memorial Symposium on Nutrition and Cancer, IARC, Lyon, France, March 1993.
9. Controversies in colon cancer prevention. ACS Symposium on Cancer Prevention and Early Detection: Practice Perspectives, Controversies and Future Directions, New York, NY September 1993.

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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.
11. Sigmoidoscopy and colonoscopy: What you see is what you get. 20th Anniversary Conference on Prevention and Early Detection of Cancer: Controversy & Progress, University of Wisconsin Comprehensive Cancer Center, Madison, WI, October 1993.
12. Can what you eat cause colon cancer? Cancer Initiative Conference Series: Causation and Prevention of Cancer, Columbia-Presbyterian Medical Center, New York, NY, October 1993.
13. Discussant, human epidemiology. Workshop on Non- Steroidal Anti-Inflammatory Agents and Cancer, American Health Foundation, New York, NY, October 1993.
14. Breast cancer screening for women under 50. Commission on Women's Health, Commonwealth Fund, New York, NY, March 1994.
15. Panel member, Controversies and concerns about cancer screening. Inaugural Conference of Industries' Coalition against Cancer, Washington, DC, April 1994.
16. Participant, Organochlorines and Cancer Workshop, Cancer Division of Health Canada, Ottawa, Ontario, September 1995.
17. Participant, Round Table on New and Emerging Technologies for the Detection and Treatment of Breast Cancer, New York State Legislative Commission on Science and Technology, Copiague NY, November 1995.
18. Participant, Workshop on Changing Mortality Rates of Tobacco-Related Diseases, American Health Foundation, New York, NY, April 1996.
19. Participant, Conference of NIA/NCI Working Group on Multiple Primary Cancers in Older-Aged Cancer Patients, National Institutes of Health, Bethesda, MD, June 1996.
20. The epidemiology of second malignancies. Moderator, Life after Cancer. Second Annual Cancer Information Exchange, Columbus CCOP, Amelia Island, Florida, February 1997.
21. Epidemiologic issues in mass tort litigation. New York Law School, New York, NY February 1997.
22. Discussant; Health Services Research/Prevention, 16th Annual Meeting of the American Society of Clinical Oncology, Denver Colorado, May 1997.
23. Participant, Banbury Center Conference on the Pathogenesis of NF1 and NF2- Therapeutic Strategies, Cold Spring Harbor NY, July 1997.
24. Participant, Conference on Practice Outcomes Monitoring and Evaluation System (POMES), Office of Alternative Medicine, Bethesda MD, August 1997.
25. Who is interested in pharmacoeconomics in Oncology? Guidelines for the Application of Pharmacoeconomic Research to Clinical Practice in Oncology: Consensus Conference. New York NY, October 1997.
26. Keynote speaker, Tumors of the small intestine. Israel Gastroenterology Association, Herzliya, Israel, June 1998.
27. Recurrence of adenomatous polyps. Medical Grand Rounds, Ichilov Hospital, Tel Aviv, Israel, June 1998.
28. Genetics of colon cancer. Regional Postgraduate Course, American College of Gastroenterology, New York NY, June 1998.
29. Epidemiology of pancreatic cancer. Pancreatic Cancer: Recent advances in staging and treatment. Memorial Sloan-Kettering Cancer Center, New York, NY, November 1998.
30. Ten dos and don'ts for success in a career in preventive oncology. Keynote speaker, Annual Meeting NCI R25/K07 Awardee, Bethesda, MD, March 2000.
31. Why is small bowel cancer so rare? Israeli-Palestinian Conference on Gastrointestinal Cancer, Palestine Council of Health/Israeli Gastroenterology Association/Middle East Cancer Consortium, Ramallah, April 2000.



