EXHIBIT 51

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

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

Expert Report of Lorelei A. Mucci, ScD, MPH

July 31, 2017

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I. SUMMARY OF QUALIFICATIONS

I am the head of the cancer epidemiology and cancer prevention program and Associate Professor of Epidemiology at the Harvard T.H. Chan School of Public Health. I am also Leader of the Cancer Epidemiology Program at the Dana-Farber/Harvard Cancer Center, the largest National Cancer Institute-designated comprehensive cancer center in the United States (US).

I received a Bachelor of Science (BS) degree in Biology in 1989 from Tufts University, Medford, Massachusetts; a Master's of Public Health (MPH) in Epidemiology and Biostatistics in 1998 from Boston University School of Public Health in Boston, Massachusetts; and a Doctor of Science (ScD) degree in Epidemiology in 2003 from the Harvard T.H. Chan School of Public Health (formerly the Harvard School of Public Health). I received post-doctoral training at the Karolinska Institutet, Sweden from 2002-2003 and at Harvard from 2003-2005.

My major research interest over the past 15 years has been in cancer epidemiology, and I have been principal investigator or co-investigator on multiple national and international investigations on this topic. My funding portfolio includes grants from the Dana-Farber/Harvard Cancer Center, the National Institutes of Health, the National Cancer Institute, the US Army Prostate Cancer Research Program, as well as foundation grants including from the Prostate Cancer Foundation and the Movember Foundation.

I have authored more than 230 original research articles in peer-reviewed literature using data from both case-control and cohort studies within the US and Europe. These studies have been published in high impact medical and oncology journals including: *Journal of the National Cancer Institute*, *Journal of the American Medical Association*, *British Medical Journal*, and *Journal of Clinical Oncology*, as well as in top-tier epidemiology journals including *Epidemiology* and *American Journal of Epidemiology*. I have also authored more than 30 book chapters and reviews, and am an Editor on two textbooks: *Pathology and Epidemiology of Cancer* (Spring Press, 2017) and *Textbook of Cancer Epidemiology, 3rd edition* (Oxford University Press, in Press).

My research portfolio includes epidemiological studies focused on assessing the causal link between a variety of exposures and cancer risk and survival. The topics cover a wide spectrum of exposures, including lifestyle factors, medications, inherited genetic factors, and other biological markers. Although a major focus of my recent research has been in prostate cancer, I have published epidemiological studies on multiple cancers including breast, colon, kidney, lung, and hematologic cancers. I am currently the co-Principal Investigator (PI) of the US Health Professionals Follow-up Study, a National Cancer Institute-funded cancer epidemiology cohort study ongoing since the 1980s. I am a founding co-PI for IRONMAN: International Registry to Improve Outcomes in Men with Advanced Prostate Cancer, an initiative jointly funded by the Movember Foundation and four pharmaceutical partners to improve outcomes and survival among men with advanced prostate cancer.

I have served on a number of editorial boards for medical journals and as an invited reviewer for leading medical, epidemiological, and oncology journals including *American Journal of Epidemiology*, *British Medical Journal*, *Epidemiology*, *Journal of the American Medical Association*, *Journal of the National Cancer Institute*, and *New England Journal of Medicine*. Since 2009, I have served as Associate Editor for *Cancer Causes and Control*, an international, multidisciplinary journal focused on studies of the causes, control, and prevention of cancer. In 2017, I joined the editorial board of *The Prostate*.

I have held numerous committee assignments and served on scientific advisory boards across Harvard University, as well as nationally and internationally. Select assignments include: Scientific Advisory Board of the Environmental Protection Agency, Acrylamide Review Panel; Scientific Advisory Board and Standing Review Committee of the Prostate Cancer Foundation; Co-Director of the Integrative Molecular Epidemiology Workshop of the American Association for Cancer Research; Project Leader of the Dana-Farber/Harvard Cancer Center SPORE in Prostate Cancer; Faculty Council Member of the Harvard T.H. Chan School of Public Health; Integration Panel Member of the Congressionally Directed Medical Research Prostate Cancer

Research Program; Grant Review Panels for the National Cancer Institute/National Institutes of Health; External Advisory Board of the Pacific Northwest SPORE in Prostate Cancer; External Advisory Board of The Sidney Cancer Center at Thomas Jefferson University; Scientific Advisory Board for the Movember Global Action Plan; and Research Advisory Council for Prostate Cancer UK. A complete listing of my publications and relevant experience is provided in my *curriculum vitae* which attached as Exhibit A.

I have not testified as an expert witness. Hollingsworth LLP is compensating me at an hourly rate of $350. Unless otherwise stated, all opinions offered in this report are to a reasonable degree of medical certainty. I reserve the right to supplement my opinions as new information becomes available.

II. OVERVIEW OF THIS REPORT

I have been asked to provide a thorough review and critical evaluation of the epidemiological literature on the association between exposure to glyphosate-based herbicides and the risk of non-Hodgkin's lymphoma (NHL). I have also been asked to review the opinions proffered by four of the Plaintiffs' experts who discuss the epidemiology of glyphosate and risk of NHL, and consider whether they have applied a scientifically reliable methodology.

In synthesizing the data on this topic, I have reviewed published studies from the literature on glyphosate epidemiology, including both case-control and cohort studies, as well as meta-analyses, pooled analyses, and reports from scientific organizations. When available, I have also reviewed unpublished manuscripts, as well as abstracts and powerpoint presentations from scientific conferences. A complete listing of the materials reviewed is attached as Exhibit B. The ultimate goal of my comprehensive review has been to provide a scientific opinion as to whether the body of epidemiological literature supports a causal association between glyphosate and risk of NHL.

Based on my evaluation, it is my opinion, to a reasonable degree of scientific certainty, that the epidemiological evidence does not provide a scientific basis to support a causal relationship between exposure to glyphosate-based herbicides and the risk of NHL.

III. EXECUTIVE SUMMARY

I have divided the remaining report to cover topics relevant to assessing the epidemiology literature related to glyphosate and NHL risk. I provide an overview of conceptual and design issues in epidemiology and discuss individual epidemiology studies as well as the Plaintiffs' expert reports.

In **Sections IV to IX**, I discuss epidemiology as a scientific discipline, and describe the two most commonly used designs in epidemiological research, the cohort and case-control studies. In observational epidemiology (the only type of epidemiology utilized to study glyphosate), the researcher observes participants. This is in contrast to randomized controlled trials where the investigator assigns the exposure. As such, there are potential biases that can occur in the design and conduct of observational epidemiology studies. Some biases are avoidable, and others we can account for in statistical analyses. A well-designed and analyzed epidemiology study can provide evidence to make inferences about associations between risk factors and disease. **It is noteworthy that a positive association estimated between a risk factor and disease does not imply cause and effect**. An epidemiologist must assess the potential for bias, confounding, or chance to underlie an association. As will be described, case-control studies are more susceptible than cohort studies to systematic bias.

In **Section X**, I discuss the epidemiology of NHL, including patterns of disease across populations and a summary of established or suspected risk factors for NHL. The latter is important because of confounding, a form of bias that occurs because exposures tend to co-aggregate in people. For example, people who smoke tend also to be less likely to exercise, eat unhealthy diets, etc. In the study of glyphosate, users of this compound may also be more likely

to use other pesticides, as well as more likely to engage in specific lifestyle factors. As such, a non-causal statistical association may be induced because the risk factor under study correlates with a factor(s) that is causing the disease. One important note is that although several risk factors for NHL have been identified, a large proportion of the etiology of NHL is unknown. As such, there is concern that there are unknown, potential confounders that underlie associations.

In **Section XI**, I review the epidemiology studies of glyphosate and NHL. The strongest data are from two analyses in the Agricultural Health Study. The Agricultural Health Study is a prospective cohort of more than 50,000 licensed pesticide applicators followed prospectively since the 1990s. It was designed specifically to investigate pesticide use and farming-related factors in relation to cancer and other health outcomes. Three-quarters of the participants were exposed to glyphosate. The first published study within the Agricultural Health Study cohort on glyphosate and NHL was in 2005[1], and it found no association between ever use of glyphosate and NHL risk adjusting for a range of lifestyle factors and other pesticide use. Moreover, there was no association between amount or intensity of glyphosate use and NHL risk. This initial cohort publication was based on 92 cases. In a subsequent analysis (Alavanja *et al*, 2013[2]) in the cohort, with 7.5 additional years of follow-up for cancer incidence, the number of cases increased about 2.5-fold (N=240). In this larger study with longer follow-up, there remained no association between glyphosate exposure and NHL risk, with no evidence of dose response analysis. The 2013 analysis also provided data that showed no association between glyphosate use and NHL B-cell subtypes.

Several case-control studies have looked at the potential association between glyphosate and NHL. They all have significant issues that may influence the validity of their findings. Still, none of the studies that presented fully adjusted odds ratios, including adjusting for use of other pesticides, showed any significant positive associations with glyphosate. Many of the early case-control studies were conducted during a time-period soon after glyphosate was introduced in the market, and as such had few exposed cases and a short latency period during

which glyphosate could influence cancer risk. In the case-control design, the investigator recruits individuals who are already diagnosed with cancer, as well as individuals who at the time are cancer-free. As such, data collection is vulnerable to recall bias, since individuals who have already been diagnosed with cancer may ruminate over what caused their disease and thus recall information about their exposures differently than controls. Some of the North-American case-control studies, including those included in pooled analyses, included a large proportion of proxy respondents, because the cases or controls were deceased. The use of proxies was shown to induce recall bias and a conflation of the odds ratios. Several of the case-control publications did not adjust for confounding due to concomitant use of other pesticides, which also may have overestimated the odds ratios. Finally, some of the studies report positive association for almost all of the pesticides, which lends support for the role of systematic bias.

The cohort study is considered to produce a higher level of evidence in observational epidemiological research compared to case-control studies. Given the internal consistency of the findings from the Agricultural Health Study cohort, the many strengths of its study design and conduct, and in light of the significant problems with the case-control studies, it is my opinion within a reasonable degree of scientific certainty that the epidemiological data shows no evidence of a causal association between glyphosate and NHL risk.

IV. EPIDEMIOLOGY AS A SCIENTIFIC DISCIPLINE

Epidemiology is a scientific discipline that involves the study of the patterns and causes of disease in human populations. Epidemiology uses established research methodologies, study designs, and analytical approaches to examine whether there are different rates or patterns of disease associated with various factors. The ultimate goal is to determine whether there exists a causal association between a specific factor and the occurrence or timing of a disease.

To assist in understanding the ideal approach to epidemiology, it is helpful to make an analogy to the ideal scientific study in determining whether an exposure (e.g. tobacco) is

causally related to a disease (e.g. oral cancer). Let's imagine that we have access to a time machine. In this imaginary study, we have a group of individuals, all of whom are exposed to tobacco (of the same form, amount, and duration), and we follow them from birth to death to examine the frequency of oral cancer. Then, we send the entire group of individuals back in time using the time machine, to live the exact same life that they lived, except now none of them are exposed to tobacco. We again assess the frequency of oral cancer, and compare the frequency of oral cancer in the population when they are or are not exposed to tobacco. Since the same individuals lived identical lives, except for the presence or absence of tobacco, any difference in the frequency of oral cancer is due to tobacco. This analogy helps to define a *cause*.

If the time machine is the idealized study design to establish causality, one can ask what approach or design would represent a realistic epidemiology strategy to achieve this same goal. Randomized trials are often considered to possess the next highest level of validity for assessing causal associations. In this approach, the investigator randomly assigns who receives the exposure. If the groups are large enough, then the group randomized to the exposure should be comparable on all other factors to the group randomized to the unexposed. For example, if one undertook a randomized trial of aspirin and colon cancer, the investigator would randomly assign participants to aspirin or to the placebo, and follow them for some amount of time to examine the frequency of colon cancer. Other than aspirin, the two groups would be similar on all other factors such as smoking, diet, genetics or other factors. By randomly assigning the participants, the two groups are comparable, and the group not exposed to aspirin is a good representation for what would have happened to the group randomized to aspirin if they had not received aspirin.

Randomized trials have several strengths. However, such trials are often impractical. For example, it can be challenging, complex, and expensive to randomize individuals to specific exposures. Particularly for diseases such as cancer that have long latency periods, individuals may need to be "exposed" to the intervention for a long period of time and followed for a decade

or more to observe the occurrence of disease. Moreover, it is unethical to randomize individuals to substances that might cause harm, such as smoking.

In the setting in which a trial is not feasible or ethical, scientists rely on observational epidemiology studies to examine associations between exposure and disease. Indeed, the essence of observational epidemiology is the study of causal associations by comparing rates of disease among exposed and unexposed individuals in which the population is naturally exposed or not exposed. For example, if the incidence of disease in exposed individuals is higher than in unexposed individuals, this may suggest a positive association between the exposure and disease. There are some challenges in using observational studies versus randomized trials. These challenges can arise since people are being observed in a real-life setting. One key feature of observational epidemiology is the importance of considering and accounting for these issues through the design of the study and its data analysis. In **Section V** below, the different study designs are described in detail.

V. EPIDEMIOLOGICAL STUDY DESIGNS

There are two primary study designs that are used in observational epidemiology to compare whether the rates of disease occurrence are different among individuals with and without the exposure under investigation: **cohort studies and case-control studies**. Cohort studies are considered more reliable and less susceptible to biases, as described below.

Cohort studies

Within the family of observational epidemiology studies, cohort studies are considered to have a higher level of validity compared to the case-control design. “Cohort” derives from the Latin *cohors*, and refers to the ancient Roman military unit of 360 soldiers. In epidemiology, a cohort refers to a group of individuals who share some common feature and are studied for the development of diseases or death for some amount of time. Because the goal is to examine

patterns of risk of developing disease, cohort studies follow people who at the start of the study have not yet been diagnosed with the disease, i.e. everyone is disease free at baseline. **Figure 1** illustrates a cohort study of pesticide use and risk of NHL, in which information on pesticides (exposure) is collected at baseline and individuals are followed prospectively for the development of NHL (disease). This has several advantages compared to a case-control approach, as described below in the section on bias.

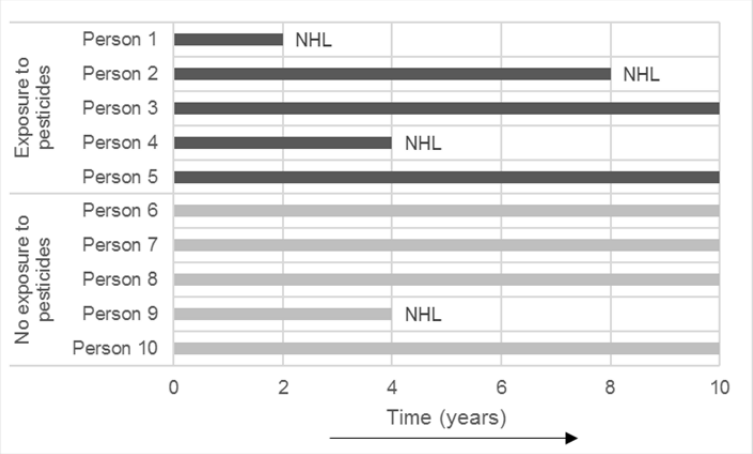

Figure 1. A hypothetical study of pesticide exposure and incidence of NHL with 10 years of follow-up.

Cohort studies allow us to compare risk or rates of disease among individuals exposed to some factor to the rate among individuals not exposed. The unexposed group is meant to represent the disease experience of what would have happened to the exposed group had they not been exposed. Thus, any excess risk in the exposed group compared to the unexposed group may be due to the exposure. However, an epidemiologist must first rule out potential bias, confounding, and chance, described in detail below, before making conclusions.

The classification of exposure can be simple, such as simply exposed or unexposed. Depending on the exposure of interest, the classification can be expanded to include consideration of current or former exposure, duration, and intensity of the exposure. Information on current and prior exposures are collected from the participants themselves at the start of the study, usually through questionnaires, interviews, or both. Some cohort studies additionally collect exposure data at follow-up points to capture exposures that may change over time. An important feature of a cohort study, as compared to a case-control study, is that the data is collected from participants prior to being diagnosed with disease. As such, the way in which

participants self-report exposures is not influenced by the experience of the disease itself. I will discuss this potential bias, known as recall bias, below in **Section VI**.

Follow-up is a key aspect in cohort studies, particularly in the study of cancer. The natural history of carcinogenesis can be long, and the time between when an exposure occurs and when a cancer is ultimately diagnosed – a concept known as the *latency period* – can be decades. As such, for most types of cancer, a cohort should be followed for many years to (1) have a sufficient number of cases, and (2) study the latency period between when an exposure happens and when cancer develops.

Case-control studies

A case-control study is another sampling strategy used in epidemiology where individuals who have a particular disease are selected and then individuals who do not have the disease, but are from the same population, are selected. In this design, individuals who have the disease are referred to as "*cases*," and individuals who do not are referred to as "*controls*. Both cases and controls are then evaluated for the presence of the exposure. The selection of the control group is a critical feature in this design. The goal is to sample the controls in a way as to represent the same population from which the cases came. Careful selection of controls will influence the validity of this study design, as the control group should provide an estimate of the exposure's prevalence in the population that gave rise to the cases.

The primary advantage of case-control studies over cohort studies is that they tend to be more time- and cost-efficient since they do not require long follow-up or as many subjects. Hence, case-control studies are often used to study diseases that are less common. However, case-control studies are more susceptible to epidemiological biases compared to cohort studies, particularly recall bias and selection bias, which will be described in detail in **Section VI**. Therefore, when feasible, cohort studies are the preferred study design when available to address a scientific question and when there are sufficient endpoints collected.

Measures of frequency and association in cohort studies

There are measures of disease frequency and association commonly used by epidemiologists in studying an exposure and disease. To illustrate these measures, I provide data from a hypothetical cohort study of 50,000 individuals on the association between pesticide exposure and risk of NHL in **Table 1**. In this hypothetical study, 10,000 individuals were exposed to pesticides at baseline and 40,000 were unexposed. During follow-up of the cohort, 600 cases of NHL were diagnosed, 400 in the exposed and 200 in the unexposed group.

	NHL cases	Disease-free	Total N
Pesticides	200	9,800	10,000
No pesticides	400	39,600	40,000
Total	**600**	**49,400**	**50,000**

Table 1. Hypothetical cohort of 50,000 individuals exposed and unexposed to pesticides.

In cohort studies, we generally consider two *measures of frequency*: **risk and rates**. "Risk" is the number of individuals newly diagnosed with a disease during a certain time divided by the number of individuals at the start of the study. In **Table 1**, this would translate to 600/50,000 = 0.012 or 1.2% over time. "Rates" are analogous to risks but integrate the number of people at the start as well as the amount of time that each person is followed in the denominator. This is an important consideration, since in many epidemiology studies, participants are followed for varying degrees of time, as illustrated in **Figure 1** above.

While measures of frequency can be useful to describe how much disease occurs in a population, epidemiologists are primarily concerned with comparing disease in different groups. *Measures of association* in cohort studies compare the risk or rate in one group relative to the risk or rate in another group. Broadly, cancer epidemiologists refer to these relative measures as the "relative risk". In the above example, we compare the risk of NHL in those exposed to pesticides (200 NHL cases /10,000 exposed at start = 0.020 = 2.0%) relative to the risk in the unexposed group (400 NHL cases / 40,000 unexposed at start = 0.010 = 1.0%): risk ratio = 2.0% / 1.0% = 2.0. The risk ratio we found tells us that the risk of developing NHL is 2-fold

higher in those exposed to pesticides compared to those not exposed to pesticides. This point estimate gives a sense of the strength (2-fold) and the direction (NHL risk was higher in the individuals exposed to pesticides) of the association.

Measures of frequency and association in case-control studies

In a case-control study, we are unable to estimate risk or rates since we are sampling on the disease itself. Instead, we estimate odds of exposure in the cases and the odds of exposure in the controls, and use these to estimate an odds ratio as a measure of association. Mathematically, the odds ratio will approximate the risk ratio if the disease is rare, and assuming no bias.

Table 2 gives a hypothetical case-control study of pesticide exposure and NHL where we included 500 NHL cases, 100 of whom report that they had been exposed to pesticides and 400 who had not, and 1,000 controls of whom 100 report the exposure. To calculate the odds ratio, we calculate the odds of exposure in the cases (100/400 = 25%) and odds of exposure in the controls (100/ 900 = 11.1%): odds ratio = 25% / 11.1% = 2.25. The odds ratio we found tells us that the odds of developing NHL is 2.25-fold higher in those exposed to pesticides compared to those not exposed to pesticides.

	NHL cases	Controls
Pesticides	100	100
No pesticides	400	900
Total	**500**	**1,000**

Table 2. Hypothetical case-control study of pesticides and NHL

Interpreting the relative risk

If the risk of disease is the same in the two exposure groups, the measure of association (relative risk) will equal 1.0, referred to as the *null value*. A finding of 1.0 for a relative risk indicates no association between the exposure and the disease since the risks are similar in the exposed and unexposed groups; a relative risk greater than 1.0 suggests positive association; a

relative risk of less than 1.0 suggests a negative association. In addition to the relative risk, we estimate the 95% confidence interval for the point estimate. This will be discussed in more detail in **Section VII**: The role of chance in epidemiology.

Definition of exposure

In cohort and case-control studies, how the exposure is defined and measured is critical to the interpretation and application of the study findings. In the earlier examples, the exposure categories were “pesticides” and “no pesticides.” In practice, this may not be a well-defined exposure because it categorizes into one group as “exposed” individuals, who may have been in contact with pesticides once during their lifetime, with those exposed regularly for a long time. In epidemiology, we often want to know not only if someone is exposed, but also about the dose, duration, or intensity of the exposure, all of which may play different roles in disease risk. For example, exposure during a day of pesticide application can vary considerably. Biomonitoring data from The Farm Family Exposure Study showed that during one day, the number of acres treated and the pounds of active compound used can vary more than 40-fold across individuals.[3]

The method in which the exposure is assessed is also a determinant of how an exposure is defined. For example, the primary way that pesticides have been assessed in epidemiology studies has been by self-report, which means that individuals answer questions about their own exposure by questionnaire or interview. An important question is how accurately one reports on details of exposure, and in case-control studies, does being a case (i.e. having cancer) influence self-reported exposures differently than controls. These issues are discussed below in the context of systematic biases in **Section VI**.

There are many possible ways in which the level of an exposure affects the process of disease development, and this concept is referred to as dose-response. Studies that can show

a clear dose-response relationship provide stronger evidence that an observed association is causal.

VI. SYSTEMATIC BIAS IN EPIDEMIOLOGY

After an epidemiological study has been designed and conducted, the data are analyzed to generate a measure of association. However, a numerical association does not necessarily imply that there is a causal association between an exposure and outcome. An epidemiologist must critically evaluate whether this association is real or if there are potentially alternative explanations that have led to a spurious finding. Before an association can be considered causal, an epidemiologist must consider what the role of confounding, bias and/or chance are, if at all.

In this section, I discuss the role of systematic biases including confounding, misclassification, recall bias, and selection bias. In **Section VII**, I discuss the concept of 95% confidence intervals and how these are used to assess the role of chance in epidemiological studies.

Bias due to confounding

Confounding derives from the Latin word *confundere,* meaning to mix together. It occurs because exposures tend to co-occur so that the effect of the exposure on an outcome is mixed with the effect of the confounder and outcome. The distortion that can result from confounding may be large, depending on the prevalence of the confounder and how strongly it is linked with both the exposure and outcome. It may cause a relative risk to be overestimated or underestimated. If a confounder is not accounted for, a false statistical association may result. Indeed, even if there is no causal association between an exposure and disease, unmeasured confounding can lead to an apparent association between a risk factor and disease.

A noteworthy example of confounding was in the initial studies that reported a positive association between coffee and heart disease risk. It was later determined that the risk for heart disease was elevated in coffee drinkers because they also tended to be smokers and smoking is strongly associated with heart disease risk.[4] It appeared that coffee was associated with heart disease, but only because of the link between coffee and smoking. It was smoking (the confounder), and not coffee, that caused the disease in the coffee drinkers.

Relevant to this report, farmers who use glyphosate are also more likely to use other pesticides and have other exposures associated with farming, some of which may have the potential to cause NHL or may be statistically associated. This is a concern, as described more fully below in **Section X**, as prior studies have found positive associations between farming and NHL risk, and these studies were done prior to the introduction of glyphosate.

Although confounding is common in epidemiology, there are appropriate methodologies in the design and analysis to limit or account for the bias. In case-control studies, investigators will often match cases and controls on factors such as age and sex; while matching does not eliminate confounding *per se*, it allows the investigator to efficiently account for confounders in the analysis. In both cohort and case-control studies, an epidemiologist should collect detailed information on known potential confounding factors. After the study is completed, epidemiologists have statistical tools including stratifying on confounding factors (i.e. conducting separate analyses on subgroups of the population who are the same with respect to confounding factor, also known as *stratification*) or the use of statistical (*multivariable*) models that enable them to account for the effect of confounding.

