STRESUPERIOR COURT OF THE STATE OF CALIFORNIA
COUNTY OF LOS ANGELES

NC, a minor

Plaintiff,

v.

Hain Celestial Group, Inc.; Beech-Nut Nutrition Company; Nurture, Inc.; Plum, PBC, d.b.a. Plum Organics; Gerber Products Company; Walmart, Inc.; Sprout Foods, Inc.; Ralphs Grocery Company; and DOES 1 through 100 inclusive

Defendants.

Case No. 21STCV22822
Judge: Hon. Amy D. Hogue

Department: 7

EXPERT REPORT OF DR. BEATE RITZ, MD, PH.D. IN SUPPORT OF GENERAL CAUSATION

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I. Background and Qualifications

I, Beate Ritz, MD, Ph.D., am Professor of Epidemiology at the UCLA Fielding School of Public Health, and served formerly as Vice Chair and Chair of the Epidemiology Department for a decade, and hold co-appointments in Environmental Health Sciences and Neurology at the UCLA, School of Medicine. I have been trained in Medicine at the University of Hamburg/Germany and received an MD in 1984 and 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 member 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). In the past two decades, I also have been the principal investigator of numerous studies that investigated the influence of pesticides and air pollutants – including metals – in environmental epidemiology studies of reproductive outcomes and autism in California. I also conducted environmental epidemiologic research based on large databases and children's cohorts 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 2007, I received the Robert M. Zweig M.D. Memorial Award (Clean Air Award) from the California South Coast Air Quality Management District and have been serving for 9 years as a scientific advisor on the Air Toxics Board of California that advises the Office of Environmental Health Hazard Assessment (OEHHA) for the state of California. I served on multiple National Academy of Sciences/Institute of Medicine (NAS/IOM) committees

evaluating Gulf War Illness – including IOM reviews of air pollutants and pesticides. I also 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 served on the editorial Board of the Journal *Epidemiology* as well as other journals (recently I edited a section of the journal *Current Environmental Health Reports*). I have been the elected President of the International Society for Environmental Epidemiology (ISEE) in 2018-19, and now serve as Past President and Fellow of the ISEE and chair of the membership committee. In 2018, was named as being in the top 1% of most cited authors in my field by Clarivate Analytics. My Curriculum Vitae is attached.

II. Charge

I have been asked to provide my opinions regarding whether early life exposure to lead, arsenic, and mercury can cause, i.e., are causally associated with, autism spectrum disorder (ASD) and whether exposure to lead can cause attention-deficit hyperactivity disorder (ADHD). This report contains a summary of my analysis and conclusions. I reserve the right to amend this report and the analysis and/or conclusions herein in light of new information, the opinions of defendants' expert witnesses, or any other reason. I also reserve the right to add new opinions regarding the relationship of baby foods that contain lead, arsenic, and mercury and their ability to cause ASD, and baby foods that contain lead and their ability to cause ADHD once this case proceeds to a stage where I will have access to information specific to the foods at issue. Finally, I also reserve the right to use demonstratives and other visual material – including animations – at any evidentiary hearing or trial in support of my opinions and testimony.

III. Summary of Opinions

A. Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to mercury, arsenic and lead during sensitive developmental periods in early childhood can cause ASD**,** and lead exposure can cause ASD at relatively low concentrations.

B. Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to lead during sensitive developmental periods in early childhood can cause ADHD, even at low levels of exposure.

IV. Prior Expert Testimony and Compensation

In the last four years, I have provided testimony as an expert witness in the following cases: *Johnson v. Monsanto Company* (Super. Ct. County of San Francisco, CGC-16-550128); *Alva and Alberta Pilliod v. Monsanto Company* (Super. Ct. County of Alameda, RG17862702); *In Re Roundup Products Liability Litigation* (N.D. Cal., MDL 2741); *Hardeman v. Monsanto Company* (N.D. Cal., 3:16-cv-00525-VC); *Hoffman, et al. v. Syngenta Corp., et al.* (Illinois Twentieth Circuit Court, No. 17-L-517); *Korein v. Syngenta Corp., et al.* (Illinois Twentieth Circuit Court). I am being compensated at a rate of 550/hr. for my time.

V. Methodology

2.0 Definitions of Statistical and Methodological Terms

(Population-based) Case-control study assembles its subjects based on their disease status. In other words, study subjects referred to as cases are enrolled because they have the disease (in this case, autism or ADHD) 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's exposures (in this case to metals) are assessed from biosample tissues (Blood or urine etc) or – if possible – exposures are reconstructed from a record system (e.g. air toxics measures). Like any study type, there are benefits and limitations with case-control studies, and I have carefully considered and weighed those issues in reviewing the relevant case-control studies in this report.

A Cohort study enrolls subjects based on their exposures (in this case metal exposures), and follows the participants closely over time to determine who develops the disease(s) of interest. At enrollment, all participants are evaluated for exposures via records or the analysis of

biosamples similar as in a case-control or cross sectional 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. A special type of 'retrospective' cohort study is possible when biosamples were obtained and stored for participants at enrolment or follow-up prior to disease onset and can be used selectively in future analysis. Like any study type, there are benefits and limitations with cohort studies, and I have considered and weighed those issues in reviewing the relevant cohort studies in this report.

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 autism risk from exposure. An OR of 3.00 would represent a 3-fold or 200% increase in the odds of disease. 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 middle 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. Alternatively, instead of calculating an OR, studies may simply compare mean or median exposures (and standard deviations) of a metal measure in a biosample between cases and controls and present statistical tests for the observed differences.

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 1.50 is a 50% 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 0.05 means that, given the null hypothesis is true, if you repeatedly conducted 100 tests of samples drawn from the same population (people), then in 5% 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. The use of the $p<0.05$ to establish statistical significance is entirely based on convention. It is inappropriate to disregard any result merely because it fails to achieve statistical significance. Rather, it is important consider all data points, and carefully consider the strength of that data, which would include considerations of statistical significance. In considering and

analyzing the studies listed in this report and reference list, I have given careful consideration to statistical significance and p-values.

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. It is inappropriate to disregard any result merely because the confidence interval crosses 1. Rather, it is important consider all data points, and carefully consider the strength of that data, which would include considerations of statistical significance. In considering and analyzing the studies discussed in this report and identified in the reference list, I have given careful consideration to statistical significance and p-values.

Statistical significance. Starting off with an assumption of no association between exposure and outcome as the "null hypothesis", when we conduct our statistical analysis, we can determine the p-value for this null hypothesis of our findings, which is the probability of obtaining an estimate at least as far from a pre-specified value (the estimates' 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, gives us the probability that the specified hypothesis is true). As a convention a $p<0.05$ is often considered "statistically significant". It is important to realize that this is simply a convention which can also be and is often replaced by other p-values such as $p<0.01$ or $p<10^{-7}$ (in genomic studies). What a p-value of 0.05 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 5% 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 exposure causes the disease. Moreover, if $p>0.05$, this doesn't "prove" that this specific null hypothesis is true

since absence of proof is not proof of absence. Similarly, when a (95%) confidence interval excludes the null value because is not inside (or covered) by the confidence interval-- it would be considered "statistically significant". As with p-values, confidence intervals can be defined differently as 95% intervals or also as 90% or 80% etc. intervals. However, confidence intervals provide additional information that a p-value does not provide. This is information about the precision of the estimate or what is also sometimes called the 'informativeness' of the data. In practice, p-values and confidence intervals that are close to the null value of the estimate (for example, if one side of the confidence interval is between 0.9 to 1.1) are sometimes considered 'marginal' in terms of statistical significance. It is however important to realize that estimates that are the least influenced by chance are not those with low p-values, but those with narrow confidence intervals.

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

In vitro. *In vitro* refers to medical procedures, tests, and experiments that researchers perform outside of a living organism. An *in vitro* study occurs in a controlled environment, such as a test tube or petri dish.

In vivo. "*In vivo*" refers to tests, experiments, and procedures that researchers perform in or on a whole living organism, such as a person, laboratory animal, or plant.

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. In considering and analyzing the studies listed in this report and reference list, I have given careful consideration to a study's statistical power.

Data Summaries. Data pooling and meta-analyses are commonly used to summarize

results across scientific investigations especially when there have been multiple small studies on a topic and results can be summarized across studies. This approach allows for results to be compared between and combined across studies such that the overall sample size is larger, and we gain statistical power to investigate associations and draw conclusions.

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 using a uniform analysis approach for the pooled data. In order to conduct data pooling, scientists need to obtain access the data from the investigators of multiple studies. Pooled studies not only have greater statistical power than the original studies from which they draw but also can conduct subgroup analyses that individual studies are not statistically powered to support.

Meta-analysis. In many instances, scientists are interested in pooling data but do not have access to the raw data from each or most studies. This is, typically, because the studies were conducted over long time frames or multiple countries and continents, or perhaps because the investigators do not want to or are not allowed to share human data due to privacy restrictions. Thus, it is likely more feasible and quicker and more efficient to conduct a meta-analysis based on reported summary estimates and other published data. A meta-analysis uses the effect measures (means, Odds Ratios or Rate Ratios) and confidence intervals or p-values which were published in the original studies to come up with a summary estimate of the relationship between exposure and disease. Meta-analyses have much greater statistical power than each study alone to support conclusions, but the authors have to decide on and provide the readers with in- and exclusion criteria for studies and follow a transparent protocol in study identification and selection. These generally include having the required data available to generate summary estimates and meeting a minimum quality criterium for study design, conduct, and analyses. Meta-analytical tools include forest plots which are visual representations of the main results of all or selected studies on a topic. The purpose of grouping them together visually is that it can give the reader a sense of the overall size and position of the effect estimates i.e. the direction and strengths of an association measures in the existing literature. Like any study type, there are

benefits and limits with using pooled analysis, and I have considered and weighed those issues in reviewing the relevant pooled analyses in this report.

Null hypothesis means no effect. The statistical tests done in studies described below aim to test the null hypothesis: they want to determine if the null hypothesis can be rejected with adequate statistical certainty and whether they can determine whether there any relationship between exposure to metal and neurodevelopmental outcomes.

Dose-response. A dose-response association represents an increasing risk with an increasing dose and/or exposure, 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 paraquat 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 autism and sex is also associated with metal exposure, we would want to adjust all effect estimates for metals by sex to remove potential confounding bias (one way to help do this is by matching cases and controls on sex i.e. select the same number of male and female cases and controls). In considering and analyzing the studies discussed in this report and identified on the reference list, I have given careful consideration to any issues of confounding.

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 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. In considering and analyzing the studies listed in this report and the reference list, I have given careful consideration to any potential biases.

A particular issue of concern for studies that use biomarkers of exposure - such as measures of metals in blood or urine - and collect these biosamples after disease onset is the potential of reverse causation i.e., it might be possible that the disease caused the exposure and not vice versa because disease caused certain behaviors or physiological states that increased exposure levels among the cases at the time of sample collection. However, as discussed below, the availability of prospective data, studies conducted on prenatal and early life exposures, refute the likelihood of reverse causation. 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 or cross sectional studies as might be the case if cases are drawn from specialty clinics while controls are drawn from a different population or neighborhood altogether. Selection-bias in cohort studies depends on large and differential (with regard to case status) loss to follow-up in cohort studies. In considering and analyzing the studies listed in this report and reference list, I have given careful consideration to any issues of reverse causality and selection bias.

2.1 Literature search

To obtain all published studies on the relationship between exposure to lead, mercury and arsenic and ASD and lead and ADHD, 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/). The use of such databases and the searching for relevant literature is commonly used by epidemiologists in assessing causality. 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. Most citations were not immediately relevant to the present question, due to their focus on topics other than those of interest. I also

reviewed and considered data in animal models as well as *in vivo* and *in vitro* studies, all of which are commonly used by epidemiologists in assessing causality.

As is typical in most published reviews or meta-analyses, I took additional steps to ensure I did not miss any relevant articles by also reviewing published papers' citations and summary reports. For these, I primarily relied on articles on the topic that were published most recently. This approach is also commonly used by epidemiologists in assessing causality. Following my literature review, I rely on the Bradford-Hill guidelines to assess causality; specifically, I will consider the applicability of the components of the Bradford Hill (Hill,1965) and the 1964 Surgeon General Reports' guidelines to interpreting the results, as follows:

Temporality: is a necessary element for inferring causality. Interpretation of evidence on temporality, that cause must come before effect, may be complicated if the sequence is not known.

Strength of Association: strength of an association (i.e., the size of an effect related to the exposure) as an important consideration for identifying causality, stronger associations weighing more strongly.

Consistency: originally, referred to the replicability or reproducibility of a finding, that is whether comparable findings were made in multiple observational studies carried out by different investigators in different populations.

Plausibility: Is the proposed effect consistent with what is known generally about the causation of the outcome of concern?

Specificity: whether or not an effect is observed in a specific population and site with no other paths to disease (i.e., a single cause linked to a single disease), has been a weak element of the causal guidelines that is often set aside as agents may cause multiple diseases and these diseases have other (multiple) causes.

Coherence: Coherence, an element of plausibility, generally refers to the complementarity of evidence, as support is generated for causation.

Experiment: if it is possible to appeal to experimental evidence it should be considered

(generally not the case for human exposures to toxicants).

Analogy: analogies or similarities between the observed association and any other associations (generally not considered a very strong argument for causality).

2.2 Reliance on peer-reviewed literature

As I teach my students, the most relevant articles, and indeed the only articles I generally review and cite in my own research, are those that have gone through peer review at a reputable journal. In each field there are different journals considered most reputable; but in general, a reputable journal is listed in the well-known and respected indexing sources such as PubMed.[1] Typically, such journals have been published for years and many are the official journals of and are backed by well-recognized and respected medical or research non-profit organizations, such as the American (or European or Asian) Medical or Pediatric Associations, the International Society for Environmental Epidemiology, or International Society for Exposure Assessment.

Peer review has been defined as "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 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." (Danick, 1991).

Independent peer review is the cornerstone of science internationally and in the United States and forms the basis for what the scientific community considers acceptable and reliable medical and scientific research. The peer review process is mostly done anonymously, that is the reviewer is mostly anonymous, although this is changing recently, while generally - but not

[1] 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."

always - the authors are identified. This system is thought to provide the intellectual rigor required to ensure that scientific papers are written and the research conducted in adherence to what is acceptable in the field with regards to placing the research into the context of the relevant literature, and the statistical methods employed, and importantly in determining whether the research protocols used widely accepted and rigorous methods, such that the reported results may be deemed valid and have avoided or accounted for biases. Finally, reviewers are asked to draw conclusions that are appropriate in the context of the study's findings. Peer reviewers provide their opinions on whether or not an article is acceptable for publication and make recommendations to the editors. To succeed within this system, authors typically will only submit their best work; i.e. work they have rigorously discussed with all co-authors and deemed of high quality after extended discussions. Secondly, authors have to subject themselves to and answer reviewers' critiques and must be willing to make changes as requested or argue against suggested changes if there is a compelling reason, and provide justifications to the editors for rejecting reviewers' suggestions. The ultimate decision is made by the editor on whether a manuscript is accepted for publication. I have personally peer reviewed on hundreds of occasions and for more than 25 different journals. I have also served on the editorial boards of three journals: Epidemiology, Epidemiologic Perspectives and Innovations, and Environmental Health.

This system of peer review has been practiced for decades. While it is not without its shortcomings, most revisions that are suggested will improve the quality of published manuscripts, and this system can avoid potential scientific fraud, while encouraging scientific honesty and publication of state-of-the-sciences papers (Dancik 1991).

