Exhibit 132

Trichloroethylene and Parkinson's disease: an examination of causal associations at Camp Lejeune

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1. Background and Qualifications of Gary W. Miller, PhD

I am a toxicologist whose career has focused on the environmental causes of neurological diseases, including Parkinson's disease. I completed my Ph.D. in Pharmacology and Toxicology in 1995 from the University of Georgia. During my training, I performed studies on a range of toxicants, including known metabolites of trichloroethylene. From 1995-1997 I was a postdoctoral fellow in the Department of Neurology in the School of Medicine at Emory University in Atlanta, GA. My postdoctoral research focused on mechanisms of dopamine neuron cell death in Parkinson's disease. I obtained a competitive research fellowship from the National Institutes of Health (NIH) to study dopamine toxicity as it relates to Parkinson's Disease. In 1997, I moved to the Duke University School of Medicine to gain additional training in neuroscience. I conducted further research on the dopamine system, especially the characterization of dopamine receptors and transporters, which are critical pathways in the development of Parkinson's Disease. My mentor was a Howard Hughes Medical Institute (HHMI) Investigator at the time, a highly prestigious designation (35 HHMI investigators have won a Nobel Prize). During my time at Duke University, I continued studies on the impact of dopamine on brain function. I published several papers on-dopamine[2-8] and continued my research on the impact of environmental chemicals on the dopamine system, including receiving a large research grant from NIH to study the impact that environmental contaminants have on brain function and disease, including the development of Parkinson's disease. In the intervening 26 years I have been continuously funded by NIH to continue my work on environmental factors in Parkinson's disease.

After four years as an Assistant Professor in Pharmacology and Toxicology at the University of Texas, I returned to Emory University as an Associate Professor to join the newly established Center for Neurodegenerative Diseases (with appointments in the Department of Environmental Health, the Department of Neurology, and the Department of Pharmacology). While at Emory, I collaborated with one of the world's leading experts on Parkinson's Disease, Tim Greenamyre MD, PhD, on several projects until the time he left for the University of Pittsburgh. I remained at Emory for 16 years, maintaining active collaborations with colleagues in the Center for Neurodegenerative Diseases. In 2009, after my promotion to Full Professor, I was asked to serve as Associate Dean for Research in the School of Public Health and continued my research on Parkinson's disease. In 2018, I was recruited to Columbia University to serve as Vice Dean for Research Strategy and

Innovation and Professor of Environmental Health Sciences and Professor of Molecular Pharmacology and Therapeutics where I continue my work on the environmental contributors to human disease. I am familiar with state-of-the-art science in determining causal links between exogenous factors and neurodegenerative diseases, including Parkinson's Disease. In addition to this, my team also studies Alzheimer's disease and many other conditions using the novel methods we have developed, including using high-resolution mass spectrometry for exposomics. At Columbia, I continue my collaborations with the neurology community continuing to publish with top neurologists.

I have been a global leader in the emerging field of exposomics. Designed as the environmental complement to genomics, exposomics works to provide a comprehensive assessment of environmental factors across a range of diseases. I have published extensively in this area.[11-39] Over the years I have taught courses in Human Toxicology, Neurotoxicology, Exposomics, Research Methods, and Responsible Conduct of Research and written numerous book chapters on toxicology and neurotoxicology, as well as two books on the exposome,[40,41] including the first ever published on the topic.

I have been actively engaged in Parkinson's disease research for over 25 years. I have received grant funding from a wide range of agencies and foundations that support Parkinson's research including the National Institute of Neurological Disease and Stroke, the National Institute of Aging, the National Institute of Environmental Health Sciences, the Michael J. Fox Foundation, the American Parkinson's Disease Association, SPARK-NS, and the Department of the Defense. I have served on advisory boards for The U.S. Veteran's Administration, the National Academy of Sciences, and the European Commission.
I served as Editor-in-Chief of the journal Toxicological Sciences from 2013-2019, which is the flagship scientific journal of the 8000-member Society of Toxicology.[42-46] In 2021, I was asked to serve as Editor-in-Chief for Exposome, a new journal published by Oxford University Press.[21,23,24,47] As an editor, I have published extensively on research integrity, reproducibility, and publication ethics.[21,23,24,42-47]

I am an elected Fellow of the American Association for the Advancement of Science (AAAS). The AAAS is world's largest multidisciplinary scientific society and a leading publisher of cutting-edge research through its *Science* family of journals. AAAS Fellows are a distinguished cadre of scientists, engineers, and innovators who have been recognized for their achievements across disciplines, from research, teaching, and technology, to administration in academia, industry and government, to excellence in communicating and interpreting science to the public. Since 1874, Fellows are elected annually by the AAAS Council and are expected to meet the commonly held standards of professional ethics and scientific integrity. I have been asked on several occasions to speak about neurotoxicology at the National Conference of Lawyers and Scientists (NCLS) conference. The NCLS was established in 1974 as a joint standing committee of the American Association for the Advancement of Science (AAAS) and the American Bar Association's (ABA) Section of Science and Technology Law. The committee has fourteen members, half appointed by AAAS and half appointed by the ABA. At these meetings

practicing judges hear from a range of academic experts about topics commonly faced in the courtroom.

The above information demonstrates that I am uniquely qualified to review data, scientific literature, and reports as they relate to environmental exposures, toxicology, and Parkinson's disease. I have served in key scientific leadership positions where scientific integrity is paramount. I have been closely engaged with the Parkinson's disease research community for many years and have worked with and interacted with the key scientists in the field. I do not have any ties to companies that manufacture TCE or related materials, nor do I have any family members that served at Camp Lejeune during the period in question.

2. Questions and Methodology

I was asked to evaluate the relationship between exposure to chemicals including trichloroethylene (TCE) and tetrachloroethylene (PCE) to Parkinson's Disease and to analyze the level(s) at which exposure to these chemicals is generally known to cause Parkinson's Disease. To address these questions, I relied upon my education, training, and experience in the field of toxicology and neuroscience while analyzing the scientific literature and studies obtained from PubMed, Endnote, and Google Scholar. I have employed a weight of the evidence approach utilizing the Bradford Hill framework in reaching my opinions, as well as state-of-the-art scientific causality analysis. This approach is the same that I would use in my academic research and professional endeavors.

The following document examines the epidemiological, animal, and mechanistic data that exists on the subject of chemical exposures and Parkinson's Disease, as well as the Camp Lejeune Justice Act.[48] This information will be placed into the context surrounding the exposure of Marine and Navy personnel, their dependents, and civilian employees who were stationed at Camp Lejeune during the time when there was significant contamination of the water supply with a range of solvents including TCE and PCE. Those present at Camp Lejeune faced potential exposure to these solvents via the water, soil, and air.

3. Summary of Opinions and Methodology

Based upon my education, training and experience in the fields of toxicology and neuroscience, and upon my independent analysis of the scientific literature on environmental exposures and their ability to cause or exacerbate Parkinson's Disease, I have formed the following opinions to a reasonable degree of scientific certainty:

1) There is overwhelming evidence that environmental factors play a role in the development and progression of Parkinson's disease
2) There is substantial evidence that industrial chemical exposures can cause Parkinson's disease.
3) Trichloroethylene (TCE) is the solvent with the strongest evidence of a causal connection to Parkinson's disease, and it is my professional opinion that TCE, is at least

as likely as not, a cause of the pathogenesis and progression of Parkinson's Disease. Additionally, PCE, a similar chlorinated solvent has the same potential toxicity to the dopamine neurons in the substantia nigra and can contribute to the pathogenesis of Parkinson's disease. It is my opinion, to a reasonable degree of scientific probability that both TCE and PCE can cause Parkinson's Disease. Further, it is my opinion, outlined in further detail below, that the levels of exposure to TCE and PCE at Camp Lejeune are known to create a significantly elevated risk of Parkinson's Disease.
4) Military personal stationed at Camp Lejeune, as well as resident family members and civilian workers, between 1953 and 1987 were exposed to unsafe levels of TCE and PCE, which are generally known to cause Parkinson's Disease.
5) This increased risk and incidence of Parkinson's Disease in the Camp Lejeune population above is already demonstrated by higher rates of the disease and there will continue to be an elevated risk for those personnel who have not yet been diagnosed with Parkinson's disease, all of which are because of exposure to TCE and PCE.
6) There is evidence that people stationed at Camp Lejeune who have Parkinson's will likely have more aggressive forms of the disease with worsened symptoms.

In evaluating the scientific literature to help determine the presence or absence of a causal association between specific exposures and disease, the Bradford Hill framework is widely used.[49] Each of these components helps strength a causal connection between a given exposure and a given outcome; however, it is not necessary to fulfill each of the components to have a causal connection. In many situations it may not be possible to provide laboratory evidence or there may not be a plausible biological explanation for a given causal association. That said, the framework provides a foundation for examining causal associations. The key components include:

Temporality: The effect must occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay).
Strength (effect size): A small association does not mean that there is not a causal effect, though the larger the association, the more likely that it is causal.
Consistency (reproducibility): Consistent findings observed by different persons in different places with different samples strengthens the likelihood of an effect.
Specificity: Causation is likely if there is a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship.
Biological gradient (dose-response relationship): Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect.
Plausibility: a plausible biological mechanism between cause and effect is helpful but often limited by current scientific knowledge.
Coherence: Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, lack of laboratory evidence does not nullify the epidemiological effect on associations.
Experiment: In some situations, it is possible to use experimental evidence to support the connection.
Analogy: The use of analogies or similarities between the observed association and any other associations.

It should be noted that Bradford Hill framework was established before many key biological mechanisms of exposure and disease were well understood, especially for Parkinson's disease. Even Sir Bradford Hill noted that the criteria were to be considered as a tool to assist in the evaluation of causality but were not intended to be strict criteria of causality.[50] Over the past fifty plus years, tremendous advances in science have been made that help inform researchers and scientists, like myself, on the causal connection between exposure and disease. In my analysis of the epidemiological literature related to TCE and Parkinson's Disease, I utilized a weight of evidence approach applying the guidance of Bradford Hill to assist in my determination of causality. In addition to addressing the epidemiology that links TCE to Parkinson's Disease, I have also analyzed the animal and mechanistic data that more firmly supports the opinion that TCE exposures, at levels which were experienced at Camp Lejeune, can cause Parkinson's Disease.

The goal of this report was to provide a rigorous scientific analysis of the available data to assist the courts in making decisions based on state-of-the-art scientific knowledge regarding whether the associations between exposures that occurred on base and health outcomes reach the level of a cause bringing about Parkinson's Disease among other factors using Bradford Hill[50] as a guide, rather than to generate an academic manuscript on the epidemiology of TCE and Parkinson's disease. I have endeavored to closely align my analysis with the standards of evidence used by the scientific and medical community to provide the court scientific confidence in the reported associations. To that effect, in addition to Bradford Hill, it is helpful to draw upon more recent frameworks regarding causation as exemplified by the work of Judea Pearl.[51,52] Dr. Pearl is considered one of the world's authorities on causal and counterfactual inference which has been instrumental in the underpinnings of artificial intelligence.[53-55] His methodology is widely used in scientific research determining these causal and counterfactual inferences with confidence intervals acceptable within the scientific community and can be considered to be complementary to Bradford Hill. Pearl refers to three steps in the Ladder of Causation starting at the bottom are association, intervention, and counterfactuals. The higher up the ladder one ascends the stronger the causal connection:

> **Association** is the independent observation. For example, one can observe that people who exercise five times a week have a lower body mass index.
> **Intervention** is using interventions or manipulations (a typical laboratory experiment in which the experimental controls the variables). For example, assigning people to a specific exercise regimen for 12-weeks and then determining if their body mass index changes.
> **Counterfactuals** address "what if?" scenarios that help explore alternate explanations. For example, what if we discovered that the people that exercised five times a week also had a gene variant that increased lean body mass and decreased body mass index? Is it the gene that caused the decreased body mass index or the exercise? What if we redesigned the study to include groups with and without the gene mutation?

By using the underlying reasoning of Bradford Hill and Pearl's Ladder of Causation to evaluate the scientific data and literature, we can be confident that the conclusions drawn are of the highest level of scientific rigor.

4. Parkinson's Disease

Parkinson's disease is the most common neurodegenerative movement disorder affecting over one million people in the U.S. It is characterized by the following hallmark features: slow movement (called bradykinesia), resting tremor, rigidity, and postural instability.[56,57] These are the primary motor-related symptoms but there are many non-motor symptoms associated with the disease including loss of the sense of smell, constipation, sleep disorders, autonomic dysfunction, and cognitive dysfunction. There are drugs that treat some of the symptoms of the disease, but unfortunately there is no cure for Parkinson's disease.

The loss of the sense of smell, constipation, and sleep disorders can appear well before a person receives a diagnosis of Parkinson's disease, which is primarily based upon the motor feature of the disease.[1] These symptoms tend to precede diagnosis of the disease (but persist throughout the course of the disease) and are referred to as prodromal symptoms. These symptoms are not exclusive to Parkinson's disease—there are other conditions that can cause them, thus they are not specific, but they are more common in people who develop Parkinson's disease. Upon diagnosis they provide a retrospective history of disease progression. If a person is diagnosed with Parkinson's disease at 65 years of age and they report losing their sense of smell at 45 then that symptom can be considered to be part of the prodromal phase of the disease. The motor symptoms of Parkinson's do not appear until 60-80% of the brain dopamine neurons are lost, but the prodromal symptoms may appear when there is only an intermediate loss of brain dopamine (see Figure 1). These prodromal symptoms are thought to be caused by dysfunction of other neurons throughout the body that are damaged through similar neurodegenerative processes and by the degeneration of the brain dopamine neurons and other neuropathological changes in the body. The point of determining the onset of prodromal symptoms is that once the Parkinson's Disease is diagnosed the timing of these symptoms helps in the retrospective analysis to determine the age of onset and progression. If environmental factors promote Parkinson's disease pathogenesis, these prodromal symptoms can provide early evidence of such an association.

5. TCE/PCE and Parkinson's Disease

The observation that some families had a higher-than-normal rate of Parkinson's led to a belief that the disease was due to genetics. While several genes have been identified that are associated with an increased risk of Parkinson's disease, especially in these rare family situations, these purely genetic causes can only explain ~10-20% of disease incidence. More importantly and over the past 25 years, it has become clearer to scientists like me who study Parkinson's Disease, that environmental exposures play a predominant role in the development Parkinson's Disease. The inability of genetic studies to explain the variable disease incidence, including twin studies that demonstrate fraternal twins are as

likely to get Parkinson's as identical twins,[58-62] weakens the argument that Parkinson's Disease is primarily due to genetics. On the other hand, more recent epidemiological studies have demonstrated that exposures to a range of chemicals, including TCE, as well as pesticides used to kill insects, plants, fish, and other nuisance species, increase disease pathogenesis, incidence, and risk.[63] These epidemiological studies are supported by hundreds of studies in laboratory animals that demonstrate that these chemicals can kill the dopamine neurons in the nigrostriatal system (for examples see[64-70]), which are the same neurons that die in Parkinson's disease. Moreover, as indicated below, the results of the studies recapitulate many of the hallmarks of Parkinson's disease. Therefore, it is scientifically valid and reasonable to conclude that these chemicals are as likely than not a cause of the initiation of the cascade of damage to selective areas of the brain and the progressive aftermath that result in the appearance of prodromal symptoms followed by the motor symptoms that are diagnostic of Parkinson's Disease.