An important step in the statistical analysis is the choice of which variables to include in a multivariable model, since failing to include an important confounding factor could result in a biased finding. A general approach is to include all factors that are associated with the exposure and with the outcome of interest (independently of the exposure). These associations can be verified empirically using the data in the study. Expert knowledge of the exposure-disease

relationship and the specific population or environment under study is also important in building the multivariable model. If many studies have been conducted looking at a specific research question, there may be scientific consensus about what factors are important confounders. In some studies, the epidemiologist will present different statistical models: the “crude” or unadjusted model, a model only adjusted for age or a small set of variables, and a “fully” adjusted model with the final set of covariates. The crude and age-adjusted models are presented to get a sense of the extent that confounding had on the exposure-disease association. This can also help to understand the potential for residual or unmeasured confounding. Because of the large role that confounding can play in an observed association, epidemiologists do not generally consider the relative risk from a crude or crudely-adjusted model as causal.

Statistical methods to adjust for confounding may be only partially successful in eliminating this bias for a couple of reasons. First, if an epidemiologist does not collect data on specific variables on the questionnaire or decides not to evaluate them in the statistical model, then confounding may still bias the relative risk, resulting in *residual confounding*. Second, residual confounding can occur if the confounder is not well measured. For example, suppose that a study is examining the association between physical activity and risk of lung cancer, and the researcher adjusts for smokers/nonsmokers. Because heavy smokers are less likely to exercise than light smokers, there may still be residual confounding due to the amount of smoking.

Misclassification Bias

Data on exposures, outcomes and confounders can be collected from participants through questionnaires, interviews, medical records, registries, and biological specimens. The quality and validity of data is essential to the validity of epidemiological studies. Epidemiological studies can be susceptible to misclassification of data, in both case control and cohort studies. For

example, if information on an exposure is collected by questionnaires, individuals may misremember how often they do something (e.g. how often they ate broccoli in the past month) or the timing of the exposure (e.g. when they had their last colonoscopy).

This misreporting leads to a misclassification such that individuals who are exposed are classified as unexposed and vice versa. If the individuals who have the disease misremember as frequently as individuals without disease, then the misclassification of the exposure is said to be random or non-differential with respect to the outcome. Even when individuals are more likely to over-report the exposure than under-report, it will be non-differential if this misreporting is independent of whether someone has the disease. When there are only two levels of an exposure, e.g. exposed or not exposed, then the misclassification will tend to bias the relative risk estimate toward the null value of 1.0. If there are more than two levels of exposure, however, then the direction of bias from misclassification is challenging to predict.

Recall Bias

Recall bias is a specific form of misclassification bias that is always differential. It occurs because the individuals who already have a disease, such as cancer, remember their exposure history differently than healthy controls. Because of the design of case-control studies, cases already have been diagnosed with cancer when they are asked to report on their prior exposures. The diagnosis of a serious illness such as cancer often leads patients to speculate on possible contributing causes of their illness, and this speculation may affect or distort their recall of details of their exposures in the past. Epidemiologists also refer to this as *rumination* by patients, whereas healthy controls do not have the same experience. Because in cohort studies individuals are reporting their exposure prior to the development of disease, recall bias is generally restricted to case-control studies. Unfortunately, there is no way to adjust for recall bias after a case-control study has been conducted.

A classic example of recall bias is in case-control studies of congenital malformations, whereby mothers of infants who have a malformation (cases) will ruminate over potential exposures they had during pregnancy and recall things differently than mothers of infants without malformations (controls)[5].

Similarly, a cancer diagnosis can represent a stressful time when a patient may look back to try and understand what caused his or her disease. An example outlining the effect of recall bias comes from a study of dietary fat and breast cancer in the Nurses' Health Study[6], a prospective cohort of women. In the cohort, investigators collected data on dietary fat intake in women in the cohort at baseline, before any cancer diagnoses, and followed them prospectively for breast cancer such that the reporting of dietary fat occurred without a woman knowing whether she would develop breast cancer in the future. The investigators then undertook a case-control study within the same cohort of women – they selected women who had developed breast cancer (cases) and those that had not (controls), and asked them to think back and report on their fat intake at baseline. The retrospective case-control analysis in the Nurses Health Study yielded an odds ratio of 1.4 for the association between total dietary fat and breast cancer. In contrast, the prospective cohort analysis in the same women when the data was collected at baseline showed no association between total fat and breast cancer. These data show the recall bias inherent in case-control studies, as cases in the case-control study were more likely to report a higher baseline intake of total fat after they were diagnosed with breast cancer than what they reported at baseline in the cohort study when they were cancer-free.

Use of proxy respondents in case-control studies

In some case-control studies, investigators cannot collect information directly from the study participants, either because the participant has since died or is infirmed. In this

case, investigators will sometimes collect information from *proxy respondents*, often spouses or next of kin of the participant. The collection of risk factor or exposure data from proxies can result in misclassification of information that can be even greater than the potential of misclassification from the study participant himself. In particular, the proxies for the cases are also more likely to ruminate about what caused the cancer or disease in their loved ones, resulting in the potential for an extreme recall bias.

As described below in **Section XI**, several publications of glyphosate and NHL included proxies: Cantor *et al* (1992)[7]; Waddell *et al* (2001)[8]; De Roos *et al* (2003)[9]; and the North American Pooled Project (NAPP, Pahwa 2015). For example, Waddell *et al* examined the association between organophosphates and NHL risk separately for data collected by individual participants versus proxies and found a strong positive association was restricted to proxy respondents with relative risk = 3.0 in proxy vs. 1.2 in direct respondents[8]. Similarly, in stratified analyses in the NAPP data (slide 26), Pahwa *et al* (2015) show that the odds ratios for associations between glyphosate and NHL are attenuated in the analysis based only on self-reported data compared to that including data from both proxy and self-report.

Selection bias

Another type of bias that can occur in epidemiological studies is selection bias. Selection bias can occur in several different ways in epidemiological research, but generally relates to a bias in the approach that an investigator takes in selecting participants into a study, or approaches of the follow-up. The key feature of selection bias is that the selection is also related to the exposure and/or outcome of the study.

Case-control studies are more susceptible to selection bias than cohort studies. The purpose of the controls in a case-control study is to give information on the frequency of the exposure in the population from which the cases come. Sometimes the

controls can be sampled in a way that over- or under-estimates the frequency of exposure, and this will then lead to a selection bias. For example, cases may be more likely to participate in an epidemiological study than controls since they are more motivated. This was the case in McDuffie *et al* (2001), in which 67% of cases and 41% of controls agreed to take part in the study.[10] If the reason for controls not being willing to participate is related to the exposure under study, then selection bias can result.

VII. THE ROLE OF CHANCE IN EPIDEMIOLOGY

Chance events, as in life, can occur in all forms of science including epidemiology. For example, we can flip a fair coin that has a $\frac{1}{2}$ or 50% chance of landing on heads and 50% tails. Let's suppose that we are interested in the probability of landing on tails. Intuitively, we know that if we flipped the coin 4 times and recorded each outcome, we would not always get a probability of 50% (2 tails, 2 heads). We could get 4 tails in a row by chance, and the probability of this occurring would be $\frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} \times \frac{1}{2} = \frac{1}{16}$ or 6.25%. However, the more coin flips we do, the more likely it is that we will get 50% tails. In other words, we can think of 50% as the long-run probability of tails, even though we might need to do hundreds of flips to get an estimate that is close to this. In this example, the probability of tails we find in our experiment is our *point estimate*, and we would say that an experiment with 1,000 coin flips has greater *precision* than an experiment with 10 coin flips.

In epidemiology, we estimate the role of chance by calculating a *95% confidence interval*. The confidence interval represents a reasonable range of values for the point estimate based on information from the study and gives us a sense of how likely chance is in finding an association. Two main features we consider when we evaluate a confidence interval include: (1) whether or not the interval includes the null value (e.g. relative risk of 1); and (2) how wide or narrow the interval. For example, in Hardell *et al* (1999)[11], the odds ratio for glyphosate and

NHL was 2.3 and 95% confidence interval was 0.4 to 13. Although the point estimate was well above 1, the confidence interval is quite wide and includes a negative association, as well as the null value of 1.0. It is important to note that implicit in the interpretation of point estimates and 95% confidence intervals is that bias and confounding have been ruled out, which is not the case in Hardell.

A related idea is that of the *power* of a study. Power is the probability that a study will find an association between the exposure and disease if there *truly is an association* (i.e. if there is not bias or confounding). The power of a study can be assessed by the width of the confidence intervals, such that wider confidence intervals imply a study that lacks power. Several factors can influence a study's power. A-) Sample size: A study with a large sample size will generally have greater power than a study with a small sample size. In addition to the total number of individuals, the number of individuals who develop the disease of interest also influences the power of a study. B-) Prevalence of the exposure: The power increases with a higher prevalence of exposure. This is one reason that epidemiologic studies may focus on individuals with a specific occupation (e.g. farmers), since exposures such as pesticides may be rare or very low in the general population. The point of prevalence of exposure is particularly critical and cannot counteract a large sample size. For example, in Hardell *et al* (1999), although there were 404 cases, only 4 were exposed.[11] This point is somewhat overlooked in the expert report of Dr. Ritz. For example, she presents only on the total number of cases in the study in table on page 15 of the report, and ironically, the study with the largest number of cases is Cocco *et al* (2013)[12] with 1,869 cases of whom only 2 were exposed to glyphosate.

If any of the biases discussed in the previous section are present, researchers will not get the correct answer when they calculate the measure of association and 95% confidence interval. Thus, if an epidemiological study has systematic bias, it is not helpful to evaluate the role of chance. In other words, a biased point estimate is not informative regardless of how precise the estimate is or how much power the study had. If all sources of bias have been

eliminated either in the study design or in the analysis of the study data, then the role of chance in the study findings can and should be evaluated.

VIII. META- AND POOLED ANALYSES

As described above, statistical power in epidemiological studies is driven by the study size, the number of endpoints, and the prevalence of the exposure. Well-designed studies individually can suffer from low power, which may result in a false positive or false negative finding. However, there are two techniques used by epidemiologists to combine the results of multiple studies together in order to increase statistical precision of an exposure-disease: meta-analysis and pooled analysis. **It is critical that the interpretation of results from meta- and pooled analyses is founded on the individual studies being free of bias and confounding.**

A meta-analysis uses a statistical approach to summarize relative risk estimates across multiple studies. The weighting considers the sample size of each study, with relative risk estimates from the larger studies and particularly the larger number of exposed cases contributing more to the meta-analysis estimate. The relative risks and 95% confidence intervals are taken from each original study manuscript. Ideally, a meta-analysis is done after a systematic review of epidemiological studies to identify all relevant studies.

A strength of meta-analyses is that they can overcome the issues of power in studies that are valid but have small sample sizes or rare outcomes. However, there are key limitations. A meta-analysis assumes that the individual studies included are devoid of bias and confounding.[13] The analytic approach used in meta-analyses cannot eliminate biases from poorly conducted individual studies. “Garbage in, garbage out” means that the validity of the summary relative risk estimate in a meta-analysis will be biased if the individual studies going into it are biased, even if it generates a very precise estimate. Meta-analyses may also be influenced by reporting bias, if it includes only published studies, since these are more likely to have positive findings.[13,14]

While a meta-analysis combines study-level data to generate a summary relative risk estimate, a pooled analysis aggregates the individual patient-level data from epidemiological studies to generate this estimate. In other words, in a pooled analysis, one pulls out study participants and their data out of the original study and develops a combined dataset. The goal of a pooled analysis is to re-analyze the primary data in order to provide a more uniform definition of exposure and outcome across studies, as well as to use similar approaches to adjust for confounding factors. As with meta-analyses, a pooled analysis can provide increased statistical power for an association between an exposure and disease. A key issue in conducting a pooled analysis is the importance of providing a clear description of what studies are being included, and whether there are inclusion/exclusion criteria for the study participants, particularly if distinct from the original publications. While it has some advantages over a meta-analysis because it can undertake a standard approach for dealing with confounding, a pooled analysis still cannot remove other biases inherent in the original study due to unmeasured confounders, selection bias, recall bias, or misclassification.

IX. CAUSAL INFERENCE

There is consensus among epidemiologists that a decision about whether an observed statistical association represents a causal relationship between exposure and disease requires more evidence than a single epidemiological study. The generally accepted first step is a systematic review of the relevant epidemiological data. This type of review should involve careful evaluation of each epidemiology study to identify potential systematic biases (**Section VI**) and assess the statistical power and role of chance (**Section VII**). It is important to reiterate that an assessment of statistical power is meaningless if there is evidence of systematic bias, since a very precise but biased estimate of association is still not meaningful.

Epidemiologists use expert subject area knowledge in combination with a close examination of the methodology and data of epidemiological studies to assess the validity of

study findings. However, even with these resources, it cannot be empirically proven that all bias has been eliminated and that an observed association in a study is causal. For this reason, we look to see if findings have been replicated in different populations. **Importantly—and this is often overlooked—this requires multiple well-conducted studies, since two studies arriving at the same conclusion through poor methodology do not augment the evidence.** Because different study designs can be prone to different types of bias, it can be useful to have evidence from a variety of types of study. Epidemiologists also use other methodologies, including pooled and meta-analysis (**Section VIII**), to compile information across studies. An observed association that has been found repeatedly in multiple, well-conducted studies is considered to have stronger evidence.

In 1965, epidemiologist Sir Austin Bradford Hill proposed a methodology, including nine characteristics, to evaluate evidence of causation[15] (**Table 3**). While these should not (and were not intended to) be used as a checklist, they provide useful questions to consider in evaluating a body of scientific evidence. A few of the proposed characteristics merit particular attention. In epidemiology, the only characteristic that is considered to be truly required for an association to be causal is temporality. Since by definition a cause must precede an effect, an exposure must occur before development of the disease in order to have caused it. This is a key reason that prospective studies, such as cohort studies, are generally given more weight than retrospective studies—because temporality can only be firmly established if the exposure is measured prior to the occurrence of disease. As part of this is the concept of latency, or the time between when an exposure occurs and when the disease results. Most epidemiologists would agree that cancer risk factors require a latency of several years to decades in order to have an effect. In the Plaintiffs' expert report of Dr. Ritz,

Strength	*How strong is the effect (ex. RR)?*
Consistency	*Has the effect been seen by others?*
Specificity	*Does exposure lead only to the disease?*
Temporality	*Did exposure precede the disease?*
Biologic gradient	*Does more exposure result in more of the disease?*
Plausibility	*Does the association make sense?*
Coherence	*Is the association consistent with available evidence?*
Experimental evidence	*Has a randomized controlled trial been done?*
Analogy	*Is the association similar to others?*

Table 3. Summary of guidelines for inferring causality proposed by Bradford Hill.

she mentions a reasonable minimum latency of 10 years for NHL risk. Strength of the study in epidemiology refers to the size of the relative risk, and larger relative risks can be more supportive of causal associations than weaker relative risks, with the idea being that bias may be less likely to explain such a large effect. Strength should be taken in the context also of the 95% confidence interval. Plausibility, such as a known biologic mechanism, is an important characteristic in evaluating epidemiologic evidence. However, such an evaluation depends on current scientific knowledge and so cannot be taken as a requirement. Consistency refers to whether the association has been seen in other studies, preferably across multiple study designs. Consistency, however, is predicated on the fact that the associations in these studies should be free of any bias or confounding. As such, consistency across multiple studies with evidence of bias does not imply causality. This is an important point, and will be discussed at the end of my Report as both Dr. Ritz and Dr. Neugut in their expert reports invoke consistency as the key metric in assessing the epidemiological literature on glyphosate and NHL risk. Finally, biologic gradient is analogous to dose response, with the idea that a stronger relative risk for higher levels of an exposure would support causality. While this may be the case, it is also critical to evaluate whether bias could have induced an apparent gradient when one does not truly exist.

X. EPIDEMIOLOGY OF NON-HODGKIN LYMPHOMA (NHL)

The most common form of blood cancer is lymphoma, which is comprised of both non-Hodgkin lymphoma (NHL) and Hodgkin lymphoma. NHL itself includes more than 60 discrete subtypes, each of which has a heterogeneous and unique biological, clinical and epidemiological[16] profile. NHL often presents clinically in the lymph nodes or other components of the lymphatic system, and is characterized by the overproduction of specific cell types of lymphocytes, including B-cells, T-cells and natural killer (NK) cells. In the US, the most common NHL subtypes, representing 90% of NHL, arise in B-cells.

In the US, NHL is the sixth most common cancer among men and women, with an estimated 72,240 cases to be diagnosed in 2017. NHL represents 4% of all cancers diagnosed, and has a lifetime risk of 1 in 50. Although certain NHL subtypes are among the most common childhood cancers, more than 95% of cases occur in adults, with a median age of diagnosis in the US of 66 years. The risk of NHL is higher among men than women, although some subtypes are more commonly diagnosed in women. Globally, 385,741 NHL cases were diagnosed in 2012, with an age standardized rate of 5.0 per 100,000 individuals. 200,000 individuals die of NHL globally including an estimated 20,140 in the US in 2017. The 5-year survival rate for all NHL subtypes combined is 70%.

The risk of NHL is higher among men than women, although this varies by subtype. In the US, whites are more likely than blacks and Asians to be diagnosed. NHL risk is higher among individuals with a family history of NHL and other lymphoid cancers in either parents or siblings. Some of this family history is attributed to inherited genetic factors.

Risk factors for NHL

There are several well-established risk factors for NHL. Many of these factors have a low prevalence, and thus do not account for a large proportion of NHL cases. As such, a large proportion of the etiology of NHL is still unknown.

Diseases leading to immunosuppression are strongly associated with risk of NHL. NHL is an AIDS-defining cancer, and is among the more common cancers occurring in recipients of organ transplants. Autoimmune conditions, such as lupus and rheumatoid arthritis, are positively associated with NHL risk.[17] Presence of allergies, hay fever, and atopic diseases have been associated with a lower risk of specific NHL subtypes in some studies, including in the Agricultural Health Study cohort.[18] Epidemiological studies have identified several infectious agents that are associated with increased risks of specific subtypes, including human T-cell lymphotropic virus-type 1 (HTLV-1) which was one of the first infections linked to a cancer[19].

As with all cancers, an understanding of the timing of exposures or the length of the latency period from when an exposure happens to when NHL is diagnosed is key. It is important to note that the epidemiology of NHL is still emerging, and many of the risk factors are either quite rare or have only more recently been identified. This latter point is important as many of the earlier epidemiological studies of NHL did not account for factors such as smoking or obesity, raising concerns for potential confounding in their associations.

Association between farming and NHL risk

Several case-control and cohort studies have examined whether farming is a risk factor for NHL, many of which were conducted prior to the introduction of glyphosate. Several of these have suggested that farmers have a small but significant increased risk of NHL.[7,20-22] Moreover, growing up on a farm is associated with a higher risk of NHL.[18] In an analysis from the InterLymph Consortium, specific types of farming were positively associated with NHL risk, with odds ratio of 1.26 (95% CI 1.05-1.51) for field crop and vegetable farmers and 1.19 (95% CI 1.03-1.37) for general farm workers, but not for animal farming.[23]

Many of these studies were conducted in populations working in farms who would have been exposed prior to the introduction of glyphosate based products.[20,24] Farmers are exposed to a variety of factors which may account for this positive association, including exposure to a range of pesticides, as well as exposures such as diesel fumes and sun exposure. These farm studies highlight the potential role of unknown confounding factors in the association of specific compounds and risk of NHL.

XI. EPIDEMIOLOGY OF GLYPHOSATE-BASED HERBICIDES AND NHL RISK

Below is an assessment of the epidemiology studies of glyphosate-based herbicides and risk of NHL. Given the superiority of the cohort over the case-control design, as well as the concerns around the quality of the case-control studies of glyphosate and NHL, I first present an overview

of the findings from cohort and then case-control studies. **Figure 2** presents a timeline of the period in which cases were diagnosed with NHL in each of the cohort and case-control studies, in relation to when glyphosate was approved for agricultural use. In addition, the grey arrow represents the window of time in which there would have been at least 10 years of sufficient latency between glyphosate exposure and cancer incidence, assuming that everyone who used glyphosate began using it on the first day of exposure. This figure helps illustrate some of the issues around sufficient latency periods in the study of Cantor *et al*, as well as the pooled analyses of De Roos *et al* (2003) and Pahwa *et al* (2015), and illustrates why it is unlikely that the cases in these studies can be attributed to glyphosate exposure. It also illustrates that the updated analysis in the only cohort study of Alavanja *et al* has the longest potential latency period of any of the studies.

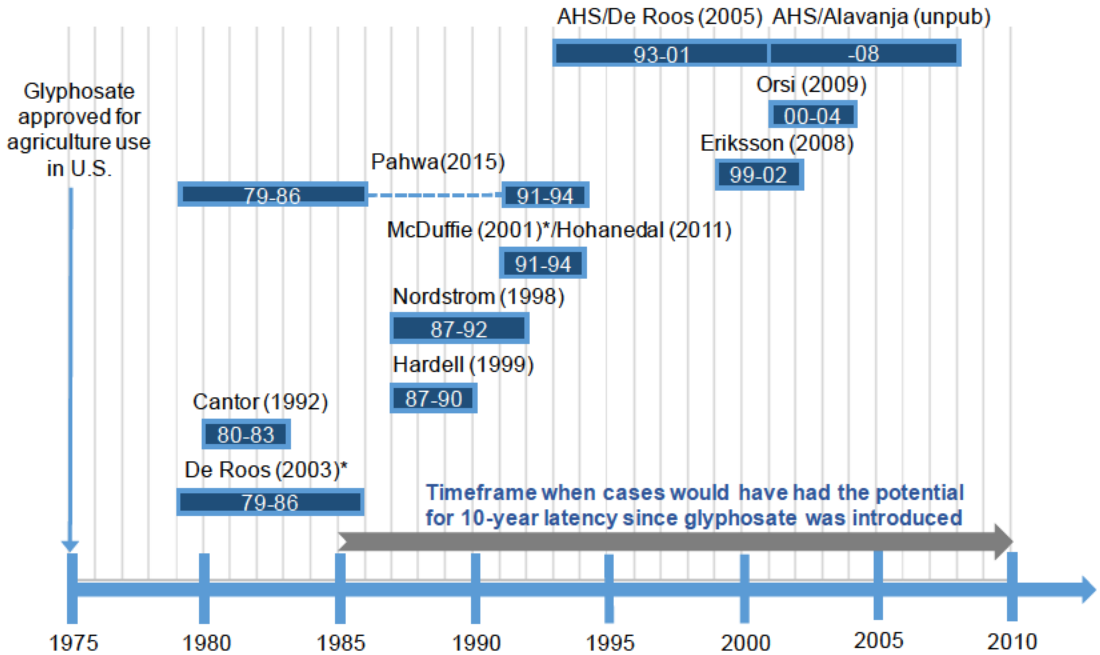

Figure 2. Comparison of timeframe for case diagnoses in epidemiology studies of glyphosate and NHL risk in reference to when glyphosate was approved for agricultural use in the U.S. The grey arrow represents the timeframe when there would have been the potential for a 10-year latency assuming individuals started using glyphosate immediately after it was introduced.

Cohort study in Agricultural Health Study

De Roos *et al*, 2005[1]. The only cohort data published on the topic to date is by De Roos *et al*, who examined the association between exposure to glyphosate and cancer risk within the US Agricultural Health Study.[1] The Agricultural Health Study is a prospective cohort of pesticide applicators residing in Iowa and North Carolina funded by the National Institutes of Health. The cohort was specifically designed to investigate and quantify potential associations between pesticide exposures and risk of cancer as well as non-cancer endpoints.[25] Participants completed a self-reported questionnaire on lifestyle factors and use of pesticides at baseline between 1993 and 1997, before any cancer diagnoses. By collecting data on pesticides and other exposures prior to the onset of any cancers, the cohort study design avoids potential recall bias which is inherent in case-control studies.[25]

The analysis included 54,315 men and women, although statistical analyses adjusting for lifestyle factors (N=49,211) and for other pesticides (N=40,719) had fewer subjects because of missing data. The questionnaire collected information on ever use of 50 pesticides. For 22 of the pesticides, including glyphosate, the questionnaire additionally collected data on extent and duration of use, number of years of use, number of days of use, and intensity of use. The enrollment questionnaire captured past and current use of glyphosate, allowing for the investigators to examine potential longer-term effects of pesticides on NHL risk.

Participants were followed prospectively for cancer incidence through linkage with state cancer registries, and for vital status using state mortality files and the National Death Index. An advantage of this approach is that there is virtually complete follow-up of participants for cancer incidence and death in the Agricultural Health Study. In the case of missing information due to loss to follow-up of study participants, there can be concerns that a selection bias could be induced,[26] illustrating the importance of complete follow-up.