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32. Colon cancer: Genetic (family) risk and the importance of early screening. Rudin Lectures for the Public on Cancer Prevention and Treatment, New York Academy of Medicine, New York, NY, June 2000.
33. Participant, Pancreatic Cancer Progress Review Group, National Cancer Institute, Chantilly, VA, September 2000.
34. Current status of colorectal cancer screening. Surgery Grand Rounds, St. Vincent's Hospital, Bridgeport, CT, November, 2000.
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.
36. AGG - An economic model for resource allocation in cancer research and preventive oncology. Keynote Presidential Address. 25<sup>th</sup> Annual Meeting of the American Society of Preventive Oncology, New York, NY, March, 2001.
37. An update on colorectal cancer prevention and screening. American Cancer Society Primary Care Physician Educational Dinner, New York, NY, March 2001.
38. Use of tumor registry data for human cancer epidemiology research. Keynote Address, Companion Animal Tumor Registry Workshop, New York, April 2001.
39. Use of large data sets in cancer research. Team Rounds, Department of Medicine, Columbia Presbyterian Medical Center, New York, May 2001.
40. Participant, Workshop on Exploring the Role of Cancer Centers for Integration of Aging and Cancer Research, National Institute on Aging/National Cancer Institute, Bethesda, Maryland, June 2001.
41. Cancer Screening. Medicine Grand Rounds, Mercy Medical Center, Rockville Centre, New York, November 2001.
42. Use of large data sets for cancer research. Epithelial Carcinogenesis Seminar Series, Strang Cancer Prevention Center, New York, NY, November 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 Malignancies can be Presented and Treated from the Bench to the Bedside. Dead Sea, Israel, January 2002.
45. What can second malignancies teach us about etiology of GI tract cancers? Dead Sea, Israel, January 2002.
46. Cancer epidemiology in humans: Overview and lessons for veterinarians. Keynote Address, 22<sup>nd</sup> Annual Conference of the Veterinary Cancer Society, New York, September 2002.
47. Who gets brain tumors and why. Brain Tumor Awareness Day. New York, November 2002. Use of large datasets in cancer research. Research Methodology Conference, Division of General Internal Medicine, Weill Cornell School of Medicine, New York, March 2003.
48. The Long Island Breast Cancer Study Project: Where do we go from here? Annual Advocacy Training Conference, National Breast Cancer Coalition Fund, Washington, DC, May 2003.
49. Increasing your awareness and reducing your cancer risk. Cancer Prevention Seminar, Weill Cornell Medical Center, New York, May 2003.
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 Cancer Institute, University of Scranton, Scranton, PA, June 2003.
52. Lessons from second malignancies about the etiology of GI tract cancer. Grand Rounds, Division of Gastroenterology, Weill Cornell Medical Center, New York, September 2003.
53. Epidemiology of malignant esophageal disease. Westchester Esophageal Disease Symposium, West Point NY, November 2003.