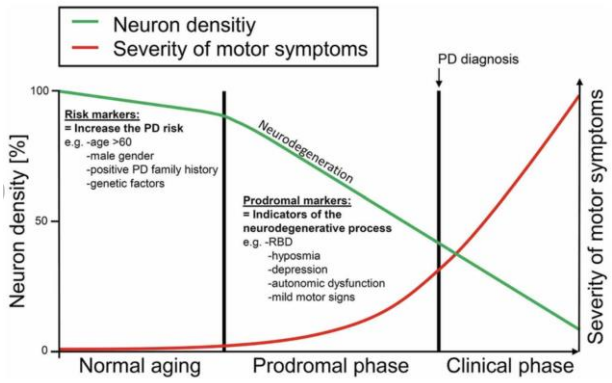

Figure 1. Graphical illustration of the prodromal phase with early neurodegenerative changes occurring years or even decades before the clinical diagnosis of Parkinson's disease (PD) can be made. Risk markers may increase the PD risk without directly being associated with neurodegenerative changes. Only slight loss of neurons due to the normal aging process can be detected in this phase. Prodromal markers represent indicators of the early neurodegenerative process in which neuronal loss is accelerated. This phase may ultimately lead to PD. Risk markers increase the likelihood that an individual may enter the prodromal phase and may finally develop motor PD. Prodromal markers may indicate the ongoing neurodegenerative process and may help to identify those individuals who will likely develop PD in the future. Reproduced from[1].

In addition, there are several occupational exposures that have been associated with Parkinson's disease and Parkinsonism in general (presentation of symptoms reminiscent of Parkinson's disease, but due to other causes). In that sense, manganism is caused by the manganese in welding fumes which have been shown to accumulate in the brains of workers and cause a condition that has many of the features of Parkinson's disease. Although the pathology of manganism is distinct from Parkinson's, those exposed to manganese have a higher incidence of classical Parkinson's disease. There have been numerous studies that show that exposure to solvents such as xylene, toluene, TCE, and PCE tend to cause a wide range of neurological dysfunction. This includes motor impairment similar to that seen in Parkinson's disease and overt Parkinson's disease in many cases. There are also a wealth of studies linking exposure to several different pesticides increases risk of developing Parkinson's disease including work from my laboratory[64,65,67-69,71-73] and others.[74-96]

Among the chemical exposures linked to Parkinson's Disease, TCE stands out as the solvent with the most compelling evidence of a direct causal link to Parkinson's disease,

based upon the epidemiological evidence, mechanistic data, and animal data (see chemical structure in Figure 2).[97-100] Ranging from acute exposures, occupational exposures, and environmental exposures, TCE has been shown to increase Parkinson's disease risk. The laboratory studies in which animals are exposed to defined amounts of TCE also provide compelling evidence for the connection.

The growing body of epidemiological evidence linking TCE to Parkinson's Disease is strong. In 2012, Sam Goldman MD, a physician and Parkinson's Disease research scientist at the University of California, initiated a study on twins, many of whom had been exposed to TCE. The study found an extremely high correlation between TCE exposure and Parkinson's disease (Relative Risk Ratio=6.1 95% confidence interval 1.2-33; this indicates a 6.1-fold increase in incidence in those exposed with the 95% confidence interval ranging from 1.2 to 33-fold higher. The 95% confidence interval suggests that if one repeated the study 100 times that the same results would be found 95% of the time).[101] In addition, the study also found a significantly elevated risk for Parkinson's Disease in those twins who were exposed to PCE (Relative Risk Ratio=10.5, 95% confidence interval .97-113; this indicates a 10.5-fold increase, but the confidence interval ranges from 0.97-fold risk to 113-fold risk—when the confidence interval falls below 1 we consider this to not be statistically significant although this doesn't rule out biological relevance). The exposure to PCE was highly associated with Parkinson's Disease, but the confidence interval indicated that the data were not robust enough to achieve statistical significance even though the data suggested up to a hundred-fold increase in risk (often a slightly larger study population would allow such an association to achieve statistical significance). Risk for Parkinson's was also significantly increased for combined exposures to TCE and PCE (Relative Risk Ratio=8.9, 95% confidence interval 1.7-47).[101] The significant increase in the combined exposure is especially germane given that in many settings these chemicals are found together as they were at Camp Lejeune.

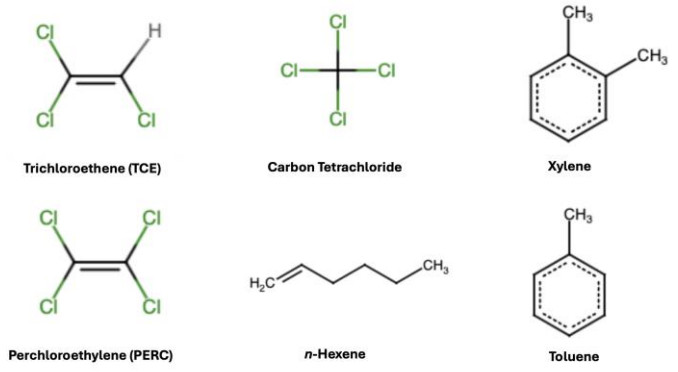

Figure 2. Common industrial solvents with known neurological effects. TCE, PERC a.k.a.PCE, and carbon tetrachloride are all solvents that have been used in dry cleaning and degreasing applications. The chlorine atoms enhance their fat solubility and likely contribute to their ability to disrupt brain function.

Dr. Goldman's population-based study revealed findings consistent with prior case reports and with a rodent model of TCE induced Parkinsonism that confirmed the biological plausibility through the loss of dopaminergic neurons in the substantia nigra and the accumulation of alpha-synuclein protein in the rodent brains. The study design, utilizing discordant twin pairs, reduced effects from genetic confouders and bias.[101]

In 2024, Frank Bove PhD, an epidemiologist at the Agency for Toxic Substances and Disease Registry at the Centers for Disease Control, analyzed mortality among personnel at Camp Lejeune, a base whose water supply was contaminated, in part, with TCE and PCE, to Camp Pendleton, another Marine base across the country that was not known at that time to be contaminated with TCE or PCE. The study revealed, in part, higher mortality ratios due to Parkinson's disease at Camp Lejeune[102,103]

In 2023, Dr. Goldman, who published the twin study noted above, performed a cohort study analyzing the risk of Parkinson's Disease at Camp Lejeune with that at Camp Pendleton. The results of his study revealed a 70% higher risk of Parkinson's for those who had been exposed to the TCE contaminated water at Camp Lejeune for longer than 3 months, during the 1975-1985 time period (Relative Risk Ratio=1.7, 95% confidence interval 1.39-2.07).[100] The 2023 Goldman study concerns portions of the very population that is the subject of this lawsuit. The study period was a ten-year period that involved modeled levels of TCE, which were calculated to be in the range of 366 ppb as an average monthly level. Of the epidemiology studies to date, this study provides the strongest evidence of the link between TCE and Parkinson's Disease, and at lower levels of exposure.[100] A key characteristic of this study was that the study team included neurologists who specialize in Parkinson's disease which increases the confidence in the diagnosis of the disease based on medical records.

As previously noted, there is strong animal and mechanistic data supporting the epidemiological studies and the conclusion that TCE is a cause of Parkinson's Disease. Mice and rats exposed to TCE via ingestion have been shown to demonstrate a significant reduction of dopamine producing neurons in the substantia nigra, a hallmark feature of Parkinson's disease.[104-108]

More recently, a study performed by Dr. Briana De Miranda, who runs a Parkinson's disease laboratory in the Department of Neurology at University of Alabama-Birmingham, tested mice and rats exposed to low doses of TCE via inhalation.[97] The team exposed the rodents to levels lower than the regulatory TCE limits, as part of a 12-week study. The experiment produced a significant loss of dopamine producing neurons, as well as the accumulation of alpha-synuclein which is a pathological feature of Parkinson's disease.

These animal studies confirm that TCE causes loss of dopamine producing neurons in the substantia nigra, a key feature of Parkinson's Disease. In addition, the animal and mechanistic data confirm that by multiple routes of exposure, TCE produces consistent dysregulation of mitochondrial function, increased oxidative stress, accumulation of pathogenic alpha-synuclein, as well as motor and behavioral changes in rodents, all of which are features of Parkinson's Disease.[97,108,109]

There are three major routes of exposure to TCE: oral, inhalation, and dermal. Recent animal studies have concluded that inhalation of TCE can be as much as 500 times the equivalence of TCE absorption via oral intake due to pharmacokinetic and metabolic

difference from the route of exposure.[108] In Camp Lejeune the military personnel, their dependents, and civilian workers were likely exposed via all three paths. With elevated TCE levels in the water, the TCE would have been ingested orally through drinking the water or other beverages made with the water or via food preparation, including the reconstitution of baby formula or other beverages. Bathing or showering in the water can cause dermal absorption through the skin as well as inhalation through the vapors. TCE can and does evaporate or volatilize from the water creating an opportunity for inhalation exposure. TCE would have volatilized into the air creating an inhalation exposure throughout the base.

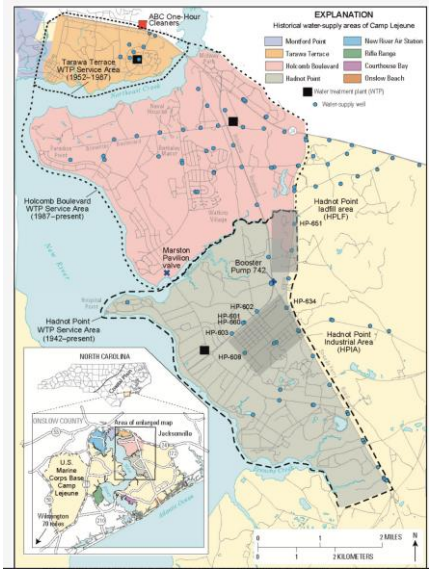

Figure 3. Source of TCE and PCE contamination at Camp Lejeune. There were multiple sources of contamination that led to TCE and PCE exposure. The sources of exposure have been mapped to the living quarters and other key areas on base.[10]

There are extensive models for how TCE gets into people and how it is distributed, metabolized, and excreted.[9] Of specific interest are the metabolites or breakdown products that selectively affect the brain tissue of the substantia nigra dopaminergic neurons in addition to the direct effect of TCE upon entering the body on these vulnerable neurons. While TCE may be eliminated from the body in a matter of hours or days, the metabolites can remain in the body for much longer. The TaClo metabolite has been hypothesized as a toxic metabolite of TCE that can cause direct damage to the mitochondria in the brain tissue.[110] These models are often developed to better understand occupational exposures to TCE, but they are typically focused on a specific type of exposure for a given workplace, e.g. dermal exposure for workers cleaning equipment. In Camp Lejeune, one must consider multiple routes, varying doses, over numerous time ranges, and various metabolites.

Human and animal studies have clearly shown that TCE and/or its metabolites get into the brain. The symptoms of acute high-dose exposures to TCE include dizziness, confusion, and lethargy. These symptoms and other biochemical data clearly show that TCE gets into the brain rapidly after exposure. The blood brain barrier is useless against a fat-soluble solvent like TCE. Chronic exposure to lower levels of TCE should follow the identical pathway. Inhalation exposures can reach the brain directly through the olfactory nerve, from lung-mediated absorption, ingestion, or dermal absorption. The fat-solubility of TCE ensures that independent of the route of exposure, TCE will enter and accumulate in the brain.

Like many solvents, a key feature of TCE if volatility. Having the solvent quickly dry can be key to their utility in consumer products or industrial applications. The majority of the parent compound will be gone within days with some metabolites persisting for a week or more. Continued and persistent intake of TCE through the routes of exposure mentioned above will lead the ongoing presence of metabolites that permeate and attack tissue in the

brain. These molecules can damage neurons, lead to a chronic inflammatory cycle, and lead to the ultimate death of dopamine neurons in the substantia nigra pars compacta. Thus, to a reasonable degree of scientific certainty from toxicology and animal research, there is a causal pathway from environmental TCE, uptake into the body, transport into the brain, deposition in brain areas affected in Parkinson's disease, damage to the dopamine neurons, and the ultimate degeneration of those neurons.

Key Conclusions

1) Individuals at Camp Lejeune between 1953 and 1987 were exposed to levels of TCE and related compounds and metabolites that exceeded currently established safety guidelines.

Studies performed by the Agency for Toxic Substances Disease Registry (ATSDR), which is part of the Centers for Disease Control and Prevention (CDC), have provided exposure models that show high levels of contamination on the base. See Figure 3 for the relative locations of contamination and water sources and Figure 4 for the estimated exposure levels. It is estimated that elevated exposure occurred between 1953 and 1987, but the data are strongest between 1975 and 1985. The TCE and PCE contamination at Camp Lejeune has been clearly established.

2) As a result of exposure to TCE and PCE, those stationed at or near Camp Lejeune have an increased risk of developing Parkinson's disease.

The study of personnel station at Camp Lejeune from 1975-1985 provides the most compelling data.[99,100] By comparing outcomes to personnel station at Camp Pendleton, the research team provided a rigorous examination. The controls of the various aspects of job descriptions, age, lifestyle allowed the study to specifically focus on the primary difference in the population. Furthermore, the documentation of the timing of the exposure and the time at which disease ascertainment was performed provided excellent temporality for a disease with a long latency, such as Parkinson's disease.

3) This increased risk of Parkinson's disease is already demonstrated by higher rates of the disease at Camp Lejeune and there will continue to be an elevated risk for those personnel who have not yet been diagnosed with Parkinson's disease.

In the normal aging process, dopamine neurons of the substantia nigra undergo neurodegeneration, but not in enough to cause Parkinson's Disease. Thus, age is a major risk factor for Parkinson's with the average window of onset being between the ages of 65-85. The etiology of Parkinson's Disease, as described above, is loss of >80% of the substantia nigra dopamine neurons, the accumulation of alpha-synuclein misfolded protein into Lewy bodies across the nervous system, and a neuroinflammatory process, all of which are progressive.

The rate of Parkinson's disease increases as populations age. The Goldman studies stopped collected incident case data after the VA announced that it would cover Parkinson's disease (January, 2017).[99,100] This exclusion was appropriate in that it prevented overestimation errors due to veterans seeking care after hearing that the condition would be covered. However, this fact doesn't mean that the Parkinson's Disease case numbers in this population are not increasing. To a reasonable degree of scientific certainty, based on the incidence of the disease, the known influence of TCE to generate the hallmarks of the disease, e.g., neuroinflammation, alpha-synuclein protein accumulation, and mitochondrial dysfunction leading to dopaminergic neurons death, it can be asserted that there are increased number of Parkinson's Disease cases unaccounted in the study population. Moreover, the Goldman study focused on exposures that occurred between 1975 and 1985 for practical reasons of obtaining the necessary records. This means that there were more than *two decades of exposures that have not been studied* simply due to lack of data. It is scientifically reasonable to conclude that Camp Lejeune veterans in their 80s undoubtedly have a higher rate of Parkinson's disease due to these exposures than their non-exposed peers, whether veterans or civilian. This highlights that the temporality of the study was good for Parkinson's disease it did not cover all of the potential windows of susceptibility and ultimate diagnosis and additional follow up will help identify more cases.

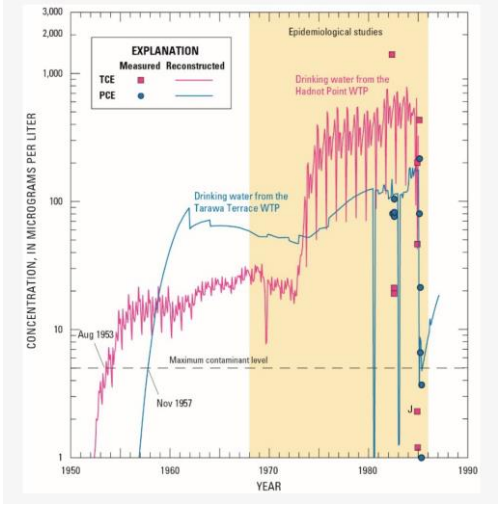

Figure 4. Estimated exposure to TCE and PCE at Camp Lejeune. This figure shows the estimated levels of TCE and PCE at Camp Lejeune. TCE levels were estimated to be higher between 1975-1985, but that PCE was still at unacceptable levels.[10]

4) There is evidence that people stationed at Camp Lejeune who have Parkinson's will likely have more aggressive forms of the disease with worsened symptoms.