Exposure to glyphosate was defined in three ways: 1-) Ever vs. never; 2-) Cumulative exposure days, defined as years of use * days per year; 3-) Intensity-weighted cumulative exposure, which weighted the cumulative exposure days * intensity level of exposure. The

inclusion of both intensity and exposure days has been shown to be essential for self-reported data on pesticide use to correlate with biological markers of exposure.[27] Moreover, the internal exposure is dependent on being present during the mixing, loading and application of the pesticide.[28] More than three-quarters (75%) of the cohort reported ever using glyphosate and 45% were heavy users defined as those in the top two tertiles of cumulative exposure days of use. This high prevalence of the exposure, particularly heavy use of glyphosate, allows for estimates of associations related to different dose-responses with meaningful statistical power.

The prevalence of use of several other pesticides was considerably higher in the participants who were heavy users of glyphosate compared to never users, which highlights the importance of potential confounding of any reported glyphosate-NHL associations. For this publication, the cohort was followed prospectively through 2001 during the follow-up period, after which time 2,088 incident cancers were reported including 92 NHL cases. In multivariable models, there was no association between ever exposure to glyphosate and risk of NHL (RR 1.1, 95% CI 0.7-1.9). In analyses of cumulative exposure and intensity weighted exposure, there was no evidence of a dose-response relationship for glyphosate with NHL risk. For example, the relative risks for NHL were 0.9 (95% CI 0.5-1.6) for the highest versus lowest cumulative exposure days and 0.8 (95% CI 0.5-1.4) for those with the highest versus lowest intensity-weighted exposure days as measured by tertiles.

Potential strengths and weaknesses. The Agricultural Health Study cohort and the study by De Roos *et al* 2005 have several strengths. First is the prospective assessment of exposure information prior to diagnosis of cancer, which eliminates recall bias. Second, the authors appropriately controlled for lifestyle factors and multiple pesticide exposures in the statistical models, reducing the potential for confounding by other farming exposures associated with NHL. Third, the number of NHL cases was sufficiently large to provide reasonable statistical power. Fourth, the study had virtually complete follow-up of the cohort for cancer incidence and mortality by leveraging the high-quality statewide cancer registries and vital records[26], which

reduces selection bias. Fifth, the investigators collected information on current and past glyphosate use which allows for a sufficient latency between an exposure and the development of cancer. Finally, the cohort had a sufficient range of exposure to investigate the extremes of exposure in relation to risk of NHL, and at levels much higher than the case-control studies below.

Potential limitations in this study include the possibility of nondifferential misclassification of glyphosate exposure. However, validation studies within the Agricultural Health Study show that these licensed applicators have been shown to be able to provide reliable self-reported information in this cohort.[29] Another concern was the amount of missing data on multiple pesticides, which resulted in 13,000 of the participants' data being excluded from models that mutually adjusted for multiple compounds and could have led to bias, which resulted in an overestimate of the relative risk in multiple myeloma, for example[30,31]. Although residual confounding could remain, the authors did adjust for multiple lifestyle factors as well as concomitant use of different pesticides, substantially reducing the likelihood for unexplained confounding.

Alavanja *et al*[2]: Follow-up of De Roos 2005. Alavanja *et al*[32] drafted a report dated March 15, 2013, which presents updated follow-up of the Agricultural Health Study cohort on the association between pesticide use, including glyphosate, and risk of lymphoma. The findings on insecticides, fungicides, and fumigants and NHL were published in 2014, but the glyphosate or other herbicide results were strangely omitted.[33]

This draft report includes an additional 7 years of follow-up for cancer incidence through December 2008, resulting in 333 incident cases of NHL, compared to 92 in De Roos *et al* 2005[1]. The report uses the same epidemiological and biostatistical methodology as De Roos *et al* 2005[1]. The 333 cases are based on an updated definition of NHL which includes both multiple myeloma and plasmacytomas, whereas De Roos *et al* 2005 used an earlier NHL definition that

did not include these subtypes. Excluding these, there were 240 NHL cases in Alavanja *et al* that were defined similarly to De Roos *et al* 2005.

Cohort participants were asked to complete a follow-up questionnaire from 1998 to 2003 to update information on pesticide use since enrollment. This was completed by 63% of the original cohort. Similar dose-exposure categories (lifetime days of use, intensity-weighted days of use) were assessed. The relative risk estimates are age-adjusted and multivariable adjusted for potential confounders.

Compared to never users, there was no evidence of a positive association for exposure to glyphosate and risk of NHL, and no evidence of dose-response in the multivariable models. For example, using the newer NHL case definition, the relative risk (95% CI) estimate for the highest vs. lowest lifetime days exposed was 1.0 (95% CI 0.7-1.4) and for the highest vs. lowest level of intensity weighted days of exposure was 0.97 (95% CI 0.7-1.4). Nor was there evidence of dose-response associations. Because of the larger sample size afforded by the additional follow-up for disease incidence, the investigators examined the association between lifetime days and intensity weighted days in relation to four of the most common B-cell subtypes. Like the overall glyphosate-NHL data, the relative risk estimates or tests for dose-response trends did not support a positive association for any specific NHL subtype. In summary, there were no positive associations for glyphosate in either the overall or dose-response analyses.

Potential strengths and weaknesses. A notable strength of this report was the additional follow-up time which led to a substantial increase in the number of NHL cases, more than 2.5 times greater than that in De Roos *et al* 2005[1]. This increase enhanced the statistical power for the dose-response associations with NHL risk, and allowed for analyses outlining potential associations between glyphosate and NHL B-cell subtypes for the first time in this cohort. The additional follow-up of the cohort also provided the authors an opportunity to consider potentially longer-term effects of glyphosate on the risk of NHL– note, there did not appear to be any association even with the longer latency in this study with longer follow-up (Figure 2). Statistical

power in De Roos *et al* 2005 was one of the sole concerns stated by the Plaintiffs' experts, and this analysis by Alavanja puts those criticisms to rest. One important observation is the high prevalence of co-exposure of pesticide use, as shown in Table 4. Indeed, 52% of the NHL cases were exposed to 5 or more herbicides. This highlights the necessity of fully adjusted multivariable models to account for potential confounding. Additional strengths include the prospective collection of exposure data, the complete cohort follow-up for NHL incidence and mortality, and the inclusion of other pesticides in the multivariable models to account for potential confounding factors.

One minor weakness is that the updated analysis on glyphosate and other herbicides has not been published to date, although the findings on insecticides, fungicides, and fumigants were published[33]. However, such concerns are ameliorated by the fact that Alavanja implemented the same study design and analysis methodology of the published De Roos *et al* 2005 study as well as multiple other publications leveraging the Agricultural Health Study[33]. Although it is unclear why Alavanja and colleagues removed the herbicide results from their updated pesticide manuscript[33], its omission could induce a publication bias that most notably impacts meta-analyses. In his publication on meta-analyses in environmental epidemiology, Dr. Blair and colleagues[13] note "Publication bias: It is valuable to seek out unpublished studies, but the quality of unpublished reports must be closely scrutinized since they presumably have not undergone the same kind of peer review as published literature". Here, that concern is minimized since the methodology is the same as those studies that have undergone peer review.

Taken together, the studies by De Roos *et al* and Alavanja *et al* in the Agricultural Health Study cohort provide the highest quality epidemiological data to date on the association between glyphosate exposure and risk of NHL.

Case-control studies including pooled analyses

There have been multiple publications from case-control study data evaluating the association between glyphosate and risk of NHL. These studies were conducted in North America, Sweden, and Europe, and varied in sample size, methods for collecting data on glyphosate and other pesticides, dose-response classification, prevalence of glyphosate use in the populations, and approaches to control for confounding.

Below, I provide an overview of the design for each case-control study, a summary of findings, and a discussion of strengths and weaknesses. Note – I have decided not to include an assessment of the study by Cocco *et al*[12]. Although it is the study with the largest overall number of NHL cases, as outlined in the Plaintiffs' expert report of Dr. Ritz (page 15), there were only 4 cases and 2 controls exposed to glyphosate, making inferences on the association between glyphosate and NHL risk meaningless.

There are several common concerns with these case-control studies. First, there is considerable potential for recall bias, since the information on pesticide exposure was collected from the cases after diagnosis. Given concerns about rumination by the cases, the effect of recall bias would likely be to inflate the relative risk by some degree. Related to this, several of the early North American and Swedish studies, including the pooled analyses leveraging these data, relied on a substantial proportion of proxy respondents potentially enhancing the likelihood that recall bias led to a systematic inflation of the odds ratios. Another key issue in many of the studies is a limited set of variables considered in the multivariable models, including the lack of mutual adjustment for different pesticides, which raises concerns about residual confounding. Moreover, several of the case-control studies are uninformative because they were undertaken too soon after glyphosate was introduced to the market, and thus the average latency is too short (e.g. Cantor *et al*,1992[7], the Swedish studies Hardell & Eriksson, 1999[11] and Hardell *et al*, 2002[34], the pooled analyses of De Roos *et al*, 2003[9] and Pahwa *et al*,

2015)[35], while others had too few exposed cases to make meaningful inferences for the associations overall or dose response (e.g. Hardell & Eriksson[11], Eriksson *et al*[36]). Finally, some of the studies (e.g. McDuffie *et al,* 2001[10]; Eriksson *et al*, 2008[36]) report positive associations for almost all of the examined pesticides, which raises a red flag that systematic bias may have artificially inflated the odds ratios due to uncontrolled confounding or study design error, such as recall bias as noted by Dr. Ritz in her expert report.

North America:

There have been several publications on the results of case-control studies from the US and Canada on the association of glyphosate and NHL. The publications include two original case-control studies (Cantor *et al* and McDuffie *et al*) that reported on the association of glyphosate and NHL risk, as well three publications that pooled different case-control studies, including two (Hoar *et al*, Zahm *et al*) that did not report individually on glyphosate-NHL risk. **Figure 3** summarizes the overlap of these publications.

Original case-control study	Pahwa 2015	Hohenadel 2011	DeRoos 2003
Cantor (Iowa/MN)	X		X
Hoar (Kansas)	X		X
Zahm (Nebraska)	X		X
McDuffie (Canada)	X	X	

Figure 3. Publications from N. America on glyphosate and NHL. Studies in pooled analyses are highlighted in **BLUE**. Hoar and Zahm are shaded in GREY as individual data on glyphosate were not published.

Cantor *et al*, 1992[7]. This case-control study from the US included men age 30 or older who were diagnosed with NHL between March 1981 and October 1983 in Iowa and between October 1980 and September 1982 in Minnesota. The study was designed to look at numerous pesticide exposures. It is noteworthy that glyphosate was approved in the US for agricultural purposes in December 1975, and thus the maximum latency period this study could assess was 5 to 8 years (with much of the exposure having

shorter latencies). The glyphosate latency issue was likely not considered by the study investigators due to the many pesticides analyzed in the study.

Both living and deceased cases were eligible, and the study included 622 men (438 living and 184 deceased cases, requiring submission of exposure information by proxies). Cases were matched to controls by age, vital status, and state of residence. Exposure information was obtained by in-person structured interview, including information on sociodemographic characteristics, smoking, medical history, occupation and residential history, and family history of cancer. The response rate was high; for cases, it was 89%, and for controls it was between 77% and 79%.

The authors estimated odds ratios (ORs) and 95% confidence intervals (CI) using multivariable models adjusted for several lifestyle factors; they did not mutually adjust for other pesticides. Compared to nonfarmers, glyphosate was not associated with an increased risk of NHL (OR 1.1, 95% CI 0.7-1.9). The authors did not report on dose-response for glyphosate and NHL. Odds ratios of 1.5 or higher were observed for several pesticides groups and insecticides, relative to nonfarmers.

Potential strengths and weaknesses. A strength of the study was that the authors collected data on potential confounding variables, including smoking and family history of cancer, and adjusted for 'use of high-risk materials', including paints, benzene, and other organic solvents. However, use of other pesticides was not included in the models, leading to the potential for residual confounding. Given the high participation rates as well as the population-based design, selection bias is unlikely.

A major weakness was the large number of proxy respondents in this study. As such, the potential for misclassification of exposure is likely high. This misclassification is likely to be differential since proxies for the cases who have died may ruminate more than proxies for the controls. This point is shown clearly in the analysis of the North American Pooling Project (NAPP, Pahwa *et al* 2015[35]) which included the Cantor dataset

and found in analyses of glyphosate and NHL stratified by respondent type. The odds ratios for "respondent only" data were attenuated to the null value compared to those with both "proxies and respondents". Another weakness noted above is that the authors did not adjust for the use of other pesticides in statistical models. Since individuals use multiple pesticides and other compounds together, and some of these appear to be associated with an increased risk, there may be the potential for residual confounding. The use of interviews is a potential limitation since the interviewer is likely not blinded to case/control status and this increases the likelihood of recall bias and misclassification of the exposure. With 26 exposed cases and 49 exposed controls, the study was somewhat limited in statistical power to detect potential small associations. As noted earlier, the timing of the case-control selection in relation to when glyphosate was introduced to the market is likely too short to see a carcinogenic effect if there were one. This point on the latency period in Cantor being inadequate is also noted in Dr. Ritz's expert report (page 19), where she comments that "one would prefer a minimum latency period of on average 10 years".

De Roos *et al* 2003, Pooled Analysis.[9] De Roos (2003) is a pooled analyses of the association between glyphosate and NHL risk using data from the US combined individual case-control data of pesticides, including Cantor *et al*[7] (Iowa/Minnesota) as well as case-controls from Nebraska[37] and Kansas[38]; individual study-level data on glyphosate and NHL from Nebraska (1983-1986) and Kansas (1979-1981) have not been published. Given the dates of case-control recruitment for all three studies, the potential latency of glyphosate exposure in this analysis is quite short. *See* Figure 2 (page 30).

The pooled analysis included 650 cases (36 exposed to glyphosate) and 1,933 controls (61 exposed). Greater than 60% of the cases were from Iowa/Minnesota (Cantor *et al*). A large proportion of cases (37%) and controls (45%) had their data

contributed by proxy respondents. In the statistical analyses, the authors considered ever vs. never exposed, and adjusted for age, study site, and multiple pesticide exposures using a hierarchical clustering analysis to account for potential confounding by use of other pesticides as well as prior data known about the variable based on pesticide class and carcinogenic potential. Ever glyphosate exposure was associated with an odds ratio of 1.6 (95% CI 0.9-2.8) in the hierarchical models, compared to 2.1 (95% CI 1.1-4.0) in the logistic regression model.

Potential strengths and weaknesses. The strengths and weaknesses of this pooled analysis are similar to those discussed above in reference to Cantor *et al*. One strength of this analysis compared to Cantor was the adjustment for potential confounding by other pesticides in the hierarchical models. However, there was considerable missing data for pesticides (25%), which could have led to selection bias.

The methodology used in the three individual studies that comprise the pooled analysis could have induced bias in the study findings in a number of ways. The number of exposed cases is fairly small, and the latency time is short in all three of the studies given the time period of case-control recruitment in relation to when glyphosate was introduced to the market. It is a notable omission that in her expert report Dr. Ritz fails to mention the issue of latency in this pooled analysis of De Roos (2003), whereas she references it in describing the Cantor dataset that comprises a majority of the cases. It is perhaps surprising that the summary odds ratio for the multivariable model in the pooled analysis (OR 2.1 for the logistic regression model) was so different from that of Cantor (OR 1.1), even though such a large proportion of cases and controls in the pooled analysis were from Cantor. It is unclear what would have led to such a difference, but the difference may reflect potential selection bias that occurred due to the missing data, if the reason the data was missing was related to case-control status. More than one-third of cases and controls had data reported by proxy respondents. In a pooled analysis of

these same three original case-control studies, Waddell *et al* (2001)[8] –who examined organphosphate pesticides only– found a significant association with NHL with pesticide exposure reported by the proxy respondents (OR 3.0) and not direct reports from participants (OR 1.2), highlighting the well-known issues in recall bias through the use of proxy respondents. This issue of recall bias using proxy respondents is also demonstrated in the powerpoint presentation of results in the pooled analyses of Pahwa *et al* (see below).

Both De Roos *et al* (2003) and Waddell *et al* (2001) are pooled analyses of the same three case-control studies from Iowa/Minnesota[7], Kansas[37], and Nebraska[38]. However, the sample sizes presented in these two reports differ. In the pooled analysis of De Roos *et al*, there were 870 NHL cases and 2,569 controls whereas in Waddell *et al* there were 748 NHL cases and 2,236 controls. This issue also arises in the the North American Pooling Project (Pahwa *et al*, see below) which included 1,117 NHL cases and 3,625 controls from these three U.S. case-control studies. As a corollary, although Waddell *et al* did not report on odds ratios for glyphosate, his findings for other pesticides differed from that of De Roos *et al* notwithstanding their use of the same study populations. For example, the odds ratio for malathion and NHL risk was 1.6 (95% CI 1.2-2.2) in Waddell but 1.1 (95% CI 0.6-1.8) in De Roos. As Dr. Neugut points out in his expert report, it is imperative that methods sections of an epidemiological study clearly outline the inclusion and exclusion criteria so that the reader can clearly understand potential selection forces. This was not done in these two reports.

Lee *et al*, 2004[39]. In 2004, Lee *et al* pooled data from Cantor *et al* of Iowa/Minnesota[7] and the Nebraska case-control study[38] to examine whether the association between pesticides and risk of NHL varied among individuals with and without asthma. The study included 872 NHL cases from these studies and 2,336 controls. Of these individuals, 45 of the cases and 132 of the

controls had been told by a doctor they had asthma. Multivariable models were minimally adjusted for age, state, and proxy status. They did not adjust for use of other pesticides.

Lee *et al* reported an odds ratio for the association between glyphosate and NHL risk among individuals without asthma of 1.4 (53 exposed cases, 91 exposed controls, 95% CI 0.98-2.1) and among individuals with asthma of 1.2 (6 exposed cases, 12 exposed controls, 95% CI 0.4-3.3). The authors report positive associations between a number of pesticides and NHL risk, particularly among individuals with asthma.

Given the extensive discussion of potential biases in pooled analysis of these two studies in De Roos *et al* 2003 above and the pooled analysis of Pahwa *et al* described below, I will not repeat these here.

McDuffie *et al*, 2001[10] and Hohenadel *et al*, 2011[40]. The first publication leveraging a population-based case-control study of Canadian men on the association between glyphosate and NHL risk was McDuffie *et al* (2001)[10]. The study included 517 NHL cases and 1,506 controls during the study period 1991-1994. Hohenadel et al (2011)[40] subsequently published on the same dataset looking at the joint effects of co-exposure to pesticides on NHL risk.

Men completed a postal questionnaire with follow-up by phone to collect additional details on pesticide use for men who reported use of 10 hours or more. The investigators undertook a pilot study to assess validation of the pesticide exposure information. Statistical models were first adjusted for age and province of residence, and additionally for family history and medical variables. The models were not mutually adjusted for other pesticides, which raises concerns for residual confounding. Exposure to glyphosate was based on ever vs. never exposed, as well as a relatively crude measure of dose response based on days per year of personally mixing or applying but not on any measure of intensity. Three categories were assessed, with the highest

category of more than 2 days of use per year (compared to the cohort study of De Roos *et al* (2005), with a much higher range in the highest category of 56 or more for cumulative exposure days and 337 or more for intensity-weighted exposure days[1]).

In McDuffie *et al*, they adjusted statistical models crudely for age, province, and several health-related variables (but not for other pesticides). They found no significant association between ever use of glyphosate and NHL risk (OR 1.20, 95% CI 0.83-1.74) based on 51 exposed cases and 133 exposed controls. The investigators also reported on days of exposure; the odds ratio was 2.12 (95% CI 1.20-3.73) in men who reported using glyphosate based herbicides on two or more days per year (23 exposed cases and 36 exposed controls), adjusting for age and province but not adjusted for other pesticides. It is noteworthy that in the dose-response analyses, several herbicides, as well as individual fumigants, insecticides, and fungicides, were positively associated with NHL risk, supporting the concern of recall bias or residual confounding in this highest category given the manner in which it was assessed.

In Hohenadal *et al*, they adjusted statistical models for age, province, and use of proxy respondents. With respect to glyphosate, they presented data on the joint effects of glyphosate and malathion and found the following odds ratios: malathion alone – 1.95 (95% CI 1.29-2.93); glyphosate alone – 0.92 (95% CI 0.54-1.55); malathion and glyphosate together 2.10 (95% CI 1.31-3.37).

Potential strengths and weaknesses: Strengths of this study include the relatively large sample size of NHL cases, the use of a population-based case-control design, and the validation pilot study to validate the collection of the exposure information.

There are several limitations to consider. First, as the investigators note, delineating putative relationships between specific compounds and NHL is challenging, since subjects are exposed to a variety of pesticides. Given this, it is unclear why McDuffie *et al* did not mutually adjust for different compounds to disentangle the

individual effects in the multivariable analyses while medical variables and family history were considered. As such, there is likely to be residual confounding across the odds ratios.

The potential for residual confounding by pesticides in this data set is highlighted in Hohenadel *et al*[40]. As I describe on page 17, stratification is one approach used by epidemiologists to assess confounding. It can also be used to assess whether there is synergy or interaction between exposures. In the analysis for glyphosate alone, in which there would be no confounding by malathion exposure, there was no association with risk of NHL. The odds ratios for malathion and glyphosate together (OR 2.10) and malathion alone (OR 1.95) are qualitatively similar and there is no evidence of a synergistic effect between the two compounds based on the two statistical approaches Hohenadel used to evaluate. In her expert report (page 18), Dr. Ritz inappropriately compares the odds ratio of malathion and glyphosate together (OR 2.10) to that of glyphosate alone (OR 0.92) to suggest evidence of synergy, without considering the main effect estimate for malathion alone. This is not a valid approach to assess synergy or interaction by epidemiologists since it does not take into account the main effect of malathion alone, which appears to be driving the association. Moreover, Hohenadel *et al* state in their discussion "[i]nteraction odds ratios should be interpreted cautiously because odds ratios for most combinations are not much larger than for malathion alone and were not statistically significant, and only the combination of malathion and carbaryl appeared to have a super-additive effect".

The participation rates in McDuffie and thus Hohenadel – particularly among the controls (48%) – were very low, raising substantial concerns for selection bias; since the goal of the controls is to represent the exposure frequency in the cohort that gave rise to the cases, low participation rates imply that the exposure frequency in this group may be biased. A somewhat higher proportion of cases than controls were administered the

telephone interview to collect more detailed data on pesticide exposure. Because the interview collected data from heavier users of pesticides, this could have enhanced the likelihood of recall bias. The definition of exposure in the dose-response association is crude. McDuffie *et al* considered categories of "days per year", with an upper cutpoint of >2 days per year, which is a very narrow range and without consideration of intensity or duration. Such a cutpoint would include individuals who have only used the compound three times in one year or several times per year in 10 years. This could result in misclassification, which given the 3-levels of exposure, could over or underestimate the true odds ratio. The number of exposed cases (N=23) and controls (N=36) in McDuffie is small, and enhances the likelihood for chance findings for some of the observed associations. Given the large number of positive associations noted overall as well as in the dose response analysis, the potential for recall bias particularly in the upper categories of exposure, and confounding due to other pesticides used, interpreting the odds ratios from this study as causal raises multiple red flags.

Pahwa *et al*, 2015 Pooled Analysis. The North American Pooling Project (NAPP) is a pooled analysis of four case-control studies, described above, including the three studies in the pooled analysis by De Roos *et al* (2003) of the US studies as well as the McDuffie *et al* (2001) in the Canadian study. In 2015, Pahwa *et al* submitted a scientific abstract to the International Society for Environmental Epidemiology (ISEE) which was accepted as an oral presentation. I reviewed this abstract, as well as two versions of powerpoint presentations for the meeting (dated 8/31/2015 and 6/3/2015).

The pooled analysis increased the overall sample size from De Roos et al (2003), with 1,690 NHL cases (533 of whom had data from proxy respondents) and 5,131 controls from the original studies; because specific dose-response data were lacking from Cantor *et al* data from Iowa/Minnesota and Hoar *et al* from Kansas, the

analyses on frequency and lifetime days were restricted to the case-control data from Nebraska (Zahm *et al*) and Canada (McDuffie *et al*, 2001); the numbers in these dose response analyses are not presented. Even with 1,690 total NHL cases, only 113 had ever used glyphosate and there are smaller numbers of cases in the dose response analyses since not all of the studies had information collected.

One should be cognizant that this pooled analysis does not supersede the limitations and biases inherent in the original case-control studies. An important additional analysis in the Pahwa *et al* August 2015 presentation slides, however, was multivariable models that adjusted for use of three pesticides: 2,4-D, malathion, and dicamba. The slides including these fully adjusted models were likely not formally presented as they were included in the end of the slide deck as additional slides. In her expert report, Dr. Ritz only provides comments related to the Pahwa *et al* scientific abstract and not the more comprehensive analyses provided in the powerpoint presentation. This is important since the odds ratios in the abstract were only adjusted for age, sex, location, proxy respondent, family history, and use of protective equipment, but not for other pesticides. In contrast, the powerpoint presentation slides present multivariable models with these factors as well as adjustment for use of 2,4-D, dicamba, and malathion. Moreover, in the slide deck, Pahwa *et al* compare the fully adjusted odds ratios comparing those from both proxy and self-respondents to those of proxy alone.

