Exhibit 9

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

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

EXPERT REPORT OF DR. BEATE RITZ, M.D., Ph.D.

IN SUPPORT OF GENERAL CAUSATION

ON BEHALF OF PLAINTIFFS

1. Beate Ritz, MD, PhD, Background and Qualifications

I, Beate Ritz, MD, Ph.D., am Professor of Epidemiology at the UCLA Fielding School of Public Health, former Chair of the Epidemiology Department, and I hold co-appointments in Environmental Health Sciences and Neurology at the UCLA, School of Medicine. I was trained in Medicine at the University of Hamburg/Germany and received a doctoral degree from the University of Hamburg in Medical Sociology in 1986. I furthermore received another doctoral degree in Epidemiology from UCLA in 1995, and subsequently was hired as a faculty at UCLA. My faculty appointment at UCLA is one of several positions specifically assigned to the Center of Occupational and Environmental Health (COEH) mandated by the State of California to conduct research, teaching, and service to communities in California on occupational and environmental health issues. Hence, my primary research interests are health effects from occupational and environmental exposures with a focus on pesticides and air pollution and chronic diseases including cancers, reproductive outcomes, neurodevelopmental disorders and neurodegenerative diseases. I served for more than a decade as the co-director of the NIEHS-

funded UCLA Center for Gene-Environment Studies in Parkinson's disease (PD) and am currently the Director of the American Parkinson's Disease Association Center for Excellence in PD Research. In the past two decades, I was the principal investigator of numerous Parkinson's disease, pesticides and gene-environment epidemiology studies in California and also conducted research based on large databases (such as cancer registries) assembled in California and Denmark. As part of my research, I developed geographic information system (GIS) based exposure assessment tools to assess chronic health effects of long-term pesticide exposures and of air pollution in California. In the early 2000s, I served as a member of the external advisory committee for the NCI/NIEHS Agricultural Health Cohort Study and for one year chaired this committee. I also was a visiting scientist at IARC/Lyon in 2006-07. In 2007, I received the Robert M. Zweig M.D. Memorial Award (Clean Air Award) from the California South Coast Air Quality Management District and in 2008 I was awarded the "Excellence in Research" award from the American Parkinson's Disease Association. I served on multiple National Academy of Sciences/Institute of Medicine (NAS/IOM) committees evaluating Gulf War Illness – including IOM reviews of cancer and of amyotrophic lateral sclerosis (ALS). Recently, I served on the NAS/IOM committee on "Incorporating 21st Century Science into Risk-Based Evaluations" and I just newly began serving on the committee to assess "Health Effects in Vietnam Veterans from Agent Orange (herbicides)". I am a CA Governor appointed member of the scientific review board for the California Air Resources Board (CARB) panel on Air Toxics. I served on the editorial Board of the Journal *Epidemiology* as well as other journals (currently I am editing a section of the journal *Current Environmental Health Reports*) and I am the newly elected President Elect of the International Society for Environmental Epidemiology (ISEE). My Curriculum Vitae is attached as Exhibit A. A list of the materials I have reviewed, in addition to those set forth in my CV, are attached as Exhibit B. Exhibit C contains my billing rate and prior testimony.

2. Methodology

2.0 Definitions of statistical and methodological terms.

(Population-based) Case-control study. A case-control study is a study where the subjects are selected for inclusion based on their disease status. In other words, study subjects referred to as

cases are enrolled because they have the disease (in this case, NHL) and controls are subjects who at the time the cases are diagnosed are not afflicted by the disease of interest; additionally, a study is considered population-based if the controls are selected without bias from the same population from which all cases arose. After study enrollment, everyone is either asked to report their past exposures (in this case, glyphosate or Roundup) or – if possible – exposures are reconstructed from a record system (e.g. sales records or application records) or by experts who evaluate job tasks and titles among all study participants (generally referred to as a job exposure matrix).

Cohort study. In a cohort study, subjects are enrolled in the study based on their exposures (in this case, to glyphosate or Roundup), and followed over time to determine who develops the disease(s) of interest. At enrollment, all participants are asked to report their past exposures or exposure is reconstructed from records, basically similar as in the case-control study, except that at enrollment no study participant is allowed to suffer from the disease of interest yet i.e. at the time of exposure assessment. In some cohorts, exposure is only assessed at enrollment (baseline) while in others exposures continue to be assessed throughout follow-up until disease occurs.

Odds Ratio (OR). An odds ratio, or OR, is a measure of association between an exposure and a disease. It represents the odds that the disease will occur in a group of people given a particular exposure, in comparison to the odds of the disease in a group of people without the exposure. An OR of 1.00 is the null, meaning no effect. Thus, an OR of 1.40 as reported in one of the studies below, for example, represents a 40% increase in NHL from exposure to glyphosate. An OR of 3.10 in one of the studies below represents a 210% increase in the odds of NHL from exposure to glyphosate. An odds ratio is a "point estimate" or the 'central' estimate of the relationship between exposure and disease, in a given study (note: the OR is in the center of the upper and lower confidence limit boundaries, see below). Odds Ratios are the statistics that are used most often to analyze case-control studies, and they are often calculated using a statistical technique called logistic regression but can also be derived by simple calculations based on a 2x2 table of data.

Rate Ratio (RR). A rate ratio is the measure of association between exposure and disease that can be calculated from cohort study data. It compares the incidence rates of disease given an exposure, to the incidence rate of disease among people without the exposure. The incidence rate allows us to take time into account and may depend on how much time has passed from the start of the study until the point in time when disease is diagnosed (or until the end of the study), thus it not only uses information based on persons but based on person times time under observation (also known as 'persontime'). Therefore, a RR different from an OR inherently relies on measures that included time under observation (i.e. rates). However, the results are interpreted in the same way: a RR of 1.00 is the null (no effect); a RR of 1.40 is a 40% increase in the rate of disease, etc.

Risk Ratio (or Relative Risk) is a ratio of the risk in the exposed divided by the risk in the unexposed in a cohort - where risks are defined as the number of (un)exposed cases divided by the total number of (un)exposed. Thus, different from rate ratios, this measure uses the number of subjects rather than the number of person-years a subject contributes during follow-up as the denominator. This method is used for well-defined (similar length) follow-up periods in the exposed and the unexposed such that the time under observation will not contribute additional information and we can substitute persons for person-time.
NOTE: under certain circumstances often met especially for rare diseases, the odds ratio (OR), risk ratio (RR) and rate ratio (RR) are the same (albeit calculated as the ratio of odds, risks, or rates) and the interpretation of the estimates is also the same.

P-value. The p-value is the probability of obtaining an estimate at least as far from a pre-specified value (in case of the null hypothesis the 'null' value) as the estimate we have obtained, if the specified value were the true value (note: no p-value, for the null hypothesis or any other hypothesis, is the probability that the specified hypothesis is true). For example, a p-value of 0.04 means that, given the null hypothesis is true, if you repeatedly conducted 100 tests of samples drawn from the same population (people), then in 4% of your tests, you would obtain the results you got solely due to random error (chance). It is a metric intended to show the likelihood of random error. It *should not* be interpreted as the probability that an agent causes an outcome.

Confidence interval (CI). A confidence interval, or CI, is given around an OR or a RR to give the likely interval which potentially includes the unobservable true measure of effect. In other words, it is an interval estimate (as compared to a point estimate) of the true underlying relationship between exposure and disease, in a given study. In practice, most published estimates are 95% confidence intervals, which means that in 95 out of 100 times when sampling your study subjects, you will find the true result (effect estimate) within the given confidence interval.

Hierarchical regression is a type of statistical analysis that was used in the 2003 De Roos study.[1] It is used when there are many correlated exposures and as a means to adjust for multiple comparisons. In De Roos, there were many different pesticides used by farmers and pesticide applicators, and therefore use of one pesticide can be strongly correlated with the use of another pesticide. For example, imagine glyphosate is often used together with another pesticide, dicamba. If the Odds Ratio that is reported between glyphosate and cancer is 2.0, then dicamba –assuming it is mostly used together with glyphosate – would be a proxy for glyphosate exposure and its OR would also be close to 2.0, just because these pesticides tend to be used together even if dicamba is not a carcinogen. However, if both pesticides truly increase risk (both are carcinogens) and we put them into the same (regression) model, we would not be able to estimate their effects properly, since they would now both have an attenuated effect estimate (this is also referred to as correlated variables 'stealing variance from each other'). De Roos used hierarchical regression to tease apart such correlations in order to determine which pesticides are the ones that are driving increases in NHL and narrow down the long list of pesticides to find the "bad actors" which were increasing risk of NHL. But, this approach makes a number of assumptions, for example that either all pesticides considered or pesticides within certain groups have similar effects on the outcome which might be incorrect.

N (number). The number of people in a study.

Statistical power is the ability of a study to estimate an effect. In essence, it is a reflection of the sample size (number of subjects in a study – in cohorts also the number of cases), the prevalence

of exposure, and the expected effect size. Large sample sizes give us generally higher statistical power, which means they have narrower and more stable confidence intervals around point estimates. Smaller sample sizes have wider confidence intervals. Thus, larger studies are much more able to find statistically significant results especially when exposures or outcomes are rare and the expected size of the effect moderate or small in size.

Data pooling or pooled analysis. To pool data is to use the raw (un-analyzed or non-summarized) data from several studies and merge them together to conduct analyses. Data pooling is often done when there have been multiple small studies on a topic, because the pooling allows for larger sample sizes and a uniform approach to the analysis of the pooled data. In order to conduct data pooling, scientists need to have permission to access the data from the investigators of multiple studies. Pooled studies have greater statistical power than the original studies from which they draw.

Meta-analysis. In some instances, scientists are interested in pooling data but do not easily have access to the raw data from each study. This is, typically, because the studies were conducted many years earlier, or perhaps because the investigators do not know/trust each other or human subject restrictions do not allow for the sharing of raw data; it is quicker and more efficient to conduct a meta-analysis based on summary estimates from published reports. A meta-analysis uses the Odds Ratios or Rate Ratios and confidence intervals which were published in the original studies, and comes up with a summary estimate of the relationship between exposure and disease. Similar to pooled analyses, meta-analyses also have much greater statistical power than each study does on its own, but the authors do not have the option of re-analyzing the original data as could be done if raw data were available (such as lagging exposures or generating different exposure categories etc.).

Null hypothesis means no effect. In the studies described below, their null hypothesis was that NHL is not related to glyphosate/Roundup exposure. The statistical tests done in the studies described below aim to test the null hypothesis: they want to determine if there the null hypothesis can be rejected with adequate statistical certainty and whether they can determine

whether there any relationship between exposure to glyphosate/Roundup and the development of NHL is suggested by a study.

A Forest Plot is a visual representation of the main results of all studies on a topic. The purpose of grouping them all together visually is that it can give the reader a sense of overall size of the effect estimates and the direction of the associations in the existing literature. See pg. 14.

Dose-response. A dose-response association represents an increasing risk with an increasing dose, such as a larger number of days per year, or a longer number of years, being related to higher Odds Ratios. For example, the overall study Odds Ratio might be 1.40, but for people who used glyphosate more often, the Odds Ratio was 2.5 while for those using it less often it might have been 1.5. This is a sign of a dose-response effect.

Incident/incidence refers to newly diagnosed cases; while prevalent cases are any existing cases at any point in time or over a certain period in time.

Confounding is a bias that occurs because a risk factor for the outcome is also a cause or precursor of the exposure of interest such that the outcome is caused by this confounder and not by the exposure one is trying to assess. For example, if sex is a risk factor for NHL and sex is also associated with occupational exposure to pesticides, we would want to adjust all effect estimates for pesticides by sex to remove potential confounding bias.

Recall bias is one type of exposure misclassification that is considered 'differential' by epidemiologists. This means that cases and controls remember or report past exposures differently because they have or do not have the disease. Generally, it has been suggested that cases may put more effort into recalling exposures since they have a need to explain their disease or are more motivated to do so to help researchers while controls are less motivated to recall past exposures. However, this is most likely a problem if the diseased subject knows or suspects an agent to cause their disease. If the subject has no way to know which pesticide might have caused a cancer for example and is asked to report all chemicals they have ever used occupationally, it is unlikely that they would only recall one and not another chemical

differentially. Thus, if recall bias existed, we would expect all pesticides they reported to the researchers to show an association with the outcome and not just one amongst many, since the tendency to recall better or more exposures than controls would not be expected to be specific to one chemical. In fact, when recall has been compared with record based evaluations, differential recall that causes recall bias has generally not been shown to be a problem. *Note:* non-differential recall error such that both cases and controls misreport their exposures is known to cause mainly bias towards the null i.e. masking any true effect rather that enhancing them. These recall biases are one type of information bias (see below).

Other biases include information bias which is characterized as mismeasurement of exposures or outcomes which can severely distort results in both case-control and cohort studies. As long as mismeasurement is non-differential (see above) i.e. the same for cases and controls or for exposed and unexposed, such biases most often cause underestimation of true effect sizes i.e. bias results towards the null that can be severe. Finally, there is selection-bias if controls are not representative of the exposures in the population that gave rise to the cases in case-control studies, or when there is a large and differential (with regard to case status) loss to follow-up in cohort studies.

2.1 Literature search

To obtain all published studies on the relationship between non-Hodgkin's Lymphoma (NHL) and glyphosate (the active ingredient in Roundup), I undertook a literature search using the same method to search for articles that I normally use in my research. This is the same method that I teach my UCLA students to use. As such, I relied upon two search engines, PubMed (https://www.ncbi.nlm.nih.gov/pubmed) and Google Scholar (https://scholar.google.com/). PubMed is an excellent resource for finding papers on the exact topic one is interested in, but it does not do as well in finding papers which were largely about a different topic but may have also briefly reported on the topic of interest. Google Scholar does well in capturing every possible paper of interest, but will often provide many articles not relevant to the subject matter at hand. I use both search engines to be as thorough as possible, but also to identify the most relevant articles. These searches initially yielded 290 articles in PubMed and 9000+ articles in Google Scholar for epidemiological studies; and over 550 articles for

animal and mechanistic literature; and over 600 citations for cancer. [Most citations were not immediately relevant to the present question, due to their focus on topics such as effects in fish resulting from runoff; effects on pregnancy and child development; or effects on other cancer types.]