The more recent paper by Goldman and colleagues in 2024 suggests that the rate of progression of Parkinson's disease is faster in those stationed at Camp Pendleton.[99] This is a logical conclusion backed by the medical records. If one is exposed to a chemical that accelerates the course of a disease it reasonable that that the chemical would also hasten the progression of the disease and exacerbate symptoms. How this is manifested can be explained by looking at the predicted loss of dopamine neurons and dopamine levels in Parkinson's disease as shown in Figure 1. The figure shows the long latency of the disease and why it must be studied decades after exposure. The observation of the motor symptoms of Parkinson's are needed to make a diagnosis.

Questions to be resolved

5) What level of TCE exposure is required to cause adverse biological effects in humans?

There is a wealth of data on TCE causing adverse biological effects, including cancer. However, for the Camp Lejeune exposure and Parkinson's disease, the focus is on neurological effects. There are several well-controlled studies from occupational exposures that can estimate the acute exposures needed to cause neurological effects, such as dizziness, confusion, and headaches.

While the scientific evidence is clear that exposure to TCE is causal for Parkinson's disease and epidemiological research informs us of the levels of exposure to TCE that can cause Parkinson's disease, it is not feasible to identify a single minimal level of exposure that any one person would eventually be diagnosed with the disease. The reason for such impracticality is that there is simply too much variation in the human population to make such a universal determination. This challenge is similar for nearly every chronic disease, for example, we know that smoking causes lung cancer, but some people who smoke will not get cancer. To be clear, we are not stating there is not a threshold at which TCE causes harm. Rather, as in any biological dependent threshold, this level is different for individuals depending on a myriad of factors. The variation in the human population makes the "minimum dose for an individual" concept, an impractical scientific endeavor. However, it is possible to estimate the average level of TCE exposure that would lead to a significant increase not just in risk of developing Parkinson's disease at some point in the future, but also in the incidence of the disease, as well as developing the early precursor symptoms of the disease (referred to as the prodromal symptoms). Indeed, the data comparing Camp Lejeune to Camp Pendleton from ATSDR and academic investigators demonstrate that the levels of TCE and PCE contamination at Camp Pendleton were sufficient to cause an increased incidence of Parkinson's disease.[99,100,102,111] Such an estimate must consider all routes of exposure and *consider the cumulative exposure over time*. In that respect, a month-long exposure to a very high concentration would give a same cumulative dose as 3 months exposure to 1/3 of that dose. It is the best scientific approach we have in analyzing epidemiological data that linking the exposure to Camp Lejeune to a significant increased risk of Parkinson's disease with the goal of determining a dose range that confers that increased risk and incidence of the disease.

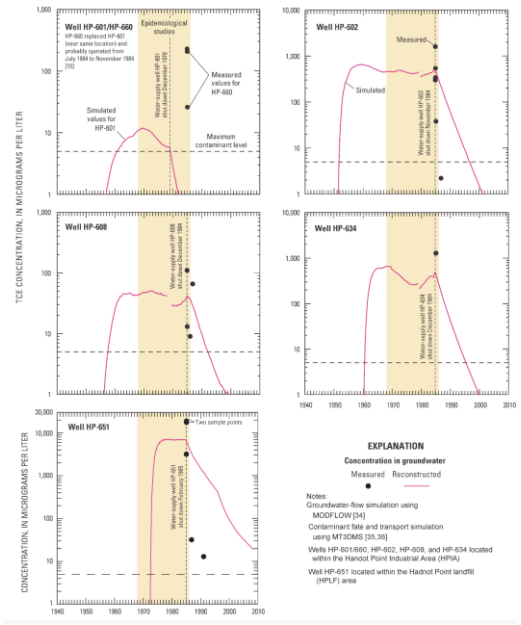

Figure 5. Documentation of TCE contamination is multiple wells that served Camp Lejeune. These models demonstrate that there was widespread contamination across the base over an extended period of time.[10]

As stated above, the study of personnel station at Camp Lejeune from 1975-1985 provides the most compelling data.[99,100] The average level of TCE contamination of the Hadnot Point supplied water has been estimated

at 366 parts per billion (ppb; mean value over the time period, Figure 7).[1-3] Parts per billion is equivalent to micrograms per liter (µg/L) Given that this has been established in the literature as a reasonable number, we can rely to estimate a minimum estimate exposure to the personnel stationed at Camp Lejeune. We must keep in mind that oral consumption is only one of the routes of exposure. It is important to recognize that adding vapor and dermal exposure, which is certain to have occurred to the Camp Lejeune population, would double the overall estimated internal dose.

The low end of the daily estimated liquid consumption is 3 liters. Liquid consumption has been reported to have been higher for certain individuals in training, and lower for other civilian population with an estimated range of 3-6 L/day. It has been estimated that oral consumption of contaminated water represents approximately 1/2 of the total dose of TCE. The vapor and dermal exposure would increase the dose by ~ two-fold. To be conservative, one could estimate that the vapor and dermal exposure would increase the dose by 2/3 or 67%. Thus, if one uses the lower end of the estimate of 3 liters per day and assumes other sources represent an additional two-thirds (67%) exposure then one can use an oral dose of 5 liters consumption/day as a proxy to represent to total exposure from combined routes (oral, vapor/inhalation, and dermal, 3 liters x 1.67 = 5 liters).

Given that the composite estimate (median) of TCE contamination on base was 366 µg/L we can simply multiply 5 liters by 366 µg/L to get an estimate of the amount of TCE consumed in a single day: 5L x 366 µg/L = 1,830 µg or 1.83 mg. Thirty days would lead to a cumulative exposure of 54.9 milligrams of TCE (this translates into 54,900 ppb/month or ~55 ppm/month). The 2023 Goldman paper concluded the PD incidence was higher in personnel who spent at least three months at Camp Lejeune, therefore, the dose of TCE that they received over a three-month period must be sufficient to cause the increased incidence.[100] If the estimated daily dose was 1,830 µg then 90 days (3 months) x 1,830 µg = 164,700 µg or 164.7 mg over the course of 90 days.[43,44,47]

Referring back to the data from Goldman that suggested that three months of exposure at Camp Lejeune is sufficient to increase the incidence of Parkinson's disease,[100] one can conclude that the estimated dose required to increase the incidence of Parkinson's disease must be equivalent to the amount of exposure that would occur over 90 days. To a reasonable degree of scientific certainty, based upon a review of the published literature and literature which I have contributed, along with the known neurotoxicity and effects of TCE, I have concluded that a cumulative dose of >**150 mg** (within 10% of the 164.7 mg calculated dose) cumulative dose is sufficient to increase the incidence of Parkinson's Disease in human beings (with a latency of up to 30-50 years).

Whether the cumulative intake exceeding 150 mg occurs over one month or six months would not likely make a difference. Thus, a person stationed at Camp Lejeune who was in a workplace setting or housing area (Figure 6) with higher-than-average exposure to TCE could achieve that level in one month, compared to the level achieved by someone on the

lower end of the exposure scale, achieving that level in six months or longer, depending on the concentration.

Dose-response relationships in experimental studies

Laboratory animal studies to model human exposures and disease. It has often been cited that the dose levels used in animal laboratory studies can be much higher than the levels to which human beings are exposed. In mouse or rat studies, much larger doses are often given in toxicology. This should not be viewed as a flaw. Humans live for over seven decades and rodents live for just a couple of years. Rodents have much higher rates of metabolism meaning that they break down chemicals faster that humans. Given the increased rate of metabolism in rodents, it is common in acute and even chronic toxicology studies to use doses much higher than shoe seen in human exposures.[36] In addition, toxicological studies are designed to identify risks and study mechanisms of disease. We increase exposure levels and often shorten duration times to generate scientific evidence faster. We observe the same pattern in toxicological studies for Parkinson's disease. In mouse or rat studies, high doses of suspected toxicants are given via routes of exposure that are amenable to laboratory studies (often orally or injected). Many of these studies have been used to cause specific dopamine neuron damage in a matter of days with compounds, including TCE. However, inhalation exposures to TCE in rats and mice, are harder to conduct, but have great value because they more closely mimic this critical route of exposures. It is notable that inhalation studies bypass the effects of higher liver metabolism seen in rodents, but rodents still have higher metabolic rates in peripheral regions including the brain. Indeed, inhalation exposures of TCE cause marked dopamine neuron degeneration in a matter of weeks. The inhalation exposure ranged from 50-100 parts per million (ppm), which is arguably higher than what occurred at camp Lejeune in terms of ingested water (Figure 7,8). However, as a toxicologist, these rodent inhalation studies are much more comparable to the effect on human beings as human occupational studies have demonstrated that TCE exposure in the workplace can range from 400ppb to 230ppm. Specifically, the fact that 50 ppm caused effects in rodents via inhalation in 8 weeks[97] conclusively establishes that the cumulative effect of TCE intake, along with inhalation via vapors is sufficient to cause the pathogenesis of the cellular damage which will eventually be diagnosed as Parkinson's disease. This is very well-aligned to the >150 mg dose calculation to cause Parkinson's disease in humans. Therefore, the doses of TCE used to cause PD toxicity in rodents is strikingly similar to what is seen in humans and this is not what is typically expected. For the majority of toxicants, it takes doses 10-100 x more than what human are exposed to in order to replicate similar symptoms or toxicity. This is not a limitation of toxicology per se, but represents the reality that modeling a disease that occurs in humans who are 70 years old is difficult in a short-lived rodent model. Such studies require more intense exposures to replicate the physiological impact of acute exposures on decades of live. The fact that the toxicity of TCE was observed at a dose similar to human exposures is rather extraordinary and suggests that at cumulative doses <150 mg are likely sufficient to cause Parkinson's disease.

6*) What are the biological mechanisms by which TCE exposure preferentially kills dopamine neurons in laboratory models or in humans?*

Illustrated below, if an environmental factor accelerates the rate of dopamine neuron death it will lead to an earlier diagnosis if Parkinson's disease. If a person was genetically predisposed to get Parkinson's disease they could have a more aggressive progression and be diagnosed as a younger age.[99-101] Although the details of the molecular mechanisms by which TCE kills dopamine neurons do not need to be known to make a connection to a disease outcome, it is still helpful to determine if there are plausible biological mechanisms to explain the toxicity. There is substantial literature that TCE impacts several molecular targets in ways that are deleterious to the health of dopamine neurons. Oxidative stress is a general term that describes the disruption of biological pathways that regulate many biological systems. Oxygen is critical to sustain life, but many of the reactions that occur in the presence of oxygen can damage cellular molecules. The mitochondrial play key roles in generating energy and signaling many biological pathways. Damage to mitochondria has been shown to cause Parkinson's disease in humans and animal models.[65,66,69,94] Activation of the Parkinson's disease-related gene, LRRK2, is known to increase Parkinson's disease incidence and TCE leads to its activation.[108,112,113] Animal studies clearly show that administration of TCE to mice or rats can specifically kill their dopamine neurons and lead to Parkinsonian hallmarks and symptoms via multiple pathogenic pathways.

7) Are there any plausible alternate explanations that would counter any of the conclusions from above?

What if it isn't TCE that is causing the death of the dopamine neurons but a related compound or a metabolite of TCE? If the source of the related compound is similar to TCE or if it is a metabolite of TCE, then nothing changes as it is the parent compound exposure that initiates the causative cascade. It is the exposure at Camp Lejeune that is responsible. What if it was the related PCE causing the adverse effects? PCE does have similar toxicity to TCE, so it is possible that PCE could be a contributing factor, but it is still a component of the contamination at Camp Lejeune. Whether it is TCE or PCE doesn't change the underlying association to the documented contamination. If future animal studies demonstrate that PCE exerts similar toxicity to dopamine neurons as TCE it would not negate the scientific evidence that TCE can kill dopamine neurons, it would only make the contamination more obviously toxic in that multiple constituents have evidence of killing dopamine neurons. This line of reasoning would apply to any related chemical that was used by the industries that caused the proximate contamination. Could it be something totally unrelated to the contamination? For example, something food-borne on base? This seems unlikely in that most food-borne illness present with rather dramatic acute symptoms and the medical staff would have readily detected that. The epidemiological evidence clearly demonstrates that being stationed at Camp Lejeune increased risk of Parkinson's disease. Might Camp Lejeune have had a different regimen of insect control on base than Camp Pendleton? Perhaps, although at that time (1950s-1970s) the persistent

organic pesticides like DDT, DDE, and dieldrin were widely used throughout the U.S. (as well as during military operations). Interestingly, these compounds have been associated with Parkinson's disease, but there is no evidence of a differential exposure between the two bases and I am not aware of any evidence of significantly different use of pesticides on a regional or national level that could explain the findings.

TABLE C-1 Plasma Half-life of Trichloroacetic Acid

Route	Administered Dose/Concentration	Species (sex)	Half-Life (h)	Reference
Intravenous injection	5-6 mg/kg TCA	Rat (male)	12	Fisher et al. 1991
		Rat (female)	7	
Intraperitoneal injection	5-10 mg/kg TCA	Mice (male)	7	Fisher et al. 1991
		Mice (female)	3	
Inhalation	42-889 ppm TCA	Mice (male)	16	Fisher et al. 1991
		Mice (female)	7	
	500-600 ppm TCA	Rat (male and female)	15	Fisher et al. 1989
	50 or 100 ppm TCE	Human	86-99	Fisher et al. 1998

ABBREVIATIONS: TCA, trichloroacetic acid; TCE, trichloroethylene.

Figure 6. Trichloroacetic acid is one of the primary metabolites of TCE. This table illustrates a major challenge for TCE exposure in humans. In studies using relatively low levels of exposure to TCE in humans the half-life is over 90 hours or nearly 4 days. Because of the ability of TCE and its metabolites to partition into lipid-rich compartments like the brain, intermittent exposures could lead to persistent levels for weeks or even months. Most of the laboratory work is focused on rodents that metabolize the compounds ten times faster. From the ATSDR ToxProfile of Trichlorethylene.

There has been speculation that soil microbes may contribute to Parkinson's disease, but there is no solid human evidence. The idea that a microbe in the soil at Camp Lejeune could be responsible seems completely unfounded. Unlike the identification of the TCE and other contamination and eventual mitigation, a microbial explanation would persist. Such a geographically distinct exposure to a microbe would have been detected over the past decades. Given the relatively short duration of the personnel and the long period of time between exposure on onset of disease, I can identify no reasonable alternative explanation for the increased incidence of Parkinson's disease in Camp Lejeune personnel. There is a notable review that was published in 2013 that was sponsored by the Halogenated Solvent Industry Alliance, Inc. This industry-sponsored paper which was prepared by reputable academic scientists concluded the paper by stating that "TCE may have etiological relevance (for Parkinson's disease)." Thus, even this industry-sponsored review had to admit that there was evidence for TCE contributing to PD.[114]

PCE and Parkinson's

Tetrachloroethylene (tetra meaning four, as in the number of chlorine atoms in the molecule; see Figure 2), also referred to as perchloroethylene, is closely related to TCE in its applications, exposures, and toxicity. In many industrial settings it is referred to as Perc or PERC; however, PCE is the preferred abbreviation and is used in this report. There have been fewer epidemiological and laboratory studies of PCE than TCE, but the chemicals are quite similar. The only difference is that PCE has four chlorines and TCE has three chlorines (see Figure 2). Also shown in Figure 7, they have common metabolic byproducts. It is also generally recognized that it is the chlorine molecule that gives these compounds their desired solvent properties, but that also leads to toxicity. Highly chlorinated molecules have been shown to be toxic to humans for decades.