In their conference abstract, Pahwa *et al* reported an odds ratio of 1.51 (95% CI 1.18-1.95) for the association between ever exposure to glyphosate and NHL risk overall in the pooled analysis. This odds ratio differs slightly from the August 2015 presentation slide deck for the ISEE meeting, with a crudely adjusted odds ratio of 1.43 (95% CI 1.11-1.83). In the model additionally adjusted for use of 2,4-D, dicamba, and malathion, the odds ratio for ever use was substantially attenuated and suggested no association with glyphosate (OR 1.13, 95% CI 0.84-1.51). Moreover, in multivariable analysis restricted to

the self-respondents, thus eliminating the recall bias associated with use of proxy respondents, the odds ratio for ever use of glyphosate was 0.95 (95% CI 0.69-1.32) in the pooled analysis. These analyses of Pahwa *et al* highlight the role of confounding in the NAPP case-control studies by concomitant use of other pesticides as well as the recall bias introduced by the high prevalence of proxy respondents.

Confounding and recall bias associated with proxy respondents are seen in other analyses. For example, in the analysis of NHL subtypes, the ISEE abstract reported an odds ratio of 2.66 (95% CI 1.61-6.00) for ever use of glyphosate and risk of the subtype DLBCL based on 45 exposed cases. In the August 2015 slide deck, the crudely adjusted odds ratio was 1.60 (95% CI 1.12-2.29) for ever use – note, what underlies the difference in the results between the abstract and the powerpoint presentation is unclear. Additionally adjusting for use of the three pesticides above attenuated the odds ratio for DLBCL to 1.23 (95% CI 0.81-1.88). After adjusting for confounding by other pesticides, the associations between ever use of glyphosate and risk of SLL (15 exposed cases, 1.79, 95% CI 0.87-3.69) and other subtypes (25 exposed cases, 1.51, 95% CI 0.87-2.60) were not statistically significant. However, the Pahwa *et al* August 2015 slide deck did not present the effect of proxy respondents for the NHL subtype analyses.

Pahwa *et al* undertook three approaches to assess potential dose-response: number of days per year (0, 1-2, 3 or more); number of years of use (0, >0-3.5, >3.5 years); and lifetime days (# years * # days/year, 0-7,>7). The number of cases and controls for these analyses were not included, nor was the number of exposed in these categories. This is important since the number of days per year and lifetime days analyses were based only on the Nebraska and Canada datasets. For the number of years of use, which included all 4 NAPP datasets, there was no association for those with the highest number of years of exposure (>3.5 years vs. 0 years) for either the crudely adjusted (1.20, 95% CI 0.82-1.75) odds ratios, those additionally adjusted for

other pesticide use (0.94, 95% CI 0.62-1.42), or additionally limiting to self-reported data only (0.78, 95% CI 0.49-1.24). The only dose-response analysis that showed a positive association with NHL risk was for number of days per year of use. Notwithstanding the limitations of this dose-response category as described above, the crudely adjusted odds ratio was 2.42 (95% CI 1.48-3.96) for >2 days per year versus 0 in the slide deck presentation (not presented in the abstract). Additionally adjusting for other pesticide use, the odds ratio was attenuated to 1.73 (95% CI 1.02-2.94). Limiting the analysis to self-respondents did not materially change the point estimate (OR 1.77, 95% CI 0.99-3.17). In the lifetime days dose-response analysis, the crudely adjusted odds ratio (OR 1.55, 95% CI 0.99-2.44) was attenuated upon adjustment for other pesticide use (OR 1.08, 95% CI 0.66-1.77) and subsequently when restricted to self-respondent data (OR 1.06, 95% CI 0.62-1.81). The number of years of use did not show evidence of positive associations for NHL risk.

In his deposition testimony, Dr. Blair recognizes the main association between glyphosate and NHL risk as null, and that the mutual adjustment for pesticides and focus on the self-respondent data was the appropriate analysis. Similarly, in her report, Dr. Ritz states “The most highly adjusted estimates (also known as the fully adjusted models) are the estimates that adjust for as many confounding variables as possible …”. As such, Dr. Ritz would likely agree that the most fully adjusted data from the Pahwa study, which adjust for confounding by other pesticides and that are restricted to the data provided by self-response, would provide the most reliable estimates. However, Dr. Ritz did not integrate these most fully adjusted estimates from Pahwa *et al*.

Potential strengths and weaknesses. The analysis by Pahwa *et al* in the NAPP attempts to undertake the most comprehensive analysis of all the North American studies. As noted in the expert report by Dr. Ritz, the pooled analyses of the four case-control studies enhances the power to look at associations between ever use of

glyphosate and risk of NHL. The power to detect dose-response is more limited, however, since the more meaningful analyses of dose-response are based solely on the data from the Nebraska and Canadian studies. One strength of the Pahwa *et al* analysis is that it sought to assess the effect of confounding due to use of other pesticides on the odds ratios for glyphosate and NHL risk; indeed, comparing the results of the fully adjusted models to those of the more crudely adjusted models shows that confounding by other pesticide use was present in the crudely adjusted results. An additional strength of Pahwa *et al* is the analysis comparing odds ratios when data from both proxy and self-respondent were included vs. self-respondent alone, to assess the potential recall bias introduced by including the proxies. Indeed, the odds ratios were attenuated to the null value (i.e. no association) in several analyses restricted to self-respondents.

As noted earlier, a pooled analysis of individual studies whose designs are inherently biased does not overcome these biases, notwithstanding the larger sample size. As shown in Figure 2 (page 30), the latency period for the three US case-control studies is short, with a maximum possible exposure time for cases of 4 to 11 years, based on years of diagnosis, but that maximum latency would assume everyone who used glyphosate had started on the day that it was approved for farming. In line with what Dr. Ritz states in her report, we would “prefer a minimum latency period of on average 10 years”. In contrast to what Dr. Ritz states, none of the three US studies can claim to have an average latency of 10 years, and only the Nebraska case-control study would have had the possibility of any cases exposed for 10 or more years.

The study with the longest possible latency is thus the Canadian study, but this is also the study most subject to selection bias, given the very low participation rates, particularly for the controls (48%). Recall bias is also a strong concern in this study, since a higher proportion of cases were administered the telephone interview to collect more detailed data on pesticide exposure.

Taken together, the Pahwa *et al* study emphasizes the bias induced in the glyphosate – NHL associations in the North American studies due to residual confounding by other pesticides, as well as recall bias due to proxy respondents.

Case-control studies in Sweden

Hardell and colleagues performed several case-control studies in Sweden and subsequently pooled analyses[11,36] that examined associations between pesticides and risk of NHL. The studies were not specifically designed to assess glyphosate exposure, and as such, the prevalence of use of glyphosate is quite low across all of the studies. In contrast to the other glyphosate studies, the Swedish studies compared those exposed to glyphosate with those who did not use any other pesticides, rather than those unexposed specifically to glyphosate. Such an approach to defining the comparison group in the Swedish studies may have led to increased residual confounding by use of other pesticides or farm exposures.

Hardell & Eriksson, 1999[11] and Hardell *et al*, 2002[34]. The first Swedish case-control study (1999) was designed to examine associations between pesticides and organic solvents and risk of NHL. The study included men from the four most northern counties and three counties in mid-Sweden. It includes 404 cases (only 4 glyphosate-exposed) diagnosed with NHL between 1987-1990. Of the cases, 192 (43%) were deceased at the time of the study initiation and required exposure information to be completed by proxy respondents. Controls (N=741, only 3 exposed) were matched to cases on age and vital status. The participants, or next of kin for deceased individuals, completed an 18-page mailed questionnaire including information on working history and exposure to different chemicals. In addition, a study interviewer supplemented the responses over the telephone, and “most subjects, both cases and controls, were interviewed in this way”.

The questionnaire was completed by 91% of cases (N=404) and a slightly lower 84% of controls, and interviews were performed 3 to 8 years after cases were diagnosed. The second Swedish publication (2002) pooled together Hardell & Eriksson (1999)[11] and a case-control study of the quite rare NHL subtype hairy cell leukemia (HCL) described in a 1998 publication[41]. This analysis included all 404 cases of NHL and their 741 matched controls, as well as 121 cases of HCL (1987-1992) and their 400 controls. Of the combined NHL and HCL cases, 8 were exposed to glyphosate and 8 controls were also exposed.

In the Hardell & Eriksson (1999) model that crudely adjusted for the matching factors, the odds ratio for ever use of glyphosate and NHL risk was 2.3 (95% CI 0.4-13), based on a very small number exposed cases (N=4) and controls (N=3) leading to very wide confidence intervals. In analyses that appeared to mutually adjust for other compounds, the odds ratios inflated and confidence intervals widened (OR 5.8, 95% CI 0.6-54), such that it is difficult to make any inferences. No dose-response relationship with glyphosate was assessed. In the crude analysis from Hardell (2002) combining NHL and HCL, glyphosate was positively associated with risk (OR 3.04, 95% CI 1.08-8.52). However, in multivariable analyses (unclear what is adjusted for), the association for glyphosate was attenuated (OR 1.85, 95% CI 0.55-6.20) with wide confidence intervals.

Potential strengths and weaknesses: Strengths of these studies are few, but include high participation rates and the use of a population-based design. The questionnaires and interviews were blinded to case/control status. However, it is unclear how many cases and controls required the telephone interview or what prompted the interview. While the interviewers were supposed to be blinded, this could introduce recall bias by probing information differently from the cases and controls. The study interviews were undertaken 3 to 8 years after cases were diagnosed. This long timeframe between diagnosis and interview enhanced the likelihood of recall bias, and also resulted in

another major limitation of the study: the large proportion of deceased cases and controls with information obtained from next of kin which can also substantially increase the likelihood of recall bias. Another major limitation is the low frequency of glyphosate exposure, with less than 1 percent of the population ever being exposed. This resulted in small number of cases and controls who reported exposure to glyphosate, an inability to investigate dose-response, and imprecise and uninterpretable estimates of association. The primary analysis only adjusted for matching factors, and as such confounding could account for some of the associations seen.

The previous study of HCL[41] found a positive association between exposure to cattle, horse, hog, poultry, and sheep with HCL risk, suggesting these may be important confounders to consider in multivariable models. However, it is unclear if these factors are adjusted for in this pooled analysis. Although latency analyses were not examined for glyphosate, the authors did examine latency for other pesticides and observed strongest associations for shorter time spans from exposure to diagnosis. For example, the association for herbicides was only statistically significant for the time period of 1-10 years from exposure to diagnosis (OR 2.53, 95% CI 1.38-4.64) and not for the time spans of 10 or more years. This consistent finding of an increased risk in the time period closest to diagnosis raises concerns about the potential for underlying systematic bias. It is unclear how many cases and controls are included and exposed in these analyses. In addition, as mentioned above, this lack of association with longer latency periods suggests that differential misclassification due to recall bias may have resulted in spurious associations.

Another limitation of these two publications is that the authors presented on these findings in previous reports and therefore multiple testing considerations should be taken into account. As currently reported, there is potential for detection of false positive findings: associations that are statistically significant due to chance. In addition,

combining the outcomes of NHL and HCL could result in misclassification of the outcome because these two disease types likely have different etiologies. HCL is generally a rare cancer, yet the number of cases and controls recruited during this time period would suggest a higher rate of HCL in Sweden than expected. This further suggests possible misclassification of HCL. Lastly, the authors examined the associations for a large number of different pesticides and observed an increased risk of NHL and HCL for all of them (though not all statistically significant). Given the likely differences in biological effects of these various compounds, this supports the role of a systematic bias in this study that may explain the positive findings.

Eriksson *et al*, 2008[36]. This case-control study from Sweden, covering 4 out of 7 health services regions, included 910 cases of B-cell, T-cell, and unspecified lymphomas diagnosed between 1999-2002. This was 78% of the 1,163 cases identified during that time period, and 91% (910/955) of eligible cases participated. 14% of cases had died or were too ill to participate, raising concerns for survivor bias, a form of selection bias. The cases were frequency-matched to controls on age and sex, with 92% of contacted controls participating in the study. Exposure was assessed similarly to the other studies in Sweden. A written questionnaire was sent to participants, with telephone interviews conducted to supplement exposure information. 29 cases (3.1%) reported any exposure to glyphosate.

In contrast with a standard epidemiological approach, the authors defined those unexposed to glyphosate as those who were unexposed to all pesticides. However, that approach does not rule out confounding, since it is likely, and as the authors state “more a rule than exception”, that individuals with glyphosate exposure also were exposed to different pesticides. In analyses not adjusted for other pesticides, there was a statistically significant increased risk of NHL for glyphosate overall (OR 2.02, 95% CI 1.10-3.71). A

number of other pesticides also showed positive associations for NHL risk overall. In multivariable analyses adjusted for other pesticides, the overall association with NHL was attenuated and no longer statistically significant (OR 1.51, 95% CI 0.77-2.94). In fact, there were no statistically significant associations for any pesticides in multivariable analyses. 17 cases (1.9%) reported more than 10 days of exposure to glyphosate. In the crudely adjusted model, the odds ratio for those with >10 days of glyphosate exposure was OR 2.36 (95% CI 1.04-5.37). The authors did not present a multivariable model fully adjusted for other pesticide use.

The authors also presented associations between glyphosate use and risk of NHL subtypes. Notwithstanding the large number of cases, the low prevalence of exposure to glyphosate is an issue for this subtype analysis – note, the number of exposed cases is not presented in the table showing the subtype results. The majority of the NHL cases were B-cell lymphomas (90%). In the crudely adjusted models, ever use of glyphosate was associated with an odds ratio of 1.87 (95% CI 0.998-3.51) of B-cell lymphoma. The authors did not present the fully adjusted models accounting for other pesticide use. It is noteworthy that the odds ratios for several of the pesticides were elevated for the B-cell lymphomas, highlighting the potential for confounding in the glyphosate analyses. For the other NHL subtypes, the results show some positive associations but need to be considered cautiously given the likely low number of exposed cases in each group (<5 cases).

Potential strengths and weaknesses: The use of population-based controls is a strength of the study and reduces the likelihood of selection bias leading to spurious results. The majority of case pathology reports underwent a histopathology review by expert pathologists, which is important given the classification of NHL subtypes. The authors comment on the high participation rates of cases and controls; however, the calculated participation rates for the cases are artificially high since they exclude deaths

and those unable to participate due to medical conditions from the denominator. Given the time period for participant recruitment, the authors have a potentially longer latency period in this time frame, although latency effects were not specifically examined. Notwithstanding this longer latency, glyphosate exposure was still quite rare in this population, with only 29 exposed cases (out of 910) and 18 exposed controls (out of 1,108).

It is important to note that most of the odds ratios for different pesticides and for different subtypes are elevated, which raises considerable concern that there is systematic bias that led to the result. As with the other case control studies mentioned in this report, recall bias is a concern. The authors state that their findings for glyphosate are "strengthened by a tendency to dose-response effect". The meaning of the dose-response analysis, however, is limited because it defined the exposure in terms of days of use rather than intensity. As I previously discussed, this method of defining exposure can lead to misclassification since the actual exposure (e.g. amount of pesticide handled) can vary widely across individuals for a given day[27]. Another limitation of this study is that the subgroup, dose-response, and multivariable analyses were not well powered as shown by the wide confidence intervals. Note: Eriksson *et al*. did not present the number of exposed cases and controls in these particular tables which makes it challenging to evaluate the reliability of these subanalyses.

The authors subdivided the data in several ways to examine various subtypes of NHL and pesticides by type of compound and duration of exposure. As noted in the other Swedish studies, the authors did not account for multiple testing in their analysis despite the large numbers of comparisons made. There are also concerns of residual confounding. Findings from this and previous studies highlight the importance of adjustment for other pesticide compounds. Eriksson *et al* showed that the association between glyphosate and NHL risk overall was attenuated and not significant after

adjustment for other pesticides. They did not adjust for other pesticides, however, in the dose response or subtype analyses for other pesticides. Although the authors collected data on other potentially important confounders, such as smoking and proximity to industrial sites, the data were not used in the present study. In summary, given the concerns that a majority of the odds ratios for diverse pesticides in this study were elevated, systematic bias in the design or conduct may have led to the observed findings in this study.

Case control study in France

Orsi *et al*, 2009. Orsi *et al*[42] investigated the association between occupational exposure to multiple pesticides and lymphoid cancers (NHL as well as Hodgkin lymphoma, lymphoproliferative syndrome, and multiple myeloma) in a hospital based case-control study. The analyses were based on 491 lymphoid cancers in men, including 244 cases of NHL, and 456 male controls (note – Dr. Ritz has the incorrect number of controls in Table 2 of her report) recruited from six hospitals in France between 2000 and 2004. Hospital-based refers to the selection of the controls from the hospitals, rather than population-based, and these were patients being seen for primarily rheumatic or orthopedic conditions.

Data collection from participants was first via self-report questionnaires followed by structured interviews, and patients and interviewers were blind to the study hypotheses. The questions included information on non-occupational exposures to pesticides, as well as an agricultural questionnaire for men who had worked as a farmer or gardener for at least 6 months in their lifetime. Of the 168 men who completed the agricultural questionnaire, 130 had insufficient information and were meant to be re-interviewed, although only 95 were able to be conducted. Odds ratios and 95%

confidence intervals were generated, crudely adjusting for age, hospital, and socioeconomic status, but not mutually adjusted for other pesticides.

Twelve NHL cases and 24 controls were exposed to glyphosate. In the crudely adjusted models, there was no association between glyphosate exposure and NHL risk overall (OR 1.0, 0.5-2.2). Orsi *et al* reported associations for the NHL subtypes diffuse large cell lymphoma (5 glyphosate exposed cases) and follicular lymphoma (3 glyphosate exposed cases). Because of the small numbers of cases and wide confidence intervals, the odds ratios for these subtypes are difficult to interpret, although also suggested no associations with glyphosate. There were no analyses of dose-response presented, notwithstanding the potential latency of more than 20 years after glyphosate was introduced.

Potential strengths and weaknesses. This study has some strengths and several weaknesses to consider. Given the time period of recruitment of cases and controls, between 2000 and 2004, there is a reasonable potential latency period to examine glyphosate and NHL risk. The data collection of use of pesticides was comprehensive, and included information on use of pesticides both at home as well as occupationally, with information collected on whether the participants had personally sprayed the pesticides, and the number and duration of applications. There are three key concerns of the study, however, that outweigh any strengths. First, the use of hospital-based controls in case-control studies can be highly problematic, as often individuals at hospitals with other diseases are more or less likely to have the exposure of interest than the actual population that gave rise to the cases. This can result in a selection bias that can over- or under-estimate an association. Related to this, the authors do not mention the participation rates of the cases and controls, and as such, it is not possible to assess the potential for selection bias to account for the results. Second, the number of NHL cases, and particularly the number of cases exposed to glyphosate, was quite small and thus

the power of the study to detect an association was reduced. Finally, although the authors acknowledge the concurrent use of multiple pesticides among participants in the study, the multivariable models are only minimally adjusted for a few factors and there is no consideration of potential confounding by pesticide use. Taken together, the limitations of this study far outweigh any strengths, thus making it difficult to make inferences.

Meta-analysis studies

As described earlier, a meta-analysis takes the odds ratios and 95% confidence intervals from individual studies and weights the results, often by the number of cases. In contrast to a pooled analysis, which has the advantage of performing similar statistical analyses across different datasets to adjust for confounding, meta-analyses must rely on relative risks from existing statistical models, including how the original authors approach confounding. At the heart of meta-analyses, as with pooled analyses, is that their validity is completely dependent on the validity in the design and conduct of the original studies.[13]

The first meta-analysis to report on the association between glyphosate and NHL risk was by **Schinasi and Leon (2014)**[43], which examined multiple pesticides and included odds ratios and 95% confidence intervals from the Agricultural Health Study (De Roos *et al*, 2005) cohort, the French case-control study of Orsi *et al*, the pooled US studies of De Roos *et al* (2003), the Canadian study of McDuffie *et al*, and two of the Swedish case-control studies (Hardell, 2002 and Eriksson *et al*, 2008). The Agricultural Health Study cohort contributed the largest number of exposed cases from any individual study. Glyphosate was associated with an odds ratio of 1.5 (95% CI 1.5, 95% CI 1.1-2.0) for NHL risk overall and 2.0 (95% CI 1.1-3.6) for B cell lymphoma based only on two studies. Although in the methods section, Schinasi and Leon state that they took the most fully adjusted odds ratios, they actually included more crudely

adjusted odds ratios for some studies, rather than those adjusted for other pesticides use when available.

In 2016, **Chang and Delzell**[44] performed a meta-analysis specifically focused on glyphosate exposure. In this meta-analysis, they expanded the analysis to integrate the more fully adjusted odds ratios when available, and also performed a qualitative assessment of the studies for potential bias. The combined relative risk for ever glyphosate and risk of NHL was 1.3 (95% CI 1.0-1.6) based on six studies. Using a statistical approach to adjust for potential publication bias, the odds ratio was 1.2 (95% CI 1.0-1.6). In contrast to the meta-analysis of Chang and Delzell, Schinasi and Leon used the crudely adjusted odds ratios from Hardell *et al* and Eriksson *et al*, and also the less well-adjusted logistic model from De Roos *et al* (2003). Interestingly, in a sensitivity analysis stratified on year of case diagnosis, the largest odds ratio for the association between glyphosate and NHL was for the cases in the 1980s (OR 1.6), i.e. those with shortest potential latency and higher use of proxy respondents, compared to those in the 1990s and 2000s (OR 1.2). The meta-analysis estimate for B cell lymphoma was similar to that of Schinasi and Leon and was 1.1 (95% CI 0.5-2.3) for diffuse large B-cell lymphoma. In evaluating the quality of the epidemiological literature, the authors comment on the validity of the relative risk estimates from the meta-analysis given that systematic errors due to bias or confounding cannot be excluded.

In 2017, **Chang and Delzell**[32] produced a technical memorandum updating their 2016 meta-analysis to include more recent data on glyphosate and NHL risk. In their primary analysis, they replaced data from the original Agricultural Health Study analysis[1] with the updated results from Alavanja *et al* which address the potential publication bias described earlier and addressed in the manuscript on meta-analyses by Dr. Blair[45]. In this approach, the meta-analysis relative risk for ever exposure to glyphosate was 1.2 (95% CI 0.9-1.6). Chang and Delzell performed a number of sensitivity analyses, including using the fully adjusted odds ratios from the NAPP study of Pahwa *et al*, 2015[35] to replace results from the US pooled analysis (De Roos 2003)[9]

and the Canadian study (McDuffie 2001)[10]. In this updated meta-analysis, the summary meta-relative risk for ever exposure to glyphosate was 1.0 (95% CI 0.86-1.2) with evidence of consistency (i.e. low heterogeneity) across the study results. For the analyses of NHL subtypes, the meta-analyses that included the updated results from Alavanja *et al* resulted in relative risks ranging from 0.8-1.4, with no statistically significant findings. These findings are notwithstanding the potential for bias and confounding remaining in the publication.

XII. COMMENTS ON PLAINTIFFS' EXPERT REPORTS

I have addressed several of the major flaws of the methodology of the Plaintiffs' experts in my presentation of the individual epidemiological studies in **Section XI** above. Below I further discuss several major points raised by the Plaintiffs' experts. I group the issues raised in the expert reports thematically, and then provide my comments in response to each issue.

Confounding. On page 16 of her report, Dr. Ritz acknowledges the importance of confounding in the epidemiological studies of glyphosate and NHL risk. She mentions the importance of using "the most highly-adjusted estimates" in order to "give the reader confidence that the findings are most likely due to glyphosate/Roundup exposure, instead of another potential cause that acts as a confounder". Dr. Neugut comments in his report about potential confounding by other herbicides, as well as specifically 2,4-D.

Response. On page 16 of her report, Dr. Ritz presents a number of the odds ratios for the association between glyphosate and NHL risk from the Pahwa pooled analysis. She presents the results from the conference abstract, rather than the more comprehensive analyses that were included in the August 2015 slide deck. It is important to note that the conference abstract only crudely adjusted for age, sex, location, proxy respondent, family history, and use of protective equipment, but not for other pesticides as Pahwa *et al* provide in her powerpoint presentation. Moreover, Pahwa shows in her powerpoint presentation the

important bias due to the use of proxy respondents, but Dr. Ritz does not present this more fully adjusted estimate. Similarly, Dr. Ritz discusses the results from Eriksson *et al* (2008), highlighting its ability to consider longer latency periods. Nowhere in her discussion of the results does Dr. Ritz mention that the odds ratios she presents for this latency analysis are only crudely adjusted.