VI. Epidemiology of ASD and ADHD and Heavy Metal Exposures

A. Autism Spectrum disorders (ASD)

ASD is a neurodevelopmental disorder diagnosed in early childhood or infancy that presents with impairments or absence of language development, social interactions, including nonverbal communication, as well as repetitive behaviors and sensitivity to changes in the

environment (Rapin 1997, American Psychiatric Association 2000). The severity of impairment on these dimensions as well as in terms of cognitive functioning can be quite variable making the autism phenotype look clinically distinct (Fombonne 1999). However, even children with ASD who are considered higher-functioning confront non-trivial challenges throughout their lives. According to reports from the Centers for Disease Control and Prevention (CDC) surveillance, ASD prevalence estimates of 1 in 150 in the year 2000 increased steadily to about 1 in 54 in 2016 and the ASD sex distribution is skewed to male predominance with a 4.3:1 male to female ratio overall.[2] Disease rate increases over time may in part be due to changes in diagnosis and recognition and inclusion of some milder forms of ASD but it cannot completely be explained by these factors (Hertz-Picciotto 2009).

B. Attention deficit hyperactivity disorder (ADHD)

ADHD is characterized by developmentally inappropriate levels of inattention, impulsivity, and hyperactivity and has become one of the most common childhood neuropsychiatric disorders, (American Psychiatric Association 2000; Biederman & Faraone 2005) with a prevalence in children of 3–8% worldwide (Froehlich et al. 2007; Remschmidt 2005). Children with ADHD are at increased risk for conduct disorder, antisocial behavior, and drug abuse later in life (Satterfield et al. 2007) and substantial costs are incurred for their medical care and education (Leibson et al. 2001, Matza et al. 2005).

C. Heavy metals (lead, mercury, and arsenic) – study design and exposure measurement

More and more research has been published in recent decades that assesses the role of toxic metal exposures as well as trace and elemental metals on human neurodevelopment including the distinct disorders known as ADHD and ASD. As autism/ASD is relatively rare in the general population, cohort studies of autism would have to be extremely large i.e., start with hundreds of thousands of pregnant women and follow all of these children to the onset of autism. As this approach would be extremely cost and labor intensive, it is rarely or never employed in

[2] (https://www.cdc.gov/ncbddd/autism/data/index.html).

human population studies. Rather, almost all studies of ASD and most ADHD studies to date relied on the much more efficient case-control or cross-sectional designs. Also, studies of ASD or ADHD have mostly measured metals in blood, urine or hair, and were relatively small in size with less than 50-100 affected children and controls. As small studies are often statistically underpowered and only can evaluate relatively large effect sizes, the estimates of effects in such studies are subject to greater random error and often do not allow firm conclusions on their own. Thus, it makes sense to combine the information from many smaller studies formally via meta-analytical tools to derive a summary effect estimate across studies. A number of such meta-analytical effect estimates have been published in recent years and, in the following, I will not present hundreds of small studies but rather concentrate on discussing the summary estimates presented in systematic reviews and meta-analytical approaches. This is a common and well-accepted approach employed in epidemiology for assessing causation. However, I will also refer to individual studies whenever necessary.

Studies of known and suspected neurotoxicants in humans, including the heavy metals lead, mercury, and arsenic, are generally based on biomarker measures of exposure. This is usually seen as an advantage as such measures are considered 'objective' or more reliable and they are not affected by recall error or recall bias as might be the case when exposures are purely self-reported. On the other hand, biomarker measures of exposure may suffer from other types of measurement error that differ between types of tissue that the biosample has been taken from, and the analytical method and its detection limits. These biomarkers of exposure also differ in terms of being able to inform about the time when the exposure occurred or the relevance of the measurement for the health effect of interest (long-term versus short term biomarkers).

Another issue that is important to consider are the detection limits of the methods applied to the different types of sample and different metals as the sensitivity of such measurements are not only different by tissue/compartment, but detection limits have also shifted over time (with more and more sensitive laboratory methods having been developed) and may be different between labs (and countries) in which analyses were conducted.

In epidemiologic studies, prenatal and early childhood exposure to heavy metals have been measured in multiple types of tissue (blood, urine, hair, brain, baby teeth, and nails) taken from pregnant women and/or children diagnosed with autism or ADHD. In children, these exposure measurements were mainly conducted after a diagnosis had been made, i.e., samples are taken from affected children and compared to those taken from unaffected children in the same way. Such biomarkers represent information about exposure levels in cases up to or after the diagnosis was made. Therefore, these studies may have to assume that measured tissue concentrations are representative of life periods most sensitive to the exposure (especially for brain development) and/or represent relevant cumulative exposures prior to diagnosis and sample collection. The assumption that these measurements are valid exposure markers for the health effect of interest are more likely to be accurate if exposure sources did not change considerably over a child's lifetime or the children studied are diagnosed at a very early age. For the metals of interest here, blood or urine measurements are commonly used and mainly reflect current exposures of study participants. There are a number of studies that measure metals in children's hair samples, and very few collect nail samples or brain samples (post-mortem). For hair, the average growth rate is roughly 1 cm per month and consecutive 1-cm segments of hair recapitulate average monthly blood levels with a ~20-day lag between the concentration of trace elements in the first centimeter next to the scalp and the corresponding average monthly blood level (Clarkson & Magos, 2006). Teeth cover the longest exposure period as baby teeth reflect exposure during both the pre- or postnatal time of development depending on which type of tooth is analyzed (incisors versus molars etc). However, studies that use ground whole teeth and do not assess the incremental microstructure of the tooth do not provide temporal information about exposure timing and, thus, only provide a cumulative exposure measure. The exception are baby teeth analyzed in a manner that provides information about the exact exposure period (see below). Most studies collect blood or urine to assess exposure levels as collecting these matrixes is common practice in medical facilities and they are convenient and easy to obtain. However, if they are collected at one point in time only they will also only reflect current exposures.

Nevertheless, the availability of large prospective studies, in addition to prenatal and early life studies – which collect samples and assessed exposure prior to outcome (here, ASD/ADHD diagnosis and/or traits) – ameliorate concerns with timing of exposure, demonstrate the etiological relevance of heavy metal exposure prior to disease onset, and support the associations observed in case-control and cross-sectional studies as being causal. Accordingly, studies that examined biomarkers reflecting more recent or current exposures should not be disregarded, and they are valuable in understanding the general trend of the associations across the literature, particularly in light of prospective, prenatal and early life data.

1. Lead

Lead has been identified by the WHO as one of ten chemicals of major public health concern requiring action by the Member States in order to protect the health of workers, children and women of reproductive age (WHO 2017). In the 20th century children in the United States have been exposed to lead through lead-based paints, water from pipes used for plumbing, and leaded gasoline at relatively high levels (Hubbs-Tait et al., 2005). When lead was removed from gasoline, soldered cans, paint, and other sources, the average blood lead levels in the U.S. population and especially in children declined considerably between the late 1970s to the early 1990s (Pirkle et al. 1994). Nevertheless, lead still contaminates water and soil, and may be found in the dust of homes from remaining lead paint (Stretesky & Lynch, 2001, Frank et al. 2019). At high blood lead levels (> 10 μg/dL) experimental and human data show persistent and deleterious effects on brain function, including lowered intelligence, behavioral problems, and diminished school performance (Baghurst et al. 1992; Bellinger et al. 1992; Cory-Slechta 1997; Dietrich et al. 1993; National Research Council 1993; Needleman & Gatsonis 1990; Pocock et al. 1994; Rice 1993; Lanphear et al 2005) based on numerous cross-sectional and prospective studies (Bellinger et al. 1987; Centers for Disease Control and Prevention (CDC) 1991; World Health Organization (WHO) 1995). As the evidence has been growing that even low-level lead exposure can affect neurocognitive development adversely and it appears that there is no safe blood-lead threshold, the CDC has progressively reduced the recommended blood lead level of

concern from 30 µg/dL in 1975, to 25 µg/dL in 1985, 10 µg/dL in 1991 and currently 5 µg/dL (CDC, 2012). Neurotoxicity of lead in children at high doses (blood lead level greater than 30 µg/dl) has been an undisputed fact for more than 40 years (CDC 1978) and as research suggests a dose response model for neurotoxic effects that has no threshold, the WHO and CDC recently agree that no level of lead in blood is safe.[3]

2. Mercury

It has been known for centuries that mercury (Hg) is neurotoxic and that it affects human behavior and mood (Clarkson & Magos 2006); specifically, the toxicity of organic methylmercury (MeHg) due to occupational exposures has long been established (see also: https://www.cdc.gov/niosh/topics/mercury/). The most important source for organic (methyl)mercury is the consumption of contaminated predator fish (Aschner et al. 2010). Metallic Hg can be released from dental amalgams into surrounding tissues. Skin creams, infant teething powders, and contaminated food are additional known sources of inorganic Hg exposure (Casarett, 2001, Counter & Buchanan, 2004). The Minamata (Japan) contamination of seafood from an industrial source made it very clear that infants exposed prenatally suffer serious neurological damage (mental retardation, cerebral palsy, deafness, blindness, and dysarthria) and at exposure levels that left the exposed mothers unaffected (Harada et al. 1995). More recently, epidemiological studies have documented many more subtle adverse effects on brain functions at lower levels of methylmercury with neuropsychological function found to be disturbed in the domains of language, attention, and memory, as well as in visuospatial and motor function (Debes et al., 2006; Grandjean et al., 1997; 2011; Murata et al., 2004; Choi et al. 2008). The National Research Council reviewed literature on animal and human studies about 20 years ago and concluded that neurodevelopmental deficits are the most sensitive, well-documented effects of mercury exposure (NRC 2000).

[3] CDC 2021, https://www.cdc.gov/nceh/lead/prevention/default.htm; WHO 2021 https://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health.

3. Arsenic

Arsenic is a metal in the earth's crust and a contaminant in many metal ores (Gochfeld, 1995). It is used in metallurgy, semiconductor, pigments and glass manufacturing industries (Hathaway et al., 1991). Some arsenic compounds were formerly used as medications (e.g. Salvarsan). Large populations living in Taiwan, Chile, Mexico, India and Pakistan have been chronically exposed and some even poisoned from naturally occurring arsenic in ground water (ATSDR, 1990, Subramanian & Kosnett, 1998, Ahsan et al., 2000). In addition, environmental arsenic exposures are attributed to the burning of fossil fuels and mining and its use in fertilizers, wood preservatives, insecticides and herbicides (Gundert-Remy 2015). In the USA, more than 2 Million residents are drinking well water considered high in arsenic (USGS 2021). In the past decade, multiple studies have detected arsenic in foods and beverages including juice, rice, milk, broth (beef and chicken), and others (Wilson 2015).

Arsenic compounds occur in various chemical states, including *trivalent, pentavalent,* and *organoarsenical* compounds (Gochfeld, 1995). Trivalent arsenic undergoes methylation to form less toxic compounds, monomethylarsenate (MMA) and dimethylarsenate (DMA), that are excreted in the urine (Johnson and Farmer, 1991); some inorganic arsenic is excreted in the urine unchanged (Hopenhayn-Rich et al., 1993). Since arsenic is cleared rapidly from the blood, blood levels may become unmeasurable when urine levels remain elevated (ATSDR, 1990). Within a month after exposure cessation, arsenic is found mainly in the skin, hair, and nails; lesser amounts are found in teeth and bone (ATSDR, 1990).

Chronic effects of arsenic include skin lesions, neurotoxicity (including developmental neurotoxicity), cardiovascular diseases, diabetes and cancer; with cancer having been of highest concern for regulating bodies thus far. Gundert-Remy et al. (2015) recently remarked that a comprehensive study on dietary exposure to inorganic arsenic in the European population gives reasons for concern (EFSA 2009), that is the body doses that European populations reach through food consumption seem to not leave a margin for exposure levels such that the benchmark dose for an increased risk of carcinogenesis may be reached.

VII. Metals and ASD

As is the case for all neurodevelopmental disorders, timing of exposure is likely as important as dose levels, thus getting the timing right for exposure assessment is crucial in order to draw valid conclusions from comparisons. Most studies collect blood or urine to assess exposure levels, or children's hair with very few studies having collected nails or baby teeth to capture longer term exposures.

That is why a study by Arora et al (2017) is particularly notable as it not only used shed baby teeth but also a measurement method that allowed the authors to assess the incremental microstructure of the tooth through which they gained important temporal information on the timing of exposure. Thus, I consider this study particularly valid in terms of exposure assessment among children with ASD as it captured the relevant time of exposure. and it greatly influences my conclusions about metals and autism development. Other studies that are addressing the temporal sequence of events and capture the appropriate time of exposure are studies that collected blood or other bio-samples prior to disease diagnosis. This includes two Korean cohort studies, the first a large study (Kim et al. 2016) that measured blood lead 4-5 years prior to assessing autistic behaviors, and another longitudinal birth cohort that measured blood mercury levels measured early and late in pregnancy, in cord blood, and at 2 and 3 years of age and assessed autistic behaviors at 5 years of age (Ryu et al 2017). Furthermore, the studies by Grandjean et al. (2014; 1997; 2012; Budtz-Jorgensen et al., 2007) in Faroese children and those of children living in the Seychelles (Wijngaarden 2013; Strain 2015) assessed pregnancy and early childhood mercury exposures and autistic behaviors. Finally, a study of mercury exposure from maternal dental amalgams that were present during pregnancy among babies born and diagnosed with either DSM-IV autism (severe) or ASD (mild) by age 6 (Geier 2009).
Another important issue concerning measures in matrices other than blood is that these metals have to be taken up by or excreted into the respective tissues/compartments. This involves some complex transport or metabolic mechanism from one tissue (blood) to others such as hair, nails, or into the urine. It has been noted that metal levels in urine and hair depend on such

mechanisms (metal ion excretion and metabolism) and alterations of these very mechanisms have been observed in autistic children and are thought to possibly be a characteristic of the disease such as altered thiol metabolism that may influence the ability of autistic children to excrete heavy metals. For example, Obrenovich (2011) found that more ASD children under 6 years of age compared with age-matched controls had abnormal markers of thiol metabolism and alterations in the deposition of several heavy metal species in hair samples, particularly arsenic, mercury, copper, and iron; this led them to suggest that children with ASD may have trouble excreting thiol-toxic heavy metal species and that this could lead to contradictory findings for metal content measured in these tissues when comparing levels in cases to normal control children. Therefore, studies that use these matrixes have to be carefully evaluated when making causal inferences especially if they do not support the results from blood-based biomarkers.

In the following, I will describe the results from epidemiologic literature on ASD and ADHD and prenatal and childhood heavy metal exposures for each metal of interest separately starting with the most important studies published to date. Later, I will integrate these studies' results into an overall assessment of the causality of the observed findings by metal and outcome.

A. Lead *and* ASD

A case-control study of autism nested within the Historic Birth Cohort at Statens Serum Institute in Denmark is particularly noteworthy in terms of accuracy of timing of exposure and estimating exposure levels during the most important and sensitive neurodevelopmental periods for ASD (Long et al. 2019). This study accessed stored amniotic fluid to determine prenatal lead levels to assess contributions to autism development. Specifically, from among Danish children born between 1995 and 1999 with stored amniotic fluid samples, the authors selected a total of 37 case children clinically diagnosed with ASD and a matched set of 50 controls (matched on child age, gender, and maternal age) for analyses. They measured concentration of a number of metals (Fe, Cu, Zn, Se, Cr, Mn, As, Cd, Pb) in the amniotic fluid via inductively coupled plasma mass spectrometry. For two metals, lead and arsenic, they found suggestive increases in estimated effects for ASD when comparing cases to controls; i.e. for Pb they estimated an OR of

1.30 (95% CI: 0.66–2.58) per 1 mg/L (results for AS see below). Due to the small sample size of this study, however, the results did not reach formal statistical significance even though they suggest that prenatal lead exposure contributes to autism risk.