With its similar structure to TCE and the fact that it contains more chlorine atoms, it is reasonable to conclude that PCE will have similar biological effects to TCE. In fact, based on my training, education, and experience as a toxicologist, my review of the current literature, my own laboratory research, it is my professional opinion that to a reasonable degree of scientific certainty, PCE has molecular features needed to produce the same biological effects as TCE, including causing the same type of damage to neurological tissue that I have outlined in this report. The same logic that I have applied to TCE is applicable to PCE, by analogy per Bradford Hill. Thus, PCE is, whether standing alone or in combination w/ other chlorinated solvents is as likely as not to cause the damage to the neurological tissue that results in Parkinson's disease, along with its complications. As noted above, the risk for Parkinson's disease at Camp Lejeune was significantly increased for combined exposures to TCE and PCE (Relative Risk Ratio=8.9, 95% confidence interval 1.7-47).[101] This supports the contention that TCE and PCE have similar effects on human biology, especially as it relates to dopamine neuron cell death and Parkinson's disease incidence. Academic researchers have focused on TCE and that is why there are more papers on TCE. That said, the observed risk for Parkinson's from combined exposures to TCE and PCE was 8.9, 95% confidence interval 1.7-47).[101] TCE is the solvent with the strongest evidence of a causal connection to Parkinson's disease, and it is my professional opinion that TCE, is at least as likely as not, a cause of the pathogenesis and progression of Parkinson's Disease. Additionally, PCE, a similar chlorinated solvent has the same potential toxicity to the dopamine neurons in the substantia nigra is also at least as likely as not to be a cause of pathogenesis and progression of Parkinson's disease.

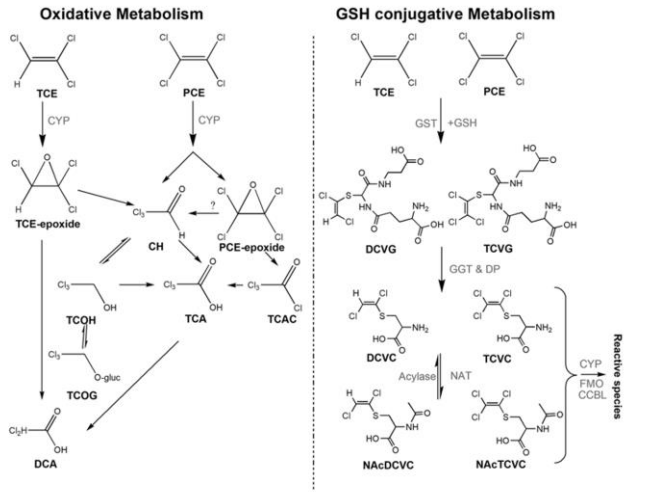

Figure 7. Overview of the metabolic pathways of TCE and PCE. Upon absorption, TCE and PCE can either be oxidized via CYPs or conjugated with GSH via GSTs. Abbreviations: TCE, trichloroethylene; PCE, tetrachloroethylene; CH, chloral hydrate; TCOH, trichloroethanol; TCA, trichloroacetate; TCAC, trichloroacetyl chloride; TCOG, trichloro-glucuronide conjugate; DCA, dichloroacetate; DCVG, S-(1, 2-dichlorovinyl)-GSH; DCVC, S-(1, 2-dichlorovinyl)-cysteine; NAcDCVC, N-acetyl-S-(1, 2-dichlorovinyl)-cysteine; TCVG, S-(1, 2, 2-trichlorovinyl)-GSH; TCVC, S-(1, 2, 2-trichlorovinyl)-cysteine; NAcTCVC, N-acetyl-S-(1, 2, 2-trichlorovinyl)-cysteine; GGT, gamma-glutamyl transferase; DP, dipeptidase; NAT, n-acetyl transferase; FMO, flavin monooxygenase; CCBL, cysteine conjugate beta-lyase. Adapted from.[9]

Summary of causative evidence

Here I will review the Bradford Hill framework, which includes temporality, strength, consistency, specificity, biological gradient, plausibility, coherence, experiment, and analogy. Most of the components of Bradford Hill have been fulfilled including temporality,

strength, consistency, biological gradient, plausibility, coherence, experiment and analogy. The study was very well-powered by studying over 300,000 people, which provided strength. The study population included people serving over the course of a decade, which means there were numerous cohorts being studied, this demonstrates consistency, in that these cohorts were present at different times of year and during different times in the decade. It also creates a level of reproducibility. Human and animal studies provide excellent support for biological gradient and dose-response. The higher exposure to TCE the worse the neurotoxicity appears to be. There is strong evidence that higher levels of exposure to TCE leads to higher levels in the brain and more severe central nervous system symptoms. The alignment of the human and animal studies, including the loss of dopamine neurons across species addresses the plausibility, coherence, and experimental aspects of the Bradford Hill criteria. The molecular and cellular pathways disrupted by TCE provide additional plausible biological explanations for the chemical can lead to death of the dopamine neurons. The fact that known TCE exposure in humans causes Parkinson's disease and TCE exposure in rodents causes death of the same neurons that die in human Parkinson's disease clearly demonstrates coherence. The dopamine neurons that reside in the substantia nigra pars compacta are quite unique. Few chemicals are known to damage these neurons across species. MPTP and rotenone have been shown to do this.[115-117] The data do not support specificity of TCE for Parkinson's disease in that the chemical has many adverse effects, e.g. increasing cancer incidence. Thus, there is not specificity for which cells in the body TCE damages. It is possible that there are common mechanisms of toxicity which may be more specific, but Parkinson's disease is not an exclusive adverse consequence of TCE exposure.

Conclusion

The scientific and medical literature linking TCE to Parkinson's disease is compelling. The human studies are of very high-quality. The laboratory studies in rodents were well-designed and of very high-quality. The data on the pharmacokinetics of TCE are extensive. The recent studies comparing personnel at Camp Lejeune and Camp Pendleton are outstanding examples of epidemiological research based on the experience of the research teams and excellent methodology. The data are robust. The temporality is well-established. The laboratory-based studies demonstrate that controlled administration of TCE in animals damages the same dopamine neurons that are lost in humans with Parkinson's disease both via oral administration and inhalation. There are several occupational studies that document the levels of exposures that cause neurological symptoms. In my professional opinion to a reasonable degree of scientific certainty that a sufficient number of the Bradford Hill components[49] have been met, as well as other characteristics one would desire in establishing causality.

Together, these lines of evidence converge on a clear line of reasoning that the cause of the increased risk of developing Parkinson's disease among military personnel stationed at Camp Lejeune during the years of 1975 and 1985 was their exposure to trichloroethylene (TCE) and the related tetrachloroethylene (PCE) on the base. There are no other plausible

explanations for the observed elevated risk. Furthermore, as these personnel continue to age, we will see this heightened risk manifest in new cases over the next several years.

Based upon a review of the scientific evidence, my training, education, and scientific experience, and to a reasonable degree of scientific certainty, it is my professional opinion that TCE is, more likely than not, a cause of Parkinson's Disease. Therefore, if one considers all of the data that has been reviewed and considered in this report, it is reasonable to conclude that the scientific community has provided ample evidence that TCE exposures are causal for dopamine neuron death in the substantia nigra of laboratory animals. Furthermore, the 2023 Goldman study revealed that levels of TCE and PCE exposure documented at Camp Lejeune were sufficient to cause a statistically significant risk and incidence of Parkinson's disease revealing a causal connection between TCE/PCE and Parkinson's disease.

Based on the known chemical similarity between TCE and PCE and the common routes of exposure, metabolism, and toxicity, my training, education, experience, and to a reasonable degree of scientific certainty, it is my professional opinion that PCE is at least as likely as not to be a cause of Parkinson's disease. Furthermore, it is my professional opinion that PCE exposure contributed to the increased incidence of Parkinson's disease observed in those stationed at Camp Lejeune.

I am being compensated at a rate of $350/hour for my time devoted to performing research, analyzing data, and preparing this report.

Dated 7 December 2024.

Gary W. Miller, PhD

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04/1995 – 01/1996 *Center for Neurological Sciences Postdoctoral Fellow*
Department of Neurology
School of Medicine
Emory University
Atlanta, GA

03/1994 – 03/1995 *Society of Toxicology Predoctoral Fellow (sponsored by Procter and Gamble)*
Department of Physiology and Pharmacology
College of Veterinary Medicine
University of Georgia
Athens, GA

03/1994 – 03/1995 *Graduate Research Assistant*
Department of Physiology and Pharmacology
College of Veterinary Medicine,
University of Georgia
Athens, GA

07/1990 – 05/1992 *Graduate Research Assistant*
Department of Biology
Old Dominion University
Norfolk, VA

08/1989 – 05/1990 *Graduate Research Assistant*
Noll Laboratory
Penn State University
University Park, PA

Education

07/1997 - 09/1998 *Postdoctoral Fellowship*
Regulation of Monoamine Transporters, Advisor- Dr. Marc G. Caron
Department of Cell Biology
Howard Hughes Medical Institute,
Duke University Medical Center
Durham, NC

04/1995 - 06/1997 Postdoctoral Fellowship
Dopamine Transporters in Parkinson's Disease, Advisor- Dr. Allan I. Levey
Department of Neurology
School of Medicine
Emory University School of Medicine
Atlanta, GA

06/1992 - 03/1995 Doctor of Philosophy in Pharmacology and Toxicology
Novel Mechanisms of Cytoprotection in Renal Proximal Tubules Proximal Tubules
Advisor: Dr. Rick G. Schnellmann
Department of Physiology and Pharmacology
College of Veterinary Medicine
The University of Georgia
Athens, GA

08/1990 – 05/1994 Master's of Science in Biology with emphasis in Immunology
Advisor: Dr. Robert Ratzlaff
Department of Biology
Old Dominion University,
Norfolk, VA

08/1985 - 05/1989 *Bachelor of Science cum laude in Exercise Physiology*
Department of Exercise Science
Old Dominion University
Norfolk, VA

Honors and Awards

Co-Chair, Banbury Conference on Integrating Exposomics into the Biomedical Enterprise, Cold Spring Harbor Laboratory (2023)
Jacob Hooisma Distinguished Lecture, International Neurotoxicology Association (2023)
Elected Fellow, American Association for the Advancement of Science (2022)
Keynote Lecture, European Union Human Exposome Project Kick Off Meeting, Brussels, Belgium (2020)
Invited Lecture, Mayo Clinic Personalized Medicine Conference, Rochester, MN (2020)
Visiting Professor, University of Paris, Descartes (2018)
Kuna-Synder Distinguished Lectureship, Rutgers University (2018)
Burroughs Wellcome Distinguished Lecture, NC State University (2015)
Daniel J. Zaffarano Lecture, Iowa State University (2014)
Asa Griggs Candler Professorship, Emory University (2012)

Georgia Research Alliance Distinguished Investigator (2011-2018)
Mentor of the Year, Emory Graduate Division of Biomedical and Biological Sciences (2010)
Achievement Award, Society of Toxicology (2010)
President, Neurotoxicology Specialty Section, Society of Toxicology (2011-2012)
Vice-President, Neurotoxicology Specialty Section, Society of Toxicology (2010-2011)
Councilor, Southeast Regional Chapter of Society of Toxicology (2009-2010)
National Institute of Environmental Health Sciences Top Papers of 2007
Chair, Gene-Environment Interactions in Neurodegeneration Session at Gordon Research Conference on Mechanisms of Toxicity, 2008.
Chair, Parkinson's Disease Poster Session, Society of Toxicology (2009)
Society of Toxicology, Neurotoxicology Specialty Section Top Abstracts of 2008
Co-Chair, Environment and Neurodegeneration Symposium at the 2007 Neurotoxicology Meeting
Co-Chair, Parkinsonism and the Environment, Lessons from the Clinic and Laboratory, Workshop at 2007 Society of Toxicology Annual Meeting
Woodruff Leadership Academy, Woodruff Health Science Center, Emory University (2005)
Co-Chair, Pesticides and Nervous System Function Symposium, 2005 Society of Toxicology Annual Meeting
President, Southeast Chapter of the Society of Toxicology (2003-2004)
Dean's Fellow, University of Texas (1999)
Society of Toxicology Neurotoxicology Specialty Section Postdoctoral Award, (1997)
National Research Service Award, National Institutes of Health, (1996 to 1998)
Center for Neurological Sciences Postdoctoral Fellowship, Emory University, (1995-1996)
Society of Toxicology Graduate Student Fellowship, - Procter and Gamble, (1994-1995)
Phi Kappa Phi, University of Georgia (1995)
Society of Toxicology Graduate Student Travel Award (1995)
First Place, Southeast Chapter of the Society of Toxicology Graduate Student Awards, (1993)
Phi Kappa Phi, Old Dominion University (1989)
Honors Program Scholarship, Old Dominion University (1985-1989)
FDA Research Award, Prince George's County Science Fair (1984)
Sigma Xi Research Award, Prince George's County Science Fair (1984)

Administrative Leadership and Academic Service

Editor-in-Chief, Exposome (published by Oxford University Press, first journal in field: 2021-present)
Editor-in-Chief, Toxicological Sciences (official journal of Society of Toxicology; 2013-2019)
Reviewer, Foundation Recherche Medicale, France (2021)
Human Biomonitoring for the European Union, Work Package Review (2017-2018)
Chair, Environmental Triggers of IBD Review Panel, Crohn's and Colitis Foundation (2017)
NIH, NIEHS Intramural Tenure Track Review Committee (2013, 2018, 2020)
Chair, Canada Foundation for Innovation Grant Review Panel (2017)
Chair, CHEAR Access Committee, Grant Review for NIH Children's Health Exposure Analysis Resource (2015-2018)
Associate Editor, Neurotoxicology (2009 to 2012)
Member, Editorial Advisory Board, Journal of Pharmacology and Experimental Therapeutics (2001 to 2010)
Member, Editorial Board, Journal of Applied Toxicology (2010-2012)
Member, Editorial Board, Toxicology Letters (2008 to 2011)
Member, Editorial Advisory Board, Toxicological Sciences (2010-2013)
Member, Editorial Advisory Board, Neurotoxicology (2007 to 2009)

Co-Chair, Environment and Neurodegeneration Study Section, National Institute of Environmental Health Sciences (2014)
Member, Neurotoxicology and Alcohol (formerly ALTX-3) Study Section, National Institutes of Health (2002 to 2007)
Ad hoc Reviewer, National Institute of Environmental Health Sciences Training Grants (2007) Review, NIEHS Superfund Basic Research and Training Program (2008)
Reviewer, NIEHS Toxicology Center Pilot Grants for Vanderbilt University (2008; 2013)
Reviewer, Collaborative Consortium for Environmental Parkinson's Disease Research Pilot Project Program (2003, 2004, 2005, 2006)
Ad hoc Reviewer, North Carolina Biotechnology Center Institutional Development Grant program. (2000)
Ad hoc Reviewer, National Institutes of Health Neuroscience Fellowship Study Section (ZRG1 F03B) (2001, 2007)
Ad hoc Reviewer, National Institutes of Health. Proteomics in Environmental Health Research (2002)
Ad hoc Reviewer, National Institutes of Health Fetal Basis of Adult Disease (2004)
Ad hoc Reviewer, National Institutes of Health Neuroscience Blueprint Centers (2006)
Ad hoc Reviewer, National Institutes of Health Neuroepidemiology (NAME) (2005, 2006)
Ad hoc Reviewer, National Institutes of Health NIEHS K12 Centers (2007)
Ad hoc Reviewer, Center for U.S. Civilian Research and Development Foundation (2001-2002)
Ad hoc Reviewer, NIH Loan Repayment Program (2007-2008)
Reviewer, Research in Ageing, United Kingdom (2008)
Reviewer, Parkinson's Disease Society, United Kingdom (2008)
Reviewer, Medical Research Council, United Kingdom (2008, 2009)
Reviewer, Beatrix Fonds Foundation, Netherlands (2009)