As is typical in most published meta-analyses and reviews, I took additional steps to ensure I did not miss any relevant articles by also reviewing other published papers to check their citations. For these, I relied on the IARC Glyphosate Monograph as well as the two meta-analyses on glyphosate and NHL, as well as other articles on the topic that were published more recently.[2-4]

Furthermore, I read the US EPA's Cancer Assessment Review Committee (CARC) report, however I disagreed with their results because they relied heavily on statistical significance in studies that were not sufficiently statistically powered to answer the question (more on this below).

2.2 Reliance on peer-reviewed literature

As I teach my students, the most relevant articles, and indeed the only articles I nearly ever review and cite in my own research, are those that have gone through peer review at a reputable journal. Each field has its own journals considered reputable; but in general, a reputable journal is a journal that is listed in the most well-known and respected indexing sources such as PubMed.[i] Typically, these journals have been published for many years and many are backed by well-recognized and respected medical or research non-profit organizations, such as the American Medical Association, the British Medical Association, the American Association for Cancer Research (AACR), the Union for International Cancer Control (UICC), or the American Cancer Society.

Peer review, as defined by Danzik, is "a system by which manuscripts submitted for publication are evaluated, using outside referees (peers), who comment on the manuscripts' merit, originality, significance, and appropriateness to the journal. The intent is to identify flaws

[i] PubMed is a service of the US National Institutes of Health (NIH). On their website (https://www.nlm.nih.gov/pubs/factsheets/j_sel_faq.html) they explain that NIH uses a committee, the Literature Selection Technical Review Committee, to review and recommend which biomedical and health- related life science journals are included. Criteria include relevant subject matter as well as journals that meet PubMed Central's scientific quality standard, described as "scientific and editorial character and quality of a journal."

in design and analysis or interpretation, to suggest improvements, to direct manuscripts to the most appropriate outlets, to discourage repetition in publishing, and to weed out poor science or scholarship."[5]

Independent peer review is the cornerstone of science in the United States and internationally, and has formed the basis for what is considered acceptable and reliable medical and scientific research. The peer review process, which is almost always done anonymously (the reviewer is nearly always anonymous, although the authors are usually not) provides the intellectual rigor required to ensure that manuscripts adhere to what is acceptable in the field with regards to reviewing the relevant literature, and examining the statistics, and determining whether research protocols apply widely accepted methods, report valid results and avoid or account for biases, and draw conclusions appropriate to the study's findings. Peer reviewers are responsible for deciding whether an article is acceptable for publication. Because of this, authors typically will first, only submit their best work; and secondly, authors have to respond to reviewer critiques and be willing to make changes as requested or argue against suggested changes if there is a compelling reason to not do so which must be explained and justified to and accepted by the journal editors. I have personally peer reviewed on hundreds of occasions and for more than 20 different journals. I have also served on the editorial boards of three journals: Epidemiology, Epidemiologic Perspectives and Innovations, and Environmental Health.

The system of peer review has been in practice for decades. Although it is not without imperfections, the revisions that are suggested improve the quality of published manuscripts, it heads off potential fraud, and its existence encourages honest and state-of-the-sciences work.[5]

It is usual that peer reviewers will assemble comments for the editors who will communicate these and the editor's own comments to the authors as requests for clarification and additional information with the intention to not only improve the manuscript but most importantly to allow them to assess research validity. When any validity issues spotted during the review process cannot be addressed sufficiently by the authors in their responses and/or a revised manuscript, the editor may decide that the manuscript is not ready for publication.

2.3 Conflicts of interest.

There have been several systematic reviews published on the role of conflicts of interest in medical research. In 2003, a review of 1140 original studies reported a strong relationship

between industry sponsorship and pro-industry conclusions, with industry-sponsored studies more than 3 times as likely to find conclusions sympathetic to industry [pooled Odds Ratio (OR): 3.60, 95% Confidence Interval (CI), 2.63-4.91].[6]

Similarly, a 2016 article in the British Medical Journal (BMJ), which analyzed the results of 190 clinical trials published in 2013, reported that the presence of a financial tie between study investigators and industry resulted in a threefold increase in a positive study result (OR=3.23, 95% CI 1.7-6.1).[7]

As these reviews show, and as is widely recognized across the medical and research communities, industry sponsorship and financial incentives are unequivocally related to study findings. For this reason, journals have increasingly required that investigators report conflicts of interest when they submit articles, and these conflicts are published for the reader to see and to take into account when drawing conclusions as to the verity of the findings or the interpretation of the presented data. This information is also made available to journal reviewers, because it may influence the choice to recommend a manuscript for publication i.e. it may contribute to assessing scientific validity of the reported research. Furthermore, this is what I as a professor teach my students, and UCLA teaches to students in bioethics courses and lectures.

I performed an analysis of the data contained in the literature review of Williams, et al. (2016) and provide my opinions on that and other data throughout this report. There is a clear conflict of interest with several of the authors, and my review of the Dr. William Heydens and Dr. John Acquavella transcripts shows that some of the authors failed to properly disclose these conflicts. Therefore, I put less weight on this group's conclusions since it suggests they lack an ability to be impartial.

2.4 Statistical significance.

If we start off a study assuming that there is no association between glyphosate/Roundup and NHL (the "null hypothesis"), then, after we do our statistical analysis, we can determine the p-value for the null hypothesis of our findings, which is the probability of obtaining an estimate at least as far from a pre-specified value (the null value in case of the null hypothesis) as the estimate we have obtained, if that specified value were the true value (note: no p-value, for the null hypothesis or any other, is the probability that the specified hypothesis is true). There is a convention to consider a $p<0.05$ as "statistically significant" however, this is simply a

convention which is sometimes replaced by other p-values such as $p<0.01$ or $p<10^{-7}$ (in genomic studies). What a p-value of 0.04 actually means is that, given the null hypothesis is true, if you repeatedly conducted 100 tests of samples drawn from the same population (people), then in 4% of your tests, you would obtain the results solely due to random error (chance). It is a metric intended to show the likelihood of random error. It *should not* be interpreted as the probability that glyphosate/Roundup causes NHL. Moreover, if $p>0.05$, this doesn't "prove" the null hypothesis; absence of proof is not proof of absence.

Similarly, when a (95%) confidence interval excludes 1.0 (such as OR=2.0, 95% CI=1.2-2.8) – because 1.0 (the null value) is outside of the confidence interval-- it would be considered "statistically significant". As with p-values, confidence intervals can be defined as 95% intervals or 90% or 80% etc. intervals. However, confidence intervals provide additional information that p-values do not provide, and this information is related to the precision of the estimates or what is also called the informativeness of the data. In practice, p-values and confidence intervals close to the null (for example, if one side of the confidence interval is between 0.9 to 1.1) are considered marginal in terms of significance. Importantly, however, the estimates least influenced by chance are not those with low p-values, but those with narrow confidence intervals.

Statistical significance testing has been widely used and often misused in the medical literature, and is use has thus been widely criticized. One journal now bans the use of all statistical tests and even confidence intervals.[8] In the last decade, there has been considerable debate on the merits and problems of significance testing,[9-29] and in many Schools of Medicine and Public Health such as UCLA, students have been taught for decades to not rely upon statistical significance to draw their conclusions in accordance with the writings of the faculty member Dr. Sander Greenland, an author of the most widely used textbook in Epidemiology Methods entitled "Modern Epidemiology."[30] At UCLA, we teach students to focus on the point estimate (e.g. the Odds Ratio or Rate Ratio) as a measure of the size of the association between exposure and disease and the confidence interval to gage the precision of this estimate and the informativeness of the data/study.

Also important to consider is the rarity of the disease, because the rarer a disease, the harder it is for a scientist to create a large enough study with enough cancer cases enrolled to have adequate statistical power. Cancer is by its nature a rare disease. The annual incidence rate

(number of new cases) of NHL is 19.7 cases per 100,000 people. This is why it is so hard to study NHL with a cohort study 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 $p<0.05$ if we assume that the effect estimate size is moderate (less 2). This is the main reason why most cancer studies are employing a case-control design which is much more efficient in terms of the necessary sample size for sufficient statistical power and in terms of costs in general.

Many of the case-control studies cited below in this review, particularly those that tried to recruit cases in rural areas, had a limited sample size simply because there are a finite number of cases of NHL in rural areas (with low population density). For example, the Nebraska study (which contributed to De Roos' pooled analysis) included 220 cases;[31] the Kansas study[32] included 200 cases. These are not large numbers, and the result is that we get wide confidence intervals, particularly when exposures are also rare (as they were in these two studies, with 6% of cases and 3% of controls reporting ever use of glyphosate).

As recognized by the US National Cancer Institute, wide confidence intervals are often seen in epidemiologic studies of rare diseases like NHL, but scientists are nonetheless encouraged to move forward and publish their results anyway. This is because smaller studies can later be used in pooled or meta-analyses, and those will have much improved statistical power to estimate precise effect estimates.

In addition, as we teach at UCLA, one study alone is never definitive. It is important for a reviewer to look at the information in the literature as a whole to understand relationships between exposure and disease. We teach students to consider point estimates (Odds Ratios) as indicators of associations and effect sizes, and to not dismiss or mis-interpret studies that have wide confidence intervals that may or may not include the null.

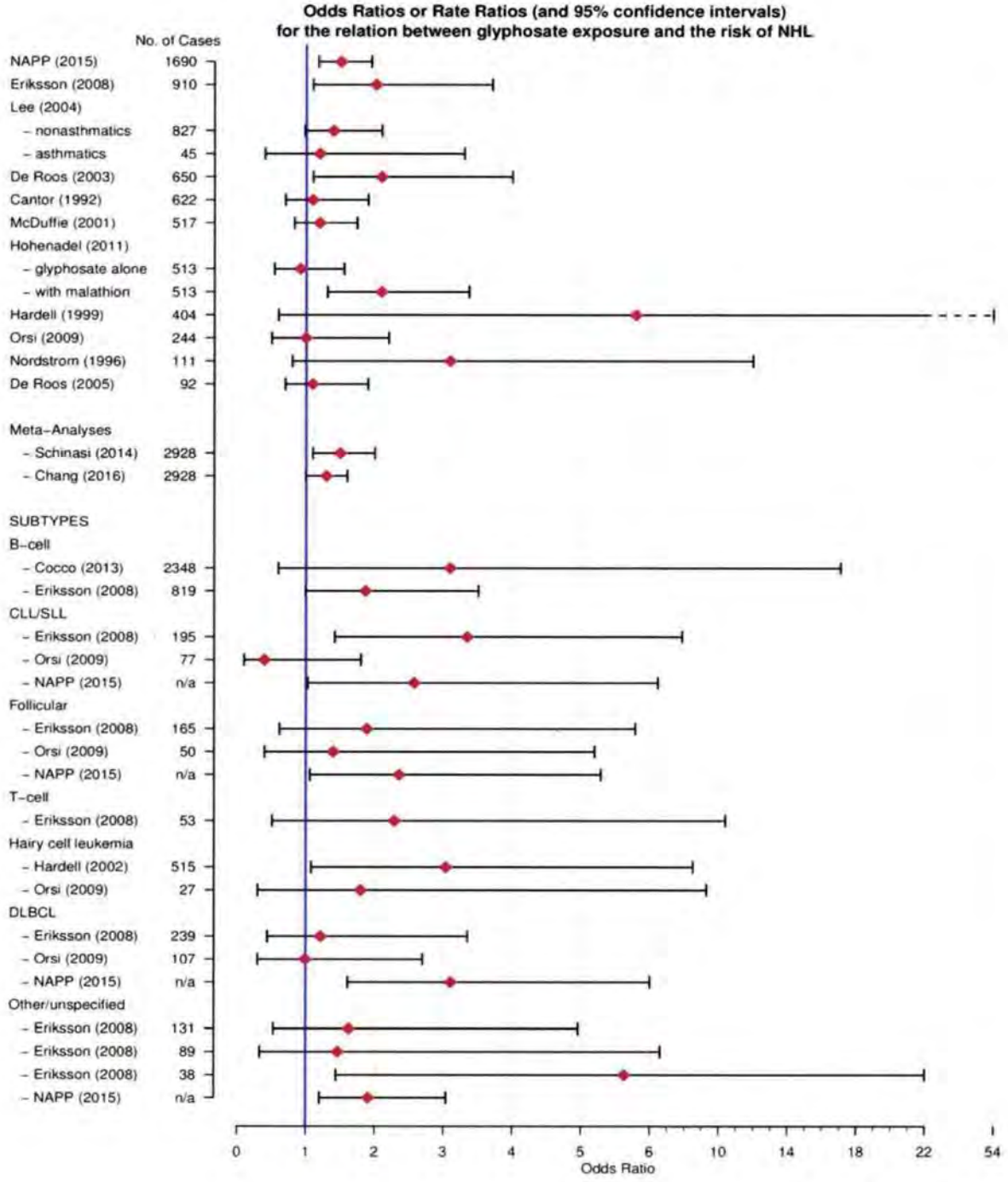
s2.5 Abstracts vs. full articles.

2.5 Abstracts vs. full articles.

Whenever possible it is preferable to examine and cite a full article over an abstract of the same study, because full articles have the space to provide a detailed overview of study methods and findings. If the full article is not yet published, however, it is common practice to cite abstracts.

3. Literature Review.

Here I summarize the findings of the epidemiologic studies on glyphosate and NHL in a forest plot, a graphical representation of all study results.



In reviewing the literature, the sample sizes and especially the number of cases should be noted, because of their bearing on 'statistical significance' and the width of confidence intervals. 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. Here I show the sample sizes of each human study of glyphosate and NHL.

First author, date	Number of cases in the study (all NHL cases combined)	Number of controls in the study
Cocco, 2013	1869	2462
Pahwa, 2015 (commonly known as the NAPP study)	1690	5131
Eriksson, 2008	910	1016
Lee, 2004	872	2336
De Roos 2003	650	1933
Cantor, 1992	622	1245
McDuffie, 2001	517	1506
Hardell, 2002	515	1141
Hohenadel, 2011	513	1506
Hardell, 1999	404	781
Orsi, 2009	244	426
Nordstrom, 1996	111	400
De Roos, 2005 (commonly known as the AHS study)	92	(54223)*

* these are the N of unaffected cohort members, however we calculate person time and generally do not use person N in analyses.