As I note on page 45 of my report, Pahwa *et al* demonstrates the important effect of confounding due to concomitant use of other pesticides. For ever/never use, Dr. Ritz highlights the abstract's odds ratio of 1.51 (95% CI 1.18-1.95) for the association between ever exposure to glyphosate and NHL risk. This odds ratio differs slightly from the August 2015 presentation slide deck with a crudely adjusted odds ratio of 1.43 (95% CI 1.11-1.83). In the model additionally adjusted for use of 2,4-D, dicamba, and malathion, the odds ratio for ever use was substantially attenuated (OR 1.13, 95% CI 0.84-1.51). Similarly, Ritz highlights the crudely adjusted odds ratio for glyphosate and diffuse large B-cell lymphoma (DLBCL) presented in the abstract (OR 3.11, 95% CI 1.61-6.00), which actually differs substantially from the crudely adjusted odds ratio from the powerpoint presentation (1.60, 95% CI 1.12-2.09), and the model additionally adjusted for other pesticides (OR 1.23, 95% CI 1.88). It is unclear why the crudely adjusted odds ratios from the abstract and powerpoint presentations actually differ. If Dr. Ritz were to rely on the most fully adjusted odds ratios from Pahwa *et al*, then her characterization of the association between glyphosate and NHL risk would have been different.

The concern of Dr. Neugut about "negative" confounding by other pesticides in the results from the Agricultural Health Study and Eriksson *et al* (2008) is illogical. Dr. Neugut posits that that any confounding by other herbicides or pesticides would have attenuated the odds ratio toward the null value rather than overestimated the relative risk. To this point, in the Agricultural Health Study, although it is true that the prevalence of 2,4-D was 53% among those never exposed to glyphosate, it was even higher among those with the lowest (75%) and highest (85%) exposure to glyphosate. As such, if 2,4-D is independently associated with NHL risk, the

confounding would have led to an over-estimate and not under-estimate of the association as Dr. Neugut states. Indeed, the prevalence of all of the commonly used pesticides in De Roos *et al* was higher and not lower in those also using glyphosate. Unless these other pesticides were associated with a protective effect on NHL risk, any confounding would have overestimated the relative risk. In fact, in his discussion of Eriksson *et al* (2008), Dr. Neugut shows the "positive" confounding associated with concomitant use of other pesticides: the crudely adjusted odds ratio for glyphosate exposure was 2.02 (95% CI 1.10-3.71) whereas including the other pesticides in the model attenuated the association towards the null 1.51 (95% CI 0.77-2.94).

Sample size and power. A major focus of the Plaintiffs' expert reports is on the importance of sample size of the epidemiological studies of glyphosate and NHL risk, and the related issue of statistical power. For example, Dr. Ritz states on page 15 of her report that because "many of the smaller studies had suggestive findings but wide confidence intervals, it is particularly important to instead consider pooled and meta-analyses that summarize across these smaller studies and not only provide a much larger sample size but may allow us to assess NHL subtypes with sufficient precision". In particular, she comments that "an informative study to consider is Pahwa's pooled analysis of the North American and Canadian studies". In her table on the same page, she presents the sample sizes from publications including the number of cases and controls for the case-control studies and the number of cases and non-cases from the De Roos *et al* 2005 cohort study. She also presents a forest plot summarizing the various odds ratios and 95% confidence intervals from the publications.

Response. In **Section VII** and **VIII** of my report, I present several concepts relevant to Plaintiffs' claims about study size and power. First, I agree that understanding the role of chance in leading to an epidemiological finding is important, and a well-powered study can reduce the role of chance. However, as I describe earlier in my report, "if an epidemiological study has systematic bias, it is not helpful to evaluate the role of chance." (page 23). This is because, as a

core epidemiological concept, the estimation of confidence intervals and assessing the role of chance implicitly assume that there is no bias or confounding present in the study. Both a pooled-analysis and meta-analysis assume that the individual studies are devoid of bias and confounding[13]. The analytic approach used in meta-analyses cannot eliminate biases from the design or conduct of poorly conducted individual studies, such as the majority of the case-control studies investigating glyphosate and NHL. "Garbage in-garbage out" means the validity of the summary relative risk estimate in either a pooled or meta-analysis will be biased if the original studies going into it are biased, even if it is a very precise estimate. In his publication on the principles of meta-analyses in environmental epidemiology, Blair *et al*[13] highlights this point. In reading Dr. Ritz's report, I was surprised at the prioritization of chance over two core concepts of epidemiological studies: confounding and recall bias.

In addition to the issue of residual confounding, the forest plot presented by Dr. Ritz is misleading, because it suggests that each of the publications represents an individual study. For example, as I described above, De Roos *et al* 2003 is a pooled analysis of three US-based case-control studies that included Cantor 1992; indeed, more than 60% of the cases in the pooled De Roos 2003 are from Cantor. Pahwa *et al* 2015 is a pooled analysis which includes the three studies in De Roos *et al* 2003 as well as the Canadian case-control study published by McDuffie 2001; Cantor is still the largest contributor of data. Hohenadel and McDuffie are analyses of the same Canadian case-control study also included in Pahwa *et al*. Dr. Ritz is in effect "double dipping" by including results from overlapping cases and controls from individual and pooled analyses.

Another important point of the forest plot is that Dr. Ritz repeatedly presents more crudely adjusted odds ratios rather than the fully adjusted odds ratios as she herself acknowledges are the more appropriate. Moreover, she does not include the updated results of Alavanja *et al* (2013) from the Agricultural Health Study.

The table on page 15 of Dr. Ritz's report is highly misleading by giving the false

impression that the studies with the larger number of cases have the greatest statistical power. Power is a function of not only the total number of cases, but importantly the prevalence of exposure. Indeed, I decided not to discuss the results of from Cocco *et al* (2013), which is the study in Dr. Ritz's table with the largest number of cases. Only 4 of the 1,869 cases and 2 of the 2,462 controls were exposed to glyphosate, and as such this is essentially an unexposed population. Several of the other case-control studies in the table similarly suffered from a low prevalence of glyphosate exposure. This is in contrast to the Agricultural Health Study, in which three-quarters of the population were ever exposed to glyphosate, allowing for meaningful analyses for both the ever/never and dose-response relationships.

Interaction between glyphosate and other pesticides. The Ritz expert report highlights data from the Canadian case-control study (McDuffie *et al* 2001 and Hohenadel *et al* 2011) to support the proposition that higher associated risk estimates resulted when glyphosate exposure was analyzed alongside another pesticide exposure than when used alone. Her report states, "McDuffie reported that when glyphosate exposure was mixed with dicamba, the risk was increased (OR 1.92, 95% CI 1.39-2.66, minimally adjusted model; OR 1.88, 95% CI 1.32-2.68; fully adjusted model) compared to dicamba exposure alone (OR 1.59 and 1.68, respectively)." Citing the Hohenadel study, Dr. Ritz further states that, "when glyphosate exposure was mixed with malathion it was stronger than when farmers only reported using glyphosate alone."

Response. The odds ratio of 1.92 in McDuffie *et al* for the "mixed" exposure is for a combination of products that include mixtures of dicamba with glyphosate but also dicamba with 2,4-D and mecoprop (phenoxyherbicides). Risk estimates for glyphosate alone are not significantly elevated, while fully-adjusted estimates are significantly elevated for 2,4-D, mecoprop, and dicamba alone. Importantly (but not included in Dr. Ritz's report), the association for malathion alone (OR 1.95, 95% CI 1.29-2.93) in Hohenadal *et al* was qualitatively similar to

the association for the combination of malathion and glyphosate (OR 2.10, 95% CI 1.31-3.37), whereas there was no association for glyphosate alone (OR 0.92, 95% CI 0.54-1.55). Rather than evidence of a super-additive effect of glyphosate, as suggested in her expert report, this finding would suggest that it is malathion exposure —and not exposure to glyphosate—which underlies the reported positive association with NHL risk observed. Although the Dr. Ritz report appears to suggest that glyphosate may act synergistically with other pesticides to cause NHL, this is not supported by the evidence.

Dose-response. Dr. Ritz's report presents evidence from McDuffie *et al*, Pahwa *et al*, and Eriksson *et al* to support a dose-response relationship between glyphosate exposure and risk of NHL. For example, using data from Pahwa *et al*, Dr. Ritz focuses on the measure of ≥2 days/year of use rather than the more meaningful estimates of dose which integrates duration. As a side note, the dose-response analyses of both days of exposure and lifetime days are limited to the case-control data from Canada and Nebraska, and not the full set of 1,690 cases and 5,131 controls from the four studies.

Response. The dose-response analyses that Dr. Ritz highlights in these studies are relatively crude in that the exposure is categorized based solely on days of exposure, potentially grouping together individuals with very different biological doses or duration of use. Moreover, the cut point is narrowly defined and it is unclear why "≥2 days/year" was chosen in McDuffie *et al* to reflect a meaningful amount of exposure. The powerpoint presentation by Pahwa *et al* shows the more meaningful dose-response trend for lifetime days of exposure. This analysis not only adjusts for concomitant use of 2,4-D, dicamba, and malathion but also considers the effect of proxy respondents on the estimates (Slide 6). The fully adjusted odds ratio for >7 lifetime days of exposure and NHL risk overall was 1.08 (95% CI 0.66-1.77). Dr. Ritz does not mention these fully adjusted results, notwithstanding her statements in her report about the importance of fully adjusting for confounding.

With respect to the dose-response analyses of Eriksson *et al*, a major concern is that the estimates are only crudely adjusted for age, sex, and date of diagnosis. The multivariable odds ratio for ever exposure to glyphosate in Eriksson *et al* clearly shows the positive confounding due to concomitant use of other pesticides. A more fully adjusted analysis of the dose-response analysis of Eriksson *et al* is needed to make meaningful inferences. Eriksson *et al* also did not provide any test for trend to evaluate evidence that the reported dose-response findings were statistically significant.

In contrast, Dr. Ritz appears to discount the De Roos *et al* (2005) study, which did not find evidence of a dose-response analysis based on either cumulative exposure days or intensity-weighted exposure days. This is important because De Roos (2005) was able to investigate a much wider range of exposure levels. Dr. Ritz criticizes the use of low exposure versus no exposure for these dose response associations. However, given that the overall association between ever use and risk of NHL was null in De Roos (2005), the use of no exposure would have resulted in a similar pattern of association. Moreover, the updated analysis of the Agricultural Health Study of Alavanja *et al* uses no exposure as the reference group in the dose-response analyses, and in multivariable models found an odds ratio of 1.0 (95% CI 0.7-1.4) for the highest vs. no exposure using lifetime days of exposure and 0.97 (95% CI 0.7-1.4) for the highest vs. no exposure using intensity weighted days of exposure.

An important issue to consider is that an apparent dose-response can also arise if there is confounding by a factor that also has a biologic gradient with the disease under study. For example, birth order appears to have a dose-response relationship with Down syndrome (i.e., compared to first-born children, risk of Down syndrome increases with second-born, and third-born, and so forth).[46] In fact, this dose-response relationship merely reflects the effect of maternal age, which increases alongside birth rank, on development of Down syndrome. Thus, a dose-response analysis is only meaningful to the extent that bias due to confounding factors, such as other pesticides particularly in dose response relationships, has been eliminated.

Short follow-up period of De Roos *et al*, 2005. In his expert report, Dr. Neugut describes as a limitation of the first publication of the Agricultural Health Study that the follow-up time was relatively short and as a corollary that the study was unable to investigate latency. Drs. Weisenburger and Portier also note this as a limitation. Dr. Neugut further dismisses the use of cohort studies in cancer epidemiological studies because they are extremely difficult and "indeed, such studies are rare" because of the need to follow individuals for such a long time.

Response. These comments and concerns around insufficient latency in the Agricultural Health Study are unequivocally wrong. First, although the information on glyphosate exposure in the Agricultural Health Study was collected on a baseline questionnaire in 1993-1997, the questionnaire collected information on both current and past exposure including the years of prior use, the number of days per year of use, as well as the intensity of use. The questionnaire used a standard epidemiological approach to collect information both retrospectively and prospectively on study participants. As such, as shown in Figure 2 (page 30), the Agricultural Health Study is poised to consider long latency periods of up to 18-26 years. The manuscript of Alavanja *et al* (2013) could consider even longer latency periods with cases followed up through 2008, 33 years after glyphosate was introduced in the U.S. Many of the case-control studies on glyphosate exposure and NHL risk used a similar approach of asking about past exposure to glyphosate, including the study by Eriksson *et al*. If Dr. Neugut is concerned about the short follow-up time of De Roos *et al*, then he should be even more concerned about the data collection of the case-control studies since the questionnaires were given to cases after their diagnosis which would imply a negative follow-up time based on the criterion of Dr. Neugut.

In his deposition, Dr. Blair addresses this specific point and states that the participants in the Agricultural Health Study had on average 15 years of exposure to pesticides prior to entry into the study.

Consistency. In their expert reports, Drs. Neugut, Ritz, and Weisenburger comment on the "consistency" of epidemiological findings on glyphosate and NHL risk, invoking Bradford Hill's framework, and use this to help support their notion of causality. Dr. Neugut's conclusion that "glyphosate in its various combinations can cause non-Hodgkin lymphoma" is based in part on his assessment that the epidemiology studies achieve consistency in finding a positive association between glyphosate exposure and development of NHL. Specifically, he states, "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."

Response. There are major flaws in these conclusions, as the epidemiological evidence evaluated does not reach the threshold of consistency as they describe. I focus on two problems with the argument put forth by Drs. Neugut and Weisenburger.

First, Drs. Neugut and Weisenburger's argument for consistency does not align with Bradford Hill's justification of this viewpoint. In Bradford Hill's description of consistency, he explains that causation is supported when observing similar results through application of different methodologies. For example, Bradford Hill states, "I would myself put a good deal of weight upon similar results reached in quite different ways, e.g. prospectively and retrospectively." Bradford Hill provides an example to illustrate that a scientific question addressed multiple times using similar but flawed methodology, and providing similar results, does not actually strengthen the case for causation. In my detailed discussion in **Section X, I** have outlined numerous sources of bias in the studies presented, many of them (such as the potential for recall bias) which are shared across studies.

Second, when one looks for the consistency of epidemiological evidence using, as Dr. Ritz suggests, "the most highly-adjusted estimates", the epidemiological evidence actually points to a fairly consistent null association for ever use of glyphosate. The associations are null for the prospective Agricultural Health Study, the fully adjusted model for the case-control studies included in Pahwa *et al*, and for the French case-control study of Orsi *et al*. The two

Swedish case-control studies of Hardell *et al* (OR 1.85, 95% CI 0.55-6.20; based on 8 exposed cases and 8 exposed controls) and Eriksson *et al* (OR 1.51, 95% CI 0.77-2.94 based on 29 exposed cases and 18 exposed controls) are also consistent with the null hypothesis given the wide 95% confidence intervals.

Temporality. Dr. Neugut claims that temporality is met by the current evidence since "exposure to glyphosate and its formulations preceded the development of NHL in all the human studies and all the animal studies."

Response. A point should be made that temporality is not established in the case-control studies in which the glyphosate exposure is ascertained after development of the NHL. The main issue concerns recall bias as discussed in **Section X**. To establish the temporality in a study requires measurement or documentation of the exposure prior to the outcome (i.e., prospectively collected exposure data). The prospective cohort studies of De Roos *et al* (2005) and Alavanja *et al* (2013) of the Agricultural Health Study are the only in which glyphosate exposure was assessed prior to the diagnosis of NHL. Related to this issue of temporality is sufficient latency. Plaintiffs' experts fail to note that De Roos *et al* (2003) and to some extent Pahwa *et al* do not have sufficient latency.

Use of the cohort study in cancer epidemiology. In their reports, both Dr. Ritz and Dr. Neugut comment on the challenges in using cohort studies to investigate associations in cancer given the relative rarity of cancer in the population. On page 4 of his report, Dr. Neugut states "From an epidemiologic perspective, this makes the use of cohort studies or intervention trials extremely difficult and expensive and indeed, such studies are uncommon." Similarly, Dr. Ritz states "This is why it is so hard to study NHL with a cohort design, because you would have to follow hundreds of thousands of people for many years in order to find any result that would give us a $p<0.05$…"

Response. As a cancer epidemiologist and co-Principal Investigator of the Health Professionals Follow-up Study, a cohort study of 50,000 U.S. men, I can state with very high confidence that these statements are flawed. Drs. Ritz and Neugut may not be aware that there are 50 high-quality cohort studies including 7 million people that are part of the National Cancer Institute's Cohort Consortium. These cohort studies, many of which have been ongoing since the 1980s or 1990s, have generated literally thousands of publications on the association between potential risk factors and the incidence of multiple cancers including NHL. While it is true that on an annual basis the incidence of a particular cancer may be relatively rare, the risk accrued over time can be quite substantial.

For NHL, the lifetime risk is 1 in 50, which would translate to an estimated number of cases in the Agricultural Health Study of 1,000. Indeed, in the publication by Alavanja *et al* (2014)[33] which assessed associations between insecticide, fungicide, and fumigant use (but not herbicides), there were 523 incident cases of NHL in the cohort with follow-up through 2010-2011. Moreover, as shown in the analysis by Chang and Delzell, the unpublished study by Alavanja *et al* (2013)[2] that included glyphosate results, contributed a relative weight of 34% to a meta-analysis because of its large number of cases and high prevalence of glyphosate exposure. Indeed, prior to Alavanja's analysis, the De Roos *et al* (2005) analysis contributed the largest number of exposed NHL cases of any individual epidemiological study. As such, the cohort analyses in the Agricultural Health Study allow for meaningful inferences about associations between risk factors and NHL risk.

Publication bias. None of the Plaintiffs' expert reports address the updated results in the Agricultural Health Study from Alavanja *et al* (2013)[2].

Response. Meta-analyses can only integrate information from studies that are available in the public domain, usually through publication in peer-reviewed medical journals or scientific conferences. As such, it is well known that meta-analyses can be susceptible to publication

bias[47-49]. As stated in the 1995 publication by Blair *et al*[13], publication bias "is a critical issue in environmental health studies, as in other fields" and "tends to push results in a positive direction (i.e. in the direction of increased risk)". He goes on to further state that **"**[u]npublished reports meeting selection criteria may be included in a meta-analysis, especially if the data upon which they are based are believed to be sound."

The updated results from the Agricultural Health Study provide important and valid additional information on the association between glyphosate and NHL risk. First, the updated analyses address one of the main concerns of the Plaintiffs' experts of the Agricultural Health Study, namely the number of NHL cases that were diagnosed during follow-up (note – notwithstanding this, the number of exposed cases in De Roos *et al*, 2005 was the largest of any of the individual epidemiological studies at the time). Second, the increased number of cases allowed for the authors to investigate both dose-response and NHL subtype analyses with considerable statistical power. Third, the follow-up of the Agricultural Health Study also addresses a concern of the Plaintiffs' experts (particularly Dr. Neugut) that participants' baseline exposure was not updated, since the analysis integrated a follow-up questionnaire. Taken together, these points highlight the impact of publication bias in the prior meta-analyses, including that of IARC, as well as discussion by the Plaintiffs' experts of the weight of the epidemiological evidence.

XIII. CONCLUSION

My conclusion on the association between glyphosate exposure and NHL risk is based on my review and evaluation of all available epidemiology literature. I considered the epidemiological findings in totality, and weighed the evidence based on the quality of the study design and statistical analysis. For each individual study, I examined the findings and considered the extent to which bias, confounding or chance could account for the findings.

Based on my evaluation, it is my opinion, to a reasonable degree of scientific certainty, that the epidemiological evidence does not provide a scientific basis to support a causal relationship between exposure to glyphosate-based herbicides and the risk of NHL.

Lorelei A. Mucci, ScD

July 31, 2017
Date

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Exhibit A

CURRICULUM VITAE

Date Prepared: July 31, 2017

NAME: Lorelei Ann Mucci

ACADEMIC TITLE: Associate Professor of Epidemiology

WORK ADDRESS: Harvard T.H. Chan School of Public Health
Department of Epidemiology
677 Huntington Avenue, 9th floor
Boston, MA 02115 United States

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HOME ADDRESS: [redacted]

EDUCATION:

1988	Certificate (Italian language and culture), American Institute for Foreign Studies, Florence, Italy
1989	BS (Biology), Tufts University, Medford, MA
1997	MPH (Epidemiology and Biostatistics), Boston University School of Public Health, Boston, MA
2003	ScD (Epidemiology), Harvard School of Public Health, Boston, MA

POSTDOCTORAL TRAINING:

2002-03	Research Fellow in Epidemiology, Karolinska Institutet, Stockholm, Sweden
2003-05	Research Fellow in Cancer Epidemiology, Harvard School of Public Health, Boston, MA

ACADEMIC APPOINTMENTS:

1998-2002	Graduate Research Assistant in Epidemiology, Harvard School of Public Health, Boston, MA

1998-2002	Graduate Research Assistant in Oral Health Policy and Epidemiology, Harvard School of Dental Medicine, Boston, MA
2000-02	Graduate Research Assistant in Medical Epidemiology, Karolinska Institutet, Stockholm, Sweden
2002-03	Research Fellow in Epidemiology, Karolinska Institutet, Stockholm, Sweden
2003-05	Research Fellow in Cancer Epidemiology, Harvard School of Public Health, Boston, MA
2003-06	Instructor of Medicine, Harvard Medical School, Boston, MA
2006-16	Assistant Professor of Medicine, Harvard Medical School, Boston, MA
2006-10	Assistant Professor in the Department of Epidemiology, Harvard School of Public Health, Boston, MA
2010-	Associate Professor of Epidemiology, Harvard T.H. Chan School of Public Health, Boston MA

HOSPITAL OR AFFILIATED INSTITUTION APPOINTMENTS:

2003-	Associate Epidemiologist, Brigham and Women's Hospital, Boston, MA
2006-	Member, Cancer Epidemiology and Prostate Cancer Programs, Dana-Farber/Harvard Cancer Center, Boston, MA
2011-	Leader, Cancer Epidemiology Program, Dana-Farber/Harvard Cancer Center
2012-	Member, Center Scientific Council, Dana-Farber/Harvard Cancer Center

OTHER ACADEMIC APPOINTMENTS:

2004	Instructor, Boston University School of Public Health, Boston, MA
2007-	Visiting Professor of Public Health, University of Iceland, Reykjavik, Iceland
2012-15	Foreign Visiting Professor, Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Sweden

OTHER PROFESSIONAL APPOINTMENTS:

2012-15	Co-Leader, Cancer Epidemiology Program, Dana-Farber/Harvard Cancer Center 2015- Leader, Cancer Epidemiology Program, Dana-Farber/Harvard Cancer Center
2012-	Head, Cancer Epidemiology and Cancer Prevention Area of Concentration, Harvard T.H. Chan School of Public Health

MAJOR ADMINISTRATIVE RESPONSIBILITIES:

2005-11	Co-Director, Channing Peer-Mentoring Program for Post-Doctoral Fellows and Instructors, Channing Laboratory, Brigham and Women's Hospital
2005-07	Organizer, Channing Seminar Series, Chronic Disease Epidemiology Group, Channing Laboratory, Brigham and Women's Hospital
2006	Harvard Medical School Leadership Development for Physicians and Scientists, Harvard Medical School
2006-	Member, Dana-Farber/Harvard Cancer Center Cancer Epidemiology Program and Prostate Cancer Program
2007-08 2008	Organizer, Epidemiology Department Seminar Series, Harvard School of Public Health Organizer, Dana-Farber/Harvard Cancer Center Interdisciplinary Workshop: Incorporating novel tumor tissue analysis into population based studies of human cancer, Harvard Medical School
2009	Science Fair Judge, New England Science Symposium, Harvard Medical School, sponsored by the Harvard Medical School Minority Faculty Development Program of the Office of Diversity and Community Partnership
2009-14	Advisory Board Member, Nurses' Health Study Tissue Laboratory, Channing Laboratory, Brigham and Women's Hospital
2010-	Scientific Advisory Board, Prostate Cancer Foundation
2011-	Movember Global Action Plan Team Member, Prostate Cancer Foundation and Movember
2011-	Executive Committee, Transdisciplinary Prostate Cancer Partnership (ToPCaP)
2012-	Executive Committee, Nordic Twin Studies of Cancer (NorTwinCan)
2012-7	Leader, Prostate Cancer Foundation "School of Public Health"
2012	Workshop organizer, mRNA profiling pilot studies in the Harvard Cohorts, Dana-Farber/Harvard Cancer Center