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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.
57. Participant, New Jersey Governor's Conference on Effective Partnering in Cancer Research 2004: Cancer Prevention. Cancer Institute of New Jersey, Jersey City, NJ, May 2004.
58. Second cancers among long-term survivors of cancer. Education session on Problems Unique to Long-Term Survivors of Cancer, 40<sup>th</sup> Annual Meeting of American Society of Clinical Oncology, New Orleans LA, June 2004.
59. Co-Chair, Cancer Prevention Categorical Course: When Does a "Positive" Prevention Trial Result in Changes in Practice? 40<sup>th</sup> Annual Meeting of the American Society of Clinical Oncology, New Orleans LA, June 2004.
60. Adjuvant therapy for colorectal cancer: efficacy and problems. Second Annual Geriatric Oncology Consortium, Washington DC, September 2004.
61. Lung cancer and NSAIDs. Ibuprofen and Cancer Prevention Roundtable: Exploring the spectrum of Evidence. Wyeth Consumer Products, New York, NY, September 2004.
62. Use of epidemiologic methods to study cancer prevention and treatment. Advances in translational treatment, Columbia University Medical Center and Science office of the Embassy of Italy, New York, October 2004.
63. Hereditary colon cancer syndromes. Community conference on Jewish genetic diseases, Holy Name Hospital, Teaneck NJ, November 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.
66. Epidemiology of pancreatic cancer. Pancreas Cancer 2005: State of the Art. Scientific Conference of the Lustgarten Foundation for Pancreatic Cancer Research, Memorial Sloan-Kettering Cancer Center, New York, NY June 2005.
67. Epidemiology and screening. Evolving strategies in Prostate Cancer. NYPH, New York NY, September 2005.
68. Racial disparities in cancer treatment. Department of Medicine Grand Rounds, UMDNJ-New Jersey Medical School, Newark, NJ, March 2006.
69. Cancer screening. Mednick-Geller Memorial Lecture, Medicine Grand Rounds, South Nassau Communities Hospital, Oceanside NY, April 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.
71. Reducing the burden of colon cancer incidence and mortality: epidemiologic approaches. Keynote address: Third Annual MD/Ph.D. Student Research Symposium, Columbia University College of Physicians and Surgeons, New York NY, November 2006.
72. Chair, Symposium on Small bowel tumours: New Approaches in Management. 15<sup>th</sup> United European Gastroenterology Week, Paris France, October 2007.
73. Small bowel adenocarcinoma: an update. Symposium on Small Bowel Tumours: New Approaches in Management. 15<sup>th</sup> Annual European Gastroenterology Week, Paris France, October 2007.
74. Decline of colorectal cancer incidence and mortality. Department of Medicine Team Rounds, Columbia University College of Physicians and Surgeons, New York NY, February 2009.
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. 33<sup>rd</sup> Annual Meeting of the American Society of Preventive Oncology, Tampa FL, March 2009.
77. Cancer Screening 2009. Medical Grand Rounds, New York Downtown Hospital, New York NY, March 2009.
78. Cancer Prevention in Primary Care. Cancer Control Series, Northeast Georgia Health System, Gainesville GA, March 2009.
79. A tribute to the Accomplishments of I. Bernard Weinstein. Advances in Cancer Research – A Tribute to the Scientific Accomplishments of I. Bernard Weinstein, Herbert Irving Comprehensive Cancer Center, Columbia University Medical Center, New York, NY, May 2009.
80. Quality of Care in Cancer Treatment. Oncology Grand Rounds, Lombardi Comprehensive Cancer Center, Georgetown University Medical Center, Washington DC, July 2009.
81. Colon cancer screening. Gastroenterology and Hepatology Update. Pri-Med New York 2009, Jacob Javits Convention Center, New York NY. August 2009.
82. Moderator, Session on Whole Soy and Breast Cancer. Soy Summit: Exploration of the Health, Safety and Nutrition of Whole Soy, Columbia University Institute of Human Nutrition, New York NY, September 2009.
83. Adherence to cancer therapy-new directions. 2009 Herbert Irving Comprehensive Cancer Center Annual Retreat, Riverdale NY, October 2009.
84. Colon cancer chemotherapy. GI grand rounds, Columbia University, New York NY, October 2009.
85. Getting ahead of the curve through early detection. 8<sup>th</sup> Annual Brain Tumor Awareness Day Conference, Brain Tumor Foundation, New York NY, November 2009.
86. Medication adherence in cancer treatment. Medicine Grand Rounds, King Edward VIII Hospital, Nelson R. Mandela School of Medicine, University of KwaZuluNatal, Durban, South Africa. January 2010.
87. Strengthening your role as an “individual contributor” as you participate in team science. Twentieth Annual Special Session, NCI-funded Cancer Prevention and Control Fellows Workshop. Bethesda MD, March 2010.
88. Alcohol and cancer. Alcohol and Health Workshop, SABMiller Corporation, London UK, May 2010.
89. Mammography screening: Why it should begin at age 50. New York Cancer Society, New York NY, November 2010.
90. Clinical vs Public Health Perspectives in Medical Decisions: Cancer Screening. Department of Pathology Grand Rounds, CUMC, New York NY, December 2010.
91. Racial disparities in cancer. Black History Month, Morgan Stanley, New York NY, February 2011.
92. Controversies in cancer screening. Department of Pathology, Faculty of Health Sciences, Wits Medical School, University of Witwatersrand, 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.
95. Colonoscopy vs sigmoidoscopy: Getting it right. Symposium on Cancer Screening through the Lifecourse – the Example of Colon Cancer. Annual Meeting of the American Society of Preventive Oncology, Las Vegas NV, 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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97. Columbia-South Africa Training Program for Research on HIV-Associated Malignancies. NCI Network Meeting: Research Capacity for Africa in HIV-Associated Malignancies. NIH Campus, Bethesda MD, January 2012.
98. Design, conduct and impact of non-interventional studies. Real World and Non-Interventional Studies Training Meeting, Pfizer, New York NY, February 2012.
99. Leading a strong cancer prevention and control program: tips for successful strategies. Annual Meeting of the American Society of Preventive Oncology, Washington DC, March 2012.
100. Cancer screening. Oncology Grand Rounds. Continuum Comprehensive Cancer Center, New York NY, April 2012.
101. Cancer prevention. Seyfarth & Shaw, LLP. New York NY, April 2012.
102. Malpractice and oncology: Cases from my files, part I. Oncology Grand Rounds, Columbia University Medical Center, New York NY, May 2012.
103. Chair, Symposium on Modern Controversies in Screening. Columbia University Epidemiology Scientific Symposium, New York NY, May 2012.
104. Update on colon screening. Panel discussion on molecular diagnostics, colonoscopy compliance, and ColonSentry. Enzo Biochem, New York NY, May 2012.
105. Invited attendee, Cancer Research in Africa Conference, Africa Oxford Cancer Foundation/AORTIC/NCI/INCA, London, England, September 2012.
106. Economics of Research in Racial Disparities. Symposium on the Economics of Addressing Disparities at Cancer Centers. Annual Meeting of the Association of American Cancer Institutes, Chicago IL, October 2012.
107. Adjuvant Therapy for Colon Cancer. Session on Colorectal Cancer. The 9<sup>th</sup> Hallym-Columbia-Weill Cornell-NYP International Symposium on Novel Anti-Cancer Treatment Modalities for Gastrointestinal Cancer. Hallym University Sacred Heart Hospital, Seoul, Korea, October 2012.
108. Medical Malpractice: What Gastroenterologists Need to Know. Update in Gastroenterology, Hepatology & Nutrition, Columbia University Medical Center, New York NY, November 2012.
109. Public Health vs. Clinical Decision-Making: Colon Cancer Screening. Grand Rounds, Department of Health Evidence and Policy, Icahn School of Medicine at Mount Sinai, New York NY, February 2013.
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 Biostatistics, Karolinska Institute, Stockholm, Sweden, October 2013.
112. Colon Cancer Screening as a Model for Public Health Practice. Ecole des Hautes Etudes en Sante Publique, Paris, France, November 2013.
113. Thoughts on new trends in the management of colon cancer. Department of Epidemiology Grand Rounds, Mailman School of Public Health, Columbia University, New York NY, April 2014.
114. Cancer screening, 2014. Lesotho Medical Society, Maseru Lesotho, May 2014.
115. Lesotho and Swaziland. HICCC Cancer Center Retreat, New York NY October 2014.
116. Co-Host, Health Outcomes Research in an Era of Cost Containment. Part I: Observational Studies. Columbia University Epidemiology Scientific Symposium, Mailman School of Public Health, Columbia University, New York NY, February 2015.
117. Co-Host, Health Outcomes Research in an Era of Cost Containment. Part II: Interventions. University of Paris Descartes – INSERM. Centre de Recherche Epidemiologie et Statistique Sorbonne, Paris, France, March 2015.
118. Genetics, genomics, and cancer prevention. Session on Cancer Prevention Research: Making the Connection between Cancer and Key Risk Factors. New York State Cancer Prevention Summit: Transforming the Cancer Agenda for the Next Generation. New York NY, May 2015.