The second study that was able to assess not only pregnancy but also early childhood lead levels accurately is the study conducted by Arora et al (2017) using shed baby teeth. These authors not only employed a validated tooth-matrix biomarker to analyze the baby teeth but conducted this study within the Swedish twin registry study relying on 32 complete twin pairs and 12 individuals from twin pairs whose sibling did not donate a tooth. This study found statistically significant divergences in metal uptake between ASD cases and control twins during discrete early developmental periods in pregnancy and infancy (first year of life). The autism cases had a higher uptake of the neurotoxin lead, and both manganese and lead were also correlated with ASD severity and autistic traits. The authors concluded that their results indicate not only that there are specific developmental windows during which lead and arsenic exposure increase ASD risk and severity but also that their findings support the hypothesis of a systemic elemental dysregulation in ASD. Two additional advantages of this twin registry-based study is that most other studies of ASD recruited patient series rather than enrolling participants from a population, i.e., representing population level exposures in cases and non-cases. And finally, the study's reliance on twins allowed them to control for genetic factor contributions to ASD which might be important in order to be able to validly assess the role of environmental factors in the etiology of ASD. This is because family members share a large percentage of genetic variation and contrasting discordant monozygotic twins – as the authors state: "is a powerful strategy for uncovering disease-associated environmental factors independent of underlying genomic sequence variation" (see also Bell, 2011). This study result contrasts with an earlier study conducted by Abdullah et al (2012) that evaluated shed baby teeth in 22 ASD cases and 22 normal control children enrolled in a US nationwide study. These authors performed laser ablation analyses of prenatal and postnatal enamel and found no differences in prenatal and early postnatal levels of lead, mercury, and manganese between the groups. However, the authors

commented that the study does not rule out the possibility of adverse effects from exposure to neurotoxicants beyond the early postnatal developmental period. Importantly, this is a small study and the authors also were not able to address genetic risk factors that may have impacted how metal exposures contributed to concentrations of these metals in the teeth or the susceptibility of children who developed ASD to these exposures.

The Arora et al (2017) investigative team more recently conducted a similar study of baby teeth from ASD patients recruited at a nationally recognized multispecialty ASD clinic who lived in one of eight US states or Canada (27 ASD cases and 7 typically developing control children). This study's goal was to investigate underlying biological mechanisms by which metals may influence neurodevelopment further with a special focus on the dysregulation of bioenergetics (Frye, 2020). Here, the investigators found that exposure to toxic metals in general and differences in nutritional (or essential) metal exposures were associated with a dysregulation of cellular bioenergetics in autistic children in line with their hypothesis that the bioenergetic mechanisms such as mitochondrial respiration and glycolysis are disrupted in ASD; specifically for lead, they observed that increased prenatal lead concentrations were related to a decreased glycolytic rate in autistic children that had experienced neurodevelopmental regression.

Finally, there are 2 cohort studies that deserve special mention and are not included in the systematic reviews or meta-analytical studies discussed below. Of note, these studies used not only a different design but also a different outcome measure. They are nevertheless significant contributions to the literature as they are uniquely able to accurately assess the timing of exposure i.e. the sample analyses all preceded the measurement of outcomes. One is the large Korean study by Kim et al. (2016) that measured blood lead 4-5 years in 7-8 year old children prior to assessing autistic behaviors 4-5 years later in 2473 Korean children with no prior history of developmental disorders. The children were assessed at follow-up when they were 11–12 years of age for autistic behaviors using two scales (Autism Spectrum Screening Questionnaire (ASSQ) and Social Responsiveness Scale (SRS)). The investigators found that even at the low blood lead concentrations recorded for these at 7–8 -year-old children (geometric mean: 1.64

mg/dL), there were more autistic behaviors at 11–12 years of age on both scales (ASSQ (b = 0.151; 95% CI: 0.061, 0.242) and SRS (b = 2.489; 95% CI: 1.378, 3.600)). They also reported associations between blood lead concentrations and social awareness, cognition, communication, motivation, and mannerisms. The authors concluded that even low blood lead concentrations at 7–8 years of age are associated with more autistic behaviors at 11–12 years of age. This study is particularly important as it establishes temporality between exposures and outcomes and has a large enough size to allow investigating associations with relatively low blood lead levels.

A second much smaller cohort relied on 371 participants of the New Hampshire Birth Cohort and measured As, Cu, Mn, Pb, Se, Zn in maternal toenail at 27 weeks' gestation (reflecting periconception/early pregnancy) and at 4 weeks postpartum (reflecting mid-pregnancy exposures) and also in infant toenails at 6 weeks (reflecting late pregnancy/ early neonatal exposure) (Doherty, 2020). At 3 years of age children were tested with the social responsiveness scale (SRS; an outcome specific to ASD). This study did not find differences for maternal or child toenail content for lead and the child's SRS-2 score. (However, they reported higher As and Se in infant toenails for the highest SRS scores (greater deficits) and children with low As and high Se had the lowest SRS score; see below). There are few studies of toenail metal content and autism in general and this study did not evaluate exposures beyond the first month of life or outcomes after age 3.

In the following I will report on the results presented in meta-analyses of ASD. The first, by Saghazadeh and Rezaei (2017) analyzed lead, arsenic, and mercury (see below for arsenic and mercury results) and a few other metals and selecting for lead 48 studies that compared patient and control whole blood, plasma, serum, red cells, hair and urine measurements and were published through Oct 2016. ASD patients were found to have higher meta-analytical estimates of erythrocyte lead levels (standardized mean difference (SMD) =1.55, CI: 0.2-2.89; summary estimate of 3 studies) and of blood lead levels (SMD =0.43, CI: 0.02-0.85; summary estimate of 6 studies). Also, in 20 studies that measured hair lead, they found lead levels to be higher in case than control children overall, but this result was attributable to concentrations of lead in the hair

of children from developing (SMD =1.58, CI: 0.80-2.36) but not developed countries. No meta-analytically statistically significant difference was seen for urine lead measures reported in 5 studies. As mentioned above, reduced hair and urine metal (including lead) levels in autistic children compared with controls have been interpreted as an inability or reduced ability of at least some children with ASD to move the metal from one compartment into another, i.e., excrete the metal and remove it from their body. There may also be larger measurement error opportunities for these matrixes (from which location on the scalp and how much hair is taken and whether samples are contaminated by dust, when urine was collected and whether or not the measures were creatinine adjusted etc) that would make it harder to find differences between groups especially when the sample size is small (less than 100). Examples of studies in which lead urine levels were found to be lower include a Turkish study of 3-12 year olds (30 children with autism, 20 healthy controls) (Yorbik 2009), a Malaysian study of children ages 3-6 years (81 ASD, 74 typically developed) (Wahil 2021), and a Spanish study of 7 year old children (35 ASD, 34 typically developing children) (Fuentes-Albero 2015).

Another recent meta-analysis by Guo et al. (2019) concentrated solely on hair lead measures and summarized the data from 20 eligible studies out of 42 citations between 1982 and 2017. The studies that met the authors' criteria of providing the data needed for this meta-analysis (mean lead levels, effect sizes, p-values comparing cases to controls) and not being an extreme outlier in terms of the effect size included a total of 941 autistic children and 846 healthy subjects, enrolled in 7 North American (Adams et al., 2006; Kern et al., 2007; Marlowe et al., 1984; Massaro et al., 1983; Obrenovich et al., 2011; Shearer et al., 1982; Wecker et al., 1985), 6 Middle Eastern (Al-Ayadhi, 2005; Al-Farsi et al., 2013; Blaurock-Busch et al., 2011; Elsheshtawy et al., 2011; Fido and Al-Saad, 2005; Mohamed Fel et al., 2015), 4 European (De Palma et al., 2012; Domingues et al., 2016; Skalny et al., 2017a, 2017b), and 3 Asian studies ((Jung et al., 2008; Lakshmi Priya and Geetha, 2011; Yasuda et al., 2005). Overall, this meta-analysis did not find formally statistically significant differences in the levels of hair lead in autistic compared with control individuals (Hedges's g=0.251; 95% CI: −0.121, 0.623; P=0.187).

However, they observed marked between-study heterogeneity ($I2 = 92.759\%$; $\tau 2 = 0.657$; $P < 0.001$), that was partially due to the varying sample sizes, the gender of the participating children, but not to differences in sampling location on the scalp, the analytic-chemistry method used to assess lead, or the country or continent where the study was performed. A sensitivity analysis indicated that the overall pooled estimate became statistically significantly and positively associated with hair lead levels when one influential study (Yasuda et al., 2005) that had the biggest sample size (n=256) and the smallest effect size was removed. This Japanese study by Yasuda et al. (2005) measured a whole panel of metals and minerals in children's hair and concluded that autistic children suffered from a mineral imbalance that contribute to lower levels of both essential and toxic metals in their hair compared with control children. The authors of this Japanese study also found a marked accumulation of some metals including lead, mercury, and cadmium in some autistic individuals, and suggested that 'autistic children may be classified into subgroups' i.e., that metals may contribute to autism in a subgroup of children who are sensitive or vulnerable to the toxic effects of metals.

Finally, a review and a meta-analysis of lead and inorganic arsenic (see below for results) from ASD case control and cross-sectional studies was published by Wang et al (2019). These authors reviewed all articles that included mean levels of lead measured in baby teeth, blood, urine, or hair – a total of 37 papers – but included only 17 in their meta-analysis as they fulfilled their criteria for in/exclusion. This meta-analysis reported statistically significantly increased overall levels of lead in cases blood and hair but not urine. The hair-based meta-analysis included only 4 studies with a total of 214 cases and 262 controls and even for these few studies the between-study heterogeneity was large. Nevertheless, when studies with extreme values were excluded they did not see a change in the observed differences between cases and controls.

Apart from the individual studies evaluated and included in the above cited meta-analyses, additional studies of lead and ASD were published after in these systematic reviews or meta-analyses were conducted and I will briefly summarize the results in the following.

A Polish study collected hair samples from 30 children diagnosed with ASD and 30

children randomly selected from the general population of the region (Filon 2020). This case control study found that the mean Pb (and As, see below) concentrations in the hair of children with ASD were statistically significantly higher than the mean concentrations in the hair of children without neurological disorders (Pb Mean ± SD: 6.028 ± 0.69 vs. 3.415 ± 1.207). A cross-sectional study of 48 ASD subjects in Italy assessed the severity of autism symptoms and cognitive levels (Fiori 2020) and reported positive correlations between hair metal burden (lead, aluminum, arsenic and cadmium levels) and severity of ASD symptoms (social communication deficits and repetitive, restrictive behaviors). Lead, molybdenum and manganese hair levels were also found to be inversely correlated with cognitive level (full intelligence quotient) in ASD individuals. A Chinese study (Qin et al 2018) compared blood plasma metals in 34 children with autism spectrum disorder (ASD) with those in 38 unaffected children from a large public kindergarten in Shenzhen; they found that children with ASD had higher average Pb levels than unaffected children (ASD: 31.9 μg/L vs. 18.6 μg/L), Hg (3.83 vs. 1.09 μg/L), and Cd (0.70 vs. 0.26 μg/L). Another Chinese study (Li et al. 2018), however, did not find a difference in lead levels in 180 cases recruited from the outpatients of a Children's Hospital (Zhejiang University School of Medicine) and from several special autism-training schools compared with 184 unrelated healthy controls recruited from outpatients of the hospital (however, they reported significantly higher level of blood arsenic (mean ± SD, 12.16 ± 13.73 vs 4.11 ± 8.02 μg/L) and mercury (mean ± SD, 55.59 ± 52.56 vs. 13.47 ± 17.24 μg/L) in the children with ASD compared with healthy controls). Finally, an Italian study (Fiore et al 2020) enrolled 48 ASD subjects ages 2 to 17 and, measuring metal content in hair, they reported a positive correlation (statistically significant) with severity of ASD symptoms and lead as well as an inverse correlation between lead and cognitive level in ASD individuals.

Most recently, a large case control study nested within a pregnancy cohort was conducted based on the Norwegian Mother, Father and Child Cohort Study (MoBa) that included 705 ADHD cases, 397 ASD cases and 1034 unaffected controls (Skogheim et al 2021). Cases were identified through linkage with the Norwegian Patient Registry and maternal concentrations of

11 metals/elements were measured in blood at week 17 of gestation including lead, total arsenic, and total mercury. For ASD and lead in mid-pregnancy they described a non-linear (U-shaped) association and increased risk at lower and higher levels of lead.

The available epidemiological literature demonstrates a consistent association between lead exposure and the development of childhood ASD. I will now turn to the Bradford Hill criteria to make an assessment of causality.

Bradford-Hill Criteria Evaluation

Strength (effect size). Most studies did not estimate effect sizes but reported mean differences in lead levels between cases and controls; however, it is remarkable that such differences were seen even at relatively low mean blood lead concentrations (see Kim et al. 2016; the large Korean cohort study). [Note, the size of an estimated effect (OR, RR) for metal biomarkers on an outcome will depend on the scale of the measure i.e. the unit used to estimate a per unit increase in risk]

Dose Response. This is met as there are larger differences reported at higher lead levels in several studies, i.e., estimates from developing countries and during earlier periods in time when lead exposure was higher. This can be considered a stronger endorsement of a causal relation; it is further supported by the dose response relationship (biological gradient such that risk increases with dose - another Bradford Hill criterion) that some studies reported with increasing lead levels (also note: a small association does not mean that there is not a causal effect, though the larger the association, the more likely that it is unbiased and thus causal).

Consistency. This criterion is met since positive associations have been reported for different populations and in different geographic regions and different time periods as well as different biologic matrices which strengthens the likelihood of a true effect.

Temporality. That disease occurred after exposure and that there is an expected delay between the cause and effect has also been reported, i.e., exposures were assessed and recorded for early infancy in baby teeth and the Korean child cohort study and the Norwegian MoBa cohort.

Specificity. This asks whether one specific exposure causes one specific outcome. Exposure to lead is associated with a host of neurodevelopmental disorders. Accordingly, this criterion is inapplicable.

Experimental evidence. This is not applicable for neurotoxin exposures in humans.

Analogy. Other neurotoxic metals have also been shown to cause autism and related neurodevelopmental abnormalities, such as the well accepted neurotoxic effects of lead on cognitive development and IQ in children.

Coherence. See below for examples from animal and cell experimental studies of mechanisms.

Biological plausibility. There is strong biological plausibility for lead causing ASD. The brain regions most vulnerable and affected by lead exposure are the frontal cortex, nucleus accumbens, basal ganglia and dorsal striatum, and hippocampus with dopaminergic and glutamatergic changes in these regions that vary by dose, age, and duration of exposure as suggested by autoradiography receptor binding studies (Cory-Slechta, 1997; Cory-Slechta et al., 1993; 1997; 1998; Jett and Guilarte, 1995; Listos et al., 2013). Specific core regions have been suggested to mediate clinical phenotypes of ASD such as the frontotemporal lobe, frontoparietal cortex, amygdala, hippocampus, basal ganglia, and anterior cingulate cortex (ACC) [Amaral 2008]. Also, lead accumulates in mitochondria leading to oxidative stress and degradation of energy metabolism (Lidsky & Schneider, 2003; Silbergeld et al., 1980) Lead depletion of mitochondria-generated ATP energy metabolism also contributes to disruption of neuronal function (Rafalowska et al., 1996) and the depletion of mitochondrial calcium stores is implicated in lead-induced apoptosis (Kapoor & van Rossum, 1984) and excitotoxicity (Beal et al., 1993; Lidsky & Schneider, 2003). Glutamate excitotoxicity is being considered as having a leading role as a biochemical mechanism of aberrant neuroplasticity accompanied by oxidative stress and mitochondrial dysfunction in ASD (Anashkina 2021). The action of Pb on glutamate release affect synaptic plasticity and contribute to impairments in hippocampus-mediated learning and memory functions (White et al. 2007). Finally, lead affects oligodendroglia that are

believed to be the most lead-sensitive cell type in the brain (Tiffany-Castiglioni, 1993; Tiffany-Castiglioni et al., 1986). Oligodendrocytes are a dominant cell type of white matter, and early white matter differences in ASD brains are thought to possibly explain the brain being connected atypically [Casanova 2004]. These cells are responsible for the production and maintenance of a protective myelin sheath that insulates axons (Tiffany-Castiglioni, 1993; Tiffany-Castiglioni et al., 1986). Lead can cause either hypomyelination or demyelination depending on concentration (Goyer, 1993; Krigman et al, 1974; Tennekoon et al., 1979; Toews et al., 1983) and damages myelin through the delay of the differentiation of oligodendroglia and impaired axonal growth and survival (Deng et al., 2001; Laterra et al., 1992; Tennekoon et al., 1979). Also, while oligodendrocyte progenitors are vulnerable to low levels of lead exposure, mature oligodendroglia are relatively resistant, suggesting a complex interaction (Deng et al., 2001).