Because sample size is so relevant in considering exposure-disease associations, an informative study to consider is Pahwa's pooled analysis of the North American and Canadian studies, the North American Pooled Project (NAPP).[33] This abstract was presented at the International Society for Environmental Epidemiology's annual conference, and hence was peer-

reviewed, as are all abstracts presented at this meeting. In this analysis of 1690 cases and 5131 controls, NAPP reported an elevated risk of all NHL with any glyphosate use (OR=1.51, 95% CI 1.18-1.95) and a dose-response effect was seen with greater use (>2 days/year, OR=2.66, 1.61-4.40). An OR of 2.66 means that glyphosate exposure increases the risk of developing NHL by more than 160%. With regards to NHL subtypes, increases were observed for small lymphocytic lymphoma (SLL; 2.58, 95% CI 1.03-6.48, among those using for more than 5 years), and for follicular lymphoma (OR=2.36, 95% CI 1.06-5.29), diffuse large B-cell lymphoma (DLBCL; OR=3.11, 95% CI 1.61-6.00), and other subtypes (OR=2.99, 95% CI 1.10-8.09) for use more than 2 days per year. These study results were published in 2014, and as such were not included in any of the meta-analyses.

There were three meta-analyses conducted on glyphosate and NHL. The first, by Schinasi and colleagues,[34] included 2928 cases from 6 studies[1,2,35-38] and reported increases in NHL risk with any glyphosate exposure (meta-RR: 1.5, 95% CI 1.1–2.0), similar to the results of the NAPP study. Particularly stronger increases were reported for B-cell lymphoma (meta-RR = 2.0, 95% CI 1.1-3.6). Notably, heterogeneity of study results was low, which means that the results across studies were highly consistent. This is important because it suggests that the increases in NHL risk were unlikely to be the result of random fluctuations of estimates across populations: when you see the same results in multiple studies across different settings, it improves confidence in the findings.

The IARC Working Group's Monograph on glyphosate[4] noted that the above meta-analysis did not always use the most "highly adjusted estimates" from each study. The most highly adjusted estimates (also known as "fully adjusted" models) are the estimates that adjust for as many confounding variables as possible, such as adjusting for age, sex, race, and also sometimes other pesticide exposures. This is relevant because it gives the reader confidence that the findings are most likely due to glyphosate/Roundup exposure, instead of another potential cause that acts as a confounder. As such IARC's Working Group conducted their own meta-analysis using solely the most highly adjusted estimates from the same studies,[1,2,35-38] and reported a meta risk-ratio of 1.3 (95% CI, 1.03–1.65), with consistent findings across studies (low heterogeneity). I concur with the IARC conclusions after conducting my own independent analysis of the studies included in the IARC review.

Also helpful to consider is the Swedish study by Eriksson,[2] which was large (N=910 cases) and in addition, this study examined cases diagnosed 1999-2002 and thus allowed for a longer time period to have elapsed between exposure and disease development (glyphosate first came on the market in 1974); this is known as the latency period between exposure and disease occurrence. Although a short latency period does not completely exclude the possibility of exposure-disease relationships in cancer, a longer latency period increases confidence in results due to increased biological plausibility i.e. typically we would generally expect a 5-10 year minimum latency between exposure and disease onset for blood system related cancers. (However, in an individual case the latency period could be as short as 1 year, and as long as 50+ years.) Eriksson reported a twofold increase in NHL risk with glyphosate exposure (OR=2.02, 95% CI 1.10-3.71). Notably, there was also evidence of a dose-response effect: with >10 days use, the risk was higher (OR=2.36, 95% CI 1.04-5.37) compared to less than 10 days of use (OR=1.69, 95% CI 0.70-4.07). This was the only study reviewed which conducted analyses and also accounted for latency (>10 years after use, OR=2.26, 95% CI 1.16-4.40) and these results are more convincing due to biologic plausibility; in the group in which less than 10 years had elapsed since exposure, the effect estimate was much lower, as would be expected since these exposures are less likely to contribute to disease onset (OR=1.10, 0.24-5.08).

Eriksson also stratified by NHL subtype; effect estimates were increased for every NHL subtype and confidence intervals overlapped, meaning that there was evidence for increased risk for all NHL types: B-cell lymphomas (OR=1.87, 95% CI 0.998-3.51); SLL/CLL (OR=3.35, 95% CI 1.42-7.89); follicular (OR=1.89, 95% CI 0.62-5.79); Diffuse large B-cell (OR=1.22, 95% CI 0.44-3.35); other specified B-cell lymphomas (OR=1.63, 95% CI 0.53-4.96); unspecified B-cell (OR=1.47, 95% CI 0.33-6.61); T-cell lymphomas (OR=2.29, 95% CI 0.51-10.4); unspecified NHL (OR=5.63, 95% CI 1.44-22.0).

An earlier Swedish study by the same research group[39] ascertained cases diagnosed 1987-1990; thus this population was distinct from those in Eriksson's analysis. This study was smaller (N=404 cases) and had few participants ever exposed to glyphosate, leading to wide confidence intervals (4 cases and 3 controls ever exposed; OR=2.3, 95% CI 0.4–13). The small sample size limits our ability to draw definitive conclusions, but it is interesting that the estimate effect size is quite similar to the one reported by the larger later study. Likely because of this limitation, authors later conducted a pooled analysis which grouped these cases with cases of hairy-cell

leukemia (a subtype of NHL), reporting a threefold increased risk of any NHL (OR=3.04, 95% CI 1.08-8.52).[36] An earlier report of only the hairy-cell leukemia cases also reported increases in risk with glyphosate exposure (OR=3.1, 95% CI 0.8-1.2), but relied on a quite small sample size (N=121 cases).[40]

The Canadian studies (McDuffie[35] and Hohenadel[41]) ascertained cases diagnosed 1991-1994 hence allowing for a latency period between first possible use of glyphosate and disease occurrence, however the sample size (N=517 cases) was smaller than that of the pooled US studies. McDuffie reported a weak increased risk of NHL with glyphosate exposure which was similar in size in minimally adjusted and fully adjusted models (OR=1.26, 0.95-1.90; OR=1.20, 0.83-1.74). This study had a variety of sources for controls and a control participation rate of 48%, which is of concern if this caused selection of controls that does not reflect the population exposure to glyphosate. To examine the accuracy of self-reported pesticide use, McDuffie conducted a validation study comparing questionnaire data from farmers to records from a local chemical supplier on pesticide purchases. They stated that concordance between self-reported and sales record based exposures was excellent, although more specific information was not provided.

Pesticides sometimes exert stronger health effects when mixed (co-exposure) with other pesticides than when used alone. 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).[35] Similarly, when glyphosate exposure was mixed with malathion (OR=2.10, 95% CI 1.31-3.37) it was stronger than when farmers only reported using glyphosate alone (OR=0.92, 95% CI 0.54-1.55).[41]

The study by Cocco was limited in how much we can glean from its results, as only 4 cases and 2 controls had ever used glyphosate. The prevalence may have been low in this study because the Cocco study included people with a range of occupations, unlike many of the other studies which focused on agricultural populations. Cocco reported increases in B-cell lymphoma with glyphosate use (OR= 3.1, 9% CI 0.6 to 17.1).[42]

Less informative for the current evaluation is the Cantor study[43] because, although it was carefully conducted, cases (in Iowa and Minnesota) were included that were diagnosed 1980-1983. Hence, only 6-10 years could have elapsed between a potential first glyphosate exposure

and NHL diagnosis, which for cancer epidemiologic studies is considered an inadequate latency period (see above) and one would want to see an at least the median latency period of 10 years. Again, for an individual the latency period may vary (1 year to many decades), but on average for a study one would prefer a minimum latency period of on average 10 years.

The Lee study[44] utilized Cantor's cohort to build upon by including subjects from Nebraska who were diagnosed July 1983 to June 1986, thus this study includes cases with a longer latency period, which improves confidence in results. Lee reported increases in NHL among non-asthmatics (OR=1.4, 95% CI 0.98-2.1, N cases=827) and a smaller elevated effect estimate in asthmatics with wide confidence intervals (OR=1.2, 95% CI 0.4-3.3) due to the small number of asthmatic cases (N=45).

De Roos 2003 reanalyzed the US studies[1] and used hierarchical regression in addition to conventional logistic regression models, a statistical technique (described above) which can account for co-exposures and correlations between pesticides but makes some strong assumptions about all pesticides or groups of pesticides having similar effects on the outcomes. Using regular logistic regression, De Roos reported an increased risk with glyphosate use (OR =2.1, 95% CI 1.1 to 4.0) and in the hierarchical regression analysis the effect estimate was smaller 1.6 and the 95% CI included the null value of 1 (95% CI =0.9-2.8). Notably, the OR for glyphosate was among the highest of 47 pesticides tested, which suggests that glyphosate may indeed be the pesticide most strongly related to NHL in these farmers among all pesticides they used. The selection of pesticides for this paper was based upon a "carcinogenic probability factor" developed for all cancers, not specific to NHL, so it is not clear whether the hierarchical regression represented the best analytic strategy for NHL since – as stated above – the model assumes that all pesticides included have a similarly strong effect on the outcome; thus we would expect the largest effect estimate to be pulled towards the null of 1 which is what happened. Also, in terms of possible exposure mismeasurement, a validation of questionnaire responses had previously been conducted which reported strong agreement between self-reported pesticide use in comparison to pesticide supplier records, and recall was similar between cases and controls.[45]

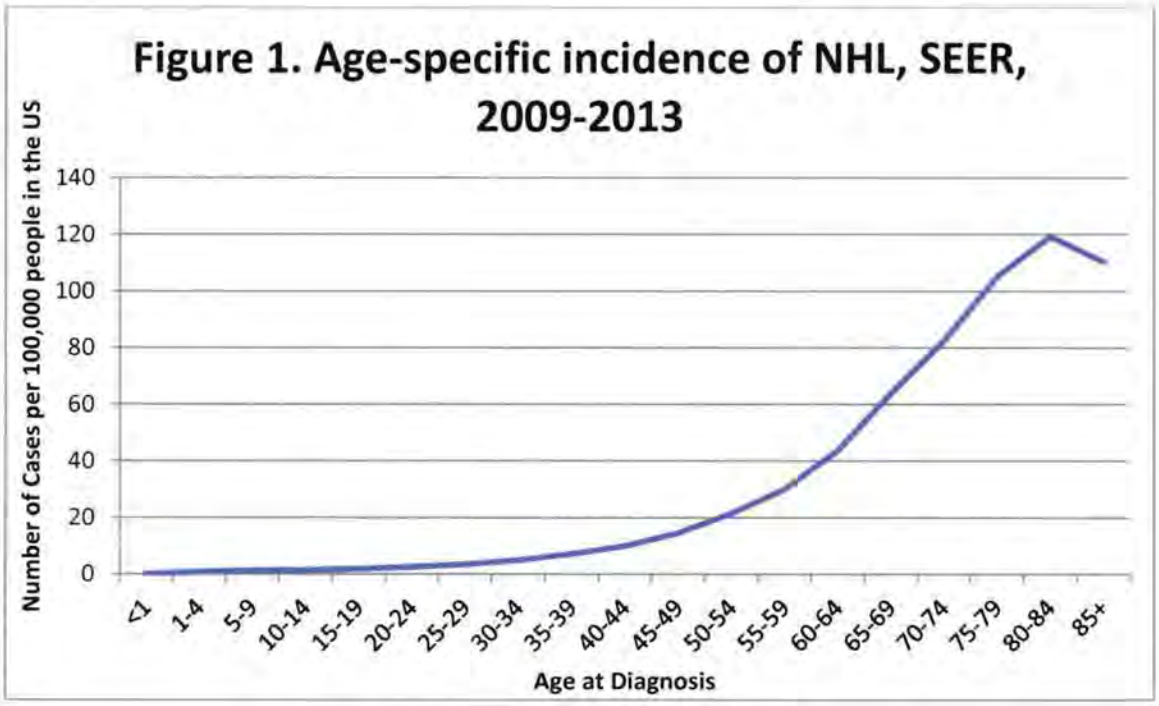
The French study by Orsi and colleagues[38] utilized a hospital-based study design, i.e. in this design cases and controls are recruited from among hospital patients. This is in contrast to nearly all of the other studies described above which used a population-based study design (with the exception of some countries within the Cocco study). Population-based studies are

considered superior to hospital based designs, because epidemiologic studies aim to select controls from the same population that gave rise to the cases, because it improves study validity. The patients who go to a hospital for NHL treatment may not live in the same area as the control patients selected; this can occur if the study hospitals are regional cancer centers which draw cases from a large geographic area. Orsi's study recruited controls who had been admitted largely from orthopedic and rheumatological admissions (mostly fractures, injuries and back pain). This may be problematic because orthopedic and musculoskeletal illnesses and injuries are conditions that typically do not require travel to a distant center for treatment, suggesting there was possible non-overlap between the case and control populations. In addition, hospital patients are an unusual group: they tend to be older, sicker, and have higher tobacco and alcohol use (and other behavioral/lifestyle differences) than the general population.[46-49] Consequently, the use of hospital controls can create unexpected and surprising findings (such as studies of cancer where the controls smoke more than the cases[48]). Further, biases can occur when the reasons for hospitalization are related to exposure. For example, if people exposed to glyphosate are more likely to be hospitalized (due to, perhaps, higher rates of time spent outdoors leading to greater injuries and back pain in farmers/gardeners) then this would bias the results. This may indeed be the case because there are known higher rates of musculoskeletal injuries among gardeners, and these people may also have higher glyphosate use.[50-52] Orsi and colleagues were unable to observe any association between glyphosate and NHL (OR=1.0, 95% CI 0.5 to 2.2; all NHL types combined). When authors examined risk by subtype, elevated risk with wide confidence intervals was reported for follicular lymphoma (OR=1.4, 95% CI 0.4-5.2) but not large diffuse large cell lymphoma (OR=1.0, 0.3-2.7). However, with 244 cases this study has only limited statistical power to conduct any subtype specific analyses.