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119. Participant, Pharmaceutical Executive Roundtable on the Rise of Pharmacoepidemiology: From Risk Profiling to Value Demonstration. Hosted by Pharmaceutical Executive magazine, New York NY, June 2015.
120. Creating successful population science programs in a cancer center. Annual Meeting of the American Society of Preventive Oncology, Columbus Ohio, March 2016.
121. Clinician vs. Population Scientist: Decision-making in Cancer. Joseph A. Fraumeni Distinguished Achievement Award Lecture, Annual Meeting of the American Society of Preventive Oncology, Columbus Ohio, March 2016.
122. The Individual vs the Population: Decision-Making in Cancer. Public Forum – Cancer Survivors: Advancing Care through Research. The 2016 Annual Retreat on Cancer Research in New Jersey, Rutgers Cancer Institute of New Jersey, New Brunswick NJ, May 2016.
123. Improving Cancer Outcomes by Improving Quality of Care. State-of-the-Art Precision Medicine for Cancer Therapy in East Asia. National Taiwan University Alumni Association of North America, New York NY, October 2016.
124. Epidemiology of Cancer Metastasis: Risk Factors and Prevention. Metastatic Research Operational Meeting, Metastatic Cancer Research Task Force, Department of Defense, McLean VA, December 2016.
125. The Physician and Cancer Finances. Symposium on Financial Consequences of a Cancer Diagnosis. Annual Meeting of the American Society of Preventive Oncology, Seattle WA, March 2017.

# 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.)

Lillian Valle, Admistratrix for Estate of Andres Valle v. U.S.A., 14-cv-3590 (NYSD)

Landry v. Midstate Radiology, NNH-CV-15-6057004S (Sup. Ct. New Haven, CT)

Camhong AN, et al., Plaintiffs, v. Takeda, et al., Defendants., No. 24C12003565 (Md.Cir.Ct.)

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

Wisniewski v. Takeda Pharmaceuticals America, Inc. Court of Common Pleas of Pennsylvania, Philadelphia County, No. 1207002272.

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