Conclusion: Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to lead during sensitive developmental periods in early childhood can cause ASD even at relatively low concentrations.

B. Arsenic *and* ASD

As mentioned above, the Historic Birth Cohort at Statens Serum Institute in Denmark analyzed amniotic fluid samples from 37 ASD cases and 50 control children (frequency-matched on child age, gender, and maternal age) born between 1995 and 1999 and measured multiple metal concentrations (Fe, Cu, Zn, Se, Cr, Mn, As, Cd, Pb) (Long, 2019). The authors reported large individual effect sizes for 1 mg/L increase in As with an OR of 1.50 (95% CI: 0.92–2.42) that failed to meet formal statistical significance but suggested adverse effects of prenatal AS exposure on ASD development.

In a cohort with 371 participants in the New Hampshire Birth Cohort researchers measured As, Cu, Mn, Pb, Se, Zn in maternal toenail at 27 weeks' gestation (reflecting periconception/early pregnancy) and at 4 weeks postpartum (reflecting mid-pregnancy exposures) and also in infant toenail at 6 weeks (reflecting late pregnancy/ early neonatal exposure) (Doherty, 2020). At 3 years of age children the children were tested with the social

responsiveness scale (SRS; an outcome specific to ASD). This study reported differences for maternal or child toenail content for high As and Se in infant toenails the highest SRS scores (greater deficits) and children with low As and high Se the lowest SRS score accounting for time-varying exposure, co-exposure mixtures, exposure interactions, and nonlinear associations.

The meta-analysis by Saghazadeh and Rezaei (2017) mentioned above identified 15 studies reporting on arsenic concentrations in most in hair samples (Skalny et al., 2016b, c; De Palma et al., 2012; Obrenovich et al., 2011; Blaurock-Busch et al., 2011; Jung et al., 2008; Adams et al., 2006; Al-Ayadhi, 2005; Kern et al., 2007; Fido et al., 2002) but also in erythrocytes (Adams et al., 2013), blood (Skalny et al., 2016a; Vergani et al., 2011; Adams et al., 2013), and urine (Blaurock-Busch et al., 2011; Adams et al., 2013; Metwally et al., 2015) for patients with ASD and control subjects. This meta-analysis found no difference in blood, urine, or hair arsenic concentrations between ASD and control subjects overall. For arsenic measurements of hair, the summary effect for 4 studies from developing countries with a total of 158 cases and 167 controls was marginally statistically significant.

A later meta-analysis conducted by Wang et al (2019), reviewed 14 reports that measured inorganic arsenic (iAS) in children ages 1 to 16 years. A total of 6 case-control and 3 cross-sectional studies compared body burdens of iAs measured in hair from ASD cases and normally developing children while 3 additional case controls studies relied on blood iAs concentrations. Meta-analytical results based on 4 studies that qualified for such an analysis found formally statistically significantly increased mean blood and hair inorganic arsenic in case compared to control children (As blood mean (+-SD) 1.95 (1.49) vs. 0.37 (0.05) $p<0.001$; arsenic hair mean (+-SD) 0.52 (0.42) vs. 0.10 (0.05) $p<0.001$).

In addition to the studies evaluated for and included in this meta-analysis another small case control study – already reviewed above for Pb - has been published that measured As and Pb in hair samples of 30 Polish children diagnosed with ASD and 30 children randomly selected from the general population of the region (Filon 2020). The mean arsenic concentration in the hair of children with ASD was statistically significantly higher than the mean concentration of

this element in the hair of children without neurological disorders (AS: Mean± SD 0.216 ± 0.09 vs. 0.061 ± 0.03). The Italian study by Fiore et al (2020) measured arsenic content in hair samples of 48 ASD subjects ages 2 to 17, but did not find any correlation with severity of ASD symptoms. Finally, an Indian study also analyzed lead in nails of low, medium and high functioning autistic children (15 in each group) and normal controls (N=50) (Priya and Geetha 2011). The level of lead were statistically significantly higher in the nail samples of autistic children when compared with control group and the mean lead levels decreased strongly from low functioning to high functioning autistic children to controls.

An ecologic study using data on 4,486 children with ASD residing in 2,489 census tracts in five sites of the Centers for Disease Control and Prevention's Autism and Developmental Disabilities Monitoring (ADDM) Network, investigated ambient arsenic concentrations measured by the US Environmental Protection Agency National-Scale Air Toxics Assessment (Dickerson et al. 2016). Adjusted for confounding factors, this study did not find an association between modelled census tract air concentrations of arsenic and ASD risk.

The recently published Norwegian case control study nested within the MoBa pregnancy cohort enrolled 705 ADHD cases, 397 ASD cases and 1034 unaffected controls (Skogheim et al 2021). As described above, the study measured maternal concentrations of total arsenic in blood at week 17 of gestation in stored blood samples from the pregnancy cohort. They reported a non-linear increased risk for ASD with prenatal exposure to mainly organic arsenic in the present study as arsenic in this population originated mainly from fish and seafood consumption of the participating pregnant women. This is an important finding as the exposure was clearly measured prior to the outcome in a large sample. Also, the MoBa study is of high validity in terms of outcome assessment as the autism diagnoses are retrieved from national disease registers and the population of Norway has universally access to health care.

The available epidemiological literature demonstrates a consistent association between arsenic exposure and the development of childhood ASD. I will now turn to the Bradford Hill criteria to make an assessment of causality.

Bradford-Hill criteria evaluation

Strength. This is partially met since the overall meta-analytical (point) effect estimates reported reflecting a weak to moderate size differences and the dose response relation-ship was non-linear at low levels of exposure in the MoBa study.

Consistency. This criterion is met since positive associations have been reported for different populations, in different places and time periods which strengthens the likelihood of a true effect.

Temporality. In the nested case control study (MoBa) and the cohort study (New Hampshire) disease occurred after exposure i.e. there is a delay between the cause and effect supporting causal inference.

Specificity. This asks whether one specific exposure causes one specific outcome. Again, as explained above with respect to lead, arsenic is associated with a host of adverse neurodevelopmental outcomes. Accordingly, this is inapplicable with these datasets.

Experimental evidence. This is not applicable for neurotoxin exposures in humans.

Analogy. Other neurotoxic metals have been shown to cause autism and related neurodevelopmental abnormalities, such as the well accepted neurotoxic effects of lead on cognitive development and IQ in children. Arsenic has also been implicated in causing more general neurodevelopmental delays and defects.

Coherence. See below for example from animal and cell experimental studies of mechanisms.

Biological plausibility. Effects of arsenic on the developing brain resulting in developmental neurotoxicity have been consistently shown in experimental studies in animals and cells. Also, epidemiological studies previously indicated that early life arsenic exposure is associated with deficits in intelligence and memory (Tolins et al. 2014). A number of different mechanisms have been reported that explain arsenic neurotoxicity and lend biological plausibility to its impact on the developing the central nervous system. Specifically, animal experiments provided evidence for oxidative stress and free radical production resulting in

neuronal death (Dwivedi et al 2011, Yen 2011), impaired hippocampal neurogenesis (Liu et al. 2012), dysregulation of the hypothalamo-pituitary-adrenal axis including reduced levels of corticosterone receptors in the hippocampus (Goggin 2012, Martinez-Finley 2009), epigenetic effects such as reduced DNA methylation in the hippocampus and frontal cortex (Martinez, 2011), reductions in brain levels of biogenic amines and other neurotransmitters (Rodriguez, et al. 2010, Zhang et al 2013, Xi et al. 2010, Liu, et al. 2013), changes in the expression of the NMDA receptor complex (Luo 2012), inhibition of neurite outgrowth (Aung 2013), structural malformation of white matter (e.g., myelin sheaths) (Zarazua et al 2010) and of hippocampal mossy fibers (Jing 2012), and endocrine disruption, including down-regulation of thyroid hormone receptor genes (Wang et al. 2013, Lindberg 2008). Animal studies also suggested that postnatal low-concentration arsenic exposure induces autism-like behavior and affects frontal cortex neurogenesis in rats (Zhou et al. 2018).

Conclusion: Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to arsenic during sensitive developmental periods in early childhood can cause ASD.

C. Mercury *and* ASD

Some particularly notable contributions to the epidemiologic literature on general child neurodevelopment stem from longitudinal cohort studies of mercury exposure from seafood in two populations that also caused an early controversy about the role of mercury on neurodevelopment. Specifically, the studies of pregnancy and early childhood mercury exposures first conducted among Faroese children (Grandjean et al. 2014; 1997; 2012; Budtz-Jorgensen et al., 2007) that assessed verbal and motor development in 7- and 14-year-olds. Secondly, a group of studies conducted in a fish eating population living in the Seychelles that documented general psychomotor development and also autistic behavior scores (Wijngaarden 2013; Strain 2015). These studies at first came to differing conclusions regarding associations between prenatal and early childhood organic mercury exposure due to fish and whale meat consumption and autistic behaviors or more general motor and verbal development. The Faroes studies found positive

associations with increasing prenatal mercury exposure. The studies conducted in the Seychelles did not find associations either for prenatal or early life exposures and autistic behavior scores and some analyses even suggested positive effects on child neurodevelopment (Wijngaarden 2013). However, data from these Seychelle studies also suggested that it may be the nutrients in fish, especially long-chained n-3 polyunsaturated fatty acids, that positively affected these outcomes and might have been confounding the results for mercury. This was later corroborated by the Seychelles Child Development Study Nutrition Cohort 2 that enrolled 1265 mother-child pairs during pregnancy and evaluated the children at 20 months of age using various neurodevelopmental scales (Strain et al 2015). While prenatal methylmercury exposure did not seem to affect neurodevelopmental outcomes on its own, the investigators found statistically significant interactions between methylmercury and poly-unsaturated fatty acids (PUFA) on the Psychomotor Developmental Index (PDI) of the Bayley Scales of Infant Development II (BSID-II). Increasing methylmercury was associated with lower (worse) PDI only in children of mothers with a higher n–6 to n–3 ratio (less optimal fatty acid composition), suggesting that maternal PUFA status may modify the associations of prenatal methylmercury exposure with psychomotor development, making it harder to evaluate risk from methylmercury exposure due to fish eating. On the other hand, in the Faroese birth cohort mercury exposure mainly originated from the ingestion of whale meat. In this cohort, mercury concentrations were assessed in maternal hair at parturition, and in cord blood, and child blood and hair at an age-7 clinical examination for more than 1000 children born in 1986–1987. Midwifes obtained information on the course of the pregnancy and nutritional habits, including the average number of fish dinners per week during pregnancy. About 90% of the cohort children participated in the follow-ups at 7 years of age (Grandjean et al. 1997) and 14 years of age (Debes et al. 2006), at which time neurobehavioral tests were administered by clinical professionals. Again, reported fish intake in pregnancy had a beneficial effect on all neurodevelopmental outcomes considered. Importantly, the previously reported mercury associations with these neurodevelopmental outcomes (Budtz-Jørgensen et al. 2002; Debes et al. 2006; Grandjean et al. 1997) became stronger for mercury

when the investigators controlled for fish consumption in pregnancy.

A more recently conducted longitudinal birth cohort enrolled 458 mother-child pairs of Korean origin recruited from 3 regions from 2006 to 2012 and measured total blood mercury concentrations early and late in pregnancy, in cord blood, and at 2 and 3 years of age and assessed autistic behaviors at 5 years of age (Ryu et al 2017). This study reported a doubling of blood mercury levels to be positively associated with autistic scores (social responsiveness score (SRS) known as a reliable ASD screening tool) in late pregnancy (β=1.84, 95% CI: 0.39, 3.29), in cord blood (β = 2.24, 95% CI: 0.22, 4.27), and at 2 years (β = 2.12, 95% CI: 0.54, 3.70) and 3 years (β = 2.80, 95% CI: 0.89, 4.72) of age. Also, the risk of receiving high scores was increased with increasing mercury levels in late pregnancy (relative risk [RR]=1.31, 95% CI: 1.08, 1.60) and in cord blood (RR = 1.28, 95% CI: 1.01, 1.63) and suggestively also at 2 years (RR = 1.20; 95% CI 0.6, 1.51) and at 3 years of age (RR = 1.26, 95% CI: 1.01, 1.57).

Finally, a study assessed mercury exposure from maternal dental amalgams that were present during pregnancy among babies born and diagnosed with either DSM-IV autism (severe, 40 children) or ASD (mild, 60 children) by age 6 (Geier et al, 2009). This study reported positive associations with the number of dental amalgams in pregnancy and severe versus mild autism i.e. the estimated adjusted ORs for up to 5 amalgams were 1.3 (not statically significant), for 6-7 amalgams the OR was 3.3 (borderline statistically significant, p=0.09), while the OR for 8+ amalgams was 4.4 (statistically significant, p=0.03). In a Seychelles cohort of 242 pregnant women recruited in 2001, researchers assessed maternal amalgam surfaces present during gestation and the child's mental (MDI) and psychomotor (PDI) developmental indices of the Bayley Scales of Infant Development-II (BSID-II) administered at 9 and 30 months. While the authors described their results for the number of maternal dental amalgam surfaces in pregnancy as 'not statistically significantly ($p > 0.05$)' associated with either PDI or MDI scores, they in fact saw a negative association with the number of amalgam (beta=-0.1; 95%CI -0.35, 0.02; p=0.075) for the mental development score at age 30 months and a smaller one at age 9 months.

The meta-analyses of mercury and ASD conducted by Saghazadeh and Rezaei (2017)

selected 38 studies published through Oct 2016 that contained mercury measurements in blood (including neonatal blood spots, plasma, serum), red blood cells (erythrocytes), hair, urine, nail, and teeth; however, they only performed meta-analysis of data from hair, blood, urine, and erythrocyte measures of mercury. They did not did not find meta-analytical differences in urinary mercury levels comparing patients with ASD and control subjects. In terms of blood measures, erythrocyte mercury concentrations were higher in patients with a summary effect size of 1.562 ($Z = 2.68$, $p = 0.007$) and in blood and plasma – but blood mercury levels did not remain statistically significant when a study considered an 'outlier' (Yassa, 2014) was dropped. For hair measurements of mercury, they evaluated 26 studies (1092 patients with ASD, 973 control subjects) and found no difference in hair mercury concentrations between patients and control subjects (effect size of 0.224; $Z = 0.44$, $p = 0.661$). When removing 4 studies considered outliers (Yassa, 2014; Elsheshtawy et al., 2011; Fido and Al-Saad, 2005; Hodgson et al., 2014) this did not change, however, in subgroup analyses, the meta-analytical summary estimate for ASD was statistically significantly higher in hair mercury concentrations in developing but not in developed countries.