Ad hoc Reviewer for the following journals:
ACS Chemical Neuroscience
Annals of Neurology
Biochemical Pharmacology
Brain Research
Brain Research Reviews
Chemical Research in Toxicology
Environmental Health Perspectives
Environmental Toxicology and Pharmacology Experimental Gerontology
Experimental Neurology
Free Radicals in Biology and Medicine
Human Molecular Genetics
Journal of Cellular and Molecular Medicine
Journal of Nanoscience and Nanotechnology
Journal of Neurochemistry
Journal of Neuroscience
Journal of Neuroscience Research
Journal of Pharmacology and Experimental Therapeutics Journal of Toxicology and Environmental Health
Journal of Toxicology and Environmental Health
Medicinal Research Reviews
Molecular Brain

Molecular Pharmacology
Neurobiology of Aging
Neuromolecular Medicine
Neuropharmacology
Neuroscience
Neuroscience Letters
Neuropsychopharmacology
Neuropsychopharmacology and Biological Psychiatry
Neurotoxicology
Neurotoxicology and Teratology
Pesticide Biochemistry and Physiology
Pharmacology, Biochemistry, and Behavior
Proceedings of the National Academy of Sciences
Reproductive Toxicology
Stem Cells
Synapse
Toxicology
Toxicological Sciences
Toxicology and Applied Pharmacology
Toxicology Letters
Transgenic Research
Trends in Pharmacological Sciences

Professional Organization and Societies

Society of Toxicology (1992 to present)
Society of Neuroscience (1995 to present)
Center for Neurodegenerative Diseases, Emory University (2002-2018)
American Society for Pharmacology and Experimental Therapeutics (2005-present)
Institute of Cellular and Molecular Biology, University of Texas (1999-2002)
Research Society on Alcoholism (2000 -2002)

COMMITTEES

Member, National Advisory Environmental Health Sciences Council (NIEHS Council; 2022-present)
Member, NEXT Working Group to NINDS Council (2024)
Member, NIH All of Us Scientific Program Advisory Board (2020-present)
Member, Scientific Advisory Board, NIH HHEAR program (2019-2024)
Member, Advisory Board Member, Human Biomonitoring for the European Union (HBM4EU) (2017-2022)
Chair, Canada Foundation for Innovation Grant Review Panel (2017)
Member, Emerging Sciences for Environment Health Decisions, Standing Committee, National Academy of Sciences (2016-present)
Member, Committee on Toxicology, National Academy of Sciences (2017-present)
Member, Woodruff Health Sciences Center Research Strategic Planning Committee (2009-2011)
Chair, Rollins School of Public Health Research Advisory Committee (2011-2012; formed committee, served as chair for first year, then transferred leadership to faculty member)
Emory Neuroscience Initiative Leadership Committee (2007-2010)
Chair, Institutional Health and Biosafety Committee, Emory University (2006-2015), Member (2004-2015)

Chair, Research Health and Safety Committee (2013-2015)
Appointment, Promotion, and Tenure Committee, Rollins School of Public Health (2007-2010)
Woodruff Health Science Center Research Advisory Council (2008-present)
Neuroscience Brainstorming Committee for Emory Strategic Plan (2005)
Parkinson's Disease Brainstorming Committee for Emory Strategic Plan (2005)
Neuroscience Strategic Planning Steering Committee (2005-2006)
Search Committee, Department of Environmental and Occupational Health for Department Chair (2006)
Search Committee, Institutional Biosafety Officer, Emory University (2005)
Search Committee, Information Technology Director, School of Public Health, Emory University (2005)
Molecular and Systems Pharmacology Graduate Program Executive Committee (2004-present) Chair, Chemical Safety for Animal Care Staff Task Force (2004-present)
Chair, Organizing Committee, Southeast Society of Toxicology Annual Meeting (2004)
Chair, Steering Committee, Collaborative Centers for Parkinson's Disease Environmental Research (2004-2005); Member (2002-present). A multi-site research program including Emory University, UCLA, and the Parkinson's Institute.
Search Committee, Department of Environmental and Occupational Health for 8 faculty members, (2002-present)
Search Committee, Center for Neurodegenerative Diseases for 4 faculty members (2002-2006) Vice President for Research Task Force on Research Website, University of Texas (2000-2001) Institutional Biosafety Committee, University of Texas (2000-2002)
Organizing Committee for Texas Neurobiology of Addiction Meeting, (2000)
Chair, College of Pharmacy Biological, Radiological, and Biohazard Safety Committee, (2000-2002)
Organizing Committee for Southcentral Society of Toxicology (2000)

EXTRAMURAL FUNDING

ACTIVE

ARPA-H D24AC00345 (Miller, PI) 9/11/2024-8/31/2029
IndiPHARM: individual metabolome and exposome assessment for pharmaceutical optimization (Miller, PI). IndiPHARM will leverage advances in mass spectrometry and exposomics to rapidly and accurately measure drugs, environmental chemicals, metabolites, dietary factors, and key components of endogenous biology simultaneously for precision medicine. We will develop combined mass spectrometry-based workflows that can be used to optimize drug therapies in individuals and populations that can be scaled for large population studies and for a range of clinical settings to ensure widespread access. Together, these goals will lead to transformative changes in the therapeutic landscape, provide improved therapy to patients in an equitable manner, and help alleviate unnecessary human suffering. Total funding $39,500,000.

Cancer Grand Challenge NCI-NIH and Cancer Research UK (Davis, PI) 4/1/2024-3/31/2029
1OT2CA297506-01 NCI-NIH/Cancer Research UK
Role: Lead of Work Package 2-Exposomics
By integrating genomics, exposomics, immune profiling, and social determinants of health data, SAMBAI will identify factors leading to disproportionate cancer rates and worse outcomes in people of

recent African descent. Total funding $25,000,000. Dr. Miller leads Work Package 2 on Exposomics ($5,000,000)

NIH/NIEHS U24ES036819 (Miller, Patel, Habre, MPI) 9/15/2024-9/14/2029
NEXUS: Network for Exposomics in the U.S.
Role: Contact MPI
NEXUS will serve as the national Center for Exposome Research Coordination for Precision Environmental Health. The team will develop and disseminate best practices, advance analytical and geospatial technologies for exposomics, and serve as a resource for the research community. Total funding $7,800,000.

SPARK-NS. Advancing Academic Discoveries in Neuroscience. 4/1/2024-3/31/2026
Synaptic Vesicle Glycoprotein 2C for Parkinson's Disease Therapeutics. SPARK-NS will provide up to $2,000,000 in non-dilutive funding for Dr. Miller's drug development efforts.

NIH1R01 ES 023839-01A0 (Miller PI) 12/1/2014-11/20/2026
NIH/NIEHS
Vesicular Modulation of Dopamine Neuron Toxicity
This R01 represents a continuation of the research performed under the expired NIEHS-funded P01 that Dr. Miller directed. The project examines key modulators of vesicular dopamine storage and vulnerability to toxicants. Role: PI Total funding $3,000,000.

NIH R01 AG067501 (Mayeux, Vardarajan, Tosto, Miller, MPI) 6/1/20-3/31/2025
NIH/NIA
Genetic epidemiology and multi-omics analyses in familial and sporadic Alzheimer's disease among secular Caribbean Hispanics and religious orders.
Role: MPI and lead of exposomics and metabolomics. Total funding $10,000,000.

NIH RF1 AG066107 (Mayeux, Vardarajan, Miller, MPI) 9/30/2020-8/31/2025
NIH/NIA
Epidemiological integration of genetic variants and metabolomics profiles in Washington Heights Columbia Aging Project. Supplement awarded 9/01/2022 to develop EXCEL AD, a platform for protocol and data sharing of exposomic data in Alzheimer's disease.
Role: MPI and lead of exposomics and metabolomic. Total funding $11,000,000.

NIH T32 ES007322 (Miller, Shaman, Factor-Litvak, MPI) 7/1/2020-6/30/2025
NIH/NIEHS
Advanced training in environmental health and data sciences: molecules to populations
This new T32 represents the consolidation of three NIEHS T32s held at Columbia. Appointees receive training in biological mechanisms of disease, environmental epidemiology, exposomics, and climate and health. Through a partnership with the Data Sciences Institute they also receive advanced data science training.
Role: Contact-MPI. Renewal received impact score of 13.

1R01 ES 023839-01A0 (Miller PI) 8/30/2018-9/30/2021
NIH/NIEHS/NIA-supplement
Vesicular Modulation of Dopamine Neuron Toxicity

This supplement expands the parent R01 by adding aims to examine the role of SV2C in cognitive decline and to examine complex environmental exposures in *C. elegans* using high-resolution exposomics and metabolomics.
Role: PI

European Commission
EIRINE Infrastructure Grant for Exposomics 10/1/2022-9/30-2027
Dr. Miller leads the only non-European component of this infrastructure program to support the development of exposomics in Europe.

INACTIVE
NIH U18 DA052498 (Miller, PI) 9/30/2020-8/31/2021
NIH/NIDA
Synaptic vesicle glycoprotein 2C (SV2C) and psychostimulant actions
This project is examining the potential of SV2C as a therapeutic target for methamphetamine abuse
Role: PI

1U2C ES030163-01 (Jones, Morgan, Li, Miller, MPIs) 8/30/2018-6/30/2022
NIH/ORD/NIEHS
Mega-scale identification tools for xenobiotic metabolism
This project was awarded of the NIH Metabolomics Common Fund
Role: Co-I, effort subcontracted to Columbia University

1RC2DK118619-01 (Lazaridis, PI) 9/1/2018-7/31/2023
NIH/NIDDK
Dissecting the pathogenesis and outcomes of PSC using multi-omics by studying the exposome and genome. This multi-site project represents a collaboration with Mayo Clinic Rochester and Emory University. Emory University will conduct the metabolomics and exposomics analysis.
Role: Co-I, effort subcontracted to Columbia University

U2CES026560-01 (Miller/Waller, PI) 9/23/2015-8/31/2020
NIH/NIEHS
National Exposure Assessment Laboratory at Emory
As part of the Children's Health Exposure Assessment Resource program established by NIH, we will provide expert services in analytical chemistry, metabolomics, and biological response to exposures to NIH-funded investigators in the U.S. Role: PI, Center Director, and Biological Response Core Lead
As of August 2018, Dr. Miller has relinquished his role as Center Director, but retained his role as Biological Response Core Lead with that core being subcontracted to Columbia University.

1R56 AG063908-01 (Mayeux, Miller, Vardarajan, MPIs) 8/30/2019-8/29/2022
NIA/NIH
Genetic epidemiology and multi-omics analyses in familial and sporadic Alzheimer's disease among secular Caribbean Hispanics and religious orders
This project will conduct multi-omic analysis on an ethnic population with a high incidence of AD
Role: MPI

P30 ES 019776-01-A1 (Miller, PI) 5/21/2013-3/31/2022

NIH/NIEHS
HERCULES Exposome Research Center
This NIEHS Core Center Grant supports exposome-level research at Emory University and Georgia Tech. The center provides cores in systems biology, analytical chemistry, metabolomics, career development, community engagement, patient-oriented research, and pilot funding.
Role: PI and Center Director
*Dr. Miller discontinued his role as Center Director as of 7/31/2018 when he left Emory University for Columbia University.

5T32 ES 012870-11 (Miller PI) 7/01/2004 - 6/30/2019
NIH/NIEHS
Graduate and Postdoctoral Training in Environmental Health Sciences and Toxicology
This is an institutional pre- and postdoctoral training grant in environmental health sciences and toxicology. Trainees receive multidisciplinary training with emphasis on modern research methods in toxicology and environmental health. Role: PI
*Dr. Miller served as Principal Investigator from 2006-2018.

HERCULES: Health and Exposome Research at Emory 4/01/2013-3/31/2017
Gary W. Miller, P.I., Center Director
National Institute of Environmental Health Sciences
Annual direct costs $700,000. Total directs costs $3,000,000. Total costs $4,500,000

Michael J. Fox Foundation 9/01/2017-8/31/2018
Interaction of SV2C and alpha-synuclein
This project is designed to determine the potential therapeutic benefit of the synaptic vesicle glycoprotein 2C in alpha-synuclein-associated neurodegeneration. Role: PI. Renewal pending.

Emory Udall Parkinson's Disease Center 9/1/2010-8/31/2015
Thomas Wichmann, P.I. Gary W. Miller, Lead Investigator Project 3.
National Institute of Neurological Diseases and Stroke
Annual direct cost budget $850,000. Total direct costs $1,300,000. Total costs $6,500,000
Annual direct cost Project 3 $175,000. Total direct costs Project 3 $875,000.

Emory Parkinson's Disease Collaborative Environmental Research Center
Gary W. Miller, P.I., Center Director, and Project 1 Lead Investigator
National Institute of Environmental Health Sciences. 9/15/08-7/30/13
This center will examine the effects of environmental toxicants on dopamine storage, mitochondrial function, and redox state as it relates to the development of Parkinson's disease.
Annual direct costs $850,0000. Total direct costs $4,300,000. Total costs $6,450,000

DJ-I and Parkinson's disease pathology
Lian Li, P.I. and Gary W. Miller, Co-I. 4/01/08-3/31/13
National Institute of Environmental Health Sciences
Annual direct costs $250,000. Total directs costs $1,250,000. Total costs $1,875,000

Nuclear integration of environmental toxic signals relevant to PD 9/01/07-8/31/12
National Institute of Environmental Health Sciences.
Zixu Mao, P.I. Gary W. Miller, Consultant

1 R01 ES015317,
Annual direct costs $250,000. Total directs costs $1,250,000. Total costs $1,875,000

Neurotoxicity of nanomaterials: evaluation of subcellular redox state
Gary W. Miller, P.I., Kurt D. Pennell, Co-P.I.
National Institute of Environmental Health Sciences.
1 R01 ES016175, 9/01/07-8/31/11. NIH/NIEHS (no cost extension)
Annual direct costs $300,000. Total directs costs $1,200,000. Total costs $1,800,000

Evaluation of therapeutics to treat Parkinson's disease 2/15/08-1/31/09
Gary W. Miller, P.I.
Neuronova, Inc. Stockholm, Sweden
Annual direct costs $30,000. Indirect costs $15,000. Total costs $45,000.

Behavioral analysis of potential therapeutics for PD 2/1/08-5/30/09
Gary W. Miller, P.I.
Omeros, Inc. Seattle, WA
Annual direct costs $30,000. Indirect costs $15,000. Total costs $45,000.

VMAT2 as Target of Environmental Toxicants (8/28/02 to 7/31/08) 1 U54 ES012068-01
Gary W. Miller, Lead Investigator Project 2
Project 2 of the Emory Collaborative Center for Environmental Parkinson's Disease Research
National Institutes of Environmental Health Sciences
Annual direct cost budget $225,000. Total direct cost of Project 2 $1,218,089. Total cost of Project 2 $1,851,495.