De Roos 2005 is an analysis of the Agricultural Health Study (AHS).[37] Pesticide applicators were recruited for this study between 1993-1997 and followed for incidence of cancers up until December 2001, therefore active follow-up ranged from 4-8 years with a median follow-up period[ii] of 6.7 years, which is considered a short latency period in cancer epidemiology. Only 92 NHL cases had developed in the cohort by end of this follow-up period,

[ii] The follow-up period is the time that elapses between the start and the end of a study. Typically, participants are followed from the start date until 1) cancer diagnosis; 2) death; 3) study end; or 4) loss to follow-up (e.g. the study investigators cannot locate them or they drop out of the study), whichever comes first.

making this the smallest case sample size of any study reviewed; this is not surprising because the mean age at AHS study enrollment was 45.3 years.[53] NHL, like most other cancers, is a disease of aging, with dramatically higher incidence as people age. Figure 1 shows the incidence of NHL among Americans, with data taken from the US National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program.[54] It is not informative to follow a group of workers that young for only 4-8 years and draw meaningful conclusions about their cancer risk, especially for a rare cancer and an expected risk of moderate size (OR or RR of 1.5 to 2.5). The estimated RR was low and the confidence intervals were wide: the risk for any NHL was 1.2 (95% CI 0.7-1.9, adjusted for age; RR=1.1, 0.7-1.9, adjusted for age, demographic and lifestyle factors, and other pesticides).



AHS investigators collected information on 50 pesticides at enrollment (in 1993-1997); as the study description states, participants were asked about ever/never pesticide exposures and years of use and frequency of use (# of days per year) for 22 pesticides at enrollment and for another 28 pesticides in a take-home questionnaire that only 44% of applicators returned. The median time of employment involving mixing and applying any pesticide was 15 years at enrollment, and therefore the pesticide exposures occurring during the most relevant time period

for cancer development may not be known.[53] Among all pesticide applicators included in the analysis, 76% had ever used glyphosate, which made it among the most common pesticide used among applicators in this study. This is in line with other research on glyphosate, which reports that as of 1999, glyphosate was the highest selling crop-protection product on the market.[55] However, it is important to note that the first year genetically engineered, glyphosate-tolerant crops were planted commercially in the U.S. is 1996, and that prior to this date glyphosate accounted for just 3.8% of the total volume of herbicide active ingredients applied in agriculture[56] while glyphosate accounted for half of the total agricultural herbicide use in 2009 [see Coupe]. Also, in a 20-year timespan covered by EPA sales and usage reports (1987–2007), glyphosate use rose faster and more substantially than any other pesticide (in 2007, usage was in the range of 81.6–83.9 million kilograms, more than double the next most heavily sprayed pesticide (atrazine: ~33.1–35.4 million kilograms) making it the most heavily applied pesticide in the U.S. with 2/3 of the share of the total volume having been applied in just the last decade.[57-59]

Given the persistence of glyphosate in soil (with a half-life of 29-60 days[60,61]), the possibility of exposure to glyphosate due to drift from fields[iii] ,[62-64] and a possibility of contaminated water supplies,[65] it is plausible that passive exposure may have ultimately been much higher among agricultural communities and pesticide applicators than the 76% who reported ever use; more importantly, the baseline exposure assessment in the AHS only covered the first two years of very intensive use of glyphosate i.e. those who were enrolled in 1996/97. When exposure to an agent is extremely high—and potentially even ubiquitous as in a cohort of pesticide applicators, who spend their days in agricultural fields—it eventually becomes impossible to study its health effects since there are little or no exposure contrast to measure at

[iii] Studies of pesticide drift suggest the distance that pesticides travel depends upon several factors: first, the method of application, with air spraying by plane or helicopter (common due to its ease of use) leading to further drift than ground spraying, because the spraying occurs higher above crops; secondly, wind speed; and thirdly, pesticide droplet size, with smaller droplets travelling further. Estimates of pesticide drift vary from 74 meters in an area with low wind, up through >2400 meters under windy conditions. Studies of glyphosate pesticide drift suggest droplets can travel upwards of 800-1000 meters. According to the US EPA, spray drift has been reported to be a problem with glyphosate, as there have been multiple reports of damage from glyphosate to non-target crops.

least at the ever/never or cruder types of classification that do not rely on biomarker assays of dose.[iv],[66]

De Roos (2005) also conducted dose-response analyses by examining intensity-weighted exposure (years of use X days per year X intensity level), grouped into 3 levels (0.1-79.5; 79.6-337.1; and 337.2-18,241); and by cumulative exposure days (years of use X days per year), categorized into 3 groups (1-20, 21-56, 57-2,678). Authors decided to compare the cancer risk in these exposed groups not to that among the never exposed, but instead compared high exposure to low exposure. While this type of comparison attempts to control for and eliminate other risk factors that may distinguish non-exposed from exposed (hence reduce potential confounding bias) this type of approach also reduces any remaining exposure contrasts even further and thus reduces the ability to estimate risk increases with exposure and make the effect estimates also less comparable to those form other studies.

Industry-sponsored studies

A meta-analysis by Chang and Delzell was sponsored by Monsanto.[67] This meta-analysis found similar results to the above meta-analyses for any increases in NHL (meta-OR: 1.3, 95% CI 1.0-1.6) and particularly elevated risks for B-cell lymphoma (meta-OR: 2.0, 95% CI 1.1-3.6). This study also found extremely low heterogeneity across studies— unusual in most meta-analyses— supporting the consistency of findings across different settings.

Bradford-Hill criteria evaluation

The strength (effect size) criterion is partially met since the overall meta-analytical (point) effect estimates reported for ever never glyphosate use are between 1.3 and 1.5 reflecting a weak to moderate size association. However, the effect estimates for longer or more extensive use in several studies were larger i.e. between 2 and 3 and this can be considered a stronger endorsement of a causal relation; it is further supported by the observed dose response (biological gradient such that risk increases with dose - another Bradford Hill criterion) that these studies found (also note: a small association does not mean that there is not a causal effect,

[iv] Rose argues that when a risk factor is ubiquitous in a population, it may strongly influence the population incidence of a disease, but may not identify high-risk individuals within a population. For example, in a society where everyone smokes, smoking will not identify high-risk individuals for lung cancer.

though the larger the association, the more likely that it is unbiased and thus causal). In terms of consistency, this criterion is met since positive associations have been reported for different populations and in different places and different time periods which strengthens the likelihood of a true effect. Temporality i.e. that the cancer occurred after exposure and that there is an expected delay between the cause and effect has been shown i.e. all exposures were assessed and recorded for the periods prior to NHL occurrence. Unfortunately, only one study examined the influence of exposure lagging i.e. considered the latency period: that study found a strong association with a 10-year lag, which further corroborates causality in terms of cancer etiology. The specificity criterion (i.e. that one specific exposure causes one specific outcome) is hard to apply in the case of herbicide or pesticide exposure since almost none of the farmers/pesticide applicators is expected to solely be exposed to glyphosate, since most farming operations require the use of multiple pesticides over time. Also in the case of blood system cancers, one could argue that different pesticides have possible carcinogenic effects on different cell types. Nevertheless, it is of interest that NHL is one cancer reported consistently among farmers for the past 2 to 3 decades, and glyphosate is consistently the most widely used herbicide in farming especially after 1995 with the advent of genetically modified crops. Finally, some studies suggested that types of NHL that are showing T14/18 translocations in lymphocytes are the ones most likely caused by external agents including some pesticides and smoking and this increases also biologic plausibility for the action of genotoxic or oxidative stress pathways (see below) with certain pesticides such as glyphosate.

Biological plausibility.

Biomonitoring studies affirm that some (not all) persons who apply glyphosate occupationally have measurable glyphosate excreted in urine, and measurable glyphosate is also seen in farming household members who reside close to treated fields.[68-70] Research on exposed agricultural workers suggests increases in genomic instability (binucleated cells, micronuclei).[71] Rodent studies report increases in DNA oxidative damage (increases in 8-OHdG in either kidney or liver; lipid peroxidation) as well as cytogenetic damage (sister-chromatid exchanges, increases in micronuclei), and DNA single-strand breaks.[72-74] Cytotoxicity and genotoxicity are also reported in studies of human cells.[75]

Roundup vs. glyphosate. One study compared the effects in rodents of glyphosate to those of Roundup, and results were similar with regards to cytotoxic and genotoxic effects.[73] While a *plausible mechanism* between cause and effect is helpful, Bradford Hill noted that knowledge of the mechanism is often limited by current knowledge; nevertheless for glyphosate two mechanisms have recently been proposed, oxidative stress and genotoxicity, and been confirmed by the laboratory experiments listed above. Finally, while *coherence* between epidemiological and laboratory findings increases the likelihood of a true effect, Bradford Hill noted that "... lack of such [laboratory] evidence cannot nullify the epidemiological effect on associations". Due to ethical concerns, there will never be any human experimental evidence for glyphosate toxicity or carcinogenicity, but human cell based studies and animal experiments can substituted as model systems and have increasingly been used in the recent past.

4. Conclusions

The epidemiologic studies as a whole support an increased risk of NHL with exposure to glyphosate or glyphosate based formulations, including Roundup. Due to the rarity of this disease, many of the earlier studies were small in size, leading to wide confidence intervals; yet findings were consistent with nearly all studies having point estimates above 1.0. In the pooled and meta-analyses, results are consistent and unequivocal. Studies that assessed dose also generally found that higher levels of exposure were associated with increased risk and importantly in the one study that did assess the importance of having been exposed more than 10 years prior to a diagnosis of cancer, the results clearly pointed to those exposures as the relevant one as compared to the more recent exposures (within 10 years) increasing plausibility of associations greatly.

In my opinion, to a reasonable degree of scientific certainty, glyphosate causes NHL. Furthermore, to a reasonable degree of scientific certainty, glyphosate based formulations, including Roundup, cause NHL.

Beate Ritz, M.D., Ph.D.

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EXHIBIT A

CURRICULUM VITAE
April 2017

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UCLA School of Public Health
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EDUCATION

1995	Ph.D. in Epidemiology, School of Public Health, UCLA
1993	M.P.H. in Epidemiology, School of Public Health, UCLA
1987	Doctoral Degree in Medical Sociology, University of Hamburg.
1983	Medical Examination Certificate, Registration as a Physician (M.D.), Board of Health in Hamburg
1977-1983	Medical School, University of Hamburg, Germany

PROFESSIONAL POSITIONS AND APPOINTMENTS

2012- 2015	Chair, Department of Epidemiology, School of Public Health, University of California Los Angeles (UCLA)
2006-current	Professor, Departments of Epidemiology, Environmental Health, and Center for Occupational and Environmental Health, School of Public Health, and Neurology, School of Medicine, UCLA
2005-2012	Vice Chair, Department of Epidemiology, School of Public Health, University of California Los Angeles (UCLA)
2004-current	Appointment in the Department of Neurology, School of Medicine, UCLA
2002-current	Co-director of the UCLA-CGEP (UCLA center for Parkinson 's Disease Environmental Research (CCPDER- CNS)
2001 -2006	Associate Professor, Department of Epidemiology, Department of Environmental Health, and Center for Occupational and Environmental Health, School of Public Health, UCLA
1995-2001	Assistant Professor, Department of Epidemiology and Center for Occupational and Environmental Health, School of Public Health, UCLA
1993-1995	Assistant Researcher, Department of Epidemiology, School of Public Health, UCLA
1989-1991	Hochschulassistentin (Assistant Professor), Institute of Medical-Sociology, University of Hamburg, Germany.
1987-1988	Research Fellow and Resident, Psychiatric University-Hospital Eppendorf, Hamburg, Germany
1984-1986	Research Fellow, Institute of Medical Sociology, University Hospital Eppendorf, Hamburg, Germany

OTHER HONORARY PROFESSIONAL APPOINTMENTS

2002-2008	Editorial Board: EPIDEMIOLOGY
2004-2009	Editorial Board: Epidemiologic Perspectives & Innovations
2007-2010	Editorial Board: Environmental Health
2001-current	Chair (since 2005) and Member (since 2001) of the external advisory committee for the NCI/NIEHS Agricultural Health Cohort Study
2001-current	Board of Directors for the 'R. Lemelson Foundation for Psychocultural Research.' Annual awards of $800,000 for research and training including a UCLA training grant for cross-disciplinary studies in anthropology, psychology and neuroscience

2001-2002	Member of the external advisory committee for the California Biomonitoring Planning Project conducted by the Environmental Health Laboratory's Biomonitoring Project (CDHS)
2002	Member of the EPA Science Advisory Board for Human Health Research Strategy (HHRS)
2002-2004	Member of the external advisory committee for the California Environmental Health Surveillance System (Governor Davis appointee to expert working group for SB 702)
2003-2006	Member of the Ethic Committee for the International Society for Environmental Epidemiology
2003-2004	Member of NAS, IOM Committee on Gulf War and Health, Phase 3: Literature Review of Selected Environmental Particulates, Pollutants, and Synthetic Chemical Compounds
2002-2004	Member of the external advisory committee for the California Environmental Health Surveillance System (Governor Davis appointee to expert working group for SB 702)
2006	Member of NAS, IOM Committee on Gulf War and Amyotrophic Lateral Sclerosis
2006	Member of the Scientific Steering Committee for Pediatric BioBank in California
2007	Robert M. Zweig M.D. Memorial Award (Clean Air Award) from the California South Coast Air Quality Management District
2007	Appointed as a Collegium Ramazzini Fellow
2007	Scientific Organizing committee for the PPTOX conference in Faroe Island
2008	Scientific Organizing committee for the ISEE conference in Pasadena
2008	Member of the Environmental Exposures Working Group conducted by RTI International for the PhenX project of GWA research at NIH
2009	Member of NAS, IOM Committee on Gulf War and Health, Phase 4
2008-09	Member of the U.S. EPA CO standard setting panel for (CASAC: *Carbon Monoxide National Ambient Air Quality Standards*)
2009-2012	Elected Councilor for the International Society for Environmental Epidemiology (ISEE)
2010-current	Member of the Conference Organizing committee of the ISEE
2009	Award from the American Parkinson's Disease Association (APDA) for outstanding contributions to the medical and scientific communities towards the advancement of Parkinson's disease research
2010-2013	Member of the External Advisory Board for the Superfund site center grant at University of Washington
2010-2013	Member of the External Review Board for the Swiss Tropical and Public Health Institute in Basel
2013	Scientific Organizing committee for the ISEE conference in Basel/Switzerland
2012-current	Member of CA-EPA Scientific Review Panel on Toxic Air Contaminants
2012	Affiliate member of the Institute of the Environment and Sustainability
2014	Scientific Organizing committee for the ISEE conference in Seattle Washington
2014-current	Member of NAS/IOM committee on Incorporating 21st Century Science into Risk-Based Evaluations

FUNDED RESEARCH

NNH12ZDA006O-EVI3
Agency: NASA (PI: Ritz)
Total Direct Costs to UCLA: $1,294,244
Multi-Angle Imager for Aerosols (MAIA) 08/01/16-11/30/25
This project will assess air pollution and adverse birth outcomes using exposure data provided by Dr. Diner's group from the MAIA NASA project. UCLA researchers will be responsible for the modeling the effects of prenatal air pollution exposures on adverse birth outcomes derived from vital statistics records for multiple locations across the world.