A second meta-analysis of mercury and ASD relied on 44 studies that met the criteria for inclusion before June 2017 (Jafari 2017). Based on the meta-analytical estimates, the authors concluded that mercury level in whole blood (Hedges =0.43, 95% CI: 0.12, 0.74, P =0.007; 16 studies), red blood cells (Hedges =1.61, 95% CI: 0.83, 2.38, P<0.001; 5 studies), and brain (0.61 ng/g, 95% CI, 0.02, 1.19, P =0.043; 3 studies) but not in urine (1.8 mg/g creatinine 95%CI, -0.21, 1.46, p=0.141; 8 studies) were statistically significantly higher in ASD patients compared with healthy subjects. Also, in 23 studies mercury levels in hair were measured, but the meta-analysis did not suggest a difference in means (−0.63 mg/g, 95% CI: −0.21, 1.46, P =0.141); when the authors excluded 3 influential studies from the middle east the levels in ASD patients were slightly lower than in healthy subjects (−0.14 mg/g, 95% CI: −0.28, −0.01, P =0.039). Notwithstanding the inconsistency with respect to hair mercury levels and the lack of significant difference in urine mercury levels between ASD cases and controls, the authors concluded that

"results of the current meta-analysis revealed that mercury is an important causal factor in the etiology of ASD." Moreover, the authors emphasized the importance of considering multiple biological markers (including urine, blood and hair) in assessing causality. I agree that the available literature, and the strengths and limitations of the various biomarkers evaluated by different studies, should be considered in totality for purposes of understanding causal relationships, as I have done in this report.

Another author group (Sulaiman, et al. 2020) performed a meta-analysis for mercury and ASD that reviewed 27 case control and cross-sectional studies from 8 different countries and reported levels of Hg in hair, urine, and blood (13 hair, 4 urine, 10 blood) to be positively and statistically significantly associated with ASD in the meta-analysis. However, these authors included fewer studies than reviewed by Jafari et al (2017) above. They also referred to one of Hg in shed primary tooth enamel (digested whole tooth), which found a positive association between Hg levels and ASD that reported a mean level of 0.15 ppm in the 15 cases compared to 0.07 ppm in the 11 controls (Adams 2007), and another in which the mean level in cases compared to controls was lower (of 1.47 ppm vs. 1.9 ppm) but this difference was not statistically significant (Abdullah 2012).

There is one study in India that analyzed mercury in nails of low, medium and high functioning autistic children (15 in each group) and normal controls (N=50) (Priya and Geetha 2011). The mercury concentration was statistically significantly higher in the nail samples of autistic children when compared with control group and the mean levels decreased strongly from low functioning to high functioning autistic children and controls.

There are also two studies that did not employ biomarkers but rather a US EPA model for generating area level hazardous air pollutant (HAP) concentrations to explore possible associations between autism spectrum disorders (ASD). One was a conducted by Windham et al. (2006) and included 284 children with ASD and 657 controls, born in 1994 in the San Francisco Bay area and assigned exposure level by census tract of birth residence for 19 chemicals including lead, mercury and arsenic from the 1996 HAPs database. The investigators reported

50% increased risk in the top quartile of heavy metals (95% CIs, 1.1–2.1) and with simultaneously adjustment for solvent the risk for metals became stronger (AORs for metals: fourth quartile = 1.7; 95% CI, 1.0–3.0; third quartile = 1.95; 95% CI, 1.2–3.1). The individual compounds that contributed most to these associations included mercury, cadmium, and nickel. A second study by Dickerson et al (2016) using data on 4,486 children with ASD residing in 2489 census tracts in five sites of the Centers for Disease Control and Prevention's Autism and Developmental Disabilities Monitoring (ADDM) Network, investigated ambient lead, mercury, and arsenic concentrations measured by the US Environmental Protection Agency National-Scale Air Toxics Assessment. Adjusted for confounding factors, census tracts with air concentrations of lead in the highest quartile had significantly higher ASD prevalence than tracts with lead concentrations in the lowest quartile (prevalence ratio (PR) = 1.36; 95 '% CI: 1.18, 1.57). In addition, tracts with mercury concentrations above the 75th percentile (>1.7 ng/m3) and arsenic concentrations below the 75th percentile (≤0.13 ng/m3) had a significantly higher ASD prevalence (adjusted RR = 1.20; 95 % CI: 1.03, 1.40) compared to tracts with arsenic, lead, and mercury concentrations below the 75th percentile. The authors concluded that ambient lead concentrations increased ASD prevalence and multiple metal exposures may have synergistic effects on ASD prevalence.

The available epidemiological literature demonstrates a consistent association between mercury exposure and the development of childhood ASD. I will now turn to the Bradford Hill criteria to make an assessment of causality.

Bradford-Hill Criteria Evaluation

Strength (effect size). This criterion is met. The overall meta-analytical (point) effect estimates reported for blood are reflecting a weak to moderate difference in mercury concentrations depending on the populations studied. The Korean study by Ryu et al (2017) that used a repeated measured of pre- and postnatal total mercury described a dose response with autistic behavior scores suggesting a biological gradient. This is further supported by studies that found higher mercury concentrations in more severe autism cases (Geier et al, 2009). [Note, the

size of an estimated effect (OR, RR) for metal biomarkers on an outcome will depend on the scale of the measure, i.e., the unit used to estimate a per unit increase in risk]

Consistency. This criterion is met since positive associations have been reported for different populations and in different places and different time periods and different matrices.

Temporality. This asks whether disease occurred after exposure and that there is a delay between the cause and effect. This criterion is met by the Korean birth cohort study and also the Faroese study but the later assessed general psychomotor development.

Specificity. This asks whether one specific exposure causes one specific outcome. This criterion is not relevant here as mercury adversely affects the brain in many different ways and also affects other organ systems.

Experimental evidence. This is not applicable for neurotoxin exposures in humans.

Analogy. Other neurotoxic metals have been shown to cause autism and related neurodevelopmental abnormalities. Mercury has also been implicated in causing more general neurodevelopmental delays and defects. The Faroese study greatly corroborates that there are adverse effects of methylmercury exposures on child neurodevelopment.

Coherence. See below for example from animal and cell experimental studies of mechanisms.

Biological plausibility. ASD development has been linked to innate and humoral immune system disturbances in the brain and the blood of ASD children (Mead et al 2015, McDougle et al 2015, Samsam et al 2014, Bjorklund et al. 2016) including the presence of activated microglia and astrocytes, increased or abnormal T cell responses, increased production of pro-inflammatory cytokines, decreased production of anti-inflammatory cytokines, the presence of autoantibodies, enhanced or diminished innate natural killer (NK) cell function and modulated monocyte responses (Mead et al 2015, Noriega et al. 2014, Estes et al 2015). A normally functioning immune system is an essential element in the regulation of synapse formation, synaptic plasticity, neuronal plasticity and synaptic pruning all very relevant to ASD etiology (Mead et al 2015, Goyal et al 2014, Meltzer et al. 2017). The presence of chronic nitro-

oxidative stress in the form of lipid peroxidation, protein and DNA oxidation and depleted levels of reduced glutathione, often coupled with general defects of the glutathione system, and mitochondrial dysfunction in the blood and brain has also been noted in children with ASD (Hendren 2014, Rossignol et al 2014, Bilbo et al 2015). As reviewed in Morris et al (2018), exposure to organic forms of mercury such a methylmercury can generate immune abnormalities, mitochondrial dysfunction, and chronic nitro-oxidative stress all mechanisms that have been reported as contributing to ASD. This review discusses the mechanisms that are potentially involved in organic mercury's neurodevelopmental neurotoxicty including its effects on sulfhydryl groups of proteins, on pericytes and cerebral endothelial cells, its accumulation within astrocytes and microglial activation and activation of immune and inflammatory pathways; and impairing mitochondrial functioning that also induces chronic oxidative and nitrative stress. Finally, there are also (epi-)genetic factors which may increase susceptibility to the toxic effects of mercury in ASD individuals with males being more strongly affected by long-term neurotoxic effects of postnatal exposure and genetic polymorphisms in glutathione transferases and other glutathione-related genes and in selenoproteins or by polymorphisms in the HLA region, and by genes encoding cytokines and receptors. Additionally, a study in prairie voles reported that mercury can induce endocrine disruption (Soto et al. 2019) and ASD has also been associated with aberrations in steroid hormones such as androgens and estrogens (Baron-Cohen et al., 2015). Overall, it is biologically plausible that mercury exposure can increase the ASD risk by causing neuronal damage and connectivity loss via mercury-induced oxidative stress and neuroinflammation.

Conclusion: Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to mercury during sensitive developmental periods in early childhood can cause ASD.

D. Lead *and ADHD*

A seminal study was published in the New England Journal of Medicine in 1979 by Needleman et al. who collected shed teeth from thousands of grade school children and

described a positive association between teacher rated childhood behavior problems and dentine lead levels. The authors concluded that these child behavior problems occurred at blood lead levels lower than 30μg/dl and were attributable to the cumulative exposures across early childhood that the teeth represented. Subsequently, similar findings were reported in studies conducted in Europe, New Zealand, and the United States with either dentine or blood lead measures in children (Winneke 1983, Yule 1984, Hatzakis 1985, Silva, 1988; Thomson et al. 1989, Needleman 1990, Sciarillo 1992, Leviton 1993). Thus, higher blood lead levels (mean levels at or above 10 μg/dL) have been reliably associated with ADHD related behaviors (Burns et al., 1999; Silva, 1988; Thomson et al. 1989). An Australian study of 322 11-13-year-old children from a lead smelting community (Burns, 1999) was unique in that it assembled a cohort and periodically assessed children prospectively for lead exposures from birth until age 7 years. Their lifetime blood lead levels were relatively high (geometric means: boys, 14.3 μg/dl; girls, 13.9 μg/dl) and so were the estimated ORs for total problem scores with a 3.2 fold increased (95%CI 1.4-6.6) in boys and 2.8 (95%CI 1.0-6.8) in girls with an increase in lifetime blood concentration from 10 to 30 ng/dl.

Others studied extended periods of lead exposure in childhood relying instead on measures of dentine, whole-tooth, and hair lead, and associated increased lead levels with children's symptoms of inattention and behavior problems reported by parents and/or teachers (Bellinger et al. 1994; Fergusson et al. 1993; Tuthill 1996). For example, Bellinger et al. (1994) found that tooth lead levels at age 6 were positively associated with ADHD- type behavior problems reported by the teachers of 1782 for 8-year-old US children. Fergusson et al. (1993) reported that higher tooth lead levels at 6-8 years were associated with an increased prevalence of emotional and behavioral problems in more than a thousand New Zealand children at age 8 years and again at age 12-13 years. Both studies described linear dose response relations between the dentine lead and these child behavior problems. In a study of 277 Massachusetts first-graders children's hair lead concentrations ranged from less than 1 to 11.3 ppm and the authors reported a clear dose-response relationship between levels of lead and negative teacher ratings of ADHD-

type behaviors with an estimated OR of 3.25 (95%CI 1.64, 6.44).95 for behavioral problems comparing low versus highly exposed children (Tuthill 1996).

As studies started to show that even lower blood lead levels (< 10 μg/dL) reduced intellectual functioning (IQ; Lanphear et al., 2005), investigations also started to focus more attention on ADHD in children with lower levels of exposure to lead. One of the first studies was conducted by Braun et al. (2006) in US children. For this nationwide cross sectional survey, the National Health and Nutrition Exam study (NHANES) 1999–2002, the investigators reported for 4,704 children 4–15 years of age that blood lead was related to parent reported diagnosis/treatment of the child for ADHD even at levels below 5 μg/dL; specifically children with blood lead > 2 μg/dl were more likely to have ADHD compared to children with blood lead levels < 0.7 μg/dl. Also, these association were very strong and exhibited a dose response trend i.e. for the 3rd, 4th and 5th quintile of lead the adjusted ORs were reported as: 3rd quintile (0.8-1 μg/dl) OR=2.1; 95% CI 0.7–6.8; 4th quintile (1.4–2.0 μg/dl) OR= 2.7 (0.9–8.4), 5th quintile (> 2.0 μg/dl) OR= 4.1 (1.2–14.0).

A large prospective birth cohort (Boston Birth Cohort) collected data for 1,479 mother-infant pairs (299 ADHD, 1180 neurotypical) relying on the child's first blood lead measurement and physician-diagnosed ADHD from electronic medical records (Ji et al 2018). This study reported a 66% increased risk of ADHD (OR=1.66, 95%CI:1.08, 2.56) with an elevated lead levels (5-10μg/dL) and this association was much stronger among boys (OR: 2.49, 95%CI:1.46, 4.26) suggesting greater vulnerability in boys than girls. Importantly, as this study was prospective the exposures clearly preceded the outcomes as the median age of the first lead measurement was 0.84 years (inter-quartile range 0.77 to 1.03 years) and the median age at first ADHD diagnosis by a physician was 6 years. This is a strong association from a prospective study design which demonstrates the etiological relevance of early life lead exposure strong, alleviating concerns about reverse causation.

Similarly, Chiodo et al. (2007) reported that in more than 506 children from Michigan with an average blood lead level of 5μg/dl (and few children above 10 μg/dl) blood lead was not

only related to teacher-rated symptoms of inattention and activity (Conners rating scales and other standard scales) but that the "examination of these non-parametric regression lines indicated linear relationships between lead and each of the neurobehavioral endpoints examined" and that there was no lower level of exposure where the associations were no longer seen. Nigg et al. (2008) reported on low-level lead exposure in formally diagnosed children with ADHD, again showing that blood lead was related to ADHD and to parent reported DSM-IV symptoms of hyperactivity. Subsequently, the same authors reported that at 'background-levels of lead' (<1μg/dL mean) exposures were associated with clinically characterized ADHD i.e. at the lowest levels of blood lead ever studied in relation to ADHD, and in both parent and teacher reports of ADHD symptoms (Nigg 2010).

Recently, Geier, et al. (2017) employed data from the 2003–2004 National Health and Nutritional Examination Survey (NHANES) dataset, which included 2,109 people aged 10-19 years. They reported a statistically significant dose-response relationship between increasing blood lead levels and the risk of a reported ADD (per μg/dL, adjusted OR = 1.29, 95% CI 1.03-1.55). The mean blood lead level among the adolescents in this study was 1.16 μg/dl (SD ± 1.27). This study corroborates the earlier results by Braun et al. (2006) for younger children in NHANES 1999-2002.

Meanwhile larger studies of children with ADHD diagnoses were also conducted in Asia. For example, Wang et al (2008) conducted a very large case–control study of 4–12 years old children (630 ADHD cases, 630 non-ADHD controls) evaluated via structured diagnostic interviews, including caregiver interviews, based on the Diagnostic and Statistical Manual of Mental Disorders, 4th ed., revised criteria (DSM-IV-R). These authors reported that ADHD cases were more likely to have been exposed to lead during childhood than the non-ADHD control subjects after adjustment for many known risk factors (children with BLLs ≥ 10 μg/dl vs. ≤ 5 μg/dl; OR = 6.0; 95% CI = 4.10–8.77, $p < 0.01$; 5–10 μg/dl vs. ≤ 5 μg/dl, OR = 4.9; 95% CI = 3.47–6.98, $p < 0.01$). They also were concerned that clinical ADHD was related to lead exposure during childhood when blood lead levels were < 10 μg/dl. In Korea, a smaller hospital-

based case-control study of 114 medically diagnosed ADHD cases and 114 controls assessed ADHD with semi-structured diagnostic interviews in children with generally low levels of blood lead on average (<2 μg/dl) (Park 2016). Nevertheless, children with ADHD had blood lead concentrations higher than controls (geometric mean 1.90 ± 086 μg/dL vs. 1.59 ± 0.68 μg/dL, p = 0.003), log transformed total blood lead concentrations were associated with a 60% higher risk of ADHD (OR: 1.60, 95 % CI: 1.04–2.45, p < 0.05), and children with blood lead concentrations above 2.30 μg/dL were at a 2.5–fold (95 % CI: 1.09–5.87, p < 0.05) greater risk of having a ADHD diagnosis. A Korean cross-sectional study measured blood lead levels in 256 Korean children aged 8–10 years and asked teachers to complete the Attention-Deficit Hyperactivity Disorder Rating Scale (T-ARS)-IV to assess inattentive and hyperactive symptoms. The geometric mean blood concentration of lead was 1.5 μg/dL (range: 0.4–4.9) (Kim et al 2010). A linear regression analysis indicated that the blood lead concentrations were associated with the inattention scores (beta=4.8, 95% CI: 1.5–8.0), the hyperactivity subscores (beta=3.1, 95% CI: 0.3–5.9), and the total score (beta=7.9, 95% CI: 2.1–13.6) on the ADHD rating scale (T-ARS). Furthermore, in a logistic regression analysis the odds of exhibiting inattentive and hyperactive symptoms was increased with higher (> 2.18 μg/dL) blood lead levels in boys with an odds ratio of 2.77 (95% CI:1.06–7.18) and no association was seen in girls.