2013	Evaluation Panel, Movember Revolutionary Team Award, Australia
2013, 2016	Expert Review Panel, Movember Translation Acceleration Grant, Prostate Cancer Canada
2013-	Organizer, Annual Celebration of Young Investigators in Cancer Research, Dana-Farber/Harvard Cancer Center
2013-	Organizer, Annual Brief Update Series in Population Sciences, Dana-Farber/Harvard Cancer Center
2014	Faculty Associate, Fulbright US Scholar Program
2014-	Scientific Advisory Board, Movember Global Action Plan 4 (GAP4), Prostate Cancer Exercise and Metabolic Health
2014-6	Co-leader, Tissue Working Group, National Cancer Institute's Cohort Consortium 2016- Co-Leader, Prostate Tissue Biomarkers Working Group, NCI Cohort Consortium
2014-	External Advisory Board, Pacific Northwest SPORE in Prostate Cancer
2014	Participant, National Cancer Institute Provocative Question Workshop, Boston MA
2014	Organizer, Second Annual Celebration of Young Investigators in Cancer Research, Dana-Farber/Harvard Cancer Center
2014	Organizer, First Annual Prostate Cancer Teach-In, Harvard School of Public Health and Massachusetts Prostate Cancer Coalition
2015	Program Committee, American Association for Cancer Research Annual Meeting
2015-	Integration Panel Member, Congressionally Directed Medical Research Programs' Prostate Cancer Research Program 2016- Executive Committee, Member at Large
2015	International Advisory Group, Pacific Rim Breast and Prostate Cancer Group
2015-2018	Research Advisory Council, Prostate Cancer UK
2015-	Co-Principal Investigator, International Registry to Improve Outcomes in Men with Advanced Prostate Cancer (IRONMAN)
2016-	Co-Director, Integrative Molecular Epidemiology Workshop, American Association for Cancer Research
2016-	Co-Principal Investigator, Health Professionals Follow-up Study

2016- External Advisory Board, The Sidney Kimmel Cancer Center at Thomas Jefferson University

COMMITTEE SERVICE:

DEPARTMENTAL/SCHOOL AND UNIVERSITY SERVICE:

2006- Admissions Committee, Epidemiology Department, Harvard T.H. Chan School of Public Health

2006- Member and Project Leader, Dana-Farber/Harvard Cancer Center Prostate Cancer SPORE

2007-10 Gender Equality Committee, Epidemiology Department, Harvard School of Public Health

2009-14 Scientific Advisory Board, Pathology Cores, Dana-Farber/Harvard Cancer Center

2010- Faculty Mentor, T32 Nutrition and Cancer Training Grant, Harvard School of Public Health

2010-14 Scientific Advisory Board, Tissue Studies, Nurses' Health Study

2010- Review Committee, Harvard School of Public Health Post-Doctoral Travel Scholarship Awards

2011-13 Organizer, Epidemiology Department Seminar Series, Harvard School of Public Health

2011-15 Grant Review Committee, David Mazzone, Dana-Farber/Harvard Cancer Center Prostate SPORE

2011-16 Post-doctoral Fellow Advisory Committee, Harvard T.H. Chan School of Public Health

2011- Head of Cancer Epidemiology Concentration, Harvard T.H. Chan School of Public Health

2012- Grant Review Committee, Dana-Farber/Harvard Cancer Center U54 Pilot Applications

2012- Member, Admissions and Financial Aid Committee, Department of Epidemiology, Harvard T.H. Chan School of Public Health

2012- Co-Leader, Cancer Epidemiology, Dana-Farber/Harvard Cancer Center
2015- Leader, Cancer Epidemiology

2013- Member, Harvard T.H. Chan School of Public Health Disciplinary Board

2014-18 Faculty Mentor, Harvard T.H. Chan School of Public Health MIRT Program

2014-	Faculty Steering Committee, John B. Little Center for Radiation Sciences, Harvard T.H. Chan School of Public Health
2015-19	Faculty Mentor, T32 Training Grant on Cancer Biostatistics, Harvard T.H. Chan School of Public Health
2015-	Methods and Substantive Exam Committee, Department of Epidemiology, Harvard T.H. Chan School of Public Health
2015-16	Independent Blue Ribbon Expert Panel, Massachusetts Prostate Cancer Action Council and Campaign for Prostate Cancer Research, Education and Awareness for High-Risk Men
2016, 2017	Harvard University Milton Fund Review Panel
2016-19	Faculty Council Member, Harvard T.H. Chan School of Public Health
2016	Chair, *Ad Hoc* Disciplinary Board, Harvard T.H. Chan School of Public Health
2017	Member, Task Force on Improving Educational Quality, Harvard T.H. Chan School of Public Health
2017	Member, Faculty Search Committee in Radiation Epidemiology, Harvard T.H. Chan School of Public Health

PROFESSIONAL SOCIETIES:

2001-05	Society for Epidemiological Research, Member
2003-	American Association for Cancer Research, Associate Member
2014-	American Society for Preventive Oncology

GRANT REVIEW ACTIVITIES:

2010	Grant Review Panel, World Cancer Research Fund
2011-	Grant Review Committee, Prostate Cancer Charity UK Research Awards
2011-6	Grant Review Committee, Prostate Cancer Foundation of Australia
2012	Ad Hoc Member, Special Study Section: Provocative Questions, National Institutes of Health/National Cancer Institute

2012-5	Ad Hoc Member, Epidemiology of Cancer (EPIC) Study Section, National Institutes of Health/National Cancer Institute
2012-	Grant Review Committee, Challenge Award Mechanism, Prostate Cancer Foundation
2013	Grant Review Committee, Health Research Board of Ireland
2013	Grant Review Committee, Irish Cancer Society Career Development Awards
2013	Grant Review Committee, Norwegian Cancer Society Team Science Award
2013	Grant Review Panel, US Army Prostate Cancer Program, Population Science Mechanism
2013-	Grant Review Panel, National Institutes of Health/National Cancer Institute, PAR Physical activity and weight control interventions among cancer survivors: effects on biomarkers of prognosis and survival
2014	Grant Review Panel, US Army Prostate Cancer Program, Idea Development Mechanism
2014-	Scientific Review Panel Member, Cancer Prevention Research Institute of Texas (CPRIT)
2015-	Grant Review Panel, Bankhead-Coley Cancer Research Program, Florida Department of Health
2015-	Member, Grant Review Panel, Fellowships: Risk, Prevention and Health Behavior, National Institutes of Health (ZRG1 F16-L 20)
2016	Member, Grant Review Panel, Basic Research in Cancer Health Disparities/ Diversity, National Institutes of Health (ZRG1 OBT-A (55) R)

EDITORIAL ROLES:

Ad Hoc Reviewer

American Journal of Epidemiology; British Journal of Cancer; British Journal of Urology International; British Medical Journal; Cancer Causes and Control; Cancer Epidemiology, Biomarkers and Prevention; Cancer Prevention Research; Cancer Research, Clinical Cancer Research; Epidemiology; European Urology; International Journal of Cancer; Journal of Clinical Oncology; Journal of Food Composition and Analysis; Journal of the American Medical Association; Journal of the National Cancer Institute; Lancet Oncology; New England Journal of Medicine; Prostate; PLOS One; PLOS Medicine

Other Editorial Roles

2006-08	Editorial Board, Menopause

2006-08	Editorial Board, The Open Epidemiology Journal
2009-	Associate Editor, Cancer Causes Control
2011-	Editorial Board, Clinical Genitourinary Cancer

HONORS AND DISTINCTIONS:

2003-10	NIH Loan Repayment Program Award, National Institutes of Health
2003	James M. Dunning Award for Research Excellence, Harvard School of Dental Medicine
2005	American Society for Clinical Oncology Merit Award
2007	American Cancer Society Travel Scholarship
2007	Harvard Dependent Care Travel Fund Award
2008-11	Michael Milken Scholar, Prostate Cancer Foundation
2009	Top Performing Young Investigator, Prostate Cancer Foundation
2010	Harvard Dependent Care Travel Fund Award
2015	Harvard Dependent Care Travel Fund Award
2015	Best of Journal of Clinical Oncology: 2015 Genitourinary Cancer
2015	Teaching Citation, Harvard T.H. Chan School of Public Health
2015	Nominated, Outstanding Post-Doctoral Mentor Award, Harvard T.H. Chan School of Public Health
2015	Mo Sista Whiska Award, Prostate Cancer Foundation
2015	Fifth Annual Alice Hamilton Award Lecture, Harvard T.H. Chan School of Public Health
2016	Teaching Citation, Harvard T.H. Chan School of Public Health
2016	Frank McGovern Lectureship Series, Massachusetts General Hospital

A. Narrative report of Research and Teaching

My major research and teaching area is cancer epidemiology. My research in cancer utilizes integrative molecular epidemiology approaches, including circulating biomarkers, inherited genetic alleles, and tumor biomarkers to study cancer risk and mortality. These projects are nested within cohorts from the US, Europe, Brazil and Australia, and a major focus area is in prostate cancer epidemiology.

Integration of tissue biomarkers in cancer epidemiology studies. I am on the Executive Committee for the Transdisciplinary Prostate Cancer Partnership (ToPCaP, www.topcapteam.org), an international, multidisciplinary effort whose objective is to integrate prostate cancer tissue biomarkers to address questions in etiology, prognosis and treatment. The projects leverage prostate cancer patients from the US and Europe, cohorts with detailed epidemiological and clinical data, histopathological and molecular pathology data, plasma and genetic biomarkers, and long-term follow-up after cancer diagnosis. The projects are undertaken in concert with the DF/HCC SPORE in Prostate Cancer, for which I lead the Population Science project and an active member of the leadership team. For the past four years, I have been a faculty member for the American Association for Cancer Research Integrative Molecular Epidemiology Workshop and joined as co-director in 2016. I also co-lead the Tissue Working Group of the National Cancer Institute's Cohort Consortium.

Twin studies of cancer. I am on the executive committee of the Nordic Twin Study of Cancer (NorTwinCan, www.nortwincan.org), a population-based cohort of 300,000 twins from Sweden, Norway, Denmark and Finland. The unique disease registers in the Nordic countries have allowed us to link data from the nationwide twin registers with cancer, mortality and disease registers, as well as questionnaire data collected on the twins. This unique population has led to a detailed understanding of the familial risk and heritability of various cancers, as well as to examine the relative contribution of genetic and environmental factors in cancer survival. We are integrating lifestyle data to address the shared genetic contributions, e.g., of genetics factors in the association between obesity and cancer risk. We are also collecting tumor tissue to examine the heritability of somatic alterations in tumors.

Physical activity interventions among prostate cancer survivors. Cardiovascular and other chronic diseases represent half of causes of death among men with locally advanced prostate cancer. Men with prostate cancer experience impairments in physical and mental quality of life, both from the cancer as well as from therapy. I am actively involved in several international studies of physical activity interventions that could improve survival, enhance the efficacy of therapeutic interventions, as well as improve overall health and quality of life would provide maximum benefit to men. We completed a pilot study of a group walking intervention in Sweden, whereby men were randomized to either usual care or SPARTACUS, a walking intervention of weekly group walks and were encouraged to maintain 10,000 steps per day on other days. We have now received funding for a trial in Boston working closely with colleagues through the DF/HCC and focusing on men who are undergoing androgen deprivation therapy. I serve on the Scientific Advisory Committee for a global exercise intervention among men with metastatic prostate cancer funded by the men's health organization, Movember. This study launched at the beginning of 2016 with the goal of enrolling 800 men with advanced prostate cancer from centers are the US, Europe, Canada and Australia with the goal to improve survival and quality of life among these patients.

Circadian rhythm. Disruption of the circadian system has been hypothesized to increase cancer risk, either because of direct disruption of the molecular machinery generating circadian rhythms or disruption of parameters controlled by the internal clock such as melatonin levels or sleep duration. As a

primary investigator, I have led studies using integrative molecular epidemiology approaches to investigate circadian disruption as measured by questionnaire data, inherited genetic variants, and urinary biomarkers for prostate cancer, with a focus on advanced and lethal disease. To date, this body of work has demonstrated in U.S. and Icelandic cohorts that disrupted sleep, genetic variants in circadian rhythm genes, and low melatonin levels are all key risk factors for advanced prostate cancer. I am PI of a recently funded grant to investigate circadian disruption and prostate cancer within the Multiethnic Cohort study.

Risk factors for advanced prostate cancer. Prostate cancer is the most common cancer among US men, yet the ratio of incidence to mortality is 6:1. There is accumulating data that the risk factors for advanced/lethal prostate cancer differ from localized, low-risk disease. For this reason, my research has focused on identifying risk factors specific to advanced prostate cancer. One such example is vasectomy, where we revisited a previous hypothesis that the vasectomy procedure may increase the prostate cancer risk. Our results supported the hypothesis that vasectomy is associated with a modest increased risk of lethal prostate cancer, but not cancer risk overall. We were able for the first time to appropriately account for detection bias due to PSA screening and other potential confounders such as infections and cancer treatment. Due to this paper's impact and clinical relevance, it was selected as the #1 paper in the Best of the Journal of Clinical Oncology in 2015.

IRONMAN: An International Registry to Improve Outcomes in Men with Advanced Prostate Cancer. I am the founding co-PI of this international disease registry of 5,000 men with advanced prostate cancer. During the past six years, the US FDA has approved six new therapies for the treatment of men with metastatic and castration-resistant prostate cancer each individually with a survival benefit. The therapies are being used clinically in different combinations and sequences, without any evidence basis for survival benefit or impacts on quality life. This international, population-based study of 5,000 men with advanced prostate cancer includes treatment information, demographic, clinical, and lifestyle data, prospective biorepository, and survival and patient reported clinical outcomes. The goal of this registry is to understand patterns of care for men with advanced prostate cancer, to identify optimal treatment sequences that improve survival and quality of life, as well as to identify biomarkers for subgroups of men who will respond well (or poorly) to specific therapy combinations. This project has funding from Movember Foundation and three pharmaceutical partners, and we are set to recruit our first patients in the fall of 2016.

Teaching. Teaching has been a core activity for me since I completed my post-doctoral training in 2013. I have served as course instructor for 5 courses based at HSPH, and currently as course instructor of Epidemiology of Cancer (EPI213), which has received an evaluation of 4.5 or higher for the past two years. I also guest lecture in several HSPH courses. For three years, I co-lead a two-week bootcamp course at HMS entitled the Molecular Pathology and Epidemiology of Cancer for the graduate students, and this has resulted in a textbook *Molecular Pathology and Epidemiology of Cancer* which is being published by Springer in summer 2016. I served as a faculty mentor for the T32 Cancer Epidemiology training grant and the Quantitative Sciences for Cancer Research at HSPH, and the R25 Post-Baccalaureate Program at Harvard University. Since 2012, I have served as a faculty member for the American Association for Cancer Research Integrative Molecular Epidemiology Workshop, and in 2016 joined as co-Director. Additional mentoring activities include formal mentoring of doctoral and MPH students at HSPH – including in epidemiology, environmental health and biostatistics, research mentoring of numerous clinical fellows in urology, medical oncology, and radiation oncology at the

Harvard teaching hospitals and the DF/HCC, creating educational opportunities, mentoring and career development for students and fellows through my role as head of the cancer epidemiology track at HSPH. Finally, I have spearheaded a number of efforts to support early-stage investigators in cancer population sciences through my role at the DF/HCC.

FUNDED GRANTS:

2002-08 National Institutes of Health/National Cancer Institute, R01CA090598 (PI Stampfer)

Growth Factors and Prostate Cancer Risk
The goal of the study was to examine biomarkers in insulin/insulin-like growth factor in prostate cancer risk and progression. Role: Co-Investigator

2003-06 US Army Prostate Cancer Program, PC031057, Idea Development Award *(PI Adami)*

A Population-Based Study of Dietary Acrylamide and Prostate Cancer Risk
This study aimed to examine the association between dietary intake of acrylamide and risk of prostate cancer. Role: Investigator

2004-07 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50CA90381, Career Development Award (PI Mucci)

A Composite Biomarker for Prostate Cancer Death
The aim of this project was to develop a molecular signature in tumors that accurately predicted prostate cancer mortality. Role: Principal Investigator

2005-08 US Army Prostate Cancer Program, PC040715, Idea Development Award (PI Rubin)

Identification of Aggressive Prostate Cancer using SNP Analysis
The goal of this study was to identify inherited genetic risk loci associated with more aggressive prostate cancer. Role: Investigator

2005-08 US Army Prostate Cancer Program, PC050696, New Investigator Award (PI Mucci)

Molecular and Clinical Predictors of Aggressive Prostate Cancer
The objective of this study was to integrate tumor biomarkers with detailed clinical and histological data in prostate cancer patients to predict risk of prostate cancer death during follow-up. Role: Principal Investigator

2006-07 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50CA90381, Development Award (PI Mucci)

Biomarkers of Angiogenesis and Development of Lethal Prostate Cancer
This project sought to characterize morphologic features of angiogenesis associated with tumors, including microvessel density and shape, and examine their relationship with prostate cancer mortality. Role: Principal Investigator

2007-09 Harvard William F. Milton Fund (PI Mucci)

Infectious origins of prostate cancer
This study aimed to investigate the association between serologic evidence of *Trichomonas vaginalis*, measured in pre-diagnostic bloods, and future risk of prostate cancer, particularly advanced disease. Role: Principal Investigator

2007-09 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50CA90381, Developmental Project Award (PI Mucci)

Genetic variation and the TMPRSS2:ETS fusion in prostate pathogenesis
This study examined the association between genetic variants in the androgen receptor and risk of prostate cancer defined by the common molecular subtype, *TMPRSS2:ERG* nested within two cohorts of men. Role: Principal Investigator

2007-10 US Army Prostate Cancer Program, Idea Development Award (PI Adami)

The Infectious Pathogenesis of Prostate Cancer
This study aimed to investigate the association between histologic markers of inflammation and atrophy, as well as presence of a novel retrovirus (XMRV) in prostate tissue as predictors of prostate cancer mortality in a Swedish cohort. Role: Investigator

2008-09 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50CA90381, Developmental Project Award (PI Stampfer)

Dietary phytoestrogens in relation to prostate cancer risk and progression
This study sought to examine the association between dietary intake of phytoestrogens and prostate cancer risk and mortality in a cohort of Swedish men. Role: Co-Investigator

2008-09 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50 CA90381, Career Development Award (PI Stark),

Proliferative inflammatory atrophy in prostate cancer: a patho-epidemiology study
This study investigated a common histologic lesion in prostate cancer, proliferative inflammatory atrophy, in prostate cancer progression as well as associations with lifestyle factors. Role: Co-Mentor

2008-11 Prostate Cancer Foundation, Young Investigators Award (PI Mucci)

Do dietary and lifestyle factors interact with the TMPRSS2:ERG fusion to predict progression
This patho-epidemiology study sought to examine the association between the *TMPRSS2:ERG* gene fusion and prostate cancer progression, as well as how lifestyle factors and genetic risk loci interact with the gene fusion to affect outcomes in men with prostate cancer. Role: Principal Investigator

2008-13 National Institutes of Health/National Cancer Institute, R01CA131945 (PI Loda)

Metabolic syndrome, fatty acid synthase, and prostate cancer
This project integrated human and experimental studies to examine the role of metabolic syndrome in prostate cancer risk and progression, and to investigate the role of the de novo lipogenesis enzyme, fatty acid synthesis, in the interplay. Role: Co-Investigator

2008-13 Harvard School of Public Health, Ellison Foundation (PI Mucci and Adami)

Genetic and environmental contributions to cancer etiology and progression among 150,000 Nordic Twins
This project allowed the creation of the Nordic Twin Study of Cancer (NorTwinCan), which included almost 300,000 twins from Denmark, Finland, Norway, and Sweden. The goal of the study was to investigate the familial risk and heritability of cancers. Role: Co-Principal Investigator

2009-12 Dana-Farber/Harvard Cancer Center Prostate SPORE, P50 CA90381, Full Project Award

TMPRSS2:ERG and SPINK1 in Lethal Prostate Cancer
This population-science project in the Dana-Farber SPORE in Prostate Cancer sought to determine clinical and etiological significance of two molecular events in prostate cancer, *TMPRSS2:ERG* and *SPINK1*. Role: Principal Investigator

2009-14 National Institutes of Health/National Cancer Institute, RO1 CA136578 (PI Mucci)
Sex hormones and the TMPRSS2:ERG fusion in prostate cancer progression
This study investigated the role of sex steroid hormones, including circulating biomarkers, genetic variants, and tissue markers on prostate cancer mortality as a function of the common gene fusion event in prostate cancer, *TMPRSS2:ERG*. Role: Principal Investigator

2010-11 Harvard Catalyst Pilot Award (PI Mucci)

Melatonin and Prostate Cancer: A biomarker study among men in the Reykjavik Cohort
The goal of this study was to examine biomarkers of circadian rhythm, including urinary melatonin levels and genetic variants in circadian clock genes, and the risk of prostate cancer in an Icelandic Cohort. Role: Principal Investigator

2010-12 Dana-Farber/Harvard Cancer Center Prostate SPORE, Full Project Award, NIH/NCI P50 CA90381 (PI Bubley)

Biguanides for the treatment of prostate cancer
This SPORE project used experimental models and epidemiological data to examine the potential of metformin for treatment of advanced prostate cancer, and assess the role of AMPK signaling as a targetable pathway. Role: Co-investigator and PI Subcontract

2010-13 US Army Prostate Cancer Program, W81XWH-10-1-0552, Idea Development Award (PI Mucci)

BRCA1 and Lethal Prostate Cancer
The goal of this study was to examine the role of tumor protein expression of BRCA1 (breast cancer 1) and risk of lethal prostate cancer, and assess its role in DNA repair and cell cycle regulation. Role: Principal Investigator

2010-13 Icelandic RANNIS Foundation (PI Sigurdardottir)

Melatonin and Prostate Cancer
This career development award supported a doctoral student at the University of Iceland to examine melatonin levels measured in prediagnostic urine and risk of prostate cancer. Role: Mentor

2010-15 National Institutes of Health/National Cancer Institute R01 CA141298 (PI Stampfer)

Growth factors and lethal prostate cancer signature
This study sought to develop a molecular signature of lethal prostate cancer using whole genome mRNA profiling data, and to assess associations of circulating levels and genetic variants in the growth factor axis and risk of lethal cancer.
Role: Co-Investigator and PI Subcontract

2011-12 Rose International Traveling Fellowship, Harvard TH Chan School of Public Health (PI Sarah Coseo Markt)

Sleep, melatonin and prostate cancer in Iceland
This travel award supported Sarah Markt to spend time with colleagues at the University of Iceland as part of her thesis project on sleep, circadian rhythm and prostate cancer. Role: Mentor

2011-13 Dana-Farber/Harvard Cancer Center SPORE in Prostate Cancer, Mazzone Career Development Award (PI Wilson)

Phosphorus and calcium intake, tumor microenvironment and prostate cancer progression
The aim of this project was to investigate the association between pre- and post-diagnostic intake of phosphorus and calcium in prostate cancer progression, and assess associations of the dietary factors on tumor biomarkers. Role: Mentor

2011-14 US Army Prostate Cancer Impact Award, PC101749 (PI Sweeney, Dana-Farber Cancer Institute)

A Systems Biology Approach to Link Nuclear Factor Kappa B Activation with Lethal Prostate Cancer

This study comprehensively examined biomarkers of nuclear factor kappa B (NFkappaB) and lethal prostate cancer in prostate cancer patient cohorts. The study integrated data on circulating biomarkers, tumor tissue expression, and genetic variants in genes and pathway defining NFkappaB activation. Role: Investigator and PI Subcontract

2012-2013 US Army Prostate Cancer Research Program, Post-doctoral Fellowship (PI Julie Kasperzyk)

Prostate Cancer Tumor Heterogeneity
The objective of this post-doctoral fellowship award was to support the training and research of the PI to investigate variability and heterogeneity of tumor tissue biomarkers in prostate cancer. Role: Mentor

2012-13 Dana-Farber/Harvard Cancer Center, Mazzone Awards Program, Disparities Research Mechanism (PI Mucci)

Estimating the Prostate Cancer Burden attributed to Lifestyle and Genetic Factors among African-American and White Men
The goal of this proposal was to quantify the extent to which differences in the prevalence of lifestyle factors and genetic variants could explain the population attributable fraction associated with prostate cancer disparities. Role: Principal Investigator

2012-13 Rose International Traveling Fellowship, Harvard TH Chan School of Public Health (PI Irene Shui)

Prostate cancer in Ireland
This travel award supported this post-doctoral fellow to spend time with colleagues at Trinity College, Dublin to undertake epidemiological studies of prostate cancer and to develop a short course on the patho-epidemiology of prostate cancer. Role: Mentor

2012-14 Prostate Cancer Foundation, Challenge Award (PIs: Loda and Mucci)

Shedding light on stromal-epithelial interactions in prostate cancer carcinogenesis and mortality
This international and multidisciplinary project sought to identify and validate gene expression patterns in epithelial and stromal tissue associated with aggressive prostate cancer, and to develop bioinformatics tools for defining the cross-talk between the two compartments. Role: Co-Principal Investigator

2012-14 Urology Care Foundation, Research Scholar Program (PI: Mark Preston)

Association between Finasteride and High-grade or Lethal Prostate Cancer
The goal of this career development award was to support Dr. Preston, a urologic oncology fellow at MGH, too examine the association between finasteride and risk of high-grade or lethal prostate cancer in the Health Professionals Follow-up Study. Role: Mentor

2012-15 Prostate Cancer Foundation, Young Investigator Award (PI: Stephen Finn, Trinity College, Ireland)