1 U01 HD087221 (PI: Devaskar/UCLA Ob-GYN)
Agency: NIH/NICHD Period: 01/01/16-12/30/19
Total Direct Costs: $2,999,640
Imaging Innovations for Placental Assessment in Response to Environmental Pollution
The objective of this proposal is to develop and evaluate a suit of cutting-edge multi-parametric magnetic resonance imaging (mp-MRI) technologies and translate these novel placental imaging modalities to assessing the impact of environmental pollution exposure on prediction of placental insufficiency.

Psychosocial stressors, air pollution and childhood respiratory health in LAFANS
Agency: NIEHS R03ES025908 (PI: Ritz) Period: 07/01/15-06/30/17
Total Direct Costs $100,000
This study will add to the previous literature by constructing a more holistic measure of the stress perceived by the child, and use that measure to determine if a child's perceived stress modifies their risk of asthma or reduced lung function from air pollution.

Pesticide Exposures and Risk of Cerebral Palsy
Agency: NIEHS R03ES025904 (PI: Ritz) Period: 07/01/15-06/30/17
Total Direct Costs $100,000
Using records from the California Department of Developmental Services (DDS), we will identify children born 1995-2007 and diagnosed with CP in California until 2010. For ~10,000 CP cases we will randomly select 1:10 matched controls from the California birth certificates. Ambient pesticide exposure estimates pre-pregnancy, during pregnancy and/or first year of life for each child will be estimated using a Geographic Information System (GIS) model we previously developed based on the California Pesticide Use Reporting (PUR) system. We will examine specific vulnerable periods in pregnancy (trimesters or months of pregnancy) to assess pesticide exposure effects on CP.

Autism, Metabolomics, and Environment (AIME)
Agency: NIEHS R21ES25573 (PI: Ritz) Period: 07/01/15-06/30/17
Total Direct Costs $275,000
We will assess whether autism risk factors can be identified using metabolomic biomarkers of exposure in stored maternal serum samples from mid-pregnancy from 200 case and 200 control pregnancies in Central California and compare biomarker exposure patterns with modelled air pollution and pesticide exposures. Metabolomics analyses will be performed in a targeted as well as untargeted manner with high-resolution metabolomics that uses mass spectrometry and advanced data extraction algorithms to quantify up to 20,000 chemicals in small biologic extracts.

Air Pollution and Childhood Autism
Agency: NIEHS R21ES024006 (PI: Ritz/Ehrenstein – multiple PI) Period: 07/01/15-06/30/17
Total Direct Costs $275,000
We use highly sophisticated modeling and analytical techniques for the detailed spatial and temporal assessment of air pollution to examine their influence on neurodevelopment in a California birth cohort linked to autistic disorder records of the CA Department of Developmental Services.

Environment and cognitive decline in older Hispanics
Multi-PI: Ritz/Haan
Agency: NIEHS Type: R01- RES023451A Period: 04/01/15-03/31/19
Total Direct Costs: $ 2,000,000
The goal of the proposed research is to investigate whether long-term exposure to two ubiquitous environmental exposures, air pollution and pesticides, contribute to cognitive decline and dementia in elderly Mexican Americans (MA) from the "Sacramento Area Latino Study on Aging" (SALSA) cohort. We capitalize upon our expertise in modeling air pollution and pesticide exposure and plan to model 1) long and short term regional, local, and traffic related air pollution using monitored criteria pollutants, CALINE4 - emissions and land use regression (LUR) models; and 2) long-term exposures to pesticides of specific chemical classes with our GIS model; and 3) assess impairment in cognitive domains and the onset of dementia longitudinally based on multiple complex environmental exposure patterns while taking into account vulnerability due to genetic and physiologic risk factors for dementia.

Air Pollution and Autism in Denmark
PI: Ritz
Agency: NIEHS Type: R21 Period: 04/01/15-03/31/17
Total Direct Costs: $ 275,000
The goal of the proposed research is to utilize Danish nationwide population-based registers and sophisticated individual-level air pollution exposure measures to assess whether early life exposure to traffic-related and particulate air pollution during critical periods of fetal development are associated with autism risk. We will use the Danish National Birth Cohort (DNBC) which enrolled pregnant women and collected extensive prospective risk factor data during pregnancy and early life for ~100,000 children

among whom 720 are already diagnosed with ASD to examine potential confounding bias for a large number of risk factors assessed in pregnancy.

Air Pollution and Cardiovascular Diseases: Identification of Novel Biomarkers
Agency: NIEHS R21 ES024560 (PI: Zhu) Period: 05/01/15-04/30/17
Total Direct Costs $275,000
Objectives: The goal of this project is to identify novel and sensitive biomarkers of cardiovascular health effects, in association to air pollution exposures.
Role: Co-I

Environmental exposure, DNA methylation, and Parkinson's disease
Agency: NIEHS 21ES024356 (PI: Ritz/ Horvath) Period: 08/06/14 – 07/31/16
Total Direct Costs: $ 250,000
Environmental exposure, DNA methylation, and Parkinson's disease
Here we use a powerful new tool and systems biology analytic methods to identify signatures for toxic exposures that evoke long-term biologic responses. Using DNA methylation we will investigate specific epigenetic markers (CpGs) correlate with toxic exposures and the role these epigenetic changes play in PD progression using epigenome wide technologies combined with analytic tools to integrate these data. We will investigate epigenetic determinants of Parkinson's disease in over 800 subjects with existing biospecimens.
Role: PI

Maternal comorbidities, prescription drug use in pregnancy, and childhood cancer (COMPAC): a record linkage study in Denmark
PI: Heck
Agency: NIH/NCI Type: R21CA175959 Period: 04/01/14-03/31/16
Total Direct Costs: $ 275,000
This study aims to link several large-scale databases in Denmark to examine maternal health and medication use in pregnancy in relation to childhood cancers. We propose to examine common pregnancy conditions that have been linked to cancers in adults and children in other studies as well as common medications taken in pregnancy which are suspected carcinogens or linked to cancer in other studies.
Role: Co-I

Inflammatory Cytokine Polymorphisms, Air Pollution, and Very Preterm Birth
PI: von Ehrenstein
Agency: NIEHS Type: R21ES022734 Period: 07/01/13 - 06/30/15
Total Direct Costs: $ 275,000
We examine the hypotheses that maternal exposure to air pollutants during pregnancy is associated with an increased risk of very preterm birth (VPTB, <32 weeks gestation), and that polymorphisms in inflammatory genes modify the influence of air pollution on the risk of VPTB. We use data from the CA Very Preterm Birth (CVPTB) Study, a nested case-control study of VPTB from 5 counties in Southern CA known for high particulate matter, ozone, and traffic exposures that has genotyped SNPs related to PTB in 26 inflammatory/immune response pathway genes in mother-infant pairs and will utilize a combination of extensive air monitoring data and air pollution modeling approaches (land use regression (LUR), CALINE4, kriging) to estimate air pollution exposures in pregnancy for CVPTB Study subjects.
Role: CO-I

Pesticide Exposure and Childhood Autism
PI: von Ehrenstein
Agency: NIEHS Type: R21ES022389 Period: 01/01/14 - 12/31/15
Total Direct Costs: $ 275,000
We examine the hypothesis that exposure to specific pesticides during vulnerable periods, particularly during fetal development, determines risks of subsequent development of autistic disorder (AD). We developed a geographic pesticide exposure assessment tool (GRAPES) that utilizes the unique California Pesticide Use Report system, in combination with agricultural land-use maps, to derive record-based estimates of historical residential exposures, and expect to identify >20,000 autism cases with diagnoses up to the age of 72

months from the CA-DDS database born in CA 1997-2009 and >1,700 from agricultural areas as well as 1:10 age-sex match controls from birth records, the largest cohort ever to address hypotheses that exposures to specific chemicals (e.g. neurotoxic or endocrine disrupting agents) contribute to AD during vulnerable periods of development.
Role: CO-I

Parkinson's Susceptibility Genes and Pesticides (PEG-Renewal)
Principal Investigator: Ritz
Agency: NIEHS/NINDS Type:R01ES010544 03/01/11-11/30/15
Total Direct Costs: $ 2,500,000
In this renewal of an epidemiologic population-based case-control study we recruit 500 additional PD patients in three rural California counties and will assessed their exposures to pesticide exposures and the effects of gene-pesticide interactions.
Role: PI

Systems genetic and reverse phenotypic analysis of age and retirement.
PI: Horvath (UCLA)
Agency: NIA Type: R01AG042511-02 07/01/13 - 06/30/17
Total Direct Costs: $ 1,000,000
We will apply/develop state of the art computational, statistical, and bioinformatic approaches with which to investigate the association between genetic data and aging- related phenotypes. Specifically, the study uses data from the Health and Retirement Study (HRS) and a systems biology approach to identifying relevant SNPs and genetic pathways and machine learning techniques and reverse phenotyping methods to better understand the complex relationship between genetics and aging outcomes including cognition and wealth.
Role: CO-I

Exposure to C8-chemicals and autism, ADHD, and cerebral Palsy in the Danish Birth Cohort
PI: Jorn Olsen (UCLA and Aarhus University, Denmark)
Agency: Danish Medical Council
Total Direct Costs (at UCLA): $ 250,000 01/01/11 - 08/31/15
The overall goal of the project is to assess the impact of C8 persistent organic pollutants in maternal serum during pregnancy and childhood outcomes of autism, ADHD and cerebral palsy in the Danish Birth cohort using follow-up data from the National Danish medical registry systems.
Role: CO-I

A Cohort Study on Air Pollution and Breast Cancer in Los Angeles County
IIR13262718 Wu (co-PI) 02/13/14-02/150/17
Susan G Komen $217,728
The overall objective is to examine the role of air pollution and risk of breast cancer among whites and non-whites in Los Angeles using the large Multiethnic Cohort Study
Role: Co-Principal Investigator

Improvements in Air Quality and Health Outcomes among California Medicaid Enrollees Due to Goods Movement Actions — Phase I: Assessing Air Quality Changes
PI: Meng , UCLA
Agency: Health Effects Institute (HEI) #: 4914-RFA11-1/2-6 09/01/12 – 08/31/15
This phase of the project will evaluate the effect of goods movement emission reduction actions on ambient air quality in goods movement corridors, non-goods movement corridors, and areas outside of these two corridors in 10 major California counties between the 2003-2007 pre-policy and 2008-2012 post-policy years.

COMPLETED RESEARCH
Assessing and Reducing Taxi Drivers' Exposure to Ultrafine Particles
PI: Yifang Zhu (UCLA) Type: R21OH10196 09/01/12–08/31/14
Agency: CDC/NIOSH
Total Direct Costs: $ 275,000

Goal: The major goals of this project are to develop ultrafine particle exposure assessment instrument and explore novel low-cost ultrafine particle exposure mitigation strategies for taxi drivers.
Role: Co-I

Air Pollution and PD in Denmark
PI: Ritz Type: R21-ES022391 12/01/12-30/11/14
Agency: NIEHS
Total Direct Costs: $ 275,000
This study will use a sophisticated and validated GIS-based dispersion model, AirGIS, to assess exposure to traffic-related air pollution in PASIDA participants; i.e. NO_2/NO_x. Specific aims are to: (1) assess the influence of long-term traffic-related air pollution exposure on PD risk for 1,867 cases and 1,920 population controls combining existing PASIDA data with new exposure measures from AirGIS; and (2) investigate the combined action of air pollution and genetic variants in inflammatory genes previously linked to PD.
Role: PI

Parental Occupation and Childhood Cancers in Denmark
PI: Heck (UCLA) TYPE: R03 ES021643 4/15/12-3/31/14
Agency: NIEHS
Total Direct Costs: $ 50,000
The specific aims of this study are: 1) Create a linked database of all childhood cancers in Denmark diagnosed 1965-2010 with recorded information on parental employment. 2) Examine the relation between parental employment and childhood cancers focusing on maternal occupational exposures. 3) Examine specific hypotheses in childhood cancer risk (occupational social contact; contact with animals; organic dust; welding fumes; bitumen fumes; outdoor work; and several associations seen in previous literature (solvents, paints and pigments, motor vehicle exhaust related occupations)).
Role: Co-I

Pesticides and Childhood Cancers
Principal Investigator: Ritz (UCLA)
NIEHS R21- ES019986 4/1/11 – 12/31/13
Total Direct Costs: $ 275,000
The specific aims of this study are to examine associations between prenatal exposure to pesticides and specific childhood cancers in California between 1980-2009 using ambient measurement data using our GIS model of pesticide exposures based on land use maps and pesticide use report (PUR) data.

UCLA Center for Centers for Neurodegeneration Science (CNS; former CGEP)
Director: Chesselet, UCLA; Co-director: Ritz
NIEHS P01ES016732 09/15/08-08/31/13
Total Direct Costs: $5,000,000
We have previously shown associations between high levels of exposure to specific environmental pesticides and Parkinson's disease and will build on this knowledge to determine the mechanisms of action that may be causing this association. We will use an integrated, multidisciplinary approach to identify additional agricultural pesticides that are disrupting similar molecular pathways, and determine whether these also increase the risk of Parkinson's. This work is expected to shed light on the pathological processes involved in sporadic Parkinson's disease, the most frequent form of the disorder, and could have public health implications for precautions in the use of some pesticides.
Project 4: Pesticides and Genes in PD: Studies in Humans
Principal Investigator: Ritz
NIEHS 09/15/08-08/31/13
Total Direct Costs: $1,250,000
This project will use the existing PEG data to test biological candidate genes and newly identified putative environmental toxicants for association with PD. We will recruit and collect biological (DNA) samples from and construct exposures estimates for 400 additional population controls. This will enable us to test new hypotheses for rarer exposures to specific toxins and will allow us to investigate gene-gene (GxG) and gene-environment (GxE) interactions with sufficient power. Targeted toxins are either (a) interfering with the ubiquitin proteasomal system (UPS), (b) altering microtubule integrity, and/or (c) inhibiting the aldehyde/alcohol dehydrogenase. Targeted genes include UBE1 and UBE1L2; PSMC2, 3, 4, and 5; HIP2; SKP1A; GSK3B; CDK5; MAPT, Sirt2, and ALDH and ADH gene clusters.