An earlier systematic review (Daneshparvar 2016) included articles on lead and ADHD up to May 2014 and reviewed all studies in which lead exposure was examined using blood samples. This review highlighted that 16 out of the 18 studies considered showed associations between blood lead levels less than 10 μg/dL in children and at least one type of ADHD. A recent meta-analysis (He 2017) indicated that even blood lead levels <3 μg/dl may be associated with ADHD symptoms in children. An even more recent systematic review of ADHD and lead (Donzelli 2019) included observational studies (cohort, case–control and cross-sectional studies) that were published in English and were carried out on children within the last 5 years. Out of 17 studies, 12 studies showed positive associations, even though not all of them were homogeneous in terms of exposure periods considered or ADHD diagnosis. Nevertheless, the authors

concluded that the studies allowed them not only to establish that there is an association between lead and ADHD but that even low levels of lead raise the risk. Additionally, the meta-analysis conducted a systematic evaluation of the methodological quality and evidence of each study using a grading scale derived from the principles of evidence-based medicine. The authors noted the poor methodological quality and high risk of bias in studies that did not observe an association between lead and ADHD compared with studies which found positive associations.

The available epidemiological literature demonstrates a consistent association between lead exposure and the development of childhood ADHD. I will now turn to the Bradford Hill criteria to make an assessment of causality.

Bradford-Hill Criteria Evaluation

Strength (effect size). This criterion is met. The effect estimates reported for blood lead and ADHD reflect strong associations with dose response trends. For example, the NHANES study (Braun et al. 2006) estimated an OR= 4.1 (95% CI 1.2–14.0) for the 5^{th} quintile of exposure (>2μg/dl) and exhibited a clear dose trend with increasing levels even below 2μg/dl.

[Note, the size of an estimated effect (OR, RR) for metal biomarkers on an outcome will also depend on the scale of the measure i.e. the unit used to estimate a per unit increase in risk]

Consistency. This criterion is met since positive associations have been reported for different populations and in different places and different time periods and different matrices.

Temporality. This asks whether disease occurred after exposure and that there is a delay between the cause and effect. This criterion is clearly met in the Ji, et al. (2018) cohort study, Burns et al (1999) Australian cohort study with repeated lead measures and the Bellinger et al. (1994) tooth lead study.

Specificity. This asks whether one specific exposure causes one specific outcome. This criterion is not relevant here as lead likely affects the brain in many different ways and other metals may also exhibit neurotoxicity.

Experimental evidence. This is not applicable for neurotoxin exposures in humans.

Analogy. Other neurotoxicants have also been shown to cause ADHD and other metals

have been related neurodevelopmental abnormalities, such as the well accepted neurotoxic effects of lead on cognitive development and IQ in children.

Coherence. See below for example from animal and cell experimental studies of mechanisms.

Biological plausibility. Lead easily crosses the blood–brain barrier and among brain regions affects the prefrontal cortex, hippocampus, basal ganglia, and cerebellum most (Costa et al., 2004; Finkelstein et al 1998; Nigg et al., 2008). Dysfunction in a cerebellar–prefrontal–striatal network have been implicated in ADHD (Valera et al 2007, Ota et al 2015). Lead also damages a number of neurotransmitter systems including dopaminergic, glutamatergic, and cholinergic pathways (Cory-Slechta, 1995). All three of these neurotransmitter systems, especially dopaminergic systems, have been linked to ADHD symptoms (reviewed in Gatzke-Kopp et al 2007; Sagvolden et al 2005). It has been shown in animal studies that lead exposure affects dopamine metabolism and decrease dopamine receptor binding in the striatum (Chang 2014, Fortune 2009) and reduced dopamine activity in striatum has been implicated as a mechanism related to the core symptoms of ADHD (Kostrzewa 2008, Spencer 2007). Specifically, hyperactivity-impulsivity may reflect effects of lead on the dopamine system (Cory-Slechta 1995, 1997; Luo et al. 2014). It is well known that dopaminergic neurons are very susceptible to oxidative stress as reactive oxygen species are generated during the dopamine degradation process (Sulzer and Zecca 2000) and lead increases oxidative stress. Lead also disrupts homeostasis of neurotransmitters, including such as dopamine (Simons 1993), by competing with calcium at its binding sites (Goering 1993) and it alters dopamine transporter activity (Akinyemi et al 2019). Finally, there is strong evidence in the animal literature that behavioral inhibition during reinforcement schedules is very sensitive to post-natal lead (Cory-Slechta 1990; Cory-Slechta et al. 2002). The inability to inhibit behaviors is one of the core symptoms of ADHD.

Conclusion: Following a review of the literature and application of the Hill guidelines, I conclude to a reasonable degree of scientific certainty that exposure to lead during sensitive

developmental periods in early childhood can cause ADHD, even at low levels of exposure.

Dated: November 12, 2021

Beate Ritz, MD, Ph.D.

Dr. Ritz Reference List

#	Author(s)	Year	Title of Article
1.	Abdullah, et al.	2012	Heavy Metal in Children's Tooth Enamel- Related to Autism and Disruptive Behaviors
2.	Adams, et al	2006	Analyses of Toxic Metals and Essential Minerals in the Hair of Arizona Children
3.	Adams, et al.	2017	Significant Association of Urinary Toxic Metals and Autism-Related Symptoms -A Nonlinear Statistical Analysis with Cross Validation
4.	Adams, et al.	2012	Toxicological Status of Children with Autism vs. Neurotypical
5.	Alabdali, et al.	2014	A key role for an impaired detoxification mechanism in the etiology
6.	Al-Ayadhi	2005	Heavy metals and trace elements in hair samples of autistic children in central Saudi Arabia
7.	Al-Farsi, et al.	2013	Levels of Heavy Metals and Essential Minerals in Hair Samples of Children with Autism
8.	Arora, et al.	2017	Fetal and Postnatal Metal Dysregulation in Autism
9.	Banerjee, et al.	2007	Environmental risk factors for attention-deficit hyperactivity disorder
10.	Bao, et al.	2009	Behavioural development of school-aged children who live around
11.	Bellinger	2013	Inorganic Arsenic Exposure and Children's Neurodevelopment A Review of the Evidence
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262.	Tripp, et al.	2008	Research review: dopamine transfer deficit: a neurobiological theory of altered reinforcement mechanisms in ADHD
263.	Tsai, et al.	2003	The effects of chronic arsenic exposure from drinking water on the neurobehavioral development in adolescence
264.	van Wijngaarden, et al.	2013	Prenatal methyl mercury exposure in relation to neurodevelopment and behavior at 19 years of age in Seychelles Child Development Study
265.	Hatzakis, et al.	1985	Blood lead and classroom behavior of children in two communities with different degree of lead exposure: evidence of a dose-related effect
266.	Adams, et al.	2007	Mercury, Lead, and Zinc in Baby Teeth of Children with Autism Versus Controls
267.	American Psychiatric Association	2000	Diagnostic and Statistical Manual of Mental Disorders Source Information, Fourth Edition (DSM-IV)
268.	Bellinger, et al.	1994	Pre-and postnatal lead exposure and behavior problems in school-aged children. Environmental Research

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#	Author(s)	Year	Title of Article
269.	Harada	1995	Minamata disease: methylmercury poisoning in Japan caused by environmental pollution
270.	Greenland & Robins	1988	Identifiability, Exchangeability, and Epidemiological Confounding
271.	Stang, et al.	2010	The ongoing tyranny of statistical significance testing in biomedical research
272.	Greenland, et al.	2016	Statistical tests, P values, confidence intervals, and power: a guide to misinterpretations
273.	Khan, et al.	2014	Disrupted brain thyroid hormone homeostasis and altered thyroid hormone-dependent brain gene expression in autism spectrum disorder
274.	EFSA Contam Panel	2009	Scientific Opinion on Arsenic in Food
275.	Wilson	2015	Arsenic Consumption in the United States
276.	Gochfeld, et al.	1995	Chemical agents: Environmental Medicine
277.	Hathaway, et al.	1991	Arsenic in arsine. In: Proctor and Hughes' Chemical Hazards of the Workplace, 5th Edition
278.	Gobierno Regional	2009	Programa Maestro de Intervención Zonas Con Presencia de Polimetales en Arica
279.	Hopenhayn-Rich & Goeden	1993	Human studies do not support the methylation threshold hypothesis for the toxicity of inorganic arsenic
280.	Johnson & Farmer	1991	Human exposures to arsenic from consumption of well water in West Bengal, India
281.	US EPA	1997	Office of Pesticide Programs, Chromated copper arsenicals and its use as a wood preservative
282.	Ahsan, et al.	2000	Associations between drinking water and urinary arsenic levels and skin lesions in Bangladesh
283.	ATSDR Agency for Toxic Substances and Disease Registry	1990	Agency for Toxic Substances and Disease Registry, Atlanta GA
284.	Shearer, et al.	1982	Minerals in the hair and nutrient intake of autistic children
285.	Wecker, et al.	1985	Trace element concentrations in hair from autistic children

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#	Author(s)	Year	Title of Article
286.	Apata, et al.	2017	Human adaptation to arsenic in Andean populations of the Atacama desert
287.	Gundert-Remy, Damn, Foth, et al.	2015	High exposure to inorganic arsenic by food: the need for risk reduction
288.	Debes, et al.	2006	Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years
289.	Grandjean, et al.	1997	Cognitive deficit in 7-year-old children with prenatal exposure to methylmercury
290.	Grandjean, et al.	2011	Methylmercury and brain development: imprecision and underestimation of developmental neurotoxicity in humans
291.	Choi & Grandjean	2008	Methylmercury exposure and health effects in humans
292.	Casarett	2001	Toxicology: The Basic Science of Poisons, 6th ed
293.	Counter & Buchanan	2004	Mercury exposure in Children: a review
294.	National Research Counsil, et al.	2000	Toxicological Effects of Methylmercury
295.	CDC	2012	Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention
296.	WHO	2017	International Lead Poisoning Prevention Awareness Campaign
297.	Hubbs-Tait, et al.	2005	Neurotoxicants, micronutrients, and social environments: Individual and combined effects on children's development
298.	Baghurst, et al.	1992	Environmental exposure to lead and children's intelligence at the age of seven years. The Port Pirie Cohort Study
299.	Bellinger, et al.	1992	Low-level lead exposure, intelligence and academic achievement: a long-term follow-up study
300.	Cory-Slechta	1997	Relationships between Pb-induced changes in neurotransmitter system function and behavioral toxicity
301.	Dietrich, et al.	1993	The developmental consequences of low to moderate prenatal and postnatal lead exposure: intellectual attainment in the Cincinnati Lead Study Cohort following school entry
302.	National Research Counsil, et al.	1993	Measuring Lead Exposure in Infants, Children and Other Sensitive Populations

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#	Author(s)	Year	Title of Article
303.	Needlemant & Gatsonis	1990	Low-level lead exposure and the IQ of children. A meta-analysis of modern studies
304.	Pocock, et al.	1994	Environmental lead and children's intelligence: a systemic review of the epidemiological evidence
305.	CDC	1991	Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control
306.	Rice	1993	Lead-induced changes in learning: evidence for behavioral mechanisms from experimental animal studies
307.	WHO	1995	Environmental health Criteria 165 - Inorganic Lead, Geneva
308.	Bellinger, et al.	1987	Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development
309.	Funahashi & Andreau	2013	Prefrontal cortex and neural mechanisms of executive function
310.	Dwivedi, et al.	2011	Reverses impaired mitochondrial energy metabolism and neuronal apoptotic cell death after arsenic exposure in rants
311.	Yen, et al.	2011	Inorganic arsenic causes cell apoptosis in mouse cerebrum through an oxidative stress-regulated signaling pathway
312.	Liu, et al.	2012	Arsenic-induced inhibition of hippocampal neurogenesis and its reversibility
313.	Goggin, et al.	2012	Perinatal exposure to 50 ppb sodium arsenate induces hypothalamic-pituitary-adrenal axis dysregulation in male C57BL/6 mice
314.	Martinez-Finley, et al.	2009	Learning deficits in C57BL/6J mice following perinatal arsenic exposure: Consequence of lower corticosterone receptor levels?
315.	Martinez, et al.	2011	Impact of early developmental arsenic exposure on promotor CpG-island methylation of genes involved in neuronal plasticity
316.	Rodriguez, et al.	2010	Chronic exposure to low levels of inorganic arsenic causes alterations in locomotor activity and in the expression of dopaminergic and antioxidant systems in the albino rat
317.	Zhang, et al.	2013	Sub-chronic exposure to arsenic disturbed the biogenic amine neurotransmitter level and the mRNA expression of synthetase in mice brains

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#	Author(s)	Year	Title of Article
318.	Xi, et al.	2010	Prenatal and early life arsenic exposure induced oxidative damage and altered activities and mRNA expressions of neurotransmitter metabolic enzymes in offspring rat brain
319.	Liu, et al.	2013	Protective effects of taurine on the decrease biogenic amine neurotransmitter levels in the brain of mice exposed to arsenic
320.	Luo, et al.	2012	Arsenite exposure altered the expression of NMDA receptor and postysnaptic signaling proteins in rat hippocampus
321.	Aung, et al.	2010	Inhibition of neurite outgrowth and alteration of cytoskeletal gene expression by sodium arsenite
322.	Zarazua, et al.	2010	Decreased arginine methylation and myelin alterations in arsenic exposed rats
323.	Jing, et al.	2012	Changes in the synaptic structure of hippocampal neurons and impairment of spatial memory in a rat model caused by chronic arsenite exposure
324.	Wang, et al.	2013	Protective effects of taurine on down-regulated expression of thyroid hormone receptor genes in brains of mice exposed to arsenic
325.	Lindberg, et al.	2008	Gender and age differences in the metabolism of inorganic arsenic in a highly exposed population in Bangladesh
326.	Valera, et al.	2007	Meta-analysis of structural imaging findings in attention-deficit/hyperactivity disorder
327.	Cory-Slechta	2003	Lead-induced impairments in complex cognitive function: Offerings from experimental studies
328.	Sagvolden, et al.	2005	A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes
329.	Potter, et al.	2006	Central nicotinic cholinergic systems: A role in the cognitive dysfunction in attention-deficit/hyperactivity disorder?
330.	Gatzke-Kopp & Beauchaine	2007	Central nervous system substrates of impulsivity: Implications for the development of attention-deficit/hyperactivity disorder and conduct disorder
331.	Marshall, et al.	2021	Risk of lead exposure, subcortical brain structure, and cognition in a large cohort of 9-to 10-year-old children