Identifying non-coding RNA repertoires of aggressive prostate cancer
This career development award supported Dr. Stephen Finn at Trinity College, Dublin, to explore the expression of non-coding RNAs in tumor tissue and risk of lethal prostate cancer. Role: Co-Mentor

2012-16 US Army Prostate Cancer Program Impact Award, PC112061 (PI: Platz, Johns Hopkins)

Telomere length and lethal prostate cancer
This study sought to develop an automated algorithm for measuring tumor and stroma-associated telomere length in prostate tissue specimens using fluorescent in situ hybridization, and to apply this platform to prostate cancer patient cohorts to assess the prognostic significance of telomere length in lethal prostate cancer. Role: Investigator and PI Subcontract

2013-15 Dana-Farber/Harvard Cancer Center, Mazzone Career Development Award (PI Jennifer Sinnott),

Impact on Prognosis of Inter- and Intratumor Heterogeneity In Prostate Cancer
This career development award supported this post-doctoral fellow to investigate the role of tissue biomarker heterogeneity, both within and across individuals, as prognostic biomarkers. Role: Mentor

2013-16 Prostate Cancer Foundation, Young Investigator Award (PI Jennifer Rider)

The Immunomodulatory and Androgen-Associated Actions of Vitamin D in Prostate Cancer
The aim of this career development award was to support the training and research of Dr. Rider focused on understanding two distinct pathways – immunomodulation and androgen signaling – as underlying the link between vitamin D and prostate cancer. Role: Mentor

2014-16 US Army Prostate Cancer Program, Post-doctoral Fellowship (PI: Ericka Noonan Ebot)

Molecular Epidemiology Investigation of Obesity and Lethal Prostate Cancer
This post-doctoral award supported the training and research of the candidate to investigate tissue-specific biomarkers associated with obesity, and examine their role in lethal prostate cancer. Role: Mentor

2014-16 American Cancer Society, Post-doctoral Fellowship (PI: Thomas Ahearn)

TMPRSS2:ERG, insulin/IGF1 signaling axis and prostate cancer progression

The goal of this post-doctoral fellowship award was to support the training and research of the candidate to investigate whether insulin and IGF1 signaling contribute to lethal prostate cancer among men whose tumors contain the common gene fusion event. Role: Mentor

2014-16 National Institutes of Health/National Cancer Institute, R21 CA185787 (PI: Svitlana Tyekucheva)

Statistical methods for tumor expression data from archival tissues in clinical and epidemiologic research
The goal of this proposal is to develop new biostatistical methods to analyze transcriptome profiling data from archival tumor materials, and to develop a publically available software tool. Finally, the aim is to test these tools in a prostate cancer patient cohort with whole genome expression profiling data. Role: Co-Investigator and PI Subcontract

2014-16 Dana-Farber/Harvard Cancer Center, Mazzone Awards Disparities Research Program

Do Baseline Prostate Specific Antigen (PSA) Levels Predict Advanced Prostate Cancer in African-American Men? (PI: Mark Preston)
The goal of this study was to examine whether pre-diagnostic levels of PSA accurately predict future risk of prostate cancer, particularly aggressive disease, in the Southern Community Cohort Study. Role: Co-Investigator

2015-16 Dana-Farber Cancer Institute Sponsored Research (PI: Mucci)

International Registry to Improve Outcomes in Men with Advanced Prostate Cancer, IRONMAN
This pilot funding supported the design and protocol development of an international registry of 5,000 men with advanced prostate cancer. Role: Principal Investigator

2013-17 National Institutes of Health/National Cancer Institute, R25 CA174664 Institutional Training and Education Grant (PI: Thomas Sellers)

Integrative Molecular Epidemiology Workshop
The goal of this training and education grant is to support an annual one-week educational workshop to train the next generation of cancer researchers with skill sets integrating biology and epidemiology. Role: Faculty Mentor

2014-17 Prostate Cancer Foundation, Young Investigator Award (PI: Kathryn Wilson)

Bone metabolism and bone metastases in prostate cancer
This career development award was focused on investigating whether prostate tumors exhibit bone like features that facilitate the development of metastatic prostate cancer. Moreover, the goal was to assess whether obesity altered the tumor environment to influence the bone homing that is common in prostate cancer metastases. Role: Mentor

2013-18 National Institutes of Health/National Cancer Institute, R01 CA174206 (PI: Giovanni Parmigiani)

Bioinformatics Tools for Genomic Analysis of Tumor and Stromal Pathways in Cancer
The focus of this project is to develop new computational methods to identify unique signals of gene expression from tumor epithelium and stroma from admixture samples, and to develop methods to identify the cross-talk of transcriptional programs of the two compartments. Finally, the aim is to apply these tools to study the role of obesity on alterations in stromal gene expression. Role: PI Subcontract and Co-Investigator

2013-18 National Cancer Institute/National Institutes of Public Health R01 CA179129 (PI: Kathryn Wilson)

Bone metabolism and bone metastases in prostate cancer
This proposal seeks to investigate tissue biomarkers and circulating markers associated with bone metabolism as predictors of lethal prostate cancer, and to study the association between lifestyle and dietary factors in relation to the biomarkers. Role: Co-Investigator

2013-18 Dana-Farber/Harvard Cancer Center SPORE in Prostate Cancer, National Cancer Institute/National Institutes of Health, P50 CA090381 (PI Kantoff)

Tumor and circulating markers as links between obesity and lethal prostate cancer
This population science project within the DFHCC SPORE in Prostate Cancer aims to understand the mechanism underlying the link between obesity and lethal prostate cancer. The proposal integrates systemic and tissue based biomarkers of metabolism and inflammation, and also integrates data on molecular subtypes of prostate cancer. Role: Project Leader

2015-22 National Institutes of Health/National Cancer Institute, P30 CA006516 (PI: Edward Benz)*

Dana-Farber/Harvard Cancer Center Support Grant
The aim of the Cancer Epidemiology Program is to support program members to undertake studies of cancer etiology and facilitate the translation of this research into prevention strategies. Role: PI Subcontract and Cancer Epidemiology Program Lead
- On P30 Renewal, Cancer Epidemiology received a score of "Exceptional Merit"

2015-17 National Institutes of Health/National Cancer Institute, UM1 CA167552 (PI: Walter Willett) *

Cancer Epidemiology Cohort of Male Health Professionals
The aim of this cohort infrastructure grant is to support the continued follow-up of the Health Professionals Follow-up Study, including biorepositories, follow-up for incidence and mortality, questionnaires, and participation in consortia. Role: Co-Investigator
2015-2016 Administrative Supplement to collect tumor tissue
* Renewal was submitted as a U01 grant with my role listed as Co-Principal Investigator. Grant was reviewed June 2016 and received an Impact Score of 14.

2016-17 Harvard TH Chan School of Public Health, Career Incubator Award (PI: Mucci)

Obesity, histone modifications and lethal prostate cancer
This pilot project seeks to test the hypothesis that excess body weight is associated with specific histone alterations that contribute to the transcriptional dysregulation of genes involved in tumor progression. Role: PI

2016-19 Movember Foundation (PI: Mucci)

IRONMAN: International registry to improve outcomes in men with advanced prostate cancer
IRONMAN is a global registry of 5,000 men with advanced prostate cancer being recruited from 8 countries. The overarching goals are to understanding the optimal patterns of drug therapies in advanced prostate cancer, to gain understanding of the quality of life detriments among these men, and to identify novel biomarkers that are associated with response to therapy and overall survival. Role: PI

2016-21 National Institutes of Health, R25 Post-baccalaureate Program at Harvard University (PI: Meredith Rosenthal)

Role: Faculty Mentor

2016-21 National Cancer Institute/National Institutes of Health, R01 R01CA202690 (PI: Mucci)

Circadian disruption and risk of prostate cancer in a multiethnic cohort
The study aims to investigate circadian disruption as a risk factor for prostate cancer in a multiethnic cohort. The study integrates a molecular epidemiology approach to evaluate common variation in circadian related genes, urinary levels of melatonin and sleep data.

2016-21 National Cancer Institute/National Institutes of Health, U01CA113913 (PI: Sanda)

Harvard and University of Washington Prostate Cancer Biomarker Center
This clinical validation center seeks to advance innovative assays to facilitate the detection of prostate cancer through meaningful application. Role: Co-Investigator and PI subcontract

2017-20 Prostate Cancer Foundation Young Investigator Award (PI: Preston)

Improving risk prediction of aggressive prostate cancer using baseline PSA during midlife and inherited genetic variants in African-American and Caucasian men
The goal of this mentored project is to devise smarter PSA screening strategies by comprehensively investigating the ability of baseline PSA along at midlife with inherited genetic variants to predict future risk of aggressive prostate cancer, with a focus on African-Americans populations. Role: Mentor

2017-20 Prostate Cancer Foundation Young Investigator Award (PI: Stopsack)

Cholesterol metabolism, statins and lethal prostate cancer
The goal of this mentored project is to investigate the role of a key gene in cholesterol metabolism, SQLE in lethal prostate cancer. Moreover, to examine the interaction between SQLE, statins and risk of lethal disease. Role: Co-Mentor

TEACHING AND TRAINING:

TEACHING IN HARVARD CHAN SCHOOL COURSES:

1999 *Introduction to Epidemiology*
Teaching Assistant
110 Students; 55 hours/year

1999-2002 *Epidemiology of Infectious Disease*
Teaching Assistant; Senior Teaching Assistant
50 Students; 55 hours/year

2000-01 *Epidemiological Analysis of Outbreaks and Infectious Disease*
Teaching Assistant
50 Students; 55 hours/year

2000-02 *Epidemiology of HIV/AIDS*
Senior Teaching Assistant
40 Graduate Students; 50 hours/year

2000-02 *Analysis of Case-control and Cohort Studies*
Senior Teaching Assistant
90 Graduate Students; 120 hours/year

2004-05 *Cancer Prevention*
Guest Lecturer
40 Graduate Students; 20 hours/year

2005 *Advanced Topics in Cancer Epidemiology*
Course Instructor (with Jing Ma)
10 Graduate Students; 60 hours/year

2006-07 *Screening*
Grading Instructor (with Monica McGrath)
40 Graduate Students; 160 hours/year

2006- *Applied Biomarkers in Cancer Epidemiology*
Guest Lecturer
15 Graduate Students; 20 hours/year

2007-13 *Practice of Epidemiology*
Course Instructor (with Meir Stampfer)
20 Graduate Students, 60 hours/year

2007- *Epidemiology of Cancer*

Grading Instructor (with Edward Giovannucci)
35 Graduate Students, 100 hours/year

2009 *Nutritional Epidemiology of Cancer*
Guest Lecturer
15 Graduate Students, 15 hours/year

2012-14 *Clinical Epidemiology, Clinical Effectiveness Program*
Workshop Leader
4 Graduate Students, 5 hours/year

2014 *Summer Program in Quantitative Sciences*
T36 Funded
Guest Lecturer

2014-16 *Summer Program in Epidemiology*
Student Mentor
3 Undergraduate Students

2014- *Gender and Health: Introductory Perspectives*
Guest Lecturer
20 Graduate Students, 10 hours/year

2015 *ID 201 Core Biostatistics and Epidemiology for Public Health Practice*
Guest Lecturer
80 Graduate Students, 10 hours/year

TEACHING IN EXECUTIVE AND CONTINUING EDUCATION COURSES:

2011- *American Association for Cancer Research Integrative Workshop on Molecular Epidemiology*
Workshop Faculty Member
2015- Workshop Co-Director
50 Graduate Students, Post-docs, junior faculty, 40 hours/year

2016 *18th Bienniel Jerome P Richie Urologic Oncology Course*
Course Faculty Member
180 Urologists, Medical Oncologists, Physicians, 10 hours/year

TEACHING IN OTHER HARVARD COURSES:

2010 *Molecular Pathology Bootcamp, Harvard Medical School*
Guest Lecturer
10 Graduate Students, 10 hours/year

2011-13 *Molecular Pathology and Epidemiology Bootcamp, Harvard Medical School*

Co-course Leader (with Dr. Massimo Loda)

2012-13 *PH207x. Health in Numbers: Quantitative Methods in Clinical and Public Health Research, edX of Harvard and MIT*
Guest Lecturer

TEACHING COURSES AT OTHER INSTITUTIONS:

2003 *Design Issues in Epidemiology, Boston University School of Public Health*
Guest Lecturer
8 Graduate Students; 15 hours/year

2004 *Cancer Epidemiology, Boston University School of Public Health*
Course Instructor/Director
10 Graduate Students; 120 hours/year

2006 *Cancer Epidemiology and Biomarkers, Modern Methods in Biostatistics and Epidemiology, Cison di Valmarino, Italy*
Course Instructor/Director
12 Graduate students, 60 hours/year

2007-08 *Design and Analysis of Case-control Studies, Modern Methods in Biostatistics and Epidemiology, Cison di Valmarino, Italy*
Course Instructor/Director
16 Graduate students, 60 hours/year

2009 *Design of Case-control Studies and Cohort, Modern Methods in Biostatistics and Epidemiology, University of Iceland, Reykjavik, Iceland*
Course Instructor/Director
25 Graduate Students, 60 hours/year

2011 *Translational Research using Bioinformatics and Epidemiology, Kings College, London, UK*
Course Instructor
20 Graduate Students, 20 hours/year

2012 *Study Design in Epidemiology Research: Case-control studies, University of Iceland, Reykjavik*
Guest Lecturer
35 Graduate Students, 20 hours/year

2013 *Integrative methods for prostate cancer research: bridging molecular and population science, Molecular Medicine Ireland, Trinity College, Dublin*
Course Instructor
50 Graduate and Medical Students, 8 hours/year

ADVISORY AND SUPERVISORY RESPONSIBILITIES:

Training	*Name*	*Current Position*
2004-06	Stephanie Bakaysa, MD MPH Student	Attending Physician Newton-Wellesley Hospital, MA
2004-07	Katja Fall, MD, PhD Post-doctoral Fellow	Associate Professor University of Orebro, Sweden
2005-	Kathryn Wilson, ScD SD Student (Secondary Mentor) Post-doctoral fellow (Mentor)	Research Scientist Harvard T.H. Chan School of Public Health
2005-08	Christine Jesser, ScD SD Student (Primary Mentor)	Epidemiologist University Medical Center, Texas
2006-07	Patravoot Vatanasapt, DMD MPH (Advisor)	Chairman of Otolaryngology, Khon Kaen University
2006-15	Jennifer (Stark) Rider, ScD SD Student (Secondary Mentor) Post-doctoral Fellow (Mentor) Junior faculty (Mentor)	Assistant Professor Boston University School of Public Health Adjunct Assistant Professor Harvard T.H. Chan School of Public Health
2007-	Irene Shui, ScD Doctoral Student (Advisor) Post-doctoral Fellow (2nd Mentor)	Senior Scientist Genzyme
2007-08	Ioannis Rigas, MD, MPH MPH Student (Advisor)	
2007-08	Julia Hayes, MD, MPH MPH Student (Advisor)	Attending Medical Oncologist Dana-Farber Cancer Institute
2007-10	Aditi Hazra, PhD Postdoctoral Fellow (2nd Mentor)	Assistant Professor Harvard Medical School
2007-12	Mara Meyer Epstein, ScD Doctoral Student (Advisor) Post-doctoral Fellow (Mentor)	Assistant Professor University of Massachusetts Medical Center
2008	Keerthana Gnanapradeepan Continuing Umbrella of Research Experience (CURE) (Mentor)	Graduate Student University of Pennsylvania School of Medicine

2008-09	Shih-Wen Lin, PhD, MPH MPH Student (Advisor)	Epidemiologist Genentech
2008-09	Michaela Cada, MD, MPH MPH Student (Advisor)	Assistant Professor of Pediatrics University of Toronto
2008-10	David Wheeler, PhD, MPH MPH Student (Advisor)	Assistant Professor Virginia Commonwealth University
2009-10	Danielle Margalit, MD, MPH MPH Student (Mentor) Clinical Fellow (Mentor)	Assistant Professor Dana-Farber Cancer Institute
2009-10	Annette Kaufman, PhD, MPH MPH Student (Advisor)	Program Director, Tobacco Control National Cancer Institute
2009-12	Wang Xiang Doctoral Student (Advisor)	Brilent, Inc Data Analytics Fellow
2009-14	Rebecca Graff, ScD SD Student (Advisor)	Post-doctoral Fellow University of California, San Francisco
2009-15	Lara Sigurdardottir, PhD PhD Student (University of Iceland; Co-Mentor)	Researcher Icelandic Cancer Society
2010-	Sarah Coseo Markt, ScD SD Student (Advisor) Post-doctoral Fellow (Mentor)	Research Associate Harvard T.H Chan School of Public Health
2010-	Jennifer Sinnott, PhD PhD Student (Thesis Committee) Post-doctoral Fellow (Mentor)	Assistant Professor of Biostatistics The Ohio State University
2010-11	Piotr Zareba, MD, MPH MPH Student (Advisor)	Attending Urologist Memorial Sloan Kettering Cancer Institute
2010-11	Tryggvi Thorgeirsson, MD, MPH MPH Student (Advisor)	Attending Physician University of Iceland
2010-12	Andreas Pettersson, MD, PhD Post-doctoral Fellow (Mentor)	Resident, Medical Oncology Karolinska Institutet, Sweden
2010-13	Elisabete Moller, PhD Doctoral Student	Nutritionist Swedish Association of Professional

	(Karolinska Inst; Co-Supervisor)	Scientists
2011-12	Henry Park, MD, MPH MPH Student (Advisor)	Resident Yale University Medical School
2011-12	Yen Chien MPH Student (Advisor)	
2011-12	Sun Mi Yoo, MD, MPH MPH Student (Advisor)	Chief Resident, Internal Medicine University of California, Los Angeles
2011-13	Jonathan Schoenfeld, MD, MPH MPH Student/Clinical Fellow (Mentor/Research advising)	Assistant Professor Dana Farber Cancer Institute
2011-14	Mark Preston MPH student and Clinical Fellow at MGH (Research advising)	Assistant Professor and Attending Physician Brigham and Women's Hospital/HMS
2012	Gregory Judson, MD MD student, Columbia (Faculty Mentor)	Chief Resident University of California, San Francisco
2012-	Ericka (Noonan) Ebot, PhD, MPH Post-doctoral Fellow (Mentor)	Research Associate Harvard T.H. Chan School of Public Health
2012-13	Ardalan Ebrahimi, MD, MPH MPH Student (Advisor)	Attending Physician Macquarie University
2012-14	Travis Gerke, ScD SD Student (Mentor)	Assistant Professor of Epidemiology Moffitt Cancer Center
2012-14	Alejandro Sanchez, MD Resident in Urology, Massachusetts General Hospital (Research Advising)	Resident in Urology
2012-16	Thomas Ahearn, PhD Post-doctoral Fellow (Mentor)	Staff Scientist National Cancer Institute
2013-	Claire Hampton Pernar, MS ScD Student (Mentor)	
2013-14	Kimberly Mak, MD, MPH MPH Student and Resident at BWH (Research advising)	Assistant Professor in Radiation Oncology Boston Medical Center

2013-14	Kazusa Ishii, MD, MPH MPH student (Mentor)	Clinical Fellow National Heart, Blood and Lung Institute
2013-14	Sigrid Carlsson, MD, PhD MPH student (Research mentor)	Associate Professor and Urologist Memorial Sloan Kettering Cancer Center
2014	Ahmed Sabri Alla Visiting Student, Alexandria University, Egypt (Research Advising)	
2014	Lorenzo Richiardi, PhD Fulbright Scholar, HSPH (Faculty mentor)	Associate Professor of Epidemiology University of Turin, Italy
2014-15	Vicente Morales Oyarvide, MD, MPH MPH Student (Advisor)	Post-doctoral Fellow Dana-Farber Cancer Institute
2014-15	Taylor Medwig Undergraduate student, Stonybrook (Research advising)	Graduate Student Stonybrook
2014-15	Christopher Allard, MD MPH Student and Urologic Oncology Fellow at MGH/DFCI (Research advising)	Attending Urologist
2014-15	Alexandra Greenberg, PhD MPH Student (Research mentor)	Research Associate Mayo Clinic, Rochester
2014-16	Reginald Tucker-Seeley, PhD Assistant Professor (K01 mentor)	Assistant Professor HSPH/Dana-Farber Cancer Institute
2014-16	Masis Isikbay Harvard Medical School student (Research mentor)	Medical Student Harvard Medical School
2014-16	Barbara Dickerman, SM SM2 Student (Advisor) 2016- PhD Student (Advisor)	PhD Student Harvard T.H. Chan School of Public Health
2014-16	Sarah Lucht, SM SM2 Student (Advisor)	PhD Student in Epidemiology University of Essen, Germany
2014-16	Lauren Barber, SM SM2 Student (Research Mentor)	PhD Student in Epidemiology Boston University School of Public Health

2014-16	Sarah Legge, ALM ALM Student, Harvard University Extension School (Thesis Director)	Teacher, MA School system
2014-5	Konrad Stopsack, MD MPH Student (Research Mentor) 2015- Medical resident Research Advising	Medical Resident Mayo Clinic, Rochester
2015-	Emma Allott, PhD Research Mentor John Fitzpatrick Fellowship Boston-Irish Prostate Cancer Collaboration	Research Assistant Professor University of North Carolina
2015-16	Vitor Moutinho da Coneicao Junior, MD SM1 Student (Advisor)	
2015-16	Hsi Yen, MD MPH Student (Advisor)	
2015-16	Cendrine Robinson, PhD MPH Student (Advisor)	Cancer Prevention Fellow National Cancer Institute
2016	Keyan Salari, MD Resident in Urology Massachusetts General Hospital (Research Advising)	
2016-	Nadine Hamieh Visiting Student, American University of Lebanon (Research Advising)	
2016-	Dana Hashim, PhD Research Advising	Post-doctoral Fellow Mt Sinai Cancer Institute
2016-17	Kristen Pluchino, PhD MPH Student (Mentor)	Cancer Prevention Fellow National Cancer Institute
2016-2017	Sabrina Tsang, PhD MPH Student (Mentor)	Cancer Prevention Fellow National Cancer Institute
2016-	Suna Park	

	SM2 Student (Mentor)
2017-	Cindy Zhou, PhD Post-doctoral Fellow
2017	Brendan Rowen MD Student, University College, Dublin Research Advising
2017-2018	Junkun Ren SM2 Student (Mentor)
2017-2018	Dongzhengyan SM2 Student (Mentor)

Other Mentoring

2009	Thesis Examiner for PhD candidate (Janneke Hogervorst), *Dietary acrylamide and human cancer risk,* Maastricht University, Maastricht The Netherlands
2010	Thesis Examiner for PhD candidate (Elizabeth Tindall), *Genetic contributions to inflammatory mediated prostate cancer*, University of New South Wales, Australia

INVITED PRESENTATIONS:

Local

2004	*Molecular Signatures of Prostate Cancer Survival* Harvard School of Public Health
2004	*Plasma Levels of Free IGF-1, Acid-labile Subunit and Prostate Cancer Risk: A Prospective Study* Channing Laboratory/Brigham and Women's Hospital Seminar Series
2004	Invited Speaker, *Acid-labile Subunit and Prostate Cancer Risk: A Prospective Study*, Slone Epidemiology Seminar, Boston University School of Public Health, Boston, MA
2005	Invited Speaker, *Identifying Molecular Markers of Indolent and Aggressive Prostate Cancer,* Boston University Medical Grand Rounds, Boston Medical Center, Boston MA
2006	*Microvessel Density and architecture: biomarkers of lethal prostate cancer* Harvard Prostate Cancer Working Group
2006	*Molecular signatures to predict lethal and indolent prostate cancer*

BWH-BRI Inaugural Cancer Research Center Retreat

2007 *Biomarkers of tumor angiogenesis and lethal prostate cancer*
Dana-Farber/Harvard Cancer Center Prostate SPORE Meeting

2007 *Incorporating tumor biomarkers into cancer epidemiology studies: the prostate cancer story* Department of Epidemiology Seminar Series, Harvard School of Public Health

2008 Invited Speaker, *Metabolism and Prostate Cancer in the PHS and HPFS cohorts*, Cancer Genome Program, Broad Institute, Cambridge, MA

2008 *Genetic susceptibility and the TMPRSS2:ERG fusion*
Dana-Farber/Harvard Cancer Center SPORE Meeting

2008 *Dietary and biochemical predictors of prostate cancer risk and progression* Harvard Urology and Prostate Cancer Seminar Series, Harvard Institute of Medicine

2008 Invited Panelist, *Work and Family Balance Panel*
Office for Women's Careers, Brigham and Women's Hospital

2008 Invited Panelist, *Women in Academe: Balancing Family and Careers*
Harvard School of Public Health

2008 *Prostate Tumor Biomarker Studies in the Health Professionals Follow-up Study*
Health Professionals Follow-up Study External Advisory Board Meeting, Harvard School of Public Health

2008 *The TMPRSS2:ERG fusion and the sex hormone milieu*
Dana-Farber/Harvard Cancer Center SPORE in Prostate Cancer Meeting, DFCI