Screening of Neurotoxicants (4/1/05-7/31/08) Supplement to 1 U54 ES012068
Gary W. Miller, P.I.
National Institutes of Environmental Health Sciences
Annual direct cost budget $130,000. Total direct cost of supplement $390,000. Total cost of supplement $600,000.

Woodruff Health Science Center Fund 9/1/05-8/31/08
Gary W. Miller, P.I., Eberhard O Voit, Co-P.I.
Emory University
Predictive algorithms of Parkinson's disease
Total costs $305,000

Disruption of Cholinergic and Dopaminergic Function in Military Deployment: Implications to Parkinson's disease. 6/01/02 to 11/30/06 DAMD 00267036
Gary W. Miller, P.I.
United States Army Medical Research Command
Annual direct cost budget $275,000. Total direct costs $1,000,000. Total costs $1,500,000.

Developmental Pesticide Exposure and Neurodegeneration (5/1/03 to 4/30/06) R21 ES-012315
Gary W. Miller, P.I
National Institute of Environmental Health Sciences.
Annual direct cost budget $100,000. Total direct costs $300,000. Total costs $450,000.

Evaluation of drug action at monoamine transporters (5/1/03-4/30/05)
Gary W. Miller, P.I.
Shire Pharmaceuticals
Annual direct costs $75,000. Total costs $90,000.

Beneficial effects of exercise in animal models of Parkinson's disease
Gary W. Miller, P.I.
Center for Complementary and Alternative Medicine, Emory University. (7/1/02-6/30/03)
Total Costs $35,000

Dopamine Transporters and Ethanol Sensitivity (1/1/01 to 12/31/02)
Gary W. Miller, P.I.
Alcoholic Beverage Medical Research Foundation.
Total costs $80,000

Sleep/Dopamine Phenotypes in Genetically Distinct Mice (8/1/99 to 7/31/03)
David Rye, P.I., Gary W. Miller, Co-I.
National Institutes of Health, NS-64276,
Total costs to Miller lab $400,000

Pesticides and Dopaminergic Function (10/1/97 to 9/30/01)
Gary W. Miller-P.I.
National Institute of Environmental Health Sciences, ES-09248
Total costs $580,000

Monoamine Transporters in Parkinsonism
Allan I. Levey, P.I., Gary W. Miller, P.I., subcontract
National Institute of Neurological Sciences and Stroke Health, NS37031
Total costs to Miller lab $353,669

Developmental Pesticide Exposure and Neurological Impairment.
Gary W. Miller, P.I.
A pilot grant from the Center for Research in Environmental Disease NIEHS 07784.
Total costs $15,000.

Regulation of Monoamine Transporters in Tourette Syndrome. (4/1/97 to 3/31/98)
Tourette Syndrome Association. Brian J. Ciliax- P.I. and Gary W. Miller-Co.I.
Total costs $40,000

Dopamine Transporters in Parkinson's Disease (3/15/96 to 3/14/98)
Gary W. Miller, P.I.
National Institute of Health, NINDS, F32 09930 ,Total costs $52,300. Terminated 9/30/97

Educational Contributions

TEACHING EXPERIENCE
Emory University

Developed a new course entitled **Genome, Exposome, and Health**. Lead instructor. Spring 2013-2015.
Developed the **Emory Exposome Summer Course** in 2016 (>130 participants)

Developed a new university-wide doctoral program in Environmental Health Sciences.
First class started August, 2011.
Served as initial Director of Graduate Studies 2011-2013.

Columbia University
Developed the Exposome Bootcamp, a 2-day summer workshop taught annually starting in 2019
Lecturer in Precision Medicine for the MSCR program, 2019-present
Lecturer in Environmental Determinants of Disease, 2021
PhD Student Journal Club, Spring 2021

Courses
Research Methods in Environmental Health Sciences (EHS697). Primary lecturer. 2012-2013 Human Toxicology (EOH 520), Course coordinator and primary lecturer 2002-2010
Neurotoxicology (EOH 523), Course coordinator and primary lecturer 2004, 2006, 2008, 2010
Drug Metabolism and Toxicology (IBS 536). Lecture on Neurotoxicology, Ion Channel Disruption (2003-2008)
Molecular Toxicology (IBS 740). Co-coordinator (2009), Lectures on renal toxicology (2005, 2007)
Public Health Preparedness. Lecture of Chemical Weapons (2004-2008).
Perspectives in Environmental Health (EOH 500). Lecture on Toxicology (2002-2008)
Participated in the Leadership Institute for Public Health Preparedness, Emory University (2004-2005)
Lectures on Chemical, Biological, and Radiological Terrorism to medical residents (2007)
Pharmacotherapeutics (PHR 375G)
Biochemical Toxicology (PHR 490N)
Advanced Toxicology (PHR 284K)
Biomedical Pharmacology II (PHR 380N)
Methods in Pharmacology and Toxicology, Course coordinator (PHR 687KA)
Principles of Neuroscience (NEU 420)
The Brain Demystified (BIO 301)

TRAINEES

Postdoctoral Fellows

Katerina Savelieva, Ph.D. (University of Texas)
Okkyung Rho, Ph.D. (University of Texas and Emory University)
Lilly Quan, M.D. (University of Texas)
Mohamed Elwan, Ph.D. (University of Texas and Emory University)
Jason Richardson, Ph.D. (Emory University)
Shannon Yancy, Ph.D. (Emory University)
Sampath Ramachandrian, Ph.D. (Emory University)
Joungil Choi, Ph.D. (Emory University)
Kennie Shepherd, Ph.D. (Emory University)
Alison Bernstein, Ph.D. (Emory University)
Chenere Ramsey, Ph.D. (Emory University)
Carlos Lazo, Ph.D. (Emory University)
James Burkett, Ph.D. (Emory University)
Megan Niedzwiecki, Ph.D. (Emory University)
Doug Walker, Ph.D. (Emory University)
Meghan Bucher, Ph.D. (Columbia University)
Faith Anderson, Ph.D. (Columbia University)
Yunjia Lai, Ph.D. (Columbia University)

Ph.D. students

Jennifer L. Tillerson, Ph.D. (University of Texas) completed 2002
W. Michael Caudle, Ph.D. (Emory University) completed 2006
Thomas Guillot, Ph.D. (Emory University) completed 2007
Jaime Hatcher, M.D., Ph.D. (Emory University) completed 2006
E. Danielle Dean, Ph.D. (Emory University) completed 2012
Tonya Taylor, Ph.D. (Emory University) completed 2011
Shawn Alter, Ph.D. (Emory University) completed 2015
Kristen Stout, Ph.D. (Emory University) completed 2016
Kelly Lohr, Ph.D. (Emory University) completed 2015
Amy Dunn, Ph.D. (Emory University) completed 2017
Rachel Cliburn, B.S., M.S., Ph.D. (Emory University) completed 2018
Carlie Hoffmann, B.S., Ph.D. (Emory University) completed 2018
Vrinda Kalia, B.S., M.P.H., Ph.D. (Columbia University)
Jocelyn Dicent, B.S. (Columbia University)

MPH students

Mary Abrams, M.P.H. (Emory University)
Gema Dumitru, M.P.H. (Emory University)
Sarah Chewning, M.P.H. (Emory University)
Margaret McLaurens, M.P.H. (Emory University)
Rod Esaw, M.P.H. (Emory University)
Brittany Holley, M.P.H. (Emory University)
Casey Brinsfield (Emory University)
Kim Richards (Emory University)
Tiffany Douthard, M.P.H. (Emory University)
Andrew Obanwanyi, M.D. (Emory University)
Susan Hobson, M.P.H. (Emory University)
Philip Jaffe, M.P.H. (Emory University)

Sharon Green, M.P.H. (Emory University)
Kristin Delea, M.P.H. (Emory University)
Stephen Hassak, M.P.H. (Emory University)
Dana Annerino, M.P.H. (Emory University)
David Kopp, M.P.H. (Emory University)
Brian Wojeck (Emory University)
Lauren Shapiro (Emory University)

M.S. students
Shannon Etheridge, M.S. (University of Texas)
Elena Rendon, M.S. (University of Texas)
Karen S. Rommelfanger, M.S. (University of Texas)
M. Elena Reveron, M.S. (University of Texas)
Ellen Heath, B.S. (Emory University)
Monica Sharma (Columbia University)
Shihan Xu (Columbia University)

Undergraduate Trainees
Craig Press (University of Texas)
Scott Edwards (University of Texas)
Beshoy Shatby, Pharm.D. (University of Texas)
Christina Harris, NIEHS Summer Minority Fellow (University of Texas) Anita Garcia, NIEHS Summer Minority Fellow (University of Texas) Curtis Keller (Emory University)
Matthew Berk (Emory University)
Abigail Harrover (Emory University)
Kenny Igzara (Emory University)
Merry Chen (Emory University)
Alexa Schlotter (Columbia University)
Irene Lee (Columbia University)

Committees
Fengui Bai, Ph.D. (University of Texas)
Sara Woolley, Ph.D. (University of Texas)
Jennifer Tillerson, M.S. (University of Texas)
Frank Lee, Ph.D. (University of Toronto) outside reader (University of Texas)
Jianhong Jiang, M.S. (University of Texas)
Julie Bratta-Kern, Proposal Committee (University of Texas)
Amanda Tang, M.S. (University of Texas)
Anne Scott, M.S. (University of Texas)
Joey Pablan, M.S. (University of Texas)
James Olzman, Ph.D. (Emory University)
Karen Rommelfanger, Ph.D. (Emory University)
Gillian Hue, Ph.D. (Emory University)
Jesse Schank, Ph.D. (Emory University)
Chad Jackson, B.S. (Emory University)
Jue Chen, B.S. (Emory University)
Lindsey Fisher, Ph.D. (Emory University)
Kevin Paavola, Ph.D. (Emory University)

Stefka Gyoneva, Ph.D. (Emory University)
Anthony Downs, B.S. (Emory University)
Laura Butkovitch, B.S. (Emory University)
Chandresh Ladva, Ph.D. (Emory University)
Elizabeth Kline, B.S. (Emory University)
Erica Landis, Ph.D. (Emory University)
Elizabeth Gibson, B.S. (Columbia University)

Rotation Students
Geoffrey Findlay, B.A. (University of Texas)
Thomas Guillot, B.S. (Emory University)
Sara Dodson, B.S. (Emory University)
Gillian Hue, B.S. (Emory University)
Jamie Hatcher, B.S. (Emory University)
Jesse Schank, B.S. (Emory University)
Shivali Dhruv, B.S. (Emory University)
Tonya Taylor, B.S. (Emory University)
Ashley Kennedy, B.S. (Emory University)
Chase Bourke, B.S. (Emory University)
Jeanne McKeon, B.S. (Emory University)
Amy Luce, B.S. (Emory University)
Jocelyn Dicent, B.S. (Columbia University)

Graduate program affiliations at Emory University
Neuroscience
Molecular and Systems Pharmacology
Environmental Health Sciences
Environmental Health MPH

Graduate program affiliations at Columbia University
Environmental Health Sciences
Pharmacology
Neurobiology and Behavior

Awards won by trainees
National Research Service Award from the National Institute of Drug Abuse to Amy Dunn (Ph.D. student in Neuroscience), 2015 (Emory University)
National Research Service Award from the National Institute of Drug Abuse to Kristen Stout (Ph.D. student in Pharmacology), 2014 (Emory University)
National Research Service Award from the National Institute of Neurological Diseases and Stroke to Kelly Lohr (Ph.D. student in Neuroscience), 2013 (Emory University)
National Research Service Award from the National Institute of Environmental Health Science to Tonya Taylor (Ph.D. student in Pharmacology and Toxicology), 2008 (Emory University) Neuroscience Scholar from the Society for Neuroscience to Tonya Taylor (2009-2012)
National Research Service Award from the National Institute of Environmental Health Science to Jaime Hatcher (M.D., Ph.D. student in Neuroscience), 2005 (Emory University)
National Research Service Award from the National Institute of Environmental Health Science to Jason

Richardson, Ph.D., 2004 (Emory University)
Postdoctoral Research Award (1st prize), International Neurotoxicology Meeting, Honolulu, Hawaii, 2004 (Emory University)
EPA/STAR Fellowship to Tommy Guillot, 2004-2007 (Emory University)
Society of Toxicology Travel Award, Tommy Guillot, 2004 (Emory University)0

PUBLISHED MANUSCRIPTS

* indicates either pre- or postdoctoral trainee of Dr. Miller

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60) Richardson, J.R., Sherer, T.B., Greenamyre, J.T., and Miller, G.W. (2004). Screening of pesticides that inhibit complex I: Implications for Parkinson's Disease. Neurotoxicology 25. (1st place Postdoctoral Research Award at Southeastern Society of Toxicology Meeting).

61) Hatcher, J.M., Richardson, J.R., Sherer, T.B., Testa, C.M., Greenamyre, J.T., and Miller, G.W. (2004). In vitro and in vivo effects of organochlorine pesticides on the dopaminergic system. American Federation for Aging Research.

62) Miller, G.W., Richardson, J.R., Caudle, W.M., Guillot, T.S., Dean, E.D., and Wang, M.Z. (2004). Perinatal exposure to pesticides alters dopaminergic neurochemistry in the developing mouse brain. NIEHS Fetal Basis of Adult Disease Grantee Meeting.

63)Richardson, J.R., Guillot, T.S., Caudle, W.M., Wang, M.Z., and Miller, G.W. (2004). Developmental pesticide exposure enhances amphetamine response and MPTP toxicity. Submitted for Society for Neuroscience Annual Meeting. 167.3.

64) Guillot, T.S., Richardson, J.R., and Miller, G.W. (2004). Pesticide exposure upregulates the dopamine transporter and increases cocaine-induced locomotor activity while abolishing place preference. Submitted for Society for Neuroscience Annual Meeting. 53.14.

65) Caudle, W.M., Richardson, J.R., and Miller, G.W. (2004). Subchronic Exposure to Low Levels of Polychlorinated Biphenyls Reduces Dopamine Transporter and Vesicular Monoamine Transporter 2 Levels. Submitted for Society for Neuroscience Annual Meeting. 94.19.

66) Hatcher, J.M., Richardson, J.R., Testa, C.M., Greenamyre, J.T., and Miller, G.W. (2004). In Vitro and in vivo effects of organochlorine insecticides on the dopaminergic system. Submitted for Society for Neuroscience Annual Meeting.

67) Hammill, C., Caudle, W.M., Richardson, J.R., Miller, G.W., and Traynelis, S. (2004). Role for protease-activated receptor 1 (PAR1) in MPTP-induced dopaminergic neurotoxicity. Submitted for Society for Neuroscience Annual Meeting. 562.6.

68) Na, H.M., Betarbet, R., Wang, M.Z., Caudle, W.M., Miller, G.W., and Greenamyre, J.T. (2004). Developmental expression of alpha-synuclein in VMAT2 hypomorph mice. Submitted for Society for Neuroscience Annual Meeting.

69) Guillot, T.S., Richardson, J.R., and Miller, G.W. (2004). Deltamethrin Increases Dopamine Transporter Expression and Enhances Basal and Cocaine-induced Locomotion. Toxicologist 78:1357.

70) Sherer, T.B., Betarbet, R, Taylor, G., Na, H.M., Caboni, P., Zhang, N., Richardson, J.R., Miller, G.W., Casida, J.E., and Greenamyre, J.T. (2004). Degeulin, a Complex I Inhibitor found in Cube Resin, is Toxic to Neuroblastoma Cells and the Nigrostriatal Dopaminergic Pathway. Society for Neuroscience Abstracts 754.8.

71) Hatcher, J.M., Guillot, T.S., Richardson, J.R., and Miller, G.W. (2005). Dieldrin Exposure Causes Oxidative Damage in Dopamine Neurons. Society of Toxicology Abstracts 84:1961.