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332.	Mead & Ashwood	2015	Evidence supporting an altered immune response in ASD
333.	McDougle, et al.	2015	Toward an immune-mediated subtype of autism spectrum disorder
334.	Samsam, et al.	2014	Pathophysiology of autism spectrum disorders: Revisiting gastrointestinal involvement and immune balance
335.	Noriega & Savelkoul	2014	Immune dysregulation in autism spectrum disorder
336.	Estes & McAllister	2015	Immune mediators in the brain and peripheral tissues in autism spectrum disorder
337.	Goyal & Miyan	2014	Neuro-immune abnormalities in autism and their relationship with the environment: A variable insult model for autism
338.	Meltzer & Van deWater	2017	The role of the immune system in Autism Spectrum Disorder
339.	Hendren	2014	Biomarkers in Autism
340.	Rossignol & Frye	2014	Evidence linking oxidative stress, mitochondrial dysfunction, and inflammation in the brain of individuals with autism
341.	Bilbo, et al.	2015	A model for the induction of autism in the ecosystem of the human body: The anatomy of a modern pandemic?
342.	Loke, et al.	2015	The role of epigenetic change in Autism Spectrum Disorders
343.	Wong, et al.	2014	Methylomic analysis of monozygotic discordant for autism spectrum disorder and related behavioral traits
344.	Pirkle, Brody, Gunter, et al.	1994	The Decline in Blood Lead Levels in the United States: The National Health and Nutrition Examination Surveys
345.	Frank, et al.	2019	Systematic review and meta-analysis of lead (Pb) concentrations in environmental media (soil, dust, water, food, and air) reported in the United States from 1996 to 2016
346.	USGS Science for a Changing World	2021	Arsenic and Drinking Water
347.	Amaral, et al.	2008	Neuroanatomy of Autism
348.	Casanova	2004	White matter volume increase and minicolumns in autism

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349.	Strain, et al.	2015	Prenatal exposure to methylmercury from fish consumption and polyunsaturated fatty acids: associations with child development at 20 mo of age in an observational study in the Republic of Sechelled, The American Journal of Clinical Nutrition, Volume 101, Issue 3
350.	Tiffany-Castiglioni	1993	Cell culture models for lead toxicity in neuronal and glial cells
351.	Goyer	1993	Lead toxicity: current concerns
352.	Krigman, et al.	1974	Lead encephalopathy in the developing rat: effect upon myelination
353.	Tennekoon, et al.	1979	Chronic lead intoxication: effects on developing optic nerve
354.	Toews, et al.	1983	Myelin deficits produced by early postnatal exposure to inorganic lead or triethyltin are persistent
355.	Listos, et al.	2013	The effects of perinatal lead exposure on dopamine receptor D2 expression in morphine dependent rats
356.	Jett, et al.	1995	Developmental lead exposure alters N-methyl-D-aspartate and muscarinic cholinergic receptors in the rat hippocampus: an autoradiographic study
357.	Cory-Slechta, et al.	1998	Nucleus accumbens dopaminergic medication of fixed interval schedule-controlled behavior and its modulation by low-level lead exposure
358.	Cory-Slechta & Widzowski	1991	Low level lead exposure increases sensitivity to the stimulus properties of dopamine D1 and D2 agonists
359.	Deng, et al.	2001	Lead exposure delays the differentiation of oligodendroglia progenitors in vitro
360.	Silbergeld, et al.	1980	Effects of lead in vivo and in vitro on GABAergic neurochemistry
361.	Lidsky & Schneider	2003	Lead neurotoxicity: Basic mechanisms and clinical correlates
362.	Rafalowska, et al.	1996	Is lead toxicosis a reflection of altered energy metabolism in brain synaptosomes?
363.	Laterra, et al.	1992	Inhibition of astroglia-induced endothelial differentiation by inorganic lead: A role for protein kinase C.

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364.	Hill, et al.	1965	The Environment and Disease: Association or Causation?
365.	Wang, et al.	2017	Genome-wide gene by lead exposure interaction analysis identifies UNC5D as a candidate gene for neurodevelopment
366.	Chen, et al.	2019	Maternal exposure to low dose BDE209 and Pb mixture induced neurobehavioral anomalies in C57BL/6 male offspring
367.	Braun, et al.	2018	Effects of Residential Lead-Hazard Interventions on Childhood Blood Lead Concentrations and Neurobehavioral Outcomes: A Randomized Clinical Trial
368.	Canfield, et al.	2004	Impaired neuropsychological functioning in lead-exposed children
369.	Adams, et al.	2013	Toxicological status of Children with Autism vs. Neurotypical Children and the association with autism severity
370.	Albizzati, et al.	2012	Normal concentrations of heavy metals in autistic spectrum disorders
371.	Cohen, et al.	1976	Pica and elevated blood lead level in autistic and atypical children
372.	Gentile, et al.	1983	Brief report: trace elements in the hair of autistic and control children
373.	Domingues, et al.	2016	Pyrethroid Pesticide Metabolite in Urine and Microelements in Hair of Children Affected by Autism Spectrum Disorder: A Preliminary Investigation
374.	Jung, et al.	2008	Study on the mineral and heavy metal contents in the ahir of pre-school aged autistic children
375.	Marlowe, et al.	1984	Decreased magnesium in the hair of autistic children
376.	Marlowe, et al.	1985	Hair mineral content as a predictor of childhood autism
377.	Massaro, et al.	1983	Trace element concentrations and behavior: clinical utility in the assessment of developmental disabilities
378.	Holmes, et al.	2003	Reduced levels of mercury in first baby haircuts of autistic children
379.	Mostafa & Refai	2007	Antineuronal antibodies in autistic children: relation to blood mercury, Egypt

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#	Author(s)	Year	Title of Article
380.	Adams, et al.	2008	Mercury in first - cut baby hair of children with autism versus typically - developing children
381.	Williams, et al.	2008	A controlled study of mercury levels in hair samples of children with autism as compared to their typically developing siblings
382.	Majewska, et al.	2010	Age - dependent lower or higher hair mercury in autistic children than in healthy controls
383.	Woods, et al.	2010	Urinary Porphyrin Excretion in Neurotypical and Autistic Children
384.	Yau, et al.	2014	Prenatal and neonatal blood mercury levels and autism spectrum disorders
385.	McKean, et al.	2015	Prenatal mercury exposure, and developmental delay, using pharmacokinetic combination of newborn blood concentrations and questionnaire data: a case control study, Environ
386.	Mostafa, et al.	2016	The levels of blood mercury and inflammatory - related neuropeptides in the serum are correlated in children with autism spectrum disorder
387.	Forns, et al.	2014	Exposure to metals during pregnancy and neuropsychological development at the age of 4 years
388.	Sioen, et al.	2013	Prenatal exposure to environmental contaminants and behavioural problems at age 7 - 8 years
389.	Chan, et al.	2015	Metallic Burden of Deciduous Teeth and Childhood Behavioral Deficits
390.	Dikme, et al.	2013	The relation between blood lead and mercury levels and chronic neurological diseases in children
391.	Stamova, et al.	2011	Correlations Between Gene Expression and Mercury Levels in Blood of Boys With and Without Autism
392.	Johnson & Farmer	1991	Use of human metabolic studies and urinary arsenic speciation in assessing arsenic exposure
393.	Subramanian & Kosnett	1998	Human exposures to arsenic from consumption of well water in West Bengal, India
394.	US EPA	1997	Chromated copper arsenicals and its use as a wood preservative

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395.	ATSDR	1990	ATSDR Case Studies in Environmental Medicine
396.	CDC	2012	CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention recommendations in "Low level lead exposure harms children: A renewed call of primary prevention"
397.	Tiffany-Castiglioni, et al.	1986	Glial culture on artificial capillaries: electron microscopic comparisons of C6 rat glioma cells and rat astroglia
398.	Baron-Cohen, et al.	2015	Elevated fetal steroidogenic activity in autism
399.	Bjorklund, et al	2016	Immune dysfunction and neuroflammation in autism spectrum disorder
400.	Anashkina & Erlykina	2021	Molecular Mechanisms of Aberrant Neuroplasticity in Autism Spectrum Disorders
401.	Beal, et al.	1993	Neurochemical and histologic characterization of striatal excitotoxic lesions produced by the mitochondrial toxin 3 - nitropropionic acid
402.	Kapoor & van Rossum	1984	Effects of Pb2+ added in vitro on Ca2+ movements in isolated mitochondria and slices of rat kidney cortex
403.	Dancik	1991	Importance of Peer Review. The Serials Librarian
404.	White, et al.	2007	New and evolving concepts in the neurotoxicology of Lead
405.	Cory-Sletcha	1990	Exposure duration modifies the effects of low level lead of fixed - interval performance
406.	Cory-Sletcha, et al.	2002	Lead exposure and dorsomedial striatum mediation of fixed interval schedule - controlled behavior
407.	Cory-Sletcha, et al.	1996	The effects of dopamine agonists on fixed interval schedule - controlled behavior are selectively altered by low - level lead exposure
408.	Sulzer & Zecca	2000	Intraneuronal dopamine - quinone synthesis: a review
409.	Simons	1993	Lead - calcium interactions in cellular lead toxicity, Neurotoxicology
410.	Goering	1993	Lead - protein interactions as a basis for lead toxicity, Neurotoxicology

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411.	Akinyemi, et al.	2019	Lead (Pb) exposure induces dopaminergic neurotoxicity in Caenorhabditis elegans: Involvement of the dopamine transporter
412.	Gochfeld, et al.	1995	Environmental Medicine: Chemical Agents
413.	Hathaway, et al.	1991	Arsenic & arsine. In: Proctor and Hughes' Chemical Hazards of the Workplace, Third ed.
414.	Stretesky & Lynch	2004	The Relationship between Lead and Crime
415.	Murata, et al.	2004	Delayed brainstem auditory evoked potential latencies in 14 - year - old children exposed to methylmercury
416.	CDC	2017	Mercury Factsheet
417.	CDC	2017	Arsenic Fact Sheet
418.	CDC	2021	Health Effects of Lead Exposure
419.	WHO (World Health Organization)	2018	Arsenic
420.	WHO (World Health Organization)	2021	Lead Poisoning
421.	WHO (World Health Organization)	2017	Mercury and health
422.	EPA	2021	Arsenic Compounds
423.	EPA	2021	Learn about Lead
424.	EPA	2021	Health Effects of Exposure to Mercury

CURRICULUM VITAE
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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

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
2012- 2015	Chair, Department of Epidemiology, School of Public Health, University of California Los Angeles (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- 2014	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
2001-2012	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)
2010-2015	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 (Governor appointed) of California state (OEHHA) Scientific Review Panel on Toxic Air Contaminants
2012	Affiliate member of the Institute of the Environment and Sustainability
2012-	Elected to the American Epidemiological Society
2014	Scientific Organizing committee for the ISEE conference in Seattle Washington
2014-2016	Member of NAS/IOM committee on Incorporating 21st Century Science into Risk-Based Evaluations
2017-18	Member of NAS/IOM committee VAO Update 11 on Health Effects of Veterans from Herbicide Exposures in Vietnam

2017-18	Section Editor for Current Environmental Health Reports (Susceptibility Factors in Environmental Health)
2017-20	President Elect/ President/Past President of the International Society for Environmental Epidemiology (ISEE)
2019	NAS invited participant preparing an interdisciplinary statement by the national academies of science of South Africa, Brazil and the United States and the United States Academy of Medicine, and the German National Academy of Natural Sciences (Leopoldina) for actions on *"Air Pollution and Health*" to the United Nations.
2020	American Journal of Epidemiology and the Society for Epidemiologic Research selected the article "The Roles of Physical Activity and Inflammation for Mortality, Cognition, and Depressive Symptoms Among Older Mexican Americans" by Dr Ritz (senior author) and I-Fan Shih (graduate student mentee of Dr Ritz) as one of the *2019 Articles of the Year*.
2019-current	Scientific Review Board member for the IUF Leibniz Institute Duesseldorf *("Environmental Contributors to Aging*")

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 work towards the advancement of Parkinson's disease research
2018	Named on the highly-cited (top 1%) authors list by Clarivate Analytics.

FUNDED RESEARCH

PENDING

Preterm Birth, Childhood Autism, Alzheimer and Parkinson's Disease and Air Pollution

CARB (PI: Ritz PI) contract # 03/01/22-2/24/24
Total costs: $1 Million

This study will conduct population-based studies the influence of air pollution on preterm birth (PTB), term low birth weight (TLBW), and autism spectrum disorder (ASD), Alzheimer and Parkinson's disease in California. Based on outcome specific risk analyses and/or the meta-analyses as well as published economic data, the study will generate estimates of exposure response and healthcare and other economic costs associated with PTB/TLBW/ASD /AD/PD from air pollution overall and for minority groups and/or socio-economically or otherwise vulnerable communities in California.

ONGOING

A Multi-Omics Approach to Environment and Depression in Parkinson's (MOOD-PD)

NIEHS(PI: Ritz) R21ES032593 09/01/2020 – 08/31/2022
Annual total costs (UCLA): $408,777

This study will assemble multi-omic data into exposure and disease-related networks to address current knowledge gaps concerning molecular mechanisms contributing to depression and anxiety in PD, a major neurodegenerative disorder of growing importance in aging societies.

Pilot Population-based Case-control Study of Environmental Risk Factors for Multiple System Atrophy (MSA)

MSA Coalition (PI: Ritz) 09/01/20-12/01/21
Total Direct Costs $ 135,000

This study will design and test case ascertainment and exposure assessment tools for a future case-control study of MSA; specifically, explore two sources for ascertaining MSA cases in California in both a rural and an urban setting. It will also mine Electronic Health Records (EHR) at UCLA Health in an effort to generate information for the creation of an EHR-based MSA registry validating through medical record review all MSA patients and generating an algorithm for an automatic search of EHR that can be transported to other settings.

Microbiome, Environment, and Parkinson's Disease(MEP)
NIEHS RO1 (PI: Ritz) R01 ES031106 01/01/19-12/31/23
Annual Direct Costs $ 499,000

This grant proposes to 1) analyze the bacterial gut flora in 400 PEG PD patients and a total of 400 age-matched household and/or community control members using 16S ribosomal RNA (rRNA)] and assess function (using the predicted metagenome) in pesticide exposed vs. non-exposed patients/controls as well as in fast vs. slow progressing PD patients. And to 2) identify and enroll 100 new-onset, medication naïve PD patients and 100 household and/or community controls and collect fecal samples for metabolomics and culture based experiments twice assess compositional biota differences over time using shotgun metagenomics. Finally, we will 3) conduct metabolomics analysis in blood or stool for the new-onset, medication naïve PD patients.

Methylation Study of PD in S4 sampling study
MJFOX FOUNDATION (PI: Ritz) 10/01/20-09/30/22
Annual direct costs: $120,00

We will investigate epigenetic age acceleration across tissues in PD, using biospecimens available through the Systemic Synuclein Sampling Study (S4); i.e. whether epigenetic aging rates are increased in colon, mandibular gland, and skin tissues of PD patients both relative to controls and also, within subjects, relative to other tissues. This project will generate cross-tissue global cytosine methylation data, using the Illumina EPIC array platform and untargeted metabolomic data to profile >15,000 metabolites.
Role: Co-I

Pesticide Exposure, Systems Biology, and Parkinson's disease
MJFOX FOUNDATION (PI: Ritz) 02/01/21-01/31/23
Annual total costs (UCLA): $ 200,000

We propose to investigate the role of pesticides in PD susceptibility and progression. Our proposed work aims to isolate novel pesticide targets of etiologic relevance in PD and use a systems biology approach to highlight biologic pathways and responses related to pesticides and PD with high-throughput omics data.
Role: PI

MICHAEL J FOX FOUNDATION FOR PARKINSON'S RESEARCH (PI: Wu) 07/01/20-12/31/21
Annual total costs (UCLA): $ 333,326

UCLA-California PD Registry-EHR (UCE-PD) Demonstration Project

The goal is to develop and demonstrate a working framework of tools, integrated within the electronic health record (EHR) at UCLA, to capture and automatically submit information about patient cases to support and enhance the potential value of the California Parkinson's Disease Registry (CPDR).
Role: Co-I

DoD (PI: V. Khurana, Harvard) 09/01/19-08/31/22
Total direct costs to UCLA: $250,000

Understanding the role of gene-environment interactions in the degeneration of human dopaminergic neurons in Parkinson's disease

This study will address major challenges in the field. Here, the epidemiologists will define quantitative environmental exposure risks (from pesticides) for Parkinson's disease and closely interact with biologists undertaking mechanistic and cellular studies. Specifically, we will ascertain whether gene-toxin (pesticide) interactions play out specifically at the level of the dopamine neuron. In order to do so we will create an approach that addresses GxE

interactions at the level of an individual patient building on a platform that, ultimately, gives us the ability to determine specific genetic and environmental factor interactions within the patient's own iPSC-derived brain cells.