2009 *The TMPRSS2:ERG fusion in prostate cancer*
Molecular Epidemiology Working Group Seminar, Harvard School of Public Health

2009 Invited Speaker, *Prostate Cancer Epidemiology and the TMPRSS2:ERG fusion*
Dana-Farber/Harvard Cancer Center Conference on Cancer

2009 Invited Speaker, *Translocations and Aberrations: a patho-epidemiology study of prostate cancer risk and progression*
Department of Epidemiology Seminar, Harvard School of Public Health

2010 *TMPRSS2:ERG and SPINK1 in Lethal Prostate Cancer*
Dana-Farber/ Harvard Cancer Center SPORE in Prostate, External Advisory Board Meeting

2011 *TMPRSS2:ERG, SPINK1 and Lethal Prostate Cancer*
Dana-Farber/Harvard Cancer Center Prostate SPORE Meeting

2011 *The Burden of Cancer from an Epidemiologist's Perspective*
Summer Program in Quantitative Methods, Harvard School of Public Health

2011 *Future Directions for Translation of Archival Tissue Studies* (Panel Discussion).
Emerging Technologies for Translation Bioinformatics: A symposium on gene expression profiling for archival tissues, Harvard School of Public Health

2011 *Shedding Light on the Heritability of Cancer: a study of 200,000 Nordic Twins*
Department of Epidemiology Seminar Series, Harvard School of Public Health

2011 Keynote Address, *Prevention of Lethal Prostate Cancer: opportunities and novel hypotheses*, Massachusetts Prostate Cancer Coalition, Newton MA

2011 Invited Speaker, *The burden of cancer from an epidemiologist's perspective*, Massachusetts College of Pharmacy CAPSTONE course, Boston, MA

2012 *Cancer: what have we learned and where do we go from here?*
Harvard School of Public Health Leadership Council Meeting

2012 *Unveiling the potential of patho-epidemiology to understand prostate cancer*
Quantitative Issues in Cancer Research Working Seminar, Department of Biostatistics, Harvard School of Public Health

2012 *The burden of cancer from an epidemiologist's perspective*, Massachusetts College of Pharmacy CAPSTONE course, Boston, MA

2012 Invited Speaker, *MicroRNA in tumor tissue: overview of design and pilots for prostate cancer*, Channing Division of Network Medicine, Brigham and Women's Hospital, Boston MA

2013 *Exploring Mechanisms Underlying the Link between Obesity, Physical Activity and Lethal Prostate Cancer*
Harvard Transdisciplinary Research in Energetics and Cancer (TREC) Annual Meeting

2013 *Tumor/Patient Genotyping Efforts within DF/HCC*
Dana-Farber/Harvard Cancer Center Scientific Council Meeting

2013 *What's hot, what's not: ongoing controversies in PSA screening for prostate cancer*
Hot Topics in Public Health, Harvard School of Public Health

2013 *Unveiling the enigma of prostate cancer epidemiology*
Genitourinary Oncology Seminar, Dana-Farber Cancer Institute

2013 Organizer, *Celebration of Young Investigators in Cancer Research*, Dana-Farber/Harvard Cancer Center

2013 Organizer and Speaker, *What's Up in Cancer Epidemiology*, Dana-Farber/Harvard Cancer Center Population Science Group

2013 Moderator, *"History and Future of Epidemiology" panel session*
Cutter Lecture Symposium, Harvard School of Public Health

2013 *Tumor/Patient Genotyping Efforts within DF/HCC*
Dana-Farber/Harvard Cancer Center Scientific Council Meeting

2014 Invited Speaker, *Gene expression profiling studies in the HSPH cohorts: tantalizing preliminary results*
Dana-Farber/Harvard Cancer Center SPORE in Prostate Cancer meeting

2014 Invited Speaker, *Metabolic consequences of obesity on cancer risk and mortality,*
Using basic and epidemiological studies to identify metabolic vulnerabilities in cancer,
Dana-Farber/Harvard Cancer Center Symposium on Metabolism and Cancer

2014 Invited Speaker, *Integrating Tissue Biomarkers into Prostate Cancer Epidemiology Research,* 2nd International Molecular Pathological Epidemiology Meeting, Dana-Farber Cancer Institute

2014 *Estimating the prostate cancer burden attributed to lifestyle and genetic factors among African-American and White men,* Dana-Farber/Harvard Cancer Center – Prostate Cancer Foundation A. David Mazzone Awards Program Scientific Retreat

2014 *Assessment of the DF/HCC Catchment Area: Massachusetts,* Dana-Farber/Harvard Cancer Center Executive Committee

2014 Invited Speaker, *Estimating the prostate cancer burden attributed to lifestyle and genetic factors among African-American and White men*, A. David Mazzone Awards Program Retreat, Dana Farber/Harvard Cancer Center

2014 Panelist, *Development and Safety Management of Cancer Drugs Workshop.* Harvard School of Public Health and Takeda Pharmaceuticals

2014 Invited Speaker, *Integrating tissue biomarkers into cancer epidemiology studies: examples from prostate cancer,* Harvard University Transdisciplinary Research on Energetics and Cancer Scientific Retreat

2014 Invited Speaker, *The prevention of lethal prostate cancer*, Boston Prostate Cancer Support Group, Beth Israel Deaconess Medical Center, Boston

2014 Invited Panelist, *Prostate Cancer Awareness Day*, Massachusetts State House

2015 Invited Speaker, *Prevention of lethal prostate cancer,* Prostate Health Education Network (PHEN) Support Group Meeting, Dana-Farber Cancer Institute

2015 Invited Speaker, *Assessment of the DF/HCC Catchment Area: Massachusetts*, 2015 Liver Cancer Incubator, Dana-Farber/Harvard Cancer Center

2015 Invited Speaker, *The prevention of lethal prostate cancers,* Boston Prostate Cancer Support Group, Beth Israel Deaconess Medical Center, Boston MA

2016 Invited Speaker, *Prevention of lethal prostate cancer*
Massachusetts General Hospital Urology Grand Rounds

2016 Invited Speaker, *Obesity and lethal prostate cancer*
DF/HCC SPORE in Prostate Cancer Monthly Meetings

2016 Invited Speaker, *Novel advances in prostate cancer prevention*, Massachusetts Prostate Cancer Coalition, Newton MA

2016 Invited Speaker, *Opportunities for research in GU Cancers in the Harvard Cohorts*, Genitourinary Oncology Research Seminar, Dana-Farber Cancer Institute

National

2004 Invited Speaker, *Dietary acrylamide and risk of cancer*, American Chemical Society, 227th National Meeting in Anaheim, CA

2005 Invited Speaker, *Dietary Acrylamide and risk of human cancer: the role of epidemiology*, Society of Toxicology, 44th Annual Meeting, New Orleans, LA

2006 Invited Speaker, *Identifying molecular signatures of indolent and lethal prostate cancer.* Prostate Cancer InterSPORE Meeting, National Cancer Institute, Houston, TX

2007 Invited Speaker, *Multigene signatures of indolent and lethal prostate cancer*, Active Surveillance for Early Stage Prostate Cancer, San Francisco, CA

2007 Panelist, *The role of acrylamide in diet and risk of cancer*, American Chemical Society, 229th National Meeting, Boston, MA

2008 Invited Speaker, *Obesity and the TMPRSS2:ERG Fusion*, Prostate Cancer Foundation Annual Retreat, Lake Tahoe, CA

2008 Invited Speaker, *Obesity and Prostate Cancer Progression in the Physicians' Health Study*, National Cancer Institute Translational Meeting, Washington DC.

2008 Invited Speaker, *Central adiposity and prostate cancer survival in relation to tumor tissue expression of sex steroid hormone receptors*, Tri-institutional Prostate Cancer Program Retreat, Newport, RI

2009 Invited Speaker, *Tomatoes, lycopene and prostate cancer: is the association with disease progression mediated through angiogenesis?* Tri-institutional Prostate Cancer Program Retreat, Baltimore, MD

2009 Invited Speaker, *Do antioxidants prevent risk of TMPRSS2:ERG fusion prostate cancer?* Prostate Cancer Foundation Annual Scientific Retreat, Lake Tahoe CA

2009 Invited Speaker, *TMPRSS2:ERG fusion and SPINK1 in prostate cancer etiology and progression,* National Cancer Institute Translational Meeting, Tyson's Corner, VA

2010 Invited Speaker, *Genetic and lifestyle factors impact prostate cancer survival through angiogenesis.* 7th Annual International M. Judah Folkman Conference Antiangiogenesis: New Frontiers in Therapeutic Development, Boston, MA

2010 Invited Speaker, *The patho-epidemiology of prostate cancer.* Multi-institutional Prostate Cancer Program Retreat, Ft Lauderdale, FL

2012 Invited Speaker, *Genetic variation in antioxidant pathway and prostate cancer progression*, SELECT Trial Symposium, Southwest Oncology Group, Dallas, TX

2012 Invited Speaker, *Promenadgruppen: a pilot walking intervention among men with prostate cancer.* Fourth annual Multi-Institutional Prostate Cancer Program Retreat, Ft. Lauderdale, FL

2012 Session Chair, Prostate Cancer: Risk, Fifth annual Multi-Institutional Prostate Cancer Program Retreat, Ft. Lauderdale, FL

2012 Organizer and Co-Leader, Sixth Annual International Prostate Cancer Patho-Epidemiology Retreat, Martha's Vineyard

2012 Invited Speaker, *Tumor Tissue Collection: The Experience of the Harvard Cohorts.* 2011 Annual Meeting of the National Cancer Institute Cohort Consortium, Boston MA

2012 Panelist, Celebration of Science, FasterCures and the Milken Institute, Washington DC

2012 Poster Discussant, American Society for Clinical Oncology Annual Meeting, Genitourinary (Prostate) Cancer, Chicago, Illinois

2014 Invited Speaker, *Chronic Diseases associated with prostate cancer*, Bienniel Prostate Cancer Forum, Prostate Cancer UK, Baltimore, MD

2014 Invited Speaker, *Where are we and where are we going: risk*, Seventh annual Multi-Institutional Prostate Cancer Program Retreat, Ft Lauderdale, FL

2014 Invited Speaker, Educational Session, *Integrative Molecular Epidemiology*, American Association for Cancer Research, San Diego, CA

2014 Invited Speaker, *Tumor Drivers of the Link between Obesity and Lethal Prostate Cancer*, Prostate Cancer Foundation Coffey-Holden Prostate Cancer Academy, Carlsbad, CA

2014 Program Committee and Moderator, 21st Annual Prostate Cancer Foundation Scientific Retreat, Carlsbad, CA

2014 Invited Speaker, *Integrative tissue biomarkers into cancer epidemiology studies: examples from prostate cancer*. Transdisciplinary Research on Energetics and Cancer (TREC) Annual Scientific Meeting, Boston, MA

2014 Invited Speaker, *Integrative molecular epidemiology of prostate cancer*, Vermont Cancer Center's Clinical & Translational Research Symposium, Burlington, VT

2015 Invited Speaker, *Precision prevention in prostate cancer: the case for TMPRSS2:ERG*, 6th International PACRIM Breast and Prostate Cancer Meeting, Stevenson, WA

2013 Session Chair, Prostate Cancer: Risk Session, Sixth annual Multi-Institutional Prostate Cancer Program Retreat, Ft. Lauderdale, FL

2013 Panelist, *Living to 1000: Impossible or in reach?* Milken Institute Global Conference, Beverly Hills, CA

2013 Invited Panelist, *Prouts Neck 2.0 Meeting on Prostate Cancer.* Beyond AR: New Approaches to Treating Metastatic Prostate Cancer, San Diego, CA

2013 Invited Speaker, *Unveiling the potential to prevent lethal prostate cancer: Integrative molecular epidemiology approaches to public health*, Northwestern SPORE in Prostate Cancer, Fred Hutchinson Cancer Institute, Seattle, WA

2013 Session chair and speaker, *The patho-epidemiology of prostate cancer: translating population science to prevention and treatment of advanced prostate cancer*, 20th Annual Prostate Cancer Foundation Annual Scientific Retreat, Washington DC

2015 Session chair and speaker, *Integrating Tissue Biomarkers into Cancer Epidemiology Studies*, National Cancer Institute's Cohort Consortium, Gaithersberg, MD

2015 Invited Speaker, *PCF5000: A Novel Disease Registry among Men with Advanced Prostate Cancer,* Eighth Annual Multi-Institutional Prostate Cancer Program Retreat, Ft Lauderdale, FL

2015 Invited Speaker, *Unraveling the mystery of prostate cancer's etiology*, Department of Epidemiology Seminar Series, University of Florida, Gainesville, FL

2015 Featured Invited Speaker, *Epidemiology of Prostate Cancer Risk and Progression,* Prostate Cancer Evidence Academy, University of Pennsylvania

2016 Presenter, *Cancer Epidemiology Cohort in Male Health Professionals*, National Cancer

Institute Tissue Supplement Webinar

2016 Invited Speaker, *Diet, lifestyle and Lethal Prostate Cancer*, Centennial Meeting of the Endocrine Society (ENDO 2016), Boston, MA

2016 Invited Speaker, *Unraveling the enigma of prostate cancer epidemiology*, H. Lee Moffitt Cancer Center, Tampa, FL

2016 Organizer and Moderator, First Annual Prostate Cancer Foundation Women's Networking Forum, Carlsbad, CA

2017 Invited Speaker, *Genomic tests in active surveillance and the role of hereditary testing,* 2017 Genitourinary Cancers Symposium, ASCO, Orlando, FL

2017 Chair, Molecular and Genetic Epidemiology Minisymposium, 2017 American Association for Cancer Research Meeting, Washington DC

International

2004 Invited Speaker, *Tissue Microarrays in Cancer Epidemiology*, 2004 Hydra Cancer Meeting, Hydra, Greece

2006 Invited Speaker, *Epidemiological studies on the relationship between acrylamide in the diet and cancer risk, Multidisciplinary approaches to reducing the levels of acrylamide in food*, Association of Applied Biologists, Hertfordshire, UK

2007 Panelist, *Icelandic Meeting on Prostate Cancer* (*Progress*), University of Iceland, Reykjavik, Iceland

2008 Invited Speaker, *Tumor angiogenesis and prostate cancer mortality*, Department of Urology, University of Orebro, Orebro, Sweden

2008 Invited Speaker, *Concepts and Principles of Cancer Screening*, University of Iceland, Reykjavik, Iceland

2008 Panelist, *Bladder Cancer-from Pathogenesis to Prevention-International Consultation*, World Health Organization International Consultation, Stockholm, Sweden

2008 Educational Review Panel for MPH and doctoral students at the Division of Public Health Sciences, University of Iceland, Reykjavik

2009 Panelist, *Prostate Cancer Retreat*, University of Orebro, Orebro, Sweden

2009 Invited Speaker, *The state of affairs of epidemiological research on acrylamide and human cancer risk*, Maastricht University, The Netherlands

2010 Invited Speaker, *Promenade Gruppen: Background and Hypothesis for the randomized trial*, Prostate Cancer at Solstice Meeting, University of Iceland, Reykjavik, Iceland

2010 Invited Speaker, *The Patho-Epidemiology of Prostate Cancer: An epidemiologist's perspective*, Società Italiana di Urologia Oncologica (SIURO), Rome, Italy

2010 Symposium Panelist and Speaker, *Bologna Patho-epidemiology Prostate Cancer Retreat*, University of Bologna, Italy

2010 Invited Speaker, *Nutrigenetics: Antioxidants and SNPs in antioxidant genes in relation to prostate cancer*, Neon Annual Meeting, Nobel Forum, Karolinska Institutet, Stockholm, Sweden

2011 Invited Speaker, *Patho-epidemiology studies of molecular signatures in the SPCG-4 trial*, Scandinavian Prostate Cancer Group Annual Meeting, Stockholm, Sweden

2011 Invited Speaker, *Molecular Markers of Lethal Prostate Cancer*, 9th Annual World Congress of Urological Research, Innsbruck Austria

2011 Invited Speaker, *Shedding Light on the Heritability of Prostate Cancer: a study of 100,000 Nordic Twins*, Invited Lecture, Trinity College, Dublin Ireland

2011 Invited Speaker, *Tumor Drivers of the Link Between Obesity and Lethal Prostate Cancer*, Research Oncology Seminar, Kings College, London UK

2012 Invited Speaker, *Tumor markers of Lethal Prostate Cancer: Links with Obesity*, Faculty Meeting Seminar, Karolinska Institutet, Stockholm, Sweden

2012 Invited Speaker, *Unveiling the heritability in cancer: an updated analysis from the Nordic twin registry of cancer*, 14th Congress of the International Society Twin Studies, Florence, Italy

2012 Invited Speaker, *Integrating mRNA profiling in prostate cancer risk prediction*, Prostate Cancer UK Action Forum, Rotterdam, the Netherlands

2013 Invited Speaker, *Obesity and TMPRSS2:ERG fusion: an example of precision patho-epidemiology*, Australian – Canadian Prostate Cancer Research Alliance, Port Douglas, Queensland, Australia

2013 Invited Speaker, *Circadian disruption: a biomarker of aggressive prostate cancer?* Australian – Canadian Prostate Cancer Research Alliance, Port Douglas, Queensland, Australia

2014 Invited Speaker, *Tumor drivers of the link between obesity and lethal prostate cancer,* Oslo Prostate Cancer Symposium 2014, Oslo, Norway

2015 Invited Speaker, *Obesity, Metabolism and Prostate Cancer Survival*, Sixth International Congress of Uro-Oncology, Sao Paolo, Brazil

2015 Invited Speaker, *PCF 500: A novel registry of men with advanced prostate cancer*, Sixth International Congress of Uro-Oncology, Sao Paolo, Brazil

2015 Invited Speaker, *The role of the circadian rhythm in prostate cancer*, Sixth International Congress of Uro-Oncology, Sao Paolo, Brazil

2015 Invited Speaker, *A Female Researcher Exploring the Male Prostate*, Medical Oncology Departmental Seminar, Kings College, London

2016 Invited Speaker, *International Registry to Improve Outcomes in Men with Advanced Prostate Cancer*, Movember Executive Board, Melbourne, Australia

2017 Invited Speaker, *Epidemiology and Genetics of Prostate Cancer,* American Association for Cancer Research International Meeting, New Frontiers in Cancer Research, Cape Town, South Africa

2017 Invited Speaker, *IRONMAN: International Registry to Improve Outcomes in Men with Advanced Prostate Cancer*, St Gallen Advanced Prostate Cancer Consensus Meeting, St. Gallen, Switzerland

2017 Invited Speaker, *Obesity, altered metabolism, and advanced prostate cancer*, Forum of Public Health and Social Medicine Webinar, University of Athens, Greece

2017 Invited Speaker, *The Integrative Molecular Epidemiology of Prostate Cancer,* 2017 John Fitzpatrick Irish Genitourinary Cancer Meeting, Dublin, Ireland

2017 Invited Speaker, *Prostate Cancer Outcomes: IRONMAN Registry*, TrueNth International Meeting, Vancouver, BC

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Original Research Articles

1. Kuper H, Tzonou A, Lagiou P, **Mucci LA**, Trichopoulos D, Stuver SO and Trichopoulou A. Diet and hepatocellular carcinoma: a case-control study in Greece. Nutr Cancer 2000;38(1):6-12.
2. Signorello LB, Kuper H, Lagiou P, Wuu J, **Mucci LA**, Trichopoulos D and Adami HO. Lifestyle factors and insulin-like growth factor 1 levels among elderly men. Eur J Cancer Prev 2000;9(3):173-8.
3. From the Centers for Disease Control and Prevention. Use of medical care, police assistance, and restraining orders by women reporting intimate partner violence - Massachusetts, 1996-1997. JAMA. 2000 Aug 2;284(5):558-9.

4. Hathaway JE, **Mucci LA**, Silverman JG, Brooks DR, Mathews R and Pavlos CA. Health status and health care use of Massachusetts women reporting partner abuse. Am J Prev Med 2000;19(4):302-7.
5. Kuper H, Hsieh C, Stuver SO, **Mucci LA**, Tzonou A, Zavitsanos X, Lagiou P and Trichopoulos D. Birth order, as a proxy for age at infection, in the etiology of hepatocellular carcinoma. Epidemiology 2000;11(6):680-3.
6. Petridou E, Giokas G, Kuper H, **Mucci LA** and Trichopoulos D. Endocrine correlates of male breast cancer risk: a case-control study in Athens, Greece. Br J Cancer 2000;83(9):1234-7. PMCID: PMC2363586.
7. Kuper H, Lagiou P, **Mucci LA**, Tamimi R, Benetou V and Trichopoulos D. Risk factors for cholangiocarcinoma in a low risk Caucasian population. Soz Praventivmed 2001;46(3):182-5.
8. Kuper H, Mantzoros C, Lagiou P, Tzonou A, Tamimi R, **Mucci L**, Benetou V, Spanos E, Stuver SO and Trichopoulos D. Estrogens, testosterone and sex hormone binding globulin in relation to liver cancer in men. Oncology 2001;60(4):355-60.
9. Lagiou A, Trichopoulos D, Tzonou A, Lagiou P and **Mucci L**. Are there age-dependent effects of diet on prostate cancer risk? Soz Praventivmed 2001;46(5):329-34.
10. Orner MB, Meehan T, Brooks DR, **Mucci LA** and McGuire JF. Support for condom availability and needle exchange programs among Massachusetts adults, 1997. AIDS Educ Prev 2001;13(4):365-76.
11. Silverman JG, Raj A, **Mucci LA** and Hathaway JE. Dating violence against adolescent girls and associated substance use, unhealthy weight control, sexual risk behavior, pregnancy, and suicidality. JAMA 2001;286(5):572-9.
12. Brooks DR and **Mucci LA**. Support for smoke-free restaurants among Massachusetts adults, 1992-1999. Am J Public Health 2001;91(2):300-3. PMCID: PMC1446536.
13. Weiderpass E, Ye W, **Mucci LA**, Nyren O, Trichopoulos D, Vainio H and Adami HO. Alcoholism and risk for endometrial cancer. Int J Cancer 2001;93(2):299-301.
14. **Mucci LA** and Brooks DR. Lower use of dental services among long term cigarette smokers. J Epidemiol Community Health 2001;55(6):389-93. PMCID: PMC1731911.
15. **Mucci LA**, Tamimi R, Lagiou P, Trichopoulou A, Benetou V, Spanos E and Trichopoulos D. Are dietary influences on the risk of prostate cancer mediated through the insulin-like growth factor system? BJU Int 2001;87(9):814-20.
16. Tamimi R, **Mucci LA**, Spanos E, Lagiou A, Benetou V and Trichopoulos D. Testosterone and oestradiol in relation to tobacco smoking, body mass index, energy consumption and nutrient intake among adult men. Eur J Cancer Prev 2001;10(3):275-80.
17. **Mucci LA**, Kuper HE, Tamimi R, Lagiou P, Spanos E and Trichopoulos D. Age at menarche and age at menopause in relation to hepatocellular carcinoma in women. BJOG 2001;108(3):291-4.
18. Demissie S, Green RC, **Mucci L**, Tziavas S, Martelli K, Bang K, Coons L, Bourque S, Buchillon D, Johnson K, Smith T, Sharrow N, Lautenschlager N, Friedland R, Cupples LA and Farrer LA. Reliability of information collected by proxy in family studies of Alzheimer's disease. Neuroepidemiology 2001;20(2):105-11.
19. Kuper H, Ye W, Broome U, Romelsjo A, **Mucci LA**, Ekbom A, Adami HO, Trichopoulos D and Nyren O. The risk of liver and bile duct cancer in patients with chronic viral hepatitis, alcoholism, or cirrhosis. Hepatology 2001;34(4 Pt 1):714-8.
20. Boffetta P, Ye W, Adami HO, **Mucci LA** and Nyren O. Risk of cancers of the lung, head and neck in patients hospitalized for alcoholism in Sweden. Br J Cancer 2001;85(5):678-82. PMCID: PMC2364115.

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22. Lagiou P, Hsieh CC, Trichopoulos D, Xu B, Wuu J, **Mucci L**, Tamimi R, Adami HO and Cnattingius S. Birthweight differences between USA and China and their relevance to breast cancer aetiology. Int J Epidemiol 2003;32(2):193-8.
23. Lagiou P, Tamimi R, **Mucci LA**, Trichopoulos D, Adami HO and Hsieh CC. Nausea and vomiting in pregnancy in relation to prolactin, estrogens, and progesterone: a prospective study. Obstet Gynecol 2003;101(4):639-44.
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26. Brawarsky P, Brooks DR and **Mucci LA**. Correlates of colorectal cancer testing in Massachusetts men and women. Prev Med 2003;36(6):659-68.
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34. **Mucci LA**, Granath F and Cnattingius S. Maternal smoking and childhood leukemia and lymphoma risk among 1,440,542 Swedish children. Cancer Epidemiol Biomarkers Prev 2004;13(9):1528-33.
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