Registry of Parkinson's Disease Study In Denmark (PASIDA)
Principal Investigator: Ritz
NIEHS RO1 - ES013717 09/01/06-08/31/13
Total Direct Costs: $5,600,000
We conduct 1) a case-control study of ~13,000 PD cases and age-gender matched controls from the Danish population via passive record linkage by unique ID between the National Patient Register, Pharmacy Database, and National Pension fund to identify risk factor information contained in these records (e.g. occupations, medication use, diseases prior to PD onset); and 2) recruit actively ~2500 of the most recently registered PD patients and population controls to collect additional risk factor information per interview and biological materials for gene-environment interaction analyses and to characterize PD patients phenotypically.

Air Pollution and Childhood Cancers
Principal Investigator: Heck (UCLA)
NIEHS R21- ES018960 4/1/10 – 12/31/13
Total Direct Costs: $250,000
The specific aims of this study are to examine associations between prenatal exposure to motor vehicle related air pollution toxics and specific childhood cancers in Los Angeles County and all of California between 1980-2009 using ambient measurement data, land use based regression (LUR) and CALINE4 models.

California Parkinson's Disease Registry Pilot Feasibility Study
Principal Investigator: Ritz
DOD 09/01/07-04/30/12
Total Direct Costs: $390,000
The primary goal is to conduct a pilot study for the legally mandated statewide population-based PD registry. We will identify PD cases in Kern, Tulare and Fresno counties from legally mandated sources (pharmacists, health care institutions, physicians and other providers). A secure prototype database will be established, and associations between PD and toxicant chemical exposure will be determined by linking to a database of toxicant chemicals established previously by UCLA based on California state data (e.g. the pesticide use databases).

UCLA UDALL Parkinson's Disease center
Principal Investigator: Chesselet, UCLA
NINDS Type: P50 NS38367 04/01/06-03/31/12
Total Direct Costs: $7,500,000
Project 6 within the center (budget of $ 500,000 annual direct costs): Progression and Health Impacts of PD Motor and Non-Motor Manifestations (C-PI Ritz)
Research goals are to assess whether development and progression of PD motor and non-motor manifestations in 300 PD patients ascertained in the PEG study (PI: Ritz see below) are influenced by environmental, behavioral, and social factors and by genetic variants of ApoE and serotonin transporter alleles; and to determine the relative contributions of progression of motor and non-motor manifestations of PD to changes in HRQOL over time.

Sunlight exposure and variations in vitamin D metabolic genes in Parkinson's disease
Principal Investigator: Ritz
NIEHS R03- ES017139 09/01/09-08/31/11
Total Direct Costs: $100,000
The goal of the proposed research based on the PEG study population is to examine the hypothesis that long-term low levels of vitamin D either through inadequate sunlight exposure or alterations in metabolic genes that influence physiological vitamin D levels increase the risk of PD. We will test associations between long-term UV exposure measures and PD and examine whether genetic alterations presumed to result in different physiological vitamin D activity in genes critical to the vitamin D pathway (VDR, CYP27B1 and CYP24A1) increase the risk of PD.

Traffic-Related Air Pollution and Ultrasound Measures of Fetal Growth
Principal Investigator: Wilhelm Turner (UCLA)

NIEHS R03- ES017314 04/01/09-03/31/11
Total Direct Costs: $100,000
The specific aims of this study are to estimate prenatal exposures to O3 and PM10 and pollutants originating from traffic (NOx) using CALINE4 air dispersion modeling and examine associations with fetal size throughout pregnancy using ultrasound measures to examine associations with weight, length, head circumference, fetal growth ratio, ponderal index, and cephalization index at birth

Ambient Air Toxics and Adverse Birth Outcomes
Principal Investigator: Wilhelm Turner (UCLA)
NIEHS R03 ES017119-01 12/15/08 – 12/30/10
Total Direct Costs: $100,000
The specific aims of this study are to: (1) examine associations between prenatal exposure to motor vehicle air toxics and low birth weight (LBW) and preterm birth in women residing in Los Angeles County, California between 1994-2006 using both ambient measurement data and land use based regression (LUR) models; and (2) gain information about how LUR models built on NOx measurements reflect exposures to specific toxins thought to have biological relevance for these outcomes.

Exposure to mobile source air pollution and adverse birth outcomes in the Los Angeles Air Basin
Principal Investigator : Jun Wu (UCI)
NIEHS R21 ES016379 9/11/08 -12/31/10
Total Direct Costs: $250,000
The overall goal of the project is to improve exposure assessment of air pollution exposure in pregnant women and investigate the impact of air pollution exposure on adverse reproductive outcomes, such as preterm birth, low birth weight, and intrauterine growth retardation.

Disparity in asthma among Californians from pollutant exposures.
Principal Investigator: Meng, UCLA
California Air Resources Board 04/22/08- 12/31/10
Direct Costs: $270,000
The goal of the research is to conduct a population-based study to examine the effects of long-term air pollution exposure near residence on chronic severe asthma and asthma-like symptoms in vulnerable populations.

Development of Exposure and Health Outcome Indicators for Those with Asthma or Other Respiratory Problems
Principal Investigator: Meng, UCLA
EPA- R833629 09/01/07-12/31/10
Direct Costs: $410,000
The goal of this research is to investigate the feasibility of combining existing environmental monitoring and health survey data to develop indicators that signal trends in exposures and health for those with asthma or other respiratory problems

Neighborhood Effects on Children's Health & Access to Care
Principal Investigator: A. Pebley, UCLA
HRSA 09/01/07- 8/31/10
Total Direct Costs: $500,000
The goal of this study is to significantly advance our knowledge about the relative importance of specific family and neighborhood characteristics in the development of major child health problems. This project is based on the Los Angeles Family and Neighborhood Survey (L.A.FANS), a longitudinal study of neighborhoods, families, adults, and children in Los Angeles County

Traffic-Related Air Pollution and Asthma in Economically Disadvantaged and High Traffic Density Neighborhoods in Los Angeles County, California (with LA F.A.N.S.)
Principal Investigator: Ritz
California Air Resources Board 01/06/05-09/30/09
Total Direct Costs: $420,000
The objectives of this research are: (1) to conduct NO_x and NO_2 monitoring at 200 locations within LA County neighborhoods with varying levels of economic disadvantage and varying exposures to air

pollution originating from vehicular sources; (2) to use these monitoring data to help inform land use-based regression (LUR) models developed to predict traffic pollutant exposures; (3) to use geostatistical models to estimate regional background concentrations of O_3 and $PM_{2.5}$; (4) to evaluate associations between exposure to NO_x, NO and NO_2 and measures of lung function and asthma prevalence, exacerbation and possibly incidence in children ages 0-17 years in conjunction with the Los Angeles Family and Neighborhood Survey (L.A. FANS) study; and (5) to evaluate whether concentrations of the more regionally distributed background pollutants (O_3 and $PM_{2.5}$) confound or modify the effects of exposure to the more heterogeneously distributed traffic-related pollutants (NO_x, NO and NO_2) on lung function and asthma.

Aggregate Exposure Assessment: Longitudinal Surveys of Human Exposure-Related Behavior
Principal Investigator: Irva Hertz-Picciotto, UC Davis
EPA 01/12/04-11/30/09
Direct Direct Costs: $388,111
This project develops data collection platforms for longitudinal assessment of exposure-related behavior. The data characterize short-term, seasonal, and long-term changes in time-activities, food consumption habits, and use of household and personal care products. We assess exposure-related behaviors at multiple collection points over time, and evaluate a number of data collection methods for validity (accuracy), precision, completion rates, cost, feasibility, and user acceptability.

UCLA Center for Gene-Environment Studies in Parkinson's Disease (CGEP-part of the NIEHS CCPDER)
Director: Chesselet, UCLA; Co-director: Ritz
NIEHS 09/01/02-08/31/09
Total Direct Costs: $7,000,000
The overall objective of this Center is to understand how the detrimental effects of pesticides, a suspected environmental risk factor for Parkinson's disease, are modulated by genetic variations that impact dopamine homeostasis in nigrostriatal neurons. The center integrates 3 RO1 research projects that investigate these questions in fly, mouse, cell culture models and applies the results also to human genetics (project 1: PI Ritz)

Research Project I within the CGEP center "Environmental toxins and genes that influence dopamine in Drosophila and humans"
Principal Investigator: Ritz
NIEHS 09/01/02-08/31/09
Total Direct Costs: $1,000,000
This project examines interindividual variability of dopamine vesicular transporter (VMAT) expression due to promoter variants in two human populations in parallel with a reporter gene assay. These populations will be genotyped for functional VMAT2 variants and association analyses of gene-environment interactions and pesticide exposures collected in the parent grant will be conducted. In addition, Drosophila genetics will be used to determine how the expression of VMAT affects dopamine-mediated toxicity and identify genes that modulate VMAT function, which will then be examined in the human population for their relevance to increase risk of PD.

Parkinson's Susceptibility Genes and Pesticides (PEG)
Principal Investigator: Ritz
NIEHS/NINDS 10/01/00-09/30/07
Total Direct Cost: $2,653,852
We are testing the gene-environment interaction hypothesis for Parkinson's disease by conducting an epidemiologic population-based case-control study of 400 newly diagnosed PD patients from three rural California counties matched to population controls; in addition we are collecting data for unaffected sibling controls. Environmental and occupational pesticide exposure estimate are derived from California pesticide-use reporting (PUR) and other data. We are examining the effects of gene-environment interactions by testing for associations of PD using multiallelic repeat markers and genotyping intragenic single nucleotide polymorphisms (SNPs) and/or deletions in 50 candidate genes.

PD Consortium: Genetic and Environmental Factors in Parkinson's Disease
Principal Investigator: L. Nelson, Stanford
MJ Fox Foundation 10/01/04-09/30/07

Total Direct Costs $50,000
We established the Consortium for the Study of Genetic and Environmental Factors in Parkinson's disease, with the goal of organizing the collaborative efforts of five investigative groups that have who have conducted (or are conducting) seven case-control studies of PD. For approximately 1700 PD cases and 2100 gender- and age-matched control subjects, we investigate how the risk of developing PD varies according to tobacco and caffeine intake, as well as variants in ten candidate genes that code for proteins that may be involved in conferring the protective effect of these agents.

Alpha Synuclein and Environmental Exposures: A Study in Humans
Principal Investigator: Langston, The Parkinson's Institute
MJ Fox Foundation 01/01/05-12/31/07
Total Direct Costs $100,000
We are investigating the joint effects of: (1) consequences of alpha-synuclein over-production and enhanced mapping of the SNCA promoter region and (2) the biologic effects specific toxicants (e.g., rotenone, paraquat, organochlorine pesticides). We take advantage of two unique cohorts at high risk for pesticide exposure currently evaluated by members of the NIEHS-funded Collaborative Centers for Parkinson's Disease Environmental Research (CCPDER) at the Parkinson's Institute (PI) and UCLA, the Agricultural Health Study cohort and a population-based study of PD and pesticide exposure in rural Central California (the PEG study).

Prostate Cancer and Pesticide Exposure in Diverse Populations in California's Central Valley
Principal Investigator: Cockburn, USC
DOD 05/01/06-12/31/07
Total Direct Costs: 250,000$
This is a pilot study bringing an innovative collaborative approach to prostate cancer research. Specifically, this study will apply novel methods of pesticide exposure assessment using Geographical Information Systems (GIS), examine whether our proposed method of recruiting and approaching cases and controls for a large population-based case-control study will result in acceptable response rates, or whether our sample will be biased with respect to socioeconomic status, race, and disease characteristics, and whether we will be able to obtain sufficient DNA from mailed (Oragene) spit collection kits to assess effect modification by known relevant genes, and have sufficient stored DNA to assess the impact of genes that may be discovered in future.

Traffic-related Air Pollution and Adverse Birth Outcomes
Principal Investigator: Ritz
NIEHS 07/15/01-06/14/07
Total Direct Costs: $641,612
The objectives of this project are to determine whether exposures to elevated and traffic-related ambient air pollution during pregnancy result in low birth weight, preterm birth, intrauterine and postneonatal mortality, or cardiac defects in infants born to women living in the South Coast Air Basin (SoCAB). We performed a cohort study of all births (between 1995 and 1999), fetal and infant deaths (between 1989 and 1997), and conducted a nested case-control study of 2600 women who delivered children in LA in 2003 to collect additional exposure, confounder, and effects modifier data.

Ergonomic Interventions for Sewing Machine Operators
Principal Investigator: Ritz
CDC/NIOSH 10/01/02-09/31/06
Total Direct Costs: $868,262
We are conducting a randomized trial of a newly developed ergonomic intervention in sewing machine operators working in LA garment shops. The ergonomic intervention package includes changes in work-station design, training of employees, and suggestions of improvement in work procedures. We are examining whether interventions can reduce rates of upper extremity, neck (and lower back) musculoskeletal disorders, severity of pain and impairment, and lost-time compared to 'placebo' (control) interventions. This study will provide employers, employees and public agencies with evidence of the effectiveness of ergonomic interventions in order to guide health and safety policy.

Traffic-Related Air Pollution and Acute Respiratory Diseases and Asthma in Children Ages 0-5 in the SoCAB From 1990-2000

Principal Investigator: Ritz
California Air Resources Board 01/06/04-09/30/05
Total Direct Costs: $55,000
The aims of this study are to estimate the transient effects of traffic related and background air pollution in the South Coast Air Basin (SoCab) on the risk for hospitalization for acute respiratory illness and asthma in children ages 0-5 using a case- crossover study design and a time-series analysis.

Assessment of In-Traffic Exposures and Human Reproductive Health
Pilot project Principal Investigator: Ritz; SCEHSC Center Principal Investigator: Froines, UCLA
EPA 07/01/04-06/30/05
Total Direct Costs Pilot Project within the PM-center: $28,000
The goal of this project is to evaluate whether maternal in-vehicle air pollutant exposures during commutes (either in passenger cases, buses or other means of public transportation) affected the risk of low birth weight (LBW) and preterm birth in infants born to women living in Los Angeles County, California between 2003-2004. Commuting behavior (travel time, mileage and/or modeled routes) will be used to evaluate exposure to motor vehicle exhaust pollutants while in-transit

Molecular Epidemiology and Gene-Environment Interaction
Principal Investigator: Zhang, UCLA
NIH/NIEHS R21 ES 011667 04/01/02-03/31/05
Total Direct Costs: $450,000
This was a planning grant for molecular epidemiology in Environmental genome. The award was to establish a molecular epidemiology research program focusing on environmental genome.