Environment, Metabolomics, and Parkinson's Disease: Multi-omics data integration

NIEHS R21 (PI: Ritz) Type: R21 ES030175 01/01/19-01/31/21
Total direct costs to UCLA: $275,000
This project will identify metabolic signatures of chronic environmental exposures and disease in 550 PD patients and 250 population controls from the PEG population-based study. We plan to integrate exposure and effect markers across multiple molecular layers (genome, epigenome, metabolome), with the goal to obtain a more holistic understanding of molecular changes due to chronic environmental exposures.

Validation and optimization of epigenetic clocks

NIH/NIA (PI:Horvath) Type: 1 U01 AG060908-01 10/01/18- 09/30/23
Annual total costs: $415,164
To address the challenge of developing biomarkers of aging, we will focus on epigenetic alterations.The overarching goals of this application are a) to realize the great promise of DNAm based biomarkers of aging (known as epigenetic clocks) for human interventional studies and b) to advance their mechanistic understanding. This application builds on our active research program surrounding DNAm based biomarkers of aging and their relationship to markers of cellular senescence.

Burroughs Wellcome Fund (PI: Ritz) 02/01/2009 - 01/31/2022
Annual total costs (UCLA): $100,000
UCLA Chronic Diseases Inter-school Training Program (BWF-CHIP)
The program provides training in laboratory and population sciences to integrate the entire continuum from molecules to populations. Chronic diseases are the number epidemic of our time in Western Nations. The etiologic complexities of chronic diseases require an equally multidisciplinary approach that goes beyond simply translating basic science discoveries into clinical practice.
Role: mPI

CARB (PI Jerrett) 01/01/18-04/31/21
Annual Direct Costs $ 184,070
Effects of Brake and Tire Wear on Particulate Matter Composition, Reactive Oxygen Species, Placental Development and Birth Outcomes in Los Angeles
This project will take extensive field measurements on speciated particle concentrations with the aims of (1) developing an exposure surface representing brake and tire wear, (2) using the particle samples to develop an exposure surface of reactive oxygen species, and (3) using these exposure estimates to assess the adverse effects on placental development and on other birth outcomes.

Multi-Angle Imager for Aerosols (MAIA)

Agency: NASA (PI: Ritz) NNH12ZDA006O-EVI3 08/01/16-11/30/25
Total Direct Costs to UCLA: $1,294,244
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.

COMPLETED RESEARCH

Imaging Innovations for Placental Assessment in Response to Environmental Pollution

Agency: NIH/NICHD 1 U01 HD087221 (PI: Devaskar/UCLA Ob-GYN) 01/01/16-12/30/19
Total Direct Costs: $2,999,640
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.

Environment and cognitive decline in older Hispanics
Multi-PI: Ritz/Haan
Agency: NIEHS Type: R01- RES023451A 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.

Environmental & Genetic Predictors of Parkinson's Progression
NIH/NIEHS 1R56ES026600-01A1 (PI:Ritz) 07/01/17-09/30/18
Annual Direct Costs $97,222
First, we will harmonize the SNP data from different GWAS and candidate gene approaches using the appropriate genomic tools for imputations and explore coding regions with programs like SIFT/polyphen /mutation taster to find possible SNPs/SNVs and interrogate the Alternative Splicing Database. Second, we will collect preliminary data on fecal sample feasibility by enrolling 50 case and household controls as pairs in a pilot study to design and test our fecal sample collection under the guidance of the UCLA microbiome core.

Air Pollution and Autism in Denmark
PI: Ritz
Agency: NIEHS R21ES024269 04/01/15-03/31/18
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.

A Cohort Study on Air Pollution and Breast Cancer in Los Angeles County
Susan G Komen (A. Wu USC) $217,728 02/13/14-02/150/18
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: Consultant - UCLA

Autism, Metabolomics, and Environment (AIME)
Agency: NIEHS R21ES25573 (PI: Ritz) 07/01/15-06/30/18
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) 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.

Air Pollution and Cardiovascular Diseases: Identification of Novel Biomarkers
Agency: NIEHS R21 ES024560 (PI: Zhu) 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

Psychosocial stressors, air pollution and childhood respiratory health in LAFANS
Agency: NIEHS R03ES025908 (PI: Ritz) 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) 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.

Environmental exposure, DNA methylation, and Parkinson's disease
Agency: NIEHS 21ES024356 (PI: Ritz/ Horvath) 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 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 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 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 01/01/11 -08/31/15
Total Direct Costs (at UCLA): $ 250,000
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

Susan G Komen (A. Wu USC) $217,728 02/13/14-02/150/17
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: Consultant - UCLA

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.

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
Annual 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

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)
2002 - 2005 Pin-Chieh Jason Wang (Ergonomic interventions and health in LA garment workers)
2003 - 2005 Kathrine Hoggatt (co-mentored with S. Greenland: Air pollution and adverse birth outcomes)
2003 - 2006 Chad Lewis (TTHM contamination in drinking water and adverse birth outcomes)
2004 - 2008 Angelika Wahner (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 (Iron genetics 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 (Pesticide exposures in pregnancy and childhood cancers)
2009 - 2014 Shilpa Narayan (Occupational pesticide exposure GxE in Parkinson's disease)
2009 - 2014 Christina Lombardi (Air pollution and childhood cancers)

2011 - 2015 Zeyan Liew: PFOA exposures in the Danish birth cohort and ADHD and autism);
2011 - 2016 Kim Paul (Gene-environment interactions in Parkinson's – PASIDA study);
2011 - 2015 Xin Cui (Bias Analyses in Population-Based Studies of Parkinson's Disease)
2011 - 2017 Andrew Park (Pesticides and childhood cancers)
2012 - 2015 Gretchen Bandoli (Stress, asthma and birth outcomes in LA)
2012 - 2015 Vivian Alonso (Assessing risk factors for adverse birth outcomes and early childhood respiratory illness: an examination of supplement initiation and participation in Special Supplemental Nutrition Program for Women, Infants and Children during pregnancy)
2013 - 2017 Yu-Hsuan Chuang (Parkinson's disease, gene methylation, and gene-environment interactions)
2013 – 2017 Xiaoqing Xu (Pharmaceuticals and childhood cancers in Denmark, NSAIDS and anti-hypertensives and age related macular degeneration)
2013 - 2017 I-Fan Shih (Parkinson's disease and physical activity)
2013 - 2017 Negar Omid (Occupational childhood cancer risk factors)
2014 - 2017 Zuelma Esquivel (Childhood cancer risk factors in Hispanic Communities)
2013 - 2018 Chenxiao Ling (Spatial tools and bias analysis in environmental epidemiology)
2015 - 2018 Clint Hall (Childhood cancers and occupational parental exosures in Denmark)
2014 - 2019 Cynthia Kuster (Parkinson's disease, endocrine disruptors and estrogen receptors)
2014 - 2019 Sam Wing (Air pollution, stress and child health)
2013 - 2020 Aline Duarte (Parkinson's disease and sleep disorders)
2015 - 2020 Yu Yu (Air pollution and metabolic syndrome in the SALSA cohort of elderly Hispanics)
2015 - 2021 Yan Qi (Metabolomic and Epigenomics Assessment of Air Pollution and Pesticides Exposure in California)
2018 - 2021 Kosuke Inoue (Estimating the Effects of Diabetes on Cardiovascular Events and Mortality:Causal Modeling and Machine Learning)
2017 - De Hi (Childhood cancers and metabolomics)
2018 - Keren Zhang (Microbiome in Parkinson's disease)
2018 - Xiwen Huang (Childhood cancers and maternal gestational diabetes)
2019- Qi Meng (Break and tirewear sources of air pollution and adverse birth outcomes)
2020- Anupong Sirirungreung (Childhood cancers and maternal infections in pregnancy)
2020- Ting Chow (Gestational diabetes and autism)
2020- Yang Cheng Hu (Omics approaches in PD)
2020- Tahmineh Romero (PD and depression)
2021- Shiwen Li (Social and physical environment and adverse birth outcomes)
2021- Yufan Gong
2021- Xiwen Chen
2021- Sanjali Mitra

Postdoctoral Fellows in Epidemiology

2005 - 2006 Pin-Chieh Jason Wang (Ergonomic interventions and health in LA garment workers)
2004 - 2008 Michelle Wilhelm (Traffic-related air pollution and pregnancy related health effects)
2008 - 2010 Angelika Wahner (Parkinson's disease, genetic factors and anti-inflammatory drug use)
2009 - 2011 Shannon Rhodes (Iron and Parkinson's disease)
2008 – 2010 Nicole Gatto (Parkinson's disease and Vitamin D)
2011 - 2013 Pei Chen Lee (Air pollution and Parkinson's disease)
2011 - 2012 Anshu Shresta (Childhood cancers and the environment)
2015 - 2017 Xin Cui (Metabolomics in autism)
2016 - 2018 Zeyan Liew (PFOA exposures in Denmark, ADHD and autism and neurodevelopment)
2017 - 2018 Yu-Hsuan Chuang (Parkinsons, epigenetics and gene-environment interactions)

2016 – 2020 Kimberly Paul (Epigenetics and metabolomics in Parkinson's disease)
2020 - 2021 Aline Duarte (MSA risk factors)

Mentees

At UCLA Dept. of Neurology

2003 - 2004 Gail Asleson Kang (*Movement Disorder Fellow*: Clinical characteristics of PD patients)

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)

At Montana University

2019-current Erin Semmens (K-awardee: Wildfire smoke and health effect)

At Arizona University

2018-2021 Melissa Furlong (K-awardee: Pesticides exposures and ADHD)

University of Umea/Sweden

2014 Opponent for doctoral student David Olsson (Air pollution and PTB and preeclampsia in Stockholm)
2017 Opponent for doctoral student Fei Yang (Parkinson's disease in Sweden)

PARTICIPATION IN GRANT, CENTER, AND INSTITUTE 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-UCLA Environmental Health Science center pilot grants, 2014-2018
Review of NIEHS conference grants July 2015
Review of Parkinson's disease grant for Parkinson's UK foundation in Great Britain 2015
Review of Genomics/Big Data fellowship applications Human Genetics/ UCLA
Review of NIH/NIEHS – River grants, R13 travel grants, NIH Director's Early Independence Award (DP5)
Review for Agency for Toxic Substance Disease Register (ATSDR) 2015
Review for Polish Science Foundation 2017 and 2018, 2019
Review for South African National Research Foundation 2017 and 2019
Review for Wellcome Trust/DBT India Alliance 2017 and 2019
Scientific Review of NIEHS RIVER grants 2018
Scientifc Reviewer for the Austrian Science Fund (FWF) 2018
Chair of Scientific Review of NIEHS RIVER grants 2019
Review of the Swiss School of Public Health and Tropical medicine SSPH+ Corona Immunitas Program 2020
Reviewer for the Outstanding New Environmental Scientists (ONES) awards at NIEHS 2020
Reviewer for a grant to the DFF / Independent Research Fund Denmark 2021
Reviewer for the Outstanding New Environmental Scientists (ONES) awards at NIEHS 2021
Reviewer for a grant to the DFG (Deutsche Forschungsgemeinschaft) / German Research Fund 2021
Scientific Reviewer for the IUF – Leibniz Research Institute for Environmental Medicine; IUF – Leibniz-Institut für Umweltmedizinische Forschung gGmbH

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/AI 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
66. Environment and Genes interacting in PD progression. APDA West Coast Forum, Anaheim Oct 2017
67. Review of Air Pollution and Birth and Child Outcomes. ISEE-Asia Chapter June2018
68. After 20-years of research on Air Pollution Health Effects on Children: how far have we come. INCHES Meeting Korea June 2018
69. Reviewing GxE and methylation in PD. Parkinson's Center Luxemburg July 2018
70. Using Genetic Risk Score in Parkinson's Disease. Invited Speaker at Annual Parkinson's Grand Challenge Event at the Van Andel Institute in Grand Rapids Michigan Aug 2019

PUBLICATIONS

PEER REVIEWED 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. PMID: 8824754
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. PMID: 9709991
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. PMID: 9884742
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 H. 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. PMID: 10069248
7. **Ritz B**. Radiation Exposure and Cancer Mortality In Uranium Processing Workers. Epidemiology, 1999, Sep; 10:531-538. PMID: 10468427
8. **Ritz B**. Cancer Mortality Among Workers Exposed To Chemicals During Uranium Processing. JOEM 1999, Jul;41(7):556-566. PMID: 10412097
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. PMID: 10529946
10. Jacob B, **Ritz B**, Heinrich J, Hoelscher B, Wichmann HE. The Effect Of Low-Level Blood Lead On hematologic parameters In Children. Environmental Research, 2000 Feb, 82 (2): 150-159. PMID: 10662529
11. **Ritz B**, Yu F. Parkinson's Disease Mortality And Pesticide Exposure In California 1984-1994. International Journal of Epidemiology, 2000 Apr, Vol. 29:323-329. PMID: 10662529
12. Hoelscher B, Heinrich J, Jacob B, **Ritz B**, Wichmann HE. Gas Cooking, Respiratory Health And White Blood Cell Counts In Children. Int. J. Hygiene and Environ Health, 2000 Mar; 203 (1): 29-37. PMID: 10956587
13. **Ritz B**, Morgenstern H, Crawford-Brown D,Young B.The Effects Of Internal Radiation Exposure On Cancer Mortality In Nuclear Workers At Rocketdyne/Atomics International. Environ Health Perspect, 2000 Aug; 108(8):743-751. PMCID: PMC1638302
14. **Ritz B**, Yu F, Chapa G, Fruin S. Effect Of Air Pollution On Preterm Birth Among Children Born In Southern California Between 1989 And 1993. Epidemiology, 2000 Sep; 11(5):502-511. PMID: 10955401
15. Morgenstern H, **Ritz B**. Effects of Radiation And Chemical Exposures On Cancer Mortality Among Rocketdyne Workers: A Review of Three Cohort Studies. Occup. Med. 2001 Apr-Jun;16(2): 219-237. PMID: 11319049
16. **Ritz B**, Yu F, Chapa G, Fruin S, Shaw G, Harris J. Ambient Air Pollution And Risk of Birth Defects in Southern California. Am J Epidemiol 2002 Jan 1;155:17–25. PMID: 11772780
17. **Ritz B**, Hoelscher B, Frye C, Meyer I, Heinrich J. Allergic sensitization owing to 'second-hand' cat exposure in schools. Allergy 2002 Apr;57(4):357-61 PMID: 11906369
18. Jacob B, **Ritz B,** Gehring U, Koch A, Bischof W ,Wichmann HE, Heinrich J for the INGA-Study group. Indoor Exposure To Molds And Allergic Sensitization. Environ Health Perspect. 2002 Jul;110(7):647-53. PMCID: PMC1240910
19. Clary T, **Ritz B**. Pancreatic Cancer Mortality And Organochlorine Pesticide Exposure In California, 1989-1996. Am J Ind Med. 2003 Mar;43(3):306-13. PMID: 12594778
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