72) Guillot, T.S., Richardson, J.R., and Miller, G.W. (2005). Sensitive Detection of Behavioral Impairments in Moderately Lesioned MPTP Mice by Automated Gait Analysis. Society of Toxicology Abstracts 1097.

73) Richardson, J.R., Guillot, T.S., Caudle, W.M., Wang, M.S., and Miller, G.W. (2005). Developmental Pyrethroid Exposure Alters Dopaminergic Neurochemistry Resulting in Hyperactivity and Enhanced Toxicity of MPTP. Toxicologist 84:973. (3rd Place Neurotoxicology Specialty Section Postdoctoral Competition)

74) Hammill, C.E., Caudle, W.M., Richardson, J.R., Miller, G.W., and Traynelis, S.F. (2005). Role for Protease-Activated Receptor 1 (PAR1) in MPTP-induced Dopaminergic Neurotoxicity. Society of Toxicology Abstracts 1529.

75) Caudle, W.M., Richardson, J.R., and Miller, G.W. (2005). Subchronic Exposure to Low-levels of Polychlorinated Biphenyls Reduces Dopamine Transporter and Vesicular Monoamine Transporter 2 Levels. Society of Toxicology Abstracts 1545.

76) Richardson, J.R., Caudle, W.M., Ramachandiran, S., and Miller, G.W. (2005). Activation of PKC and Down-regulation of the Dopamine Transporter Following In Vivo Exposure to Polychlorinated Biphenyls. FASEB J.

77) Ramachandiran, S., Richardson, J.R., and Miller, G.W. (2005). Dopamine exacerbates toxicity of paraquat but not MPTP or rotenone in neuroblastoma cells stably expressing dopamine transporter. Society for Neuroscience Abstracts.

78) Richardson, J.R., Caudle, W.M., Wang, M.Z., Pennell, K.D., and Miller, G.W. (2005). Developmental Dieldrin Exposure Alters the Dopaminergic System and Increases MPTP Toxicity. Society for Neuroscience Abstract.

79) Caudle, W.M., Richardson, J.R., Wang, M.Z., and Miller, G.W. (2005). VMAT2 Reduction Causes Age-Related Neurodegeneration. Society for Neuroscience Abstracts.

80) Hatcher, J.M., Richardson, J.R., Guillot, T.S., Pennell, K.D., and Miller, G.W. (2005). Dieldrin Causes Oxidative Damage and Decreases Dopamine Transporter Levels in Mouse Striatum. Society for Neuroscience Abstracts.

81) Caudle, W.M., Richardson, J.R., Wang, M.Z., Pennell K.D., and Miller, G.W. (2006). PCBinduced alterations in the dopamine transporter as a precursor to nigrostriatal dopamine damage. Society of Toxicology Abstracts 2441

82) Hatcher, J.M., Richardson, J.R., Guillot, T.S., McCormack, A.L., DiMonte, D.A., Pennell, K.D., and Miller, G.W. (2006). Oxidative Damage and Nigrostriatal Dopamine Dysfunction Following Exposure to the Organochlorine Pesticide Dieldrin. Society of Toxicology Abstracts 1112

83) Ramachandiran, S., Richardson, J.R., and Miller, G.W. (2006). Mechanism of action of paraquat is distinct from that of MPP+ or rotenone in neuroblastoma cells stably expressing dopamine transporter. Society of Toxicology Abstracts 1106

84) Pennell, K.D., Hatcher, J.M., Caudle, W.M., Richardson, J.R. Gearing, M., Levey, A.I., Jones, D.P., and Miller, G.W. (2006). Elevated levels of dieldrin are associated with Parkinson's disease. Accepted for presentation at the 232nd American Chemical Society Meeting.

85) Richardson, J.R., Guillot, T.S., Caudle, W.M., and Miller, G.W. (2006). Developmental Pesticide Exposure Reproduces Features of ADHD. Society for Neuroscience Abstracts.

86) Caudle, W.M., Richardson, J.R., Wang, M.Z., McCormack, A.L., Di Monte, D.A., Colebrooke, R., Emson, P.C., and Miller, G.W. (2006). Reduced vesicular storage of dopamine causes age related neurodegeneration. Society for Neuroscience Abstracts.

87) Caudle, W.M., Richardson, J.R., Shepherd, K.R., Wang, M.Z., Guillot, T.S., McCormack, A.L.,

Colebrooke, R.E., Di Monte, D.A., Emson, P, and. Miller, G.W. (2007). Reduced vesicular storage of dopamine causes progressive nigrostriatal neurodegeneration. T Society of Toxicology Abstracts 794.

88) Watson, J.L., Caudle, W.M., Richardson, J.R., and Miller, G.W. (2007). Polychlorinated Biphenyl-Mediated Loss of Striatal Dopamine Terminal Markers: Possible Role of Vesicular Monoamine Transporter 2 Inhibition. Society of Toxicology Abstracts 1046.

89) Ramachandiran, S., Hansen, J.M., Jones, D.P., Richardson, J.R., and Miller, G.W. (2007). Mechanisms of MPP+, rotenone, and paraquat toxicity: thioredoxin oxidation, and activation of cell death pathways. Society of Toxicology Abstracts 902.

90) Taylor, T.N., Caudle, W.M., Wang, M.Z., Hansen, J.M., Richardson, J.R., Jones, D.P., and Miller, G.W. (2007). Altered redox status in a mouse model of Parkinson's disease based on reduced vesicular storage of dopamine. Society for Neuroscience Abstracts 794.11.

91) Hatcher JM, Gearing M, Levey AI, Pennell KP, Miller GW (2006) Elevated Levels of the Pesticide Dieldrin are Associated with Parkinson's Disease. Society for Neuroscience, 37th Annual Meeting; Atlanta, GA.

92) Hatcher JM, Gearing M, Levey AI, Pennell KP, Miller GW (2007) Elevated levels of chlorinated pesticides in the brain are associated with Parkinson's disease. Society of Toxicology, 46th Annual Meeting; Charlotte, NC.

93) Hatcher JM, Gearing M, Levey AI, Pennell KP, Miller GW (2007) Elevated levels of chlorinated pesticides in the brain are associated with Parkinson's disease. Collaborative Center for Parkinson's Disease Environmental Research Annual Conference; Asilomar, CA.

94) Miller GW, Hatcher JM, Gearing M, Levey AI, Pennell KD. (2007) Cyclodiene insecticides and Parkinson's disease: evidence from mice and man. Neurotoxicology. 24th Annual Meeting; San Antonio, TX.

96) Taylor TN, Caudle WM, Wang MZ, Hansen JM, Richardson JR, Jones DP, Miller GW. Altered redox status in a mouse model of Parkinson's disease based upon reduced vesicular storage of dopamine. Society for Neuroscience Annual Meeting, San Diego CA, 2007.

97) Dean, E.D., Torres, G.E., Miller, G.W. alpha-Synuclein Interacts with VMAT2 to Regulate VMAT2 Activity. March 2007 Society of Toxicology in Charlotte, NC.

98) Dean, E.D., Torres, G.E., Miller, G.W. alpha-Synuclein Interacts with VMAT2 to Regulate VMAT2 Activity. April 2007 CCPDER meeting in Asilomar, CA.

99) Dean, E.D., Shepherd, K.R., Li, Y., Torres, G.E., Miller, G.W., Identification of a novel interaction between alpha-synuclein and VMAT2. April 2008 FASEB meeting in San Diego, CA.

100) Taylor TN, Caudle WM, Wang MZ, Schank JR, Mitchell HA, Weinshenker D, Miller GW. Contribution of locus coeruleus degeneration to the Parkinsonian symptoms in VMAT2 deficient mice. Society of Toxicology Annual Meeting, Seattle WA, March 16-20, 2008. *This abstract was

recognized at one of the top five abstracts in Neurotoxicology at this annual meeting.

101) Shawn P. Alter, Tonya N. Taylor, Roy Sutliff, David S. Goldstein, Gary W. Miller. Noradrenergic denervation in the central and peripheral nervous systems in the VMAT2 - deficient mouse model of Parkinson's disease. Society for Neuroscience. Washington, DC , November 2011.

102) Dean, E.D., Mexas, L., Wang, M.Z., Doorn, J., and Miller, G.W. Reduced Vesicular Monoamine Transporter 2 Protein Expression Prevents Age-Associated Glucose Intolerance. March 2011. Molecular and Systems Pharmacology Symposium- Atlanta, GA.

103) AI Bernstein, KA Stout and GW Miller. Development of a real time, spatially resolved fluorescent assay for vesicular packaging of monoamines. Society for Neuroscience Annual Meeting, 2011, Washington DC

104) A. I. Bernstein, K. A. Stout, J. D. de Gastyne and G. W. Miller. Development of a highthroughput screening platform for monoamine toxicity. Society of Toxicology Annual Meeting, 2011, Washington DC.

105) EN Heath, AI Bernstein, TS Guillot, KM Lohr, KA Stout, MZ Wang, YJ Li, A Salahpour and GW Miller. Generation of mice with overexpression of the vesicular monoamine transporter 2 (VMAT2;Slc18a2). Society for Neuroscience Annual Meeting, 2012, New Orleans, LA

106) KM Lohr, AI Bernstein, TS Guillot, KM Lohr, EN Heath, KA Stout, MZ Wang, YJ Li, A Salahpour
and GW Miller. Behavioral and neurochemical characterization of mice with overexpression of the vesicular monoamine transporter 2 (VMAT2; Slc18a2). Society for Neuroscience Annual Meeting, 2012, New Orleans, LA

107) KA Stout, AI Bernstein, Y Li, TS Guillot, GW Miller. A cellular model to assess plasma membrane and vesicular transport of norepinephrine. Society for Neuroscience Annual Meeting, 2012, New Orleans, LA

108) Gary W. Miller,Ali Salahpour, Alison I. Bernstein, Thomas S. Guillot, Kelly Lohr, Ellen N. Heath, Kristen Stout, Minzheng Wang, and Yingjie Li. Generation and characterization of mice with elevated expression of the vesicular monoamine transporter 2 (VMAT2: Slc18a2). The Tenth International Catecholamine Symposium, 2012, Pacific Grove, California

109) Alison I. Bernstein, Kristen A. Stout, and Gary W. Miller. A fluorescent-based assay for live cell, spatially resolved assessment of vesicular monoamine transporter 2-mediated neurotransmitter transport. The Tenth International Catecholamine Symposium, 2012, Pacific Grove, CA

110) A. I. Bernstein, K. A. Stout, T. S. Guillot, and G. W. Miller.Using a Fluorescent substrate to define effects of Polychlorinated Biphenyls on Monoamine Transporters. Society of Toxicology Annual Meeting, 2012, San Francisco, CA

111) Lazo C, Alter S, Kim HM and Miller GW (2012) Development of a reserpine dosing regimen to

study the effects of chronic impairment of VMAT2 on Parkinson's disease-related pathogenesis". New Orleans: Society for Neuroscience Meeting.

112) Betarbet RS, Dalal NV, Herskowitz J, Lazo C, Miller G, Lah J, Levey A, Pranski E. (2012) "Neuronal RING finger protein 11 (RNF11) modulates susceptibility to 6-OHDA-induced nigral degeneration and behavioral deficits". New Orleans: Society for Neuroscience Meeting.

113) Lazo C, Miller G. (2012) Use of in vivo reserpine inhibition of vesicular monoamine transporter to study Parkinson's disease. San Francisco: Society of Toxicology Meeting.

114) Lohr, KL, Bernstein, AI, Goldstein, DS, Guillot, TS, Stout, KA, Lazo, CR, Heath, EN, Wang, MZ, Li,Y, Salahpout, A., Miller, GW. Generation and characterization of mice with elevated expression of VMAT2. Southeastern Society of Toxicology Annual Meeting, 2012. Athens, GA. (abstract)

115) Shawn P. Alter, Tonya N. Taylor, Patricia Sullivan, David S. Goldstein, Gary W. Miller Progressive noradrenergic degeneration in mice with reduced expression of the vesicular monoamine transporter (VMAT2). (Oral presentation and poster). Tenth International Catecholamine Society, Monterey, CA, August 2012.

116) Alter SP, Taylor, TN, Goldstein DS, Miller GW. VMAT2 hypomorphy causes noradrenergic loss preceding nigral-striatal degeneration in a mouse model of Parkinson's disease. Society of Toxicology Annual Meeting, San Francisco, CA, March 2012

117) Shawn P. Alter, Tonya N. Taylor, Patricia Sullivan, David S. Goldstein, Gary W. Miller Progressive noradrenergic degeneration in mice with reduced expression of the vesicular monoamine transporter (VMAT2). Society for Neuroscience Annual Meeting. New Orleans, LA, October 2012.

118) Dunn, A.R., Stout, K.S., Lohr, K.M., Bernstein, A.I., Guillot, T.S., Yi, H., Wang, M-Z., Li, Y., Salahpour, A., Miller, G.W. (2013, October). Genetic manipulation of vesicle function as a potential mediator of neurotoxicant vulnerability. Abstract submitted for a poster at the Annual Meeting for the Southeast Chapter of the Society for Toxicology, Atlanta, GA, USA.

119) Luce, A.R., Lohr, K.M., Stout, K.A., Bernstein, A.I., Guillot, T.S., Wang, M-Z., Li, Y., Salahpour, A., Miller, G.W. (2013, November). Genetic manipulation of vesicular transport machinery mediates dopamine neurochemistry and release dynamics. Abstract submitted for a poster at the Annual Meeting for the Society for Neuroscience, San Diego, CA, USA.

120) Lohr KM, Bernstein AI, Stout KA, Dunn AR, Wang M, Salahpour A, Miller GW. The vesicular monoamine transporter 2 (VMAT2) as a mediator of vesicular function, neurotoxicity, and behavior. Society for Neuroscience Annual Meeting 2013, San Diego, CA.

121) Lohr KM, Bernstein AI, Guillot TS, Stout KA, Lazo CR, Heath EN, Wang M, Li Y, Salahpour A., Miller GW. Characterization of mice with overexpression of the vesicular monoamine transporter 2 (VMAT2). Annual Meeting of the Society of Toxicology 2013, San Antonio, TX.

122) Dunn, A.R., Stout, K.A., Ozawa, M., Wang, M., Li, Y., Guillot, T.S., Miller, G.W. (2014, November). Effects of genetic deletion of the synaptic vesicle glycoprotein 2C (SV2C). Abstract

submitted for a poster at the annual meeting for the Society of Neuroscience, Washington, D.C, USA.

123) Lohr KM, Wang M, Salahpour A, Guillot TS, & Miller GW. Increased vesicular monoamine transporter 2 (VMAT2) expression opposes dopaminergic neurotoxicity in the nigrostriatal pathway. Society for Neuroscience Annual Meeting 2014, Washington DC.

124) R.A. Cliburn, K.M. Lohr, T.S. Guillot, G.W. Miller (2015) The effects of increased dopaminergic transmission on cognitive bias in mice: VMAT2 and behavior. Society for Neuroscience (Washington, D.C.).

125) Dunn, A.R., Stout, K.A., Ozawa, M., Wang, M., Li, Y., Caudle, W.M., Miller, G.W. (2015, March). The role of synaptic vesicle glycoprotein 2C (SV2C) in Parkinson's disease. Abstract submitted for a poster at the annual meeting for the American Society of Neurochemistry, Atlanta, GA, USA.

126) Dunn, A.R., Stout, K.A., Ozawa, M., Wang, M., Caudle, W.M., Miller, G.W. (2015, August). The synaptic vesicle glycoprotein 2C (SV2C) is disrupted in Parkinson's disease. Abstract submitted for a poster at the Catecholamines Gordon Research Conference & Seminar, Sunday River, ME, USA.