Uncontrolled Asthma and Exposure to Air Pollutants: Linking Chronic Disease and Environmental Data Sources
Principal Investigator: Meng, UCLA
CDC/NIOSH/ 10/01/02-09/01/05
Total Direct Costs: $600,000
Based on the California Health Interview Survey (CHIS 2001) data, an extensive air monitoring network, and detailed information on traffic density we are conducting a population-based epidemiologic case-control study to: (1) ascertain the relationship between control of asthma and exposure to air pollutants in Los Angeles County and San Diego County, California; and (2) build and enhance the partnerships between public health and environmental agencies and local communities.

Center of Excellence for Environmental Public Health Tracking
Principal Investigator: Balmes, UCSF
CDC/ATSDR 10/01/02-09/01/05
Total Direct Costs (UCLA only): $300,000
The UCLA part of this center grant uses the data from 5,200 California Health Interview Survey (CHIS 2001) respondents who reported having been diagnosed with asthma at some point in their lives and live in the Greater Bay Area, San Joaquin Valley, and Los Angeles County. Criteria pollutant averages are employed as measures of background ambient air quality and linked with sociodemographic information and data on asthma management, access to care, and risk behaviors collected through CHIS for each targeted respondent.

Community Response to Maternal/Child Heath Disparities
Principal Investigator: Hobel, Cedars Sinai
NIH 04/1/03-9/30/05
The major goals of this study are to examine the interrelating biological and social-behavioral factors that contribute to health disparities in pregnancy outcomes and infant and early childhood mortality and morbidity. We will participate as one of five selected sites in the nation to plan for a multi-centered, community-based study examining the relationship between environmental factors and child health disparities.

Extension of the Rocketdyne/Al Worker Cohort Through 1999
Principal Investigator: Ritz
California Cancer Research Program 07/01/00-06/30/04

CRP award #00-00781V-20218
Total Direct Cost: $324,508
We extended the mortality follow-up of two previously established cohorts of workers employed at Rocketdyne/Atomics International (now Boeing North American) facility for an additional 5 years and added a cancer incidence component for the period 1972-1998. This study allowed evaluating the impact of radiation and some known animal carcinogens on cancer mortality and morbidity.

Assessment Scale for End-of-Life Care in End-Stage Dementia
Principal Investigator: Ackerman, UCLA
Alzheimer's Association 10/01/00-09/30/03
Total Direct Costs: $217,583
This pilot project developed a scale to assess end-of-life care for end-stage dementia patients and evaluated its performance using mortality data.

Pilot grant from Southern California Center for Airborne Particulate Matter (SCCAPM)
Principal Investigator: Froines, UCLA; Pilot grant Principal Investigator: Ritz
U.S.-EPA-Star grant 07/01/01-12/31/02
Total Direct Cost: $12,000
The pilot grant supported exposure assessment for an epidemiologic study of traffic related adverse birth outcomes.

Evaluation and Validation of Pesticide Use Reporting in California
Principal Investigator: Ritz
UC Toxic Substances Research & Teaching Program 07/01/99-06/30/01
Total Direct Costs: $ 50,000
The goal of this pilot grant was to use biomarker data to evaluate the validity of pesticide exposures estimates derived from geographic models of environmental exposure based on pesticide use reports and land use maps in California residents.

Identify and Reduce Work Hazards in Home Health Care Workers
Principal Investigator: Ritz
Institute of Labor and Employment Pilot Study 02/01/01-30/08/01
Total Direct Costs: $ 7,500
This pilot project developed and tested a survey instrument and collected preliminary data for a study of job hazards in 74,000 home health care workers in LA county.

Pilot Study for Gene-Environment Interaction and Parkinson's Disease Study
Principal Investigator: Ritz
APDA Center Pilot Grant 03/01/99-12/31/00
Total Direct Costs: $35,000
This pilot project involved establishing data resources to improve exposure measures for pesticides, and setting up of a county-wide networks to reach incident Parkinson's cases in rural California.

Development of a Temporary Parkinson's Disease Registry for Southern California
Principal Investigator: Ritz
APDA/Pilot Grant from the PD-center at UCLA 03/01/99-12/31/00
Total Direct Costs: $10,000
This pilot project established mechanisms to obtain incident Parkinson's cases in rural California using information provided by local health care providers, Parkinson's disease foundations, clinics, and Medicare, and to determine which data sources exist for the application of capture-recapture methods to validate coverage of a future PD registry.

Modeling Air Pollution and Birth Defects
Principal Investigator: Ritz
CBDMP Grant/SCEHS/NIEHS Pilot Grant 07/01/00-09/30/00
Total Direct Costs: $5,600
The objective of this project was to examine the usefulness of some advanced statistical modeling procedures in order to determine whether exposures to elevated levels of ambient air pollutants (PM10,

CO) at the levels found in the South Coast Air basin (SoCAB) basin caused defects of the cardiac system of fetuses.

Pesticide Exposure Modeling Based on Historical Use Reporting in California to Investigate Long-Term Health Effects
Principal Investigator: Ritz
UCLA-USC NIEHS-Center Pilot Grant 05/01/99-04/30/00
Total Direct Costs: $18,000
The objectives of this pilot grant were to develop a geographic model for pesticide exposure of California residents between 1950 and 1990 using satellite images of crops, aerial photographs, and Pesticide Use Reporting Data from the California Department of Pesticide Regulations.

Epidemiologic Study to Determine Possible Adverse Health Effects on Rockwell/Rocketdyne Workers from Exposure to Radioactive and Hazardous Substances
Principal Investigator: Morgenstern, UCLA
CPHF/DOE/DE-FG-03-91SF18983 01/10/93-03/31/99
Total Direct Costs: $740,000
The major goal of this study was to test the hypothesis whether exposure to toxic chemicals and ionizing radiation among Rockwell/Rocketdyne workers caused an excess of cancer mortality.

Hazard Surveillance in the Defense Nuclear Industry
Principal Investigator: Froines, UCLA
CDC/NIOSH/R01-CCR912034 09/01/95-08/31/99
Total Direct Costs: $1,244,745
The major goals of this project were to develop an integrated theory, approach, and methodology to exposure assessment and hazard surveillance in the U.S. defense nuclear industry.

The Influence of Air Pollution in the Los Angeles Metropolitan Area on the Occurrence of Birth Defects, 1990-1993
Principal Investigator: Ritz
SCEHSC/NIEHS/UCLA-USC NIEHS-Center Pilot Grant 09/01/97-09/30/98
Total Direct Costs: $24,000
The objective of this pilot project were to examine whether the exposure of pregnant women to elevated levels of ambient air pollutants (Ozone, NO2, PM10, CO) at the levels found in the Los Angeles Metropolitan Area or the South Coast Air basin (SoCAB) basin cause low birth weight or preterm birth.

RESEARCH CONDUCTED IN GERMANY (1984-1989)
Health effects of airborne-dioxin exposure in Hamburg nursery schools
Rheumatic disorders, working conditions and coping behaviors in female office workers
Work-related knee-joint and elbow injuries in pipe-fitters and welders
Back and neck pain, psycho-social and ergonomic stresses in nursing professions

HONORS AND AWARDS

1999	UCLA Faculty Career Development Award
1999	'Rothman' award presented at SER by C. Poole
1989-1992	Post-doctoral fellowship received from DAAD ("German Academic Exchange Office of the Ministry of Research and Technology")
2001	Delta-Omega Award
2007	Robert M. Zweig M.D. Memorial Award (Clean Air Award) from the South Coast Air Quality Management District (AQMD)
2009	Award from the American Parkinson's Disease Association for outstanding contributions to the medical and scientific communities and for my work towards the advancement of Parkinson's disease research

TEACHING

UCLA, School of Public Health, graduate courses, 1995-present
Epidemiology Methods (Core methods course (200B) in the UCLA Epidemiology program)
Environmental Epidemiology
Occupational Epidemiology
Advanced Methods in Occupational and Environmental Epidemiology
Seminar: Occupational and Environmental Cancers
Seminar: Policy Issues in Occupational and Environmental Health

University of Hamburg, Medical School, 1984-89
Lectures and seminars in Medical Sociology for medical students
Lectures and seminars in Psychiatry for medical students

ADVISING AND MENTORING OF DOCTORAL STUDENTS (PH.D) AND POSTDOCTORAL FELLOWS (SUBJECT OF DISSERTATION OR FELLOWSHIP)– *note: this list only includes primary advisees (i.e. chair of committee and not member of dissertation committee) and does not include master level students*

At UCLA:

1997 - 2001	Kurt Straif (Cancer mortality in the German rubber industry)
1998 - 2000	Timothy Clary (Pancreatic cancer mortality and pesticide use in California)
1998 - 2004	Michelle Wilhelm (Traffic-related air pollution and pregnancy related health effects)
1998 - 2004	Rudy Rull (GIS modeling of pesticide exposure and neural tube defects)
1998 - 2004	Anusha Krishnadsan (Occupational physical activity and prostate cancer incidence)
2001 - 2004	Yingxu Zhao (Work place exposures to chemicals and cancer incidence)
2003 - 2004	Gail Asleson Kang (*Movement Disorder Fellow*: Clinical characteristics of PD patients)
2002 - 2006	Pin-Chieh Jason Wang (Ergonomic interventions and health effects in LA garment workers)
2003 - 2006	Chad Lewis (TTHM contamination in drinking water and adverse birth outcomes)
2003 - 2005	Kathrine Hoggatt (co-mentored with Dr Greenland: Air pollution and adverse birth outcomes)
2004 - 2008	Angelika Wahner (Doctoral student & postdoctoral fellow: Parkinson's disease, genetic factors and anti-inflammatory drug use)
2004 - 2008	Marie Sharp (The Latina Paradox in Birth Outcomes)
2004 - 2008	Sadie Costello (Parkinson's disease and life style factors)
2005 - 2008	Shannon Rhodes (Doctoral student & postdoctoral fellow: Iron genetics and Parkinson's disease)
2008 - 2010	Nicole Gatto (Postdoctoral fellow: Vitamin D, sunlight and Parkinson's disease)
2004 - 2008	Amanda Colligan (Residential pesticide exposure and Parkinson's disease)
2005 - 2012	Anthony Wang (Occupational pesticide exposures and Parkinson's disease)
2007- 2011	JoKay Ghosh (Air toxics and adverse birth outcomes)
2008- 2013	Tracy Becerra (Autism and race ethnicity in Los Angeles)
2008- 2013	Erin Jacob-Marcotte (Pesticides in pregnancy and childhood cancers)
2011-2012	Anshu Shresta; post-doctoral fellow (Childhood cancers and the environment)
2011-2013	Pei Chen Lee; postdoctoral fellow (Air pollution and pregnancy biomarkers)
2009-2014	Shilpa Narayan (Progression in Parkinson's disease)
2009-2014	Christina Lombardi (Air pollution and childhood cancers)
2011-2014	Zeyan Liew: PFOA exposures in the Danish birth cohort and ADHD and autism)
2012 -present	Gretchen Bandoli (Stress, asthma and birth outcomes in LA)
2012 -present	Kristina Vanderwaal Hool (breast cancer and methylation patterns)
2011- present	Kim Paul (Gene-environment interactions in Parkinson's – PASIDA study)
2011- present	Xin Cui (Bias analysis in the PASIDA study of Parkinsons)
2011- present	Andrew Park (Pesticides and childhood cancers)
2012- present	Vivian Alonso (Nutrition, vitamins use and reproductive health)
2013- present	Yu-Hsuan Chuang (Parkinsons, gene methylation, and gene-environment interactions)
2013- present	Xiaoqing Xu (Pharmaceuticals and childhood cancers in Denmark)
2013- present	Matt Feaster (Occupations risk factors for childhood cancers)
2013- present	I-Fan Shih (Parkinsons and physical activity)
2013- present	Negar Omid (Childhood cancer risk factors)

2013- present Aline Duarte (Parkinson's non-motor symptoms)
2013- present Chenxiao Ling (Bias analysis in environmental epidemiology)
2014- present Cynthia Kuster (Parkinsons' and estrogen receptors)
2014- present Zuelma Esquivel (Childhood cancer risk factors)

At University of Washington:

2004-2006 Kathrine Carr (*Postdoctoral Fellow*: Bronchiolitis and air pollution in LA infants)

At UCI:

2011-2013 Jun Wu (junior faculty mentor for W. Rosenblith award given by HEI)

At the University of Copenhagen, Denmark:

2008-present Line Kenborg (Parkinson's disease and outdoors work and sunlight exposures)
2007-2009 Kathrine Rugbjerg (Parkinson's disease and head trauma and auto-immune diseases)

University of Umea/Sweden

2014 Opponent for doctoral student David Olsson (Air pollution and PTB and preeclampsia in Stockholm)

PARTICIPATION IN GRANT AND CENTER REVIEWS

Reviewer on a NCI Special Emphasis Panel "Improving Exposure Assessment in Environmental and Occupational Epidemiology of Cancer", May 2001
Reviewer of the NIEHS-funded Columbia University Environmental Health Sciences Center, May 2002
Reviewer of the Charles Harkin Award Application for Research in Thyroid Cancer, NIH, April 2003
Reviewer of the Wellcome Trust Application "Pre and post-natal exposure to particulate matter and pregnancy and infant outcomes: an historical cohort study", 2003
Reviewer of the Health Effects Institute's (HEI) Walter Rosenblith New Investigator Award application, April 2003
Reviewer of pilot grants for the Southern California NIEHS center grant (2004 and 2005)
Reviewer of pilot grants for the UCLA-CCPDER center (NIEHS funded) (2003 and 2005 and 2008)
Reviewer for NCI, Epidemiology of Cancer (2004/05 Council EPIC)
Reviewer for several NIH, Department of Health & Human Services meeting applications, 2003-2005
Reviewer (Chair of Review Committee) for a NIEHS-PO1 application (2004)
Appointment to Review Committee of the European Science Foundation (ESF) (2005)
Annual Review of SCEHSC Pilot Project Submission (permanent member 2004-current)
Institutional Patient-Oriented Career Development Programs in the Environmental Health Sciences [K12] (ES06-005). (2007)
Conference grant applications (2004-2007)
NIH reviewer for Outstanding New Environmental Scientist (ONES) award in the Environmental Health Sciences (2006)
Member of the EPA's Clean Air Scientific Advisory Committee (CASAC) Carbon Monoxide (CO) Review Panel (2008-current)
Grant review for an internal NIEHS scientist's application (Dr. Chen) (2007 and 2008)
Grant review for NIEHS special emphasis panels 2009-2010
Grant review for NIH-BCHI 2011
Pilot grant review for the Northern California Center for the National Children's Study –Pilot Projects Program August 2011
External Review of the Neurology Department at Columbia (NY), 2011
Scientific Review of Superfund Site Projects as EAC member for University of Washington, 2012
External Review of the Swiss Tropical and Public Health Institute (TPH), 2012 and 2013
External Review of the Epidemiology Branch at NIEHS, 2013
Review for Harvard NIEHS center pilot grant, 2014
Review of applications for Health Effects Institute (HEI Boston), Rosenblith awardees, 2014
Review for Mount Sinai (NY) NIEHS center pilot grants, 2014
Review for NIEHS USC-UCLAEnvironmental Health Science center pilot grants, 2014
Review of NIEHS conference grants July 2015
Review of Parkinson's disease grant for Parkinson's UK foundation in Great Britain

JOURNAL REVIEWER FOR:

American Journal of Epidemiology
Epidemiology
International Journal of Epidemiology
Annals of Epidemiology

Environmental Health Perspectives
Environmental Health
Occupational and Environmental Medicine
Archives of Neurology
Annals of Neurology
Neurology
Movement Disorders
Pediatrics
JAMA
Lancet
Parkinson's and Related Disorders
Pharmacogenetics and Genomics
Journal of the Air & Waste Management Association
Journal of Exposure Analysis and Environmental Epidemiology
Chemosphere
Zeitschrift Sozial- und Präventivmedizin (SPM)
Human Reproduction
Women & Health
Etc.

INVITED SEMINARS AND LECTURES (SELECTED)

1. The Health Effects of Low-level Ionizing Radiation, USC, Health Sciences 1996
2. Work Environment and Health, UCLA Health Sciences 1996
3. The Effects of Carbon Monoxide Exposure on Low Birth Weight in the LA Metropolitan Area, 1989-1993, USC, Southern California Environmental Health Sciences, 1997
4. Cancer Mortality in Radiation Workers, USC Southern California Environmental Health Sciences, 1997.
5. Basic Principles of Reproductive Epidemiology, European School of Risk Assessment in Reproduction" in Florence/Italy December, 1997.
6. The Rocketdyne/Al Worker Health Study: Results and Lesson's Learned, California Department of Health Services, Occupational Health Branch, 1998
7. Air Pollution and Low Birth Weight in Southern California, GSF Munich Germany, 1998.
8. Air Pollution and Adverse Birth Outcomes: Methodological Issues and First Results, Southern California Environmental Health Science Center, USC, 1998.
9. Gene-Environment Interaction and Parkinson's Disease, Neurology Grand Rounds, UCLA 1998
10. Air Pollution and Adverse Birth Outcomes in Southern California, Dept. of Reproductive Epidemiology, University of Michigan, East Lansing, 1999.
11. Methodologic Issues in Studying of Gene-Environment Interaction, GSF Munich Germany, 1999
12. Methodologic Aspects of Studying Cancer Mortality in Radiation Workers, Dept. of Epidemiology, University of Michigan, East Lansing, 2000.
13. Cancer Mortality in Fernald Uranium Workers, NIOSH, Cincinnatti, 2000.
14. GIS Modeling of Pesticide Exposures in California, Dept. Environmental Epidemiology, GSF Munich Germany, 2000
15. Traffic-related Air Pollution and Adverse Birth Outcomes in Southern California, Dept. Environmental Epidemiology, GSF Munich Germany, 2000
16. Studying Parkinson's disease in Populations; American Parkinson's Disease Association conference for patients and care providers at UCLA, 2001
17. From the Epidemiology of Parkinson's Disease to Gene-Environment Interactions, VA-PD conference, Woodland Hills, 2001
18. GIS Modeling of Air Pollution and Pesticide Exposures in California, USC-UCLA NIEHS Town hall meeting; Dec, 2001
19. GIS Modeling in the context of a Gene-Environment Interaction study of Parkinson's disease, Dept. Environmental Epidemiology, GSF Munich Germany, 2001
20. The Epidemiology of Parkinson's Disease, Conference of the Society for Research on Amyotrophic Lateral Sclerosis, Colorado May 2002
21. Traffic-related Air Pollution and Reproductive Health Effects: An Overview; Environmental Health Sciences seminar at UC Riverside, Feb. 2002
22. Reproductive Health Effects due to Carbon Monoxide Air Pollution in Southern California, NRC

Subcommittee on Health Effects from CO pollution meeting at UC Irvine, April 2002
23. Traffic-related Air Pollution and GIS Modeling in Southern California, USC-GIS Workshop Pasadena, May 2002
24. Health Effects Modeling with GIS, USC-GIS Workshop Public Forum at USC, May 2002
25. Dopamine Imbalance and Oxidative Stress in Parkinson's Disease, VA Research Conference on PD and Movement Disorders, Los Angeles 2002
26. The Center for Gene Environment Interaction in Parkinson's disease (CGEP) at UCLA: Dopamine Imbalance in Parkinson's Disease, Inaugural NIEHS Conference at the Parkinson's Institute in Sunnyvale CA, August 2002
27. Air pollution effects on birth outcomes: An overview. Health Effects Institute, Annual conference held at Georgetown University; 2003
28. Linking air pollution effects and adverse birth outcomes in the Los Angeles basin throughout the 1990s. U.S. EPA, Chapel Hill, NC; 2003
29. Air Pollution and Adverse Birth Outcomes in the South Coast Air Basin, 1989-2000; Conference of the Czech NAS meeting on air pollution effects (Dr. Sram), Prague, 2003.
30. Air pollution and adverse birth outcomes, an update on recent developments. Department of Preventive Medicine at the University of Southern California, 2003
31. GIS modeling of environmental exposures: applications to air pollution and pesticide exposures. Department of Environmental Health, Harvard, 2004
32. Air pollution models of adverse birth outcomes. Department of Epidemiology at the University of North Carolina, 2004
33. Parkinson's disease, metals and pesticides. Department of Toxicology, Symposium on Toxics Risks and Aging, Duke 2005
34. Air pollution and adverse birth outcome research in the SoCAB from 1995-2005. California Air Resources Board, Sacramento, Sept 2005
35. Parkinson's disease and pesticide exposure assessment in farming communities in the California Central Valley. Symposium of the Ramazzini Conference, Bologna, Italy Sept. 2005
36. Parkinson's disease and aging. UCLA Center on Aging Research Conference on Aging 2006.
37. Air Pollution and Asthma in Children. AQMD Asthma Impacts of Air Pollution Conference Los Angeles, Feb. 2006
38. Parkinson's disease and pesticides in the Central California Valley. NIEHS center at Columbia University, NY 2007
39. Assessing pesticides exposures for prostate cancers in the Central California Valley. IARC, Lyon 2007
40. Air pollution and adverse birth outcomes in LA. INSERM, Paris 2007
41. Gene Environment Interactions in Parkinson's disease. CREAL Institute, Barcelona 2008
42. Latest results on Gene Environment Interactions in Parkinson's disease. INSERM, Paris 2008
43. Re-assessing Gene Environment Interactions in Parkinson's disease. MDS conference symposium, Chicago 2008
44. Methodological Issues in studying risk factor for Parkinson's disease in populations. MDS conference symposium, Chicago 2008.
45. Environmental and occupational health studies in California. University of Dublin 2008
46. Air pollution, pregnancy and child health; Healthy Development and Ageing Workshop; British Foreign & Commonwealth Office, LA 2009
47. Air pollution, pregnancy and child health; Physician's for Social Responsibility Environmental training 2009
48. Air pollution and adverse pregnancy outcomes in LA; Annenberg School of Journalism 2009
49. Parkinson's disease and pesticides. George Washington University Environmental Health Program 2009
50. LUR model for traffic related exposures and adverse birth outcomes in LA. Helmholtz Center Munich 2010
51. Parkinson's disease and gene–pesticide interactions. Symposium on Predictive Health, Human Health: Molecules to Mankind. Emory University Atlanta Dec 2010
52. Air Pollution and Adverse Birth Outcomes, invited speaker at HEI annual conference Boston 2011
53. Parkinson's disease in Denmark; the PASIDA study; University of Odense Denmark, May 2011
54. Gene-environment interactions in Parkinson's disease, invited symposium speaker at the International Society for Environmental Epidemiology (ISEE), Barcelona 2011
55. Air Pollution and the Brain; invited plenary speaker at the annual conference of the International Society for Environmental Epidemiology (ISEE), South Carolina 2012

56. Air Pollution and Autism; invited speaker at the University of Aarhus, Denmark 2012
57. Air Pollution, Children and Women's Health in LA; invited speaker at the SCAMQD conference for stakeholders, LA 2013
58. How to be an Epidemiologist, invited speaker at SER, Boston 2013
59. Pesticides and Neurodegeneration; invited speaker at the Conference on safety of fumigated container shipping in Berlin, Germany 2014
60. History of Environmental and Occupational Epidemiology, invited speaker at SER, Seattle 2014
61. History of Air Pollution, Adverse Birth Outcomes and Children's Health in California; Invited Plenary Speaker for the ISEE Young Researcher Conference, Barcelona 2014
62. Environmental Causes of Adverse Neurodevelopment; Invited Speaker at the B-Debate Barcelona (Environment and Child Brain Development: the Challenges in the Global Context) Conference, Barcelona 2014
63. Autism Epidemiology; invited speaker at the annual CART meeting UCLA 2014
64. Epidemiology of Parkinson's disease, invited speaker at annual GEO-PD meeting Vancouver CA, 2014
65. Parkinson's Disease Epidemiology: a Gene-Environment Perspective, invited speaker at the Neurogenetics Institute of Luebeck/Germany, 2015

PUBLICATIONS

PEER REVEIWED JOURNAL ARTICLES (*indicates mentored students/fellows)

1. **Ritz B**. Humeral Epicondylitis Among Gas- And Waterworks Employees. Scandinavian Journal of Work, Environment and Health, 1995 Dec, 21(6): 478-86.
2. **Ritz B**, Heinrich J, Wjst M, Wichmann E, Krause C. Effect Of Cadmium Body Burden On Immune Response Of School Children. Archives of Environmental Health 1998,Jul-Aug; Vol 53: 272-280
3. **Ritz B**, Morgenstern H, Froines J, Young B. Effects Of Exposure To External Ionizing Radiation On Cancer Mortality In Nuclear Workers Monitored For Radiation At Rocketdyne/Atomics International. AJIM 1999, Jan; Vol 35: 21-31.
4. **Ritz B**, Yu F. The Effect Of Ambient Carbon Monoxide On Low Birth Weight Among Children Born In Southern California Between 1989 and 1993. Environmental Health Perspectives 1999 Jan, 107(1):17-25. PMCID: PMC1566307
5. Heinrich J, Hoelscher B, Wjst M, **Ritz B**, Cyrys J, Wichmann HE. Respiratory Diseases And Allergies In Two Polluted Areas In East Germany. Environmental Health Perspectives 1999,Jan; 107(1):53-62. PMCID: PMC1566314
6. **Ritz B**, Morgenstern H, Moncau J. Age At Exposure Modifies The Effects Of Low-Level Ionizing Radiation On Cancer Mortality In An Occupational Cohort. Epidemiology 1999, Mar; 10(2):135-140.
7. **Ritz B**. Radiation Exposure and Cancer Mortality In Uranium Processing Workers. Epidemiology, 1999, Sep; 10:531-538
8. **Ritz B**. Cancer Mortality Among Workers Exposed To Chemicals During Uranium Processing. JOEM 1999, Jul;41(7):556-566.
9. **Ritz B**, Morgenstern H, Froines J., Moncau J. Chemical Exposures Of Rocket Engine Test Stands Personnel And Cancer Mortality In A Cohort Of Aerospace Workers. JOEM, 1999 Oct; 41(10): 903-910.
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EXHIBIT B

Studies excluded from the present review and the reasons for exclusion

Brown et al, "Pesticide exposures and multiple myeloma in Iowa men."[1]	Only provided results for multiple myeloma.
Fritschi et al, "Occupational exposure to pesticides and risk of non-Hodgkin's lymphoma." [2]	This paper did not report an effect estimate specific to glyphosate
Flower et al, "Cancer risk and parental pesticide application in children of Agricultural health study participants."[3]	Study took place in children; no specific glyphosate- lymphoma associations were reported.
Hoar et al, "Agricultural herbicide use and risk of lymphoma and self-tissue sarcoma."[4]	Results specific to glyphosate were not reported.
Kachuri et al, "Multiple pesticide exposures and the risk of multiple myeloma in Canadian men."[5]	Results only reported for multiple myeloma.
Landgren et al, "Pesticide exposure and risk of monoclonal gammopathy of undetermined significance in the Agricultural Health Study."[6]	Monoclonal gammopathy of undetermined Significance (MGUS) is a precursor condition to multiple myeloma.
Sorahan, "Multiple Myeloma and Glyphosate Use: A Re-Analysis of US Agricultural Health Study (AHS) Data."[7]	Only provided results for multiple myeloma.
Waddell et al, "Agricultural use of organophosphate pesticides and the risk of non-Hodgkin's lymphoma among male farmers (United States)."[8]	This study did not report on glyphosate.
Zhang et al, 2016, "Health effect of agricultural pesticide use in China: implications for the development of GM crops."[9]	This article examined blood chemistry measures in relation to glyphosate, (markers for renal and hepatic function such as electrolytes, B vitamins, serum glucose, C-reactive protein, and peripheral nerve conduction). Not directly relevant for NHL

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EXHIBIT C

Compensation

My rates for expert work are $550.00/hour and $5,000.00/day for deposition and trial testimony.

Prior Testimony

I have not given a deposition or trial testimony in the last four years.