127) Dunn, A.R., Stout, K.A., Wang, M., Li, Y., Cai, H., Caudle, W.M., Miller, G.W. (2015, October). Disruption of the synaptic vesicle glycoprotein 2C (SV2C) in Parkinson's disease. Abstract submitted for a poster at the annual meeting for the Society of Neuroscience, Chicago, IL, USA.

128) Stout KA, Ozawa M, Dunn AR, Hoffman CA, Wang M, Miller GW. Glycosylation of synaptic vesicle glycoprotein 2C (SV2C) affects vesicular packaging of dopamine. Poster presentation. Society for Neuroscience. Chicago, IL. October 2015.

129) R.A. Cliburn, K.M. Lohr, L.Rajan, G.W. Miller (2015) Relationship between altered vesicular monoamine function and complex behavior. Society for Neuroscience (Chicago, IL).

130) Lohr KM, Chen M, Wang M, & Miller GW. Increased vesicular function improves synaptic dopamine handling and opposes MPTP neurotoxicity, Society for Neuroscience Annual Meeting 2015, Chicago, IL.

131) Lohr KM, Stout KA, Dunn AR, & Miller GW. Increased vesicular function improves synaptic dopamine handling and opposes neurotoxicity. American Society of Neurochemistry Annual Meeting 2015, Atlanta, GA.

132) Niedzwiecki MM, Samant P, Tran V, Banton B, Jones DP, Miller GW, Prausnitz M. Towards minimally-invasive exposure monitoring: high-resolution, untargeted metabolomic profiling in interstitial fluid samples. Emory Exposome Summer Course, Atlanta, GA, USA, June 2016.

133) Dunn, A.R., Stout, K.A., Bernstein, A.I., Wang, M., Li, Y., Caudle, W.M., Miller, G.W. (2016, September). Disruption of the synaptic vesicle glycoprotein 2C (SV2C) in Parkinson's disease. Abstract submitted for a poster at the Dopamine 2016 meeting, Vienna, Austria.

134) Hoffman CA, Stout KA, Wilson B, Jonas L, Miller GW. Development of a high-throughput assay to measure VMAT2-mediated vesicular dopamine transport. Poster presentation. Dopamine 2016. Medical University of Vienna, Vienna, Austria. September 2016.

135) R.A. Cliburn, K.M. Lohr, L.Rajan, E, Winokur, J. Schroeder, D. Weinshenker, G.W. Miller (2016) Neurochemical and behavioral response to psychostimulants in mice with altered VMAT2 function. Dopamine 2016 (Vienna, Austria).

Invited and/or Peer-Selected Presentations at Regional, National or International Levels

1) University of Texas Neuroscience Symposium. Parkinson's disease: dopamine transport and neuronal susceptibility. April 17, 1999

2) National Institute of Environmental Health Sciences Meeting "Apoptosis, Growth Factors, and Signal Transduction Pathways: Basic Biology and Toxicology." Pesticides and dopaminergic function. April 19-21, 1999.

3) National Institute of Environmental Health Sciences "Concept Forum on the Role of the Environment in the Etiology of Parkinson's Disease." July 22, 1999.

4) Department of Veterinary and Biomedical Sciences, Pennsylvania State University "Dopamine Transporters and Neuronal Injury." Feb. 2000.

5) The Parkinson's Institute, Sunnyvale, CA. Dopamine transporters: link between pesticide exposure and Parkinson's disease. Feb. 1, 2000.

6) Institute of Pharmacology, Russian Academy of Medical Sciences, Moscow, Russia. Dopamine transporters and neuronal injury. June, 2000

7) Annual Institute of Alcohol and Drug Studies, Austin, TX. Neurobiology of Addiction, July, 2000.

8) Department of Pharmacology and Toxicology, University of Texas Medical Branch. Role of dopamine transporters in addiction and neurodegeneration, September, 2000.

9) IEEE-EMBS Asia-Pacific Conference on Biomedical Engineering, Hangzhou, China. Neurotransmitter transporters and cocaine: insights from genetically altered mice. Sept., 2000.

10) Pfizer, Groton, CT. CNS Drug Discovery. Monoamine Transporters in Neurodegenerative and Neuropsychiatric Disorders, October, 2000.

11) Pfizer, Groton, CT. Drug Safety Evaluation. Mediators of Dopamine Toxicity. October, 2000.

12) Ambion, Austin, Texas. Dopamine Transporters and Pesticides in Parkinson’s disease. March, 2001

13) National Institute of Environmental Health Sciences Meeting "Mechanisms of Apoptosis, Growth Factors, Signal Transduction, and Oxidative Stress.” Dopamine transporters, apoptosis, and Parkinson’s disease. April 19-21, 2001.

14) The Section of Neurobiology, School of Biological Sciences, University of Texas at Austin. Parkinson's disease and Pesticides: What's the link? April 25, 2001.

15) Neurogenomics: Building a Better Brain. "Development of the TEXANeurochip" Vanderbilt University, May, 2001

16) Department of Physiology and Pharmacology, "Dopamine Transporters, Parkinson's Disease, and Alcoholism" Wake Forest University May 2001.

17) Department of Pharmacology and Toxicology, University of Arkansas for Medical Sciences. "Dopamine Transporters and Neurodegenerative Disease" June, 2001.

18) Department of Psychiatry, Yale University. "DAT and VMAT2 as Predictors of Neurological Disorders" June, 2001.

19) 19th Annual International Neurotoxicology Conference, Colorado Springs, CO. Dopamine Transporters as Targets of Insecticides. August, 2001.

20) Center for Neuroscience, Department of Psychiatry, University of Texas Southwestern Medical School. "Dopamine Transporters in Neurodegenerative Disease." October, 2001.

21) FASEB-American Society for Experimental Biology. New Orleans, LA. Dopamine Transporters in Toxicology and Disease. April, 2002.

22) Department of Medical Pharmacology and Toxicology, Texas A&M University. College Station, TX. Environmental and Genetic Factors in Parkinson's Disease. October, 2002.

23) Department of Pharmacology, Emory University. Dopamine Transporters, Environmental Agents, and Parkinson's Disease. April, 2003.

24) Collaborative Consortium on Environmental Parkinson's Disease Research, Napa, CA Vesicular Monoamine Transporter as Target of Environmental Toxicants. July, 2003.

25) Shire Pharmaceuticals Workshop, Washington, D.C. "Amphetamines and monoamine transporters." October, 2003.

26) Frontiers in Neuroscience, Emory University. "Dopamine Transporters as Targets of Environmental Toxicants." October, 2003.

27) Society of Toxicology Symposium on Methods of Evaluating Neurotoxicity. Baltimore, MD. "Analysis of Neurotoxicity in VMAT2 Knockout Mice. March, 2004.

28) Collaborative Consortium on Environmental Parkinson's Disease Research, Atlanta, GA. Update on interactions between VMAT2 and environmental agents. May, 2004.

29) Department of Chemistry, Emory University. Environmental factors in Parkinson's. April 2005.

30) Environmental Protection Agency. Pesticides, Neurodegeneration, and Hyperactivity, April 2005.

31) Collaborative Consortium for Parkinson's Disease Environmental Research. VMAT2 as a Target of Environmental Toxicants. Asilomar, CA. May 2005.

32) Department of Environmental Health Sciences, University of Georgia. Pesticides, PCBs, and Parkinson's disease. September, 2005.

33) Department of Civil and Environmental Engineering, Georgia Tech. Persistent organic pollutants and Parkinson's disease. November, 2005.

34) Department of Pharmaceutical Sciences. University of Wisconsin, Madison. Pesticides and Parkinson's disease. December, 2005.

35) Collaborative Consortium for Parkinson's Disease Environmental Research. Environmental factors in Parkinson's disease. Asilomar, CA. April, 2006.

36) National Academies Meeting on Science and Security, Southeast Regional Meeting. Role of Institutional Biosafety Committees in Security Issues in Academic Settings, June, 2006.

37) Collaborative Consortium for Parkinson's Disease Environmental Research. Pesticides and Parkinson's disease: an update. Asilomar, CA. April, 2007.

38) Keynote Speaker, North Carolina Chapter of the Society of Toxicology. Parkinson's disease: a toxicological perspective. Research Triangle Park, NC, April, 2007.

39) Symposium speaker, Society of Toxicology Annual Meeting. Toxicological models of Parkinson's disease and their impact on clinical care. Charlotte, NC, April, 2007.
40) Neuronova. VMAT2 as a therapeutic target. Stockholm, Sweden. June, 2007.

41) Invited Speaker, Cyclodiene insecticides and Parkinson's disease. 24th Annual International Neurotoxicology Meeting, San Antonio, TX. November, 2007.

42) Department of Environmental Medicine. University of Rochester. Pesticides and Parkinson's disease: a toxicological perspective. Rochester, NY. December 2007.

43) Frontiers in Neuroscience, Emory University. Should a toxicologist be studying Parkinson's disease? Atlanta, GA. March, 2008.

44) Neuroscience Seminar Series, Colorado State University. Altered vesicular storage of monoamines in Parkinson's disease. May, 2008.

45) Gordon Research Conference. Mechanisms of Toxicity. Gene Environment Interactions in Neurodegenerative Disease. July, 2008.

46) National Institute of Environmental Health Sciences. Centers for Neurological Diseases Annual Meeting. An animal model of the non-motor symptoms in Parkinson's disease. October, 2009.

47) Department of Pharmaceutical Sciences. Auburn University. Vesicular storage of monoamines in neurodegenerative and neuropsychiatric disorders. November, 2010.

48) Old Dominion University. Keynote speaker, Undergraduate Research Symposium. Blood, sweat, and fears: the challenges of undergraduate research. February, 2011.

49) University of Missouri, Translational Neuroscience Symposium, Invited Speaker. Vesicular monoamines and Parkinson's disease. February 2011.

50) Department of Health and Kinesiology, Texas A&M, Distinguished Lecture Series. Parkinson's disease and Aging. April, 2011.

51) Emory University Department of Neurology, Grand Rounds. Vesicular storage of monoamines and Parkinson's disease. October, 2011.

51) Program in Toxicology, University of California, Los Angeles. Vesicular neurotransmitters and neurotoxicity. October, 2011.

52) National Institute of Neurological Disease and Stroke, Grand Rounds. Bethesda, MD. Vesicular storage of monoamines and Parkinson's disease. November, 2011.

53) University of Iowa, College of Pharmacy. Neurotransmitter storage as a target of toxicity. May 2012.

54) National Institute of Environmental Health Sciences. Centers for Neurodegenerative Sciences. Emory Parkinson's Disease Collaborative Environmental Research Center. May 2012.

55) National Institute of Environmental Health Sciences. Premotor Symptoms of Parkinson's Disease Symposium. Non-motor symptoms of PD in VMAT2-deficient mice. June, 2012.

56) Vanderbilt Molecular Toxicology Center. Neurotransmitter transporters and neurotoxicity. November, 2012.

57) Alzheimer's Disease Research Center. Emory University. The Exposome and Neurodegeneration. February, 2013.

58) Society of Toxicology. Workshop on Environmental Factors in Neurodegeneration. Industrial Toxicants and Parkinson's Disease. March, 2013.

59) Yale School of Public Health. This is Your Brain on the Environment: Parkinson's, Pesticides, and PCBs. September, 2013

60) Mailman School of Public Health, Columbia University. PCBs, Pesticides, and Parkinson's: a story of storage. October 2014.

61) Iowa State University. PCBs and Parkinson's disease. November 2014.

62) National Institute of Environmental Health Sciences. The Importance of the Biological Response to the Exposome. Exposome Workshop. January 2015.

63) University of Michigan School of Public Health. The Exposome. January, 2015.

64) Midland Society of Toxicology. The Exposome and Toxicology. March, 2015.

65) University of Minnesota, Duluth. The Toxicology of Parkinson's Disease. March, 2015

66) Society of Toxicology, Continuing Education Course. An Introduction to the Exposome. March 2015.

67) Society of Toxicology. Crafting High Impact Manuscripts. March 2015.

68) Michigan State University. Using the exposome to expand toxicology. May, 2015.

69) International Society of Exposure Science. The importance of biological impact to the concept of the exposome. October, 2015.

70) University of California at Los Angeles. The Exposome as an Opportunity for Toxicology. October 2015.

71) North Carolina State University. Burroughs Wellcome Distinguished Lecture. The Exposome as an Opportunity for Toxicology October, 2015.

72) Workshop on the Exposome. Establishing an exposome infrastructure in academia. Leipzig, Germany, December 2015.

73) University of California at Davis. The Exposome: do we really need another –ome? January, 2016.

74) Wright Patterson Air Force Base, Human Performance Directorate. The exposome as a platform for toxicology. February, 2016.

75) University of Washington. The exposome in environmental health sciences. May, 2016.

76) National Institute for Environmental Studies. The Exposome: Shifting the Paradigm in Environmental Health Sciences. Tsukuba, Japan. June, 2016.

77) Satellite meeting of the China C. elegans meeting. The Exposome: Shifting the Paradigm in Toxicology and Environmental Health. Beijing, China. July, 2016

78) International Transporter Biology Meeting. SV2C as a mediator of dopamine transport. Vienna, Austria. September 2016.

79) International Society of Environmental Epidemiology. The exposome: biological responses. Rome, Italy, September 2016.

80) Vesicular storage of dopamine and Parkinson's disease. Dopamine 2016. Vienna, Austria. September, 2016.

81) Weill-Cornell Medical School. Beyond the genome: using the exposome to examine environmental influences of disease. New York, NY. October 2016.

82) University of New Mexico. Dopamine and Parkinson's Disease: A Story of Storage. Albuquerque, NM, October, 2016.

83) EHS FEST-50th Anniversary of NIEHS. G x E: dichotomy or synergy? Durham, NC. December, 2016.

84) The Exposome: Toxicology and beyond. Department of Environmental Health Science, College of Public Health, University of Georgia. Athens, GA. February 2017.

85) Parkinson's disease: vesicular storage and environmental influences. Florida International University. Miami, Florida. February 2017.

86) Using the exposome to study complex diseases. Department of Medicine. Mayo Clinic, Rochester, Minnesota. March 2017

87) Parkinson's disease, aging, and the environment. Biology of Aging Conference. Morehouse School of Medicine. Atlanta, Georgia. May, 2017.

88) Advancing Parkinson's disease using clues from the environment. Parkinson's Disease Gordon Conference. Newry, Maine. June 2017

89) Environmental factors in Parkinson's disease. National Institute of Environmental Studies. Tsukuba, Japan. July 2017

90) Using the exposome to advance toxicology. 44th Annual Japanese Society of Toxicology. Yokohama, Japan. July 2017

91) Metabolomics and exposomics in primary sclerosing cholangitis. Carlos Annual Meeting. Mayo Clinic. Rochester, Minnesota. July 2017.

92) The exposome as a framework for toxicology. Central States Society of Toxicology Annual Meeting. Ames, Iowa. September, 2017.

93) Parkinson's disease and environmental insights. Brain Institute, Florida Atlantic University, October, 2017

94) The exposome: a framework for toxicology. Chinese Society of Toxicology. Jinan, China. November, 2018.

95) Update on activities in the HERCULES Exposome Research Center. HELIX Annual Meeting. Venice, Italy. October, 2018

96) HERCULES Exposome Research Center. HELIX Annual Meeting. Barcelona, Spain. October, 2018

97) The exposome in aging and neurodegeneration. Center for Immunity and Inflammation. Stanford University. November, 2017.

98) Parkinson's disease: environmental clues and transporter blues. Oxford University Parkinson's Disease Center. Oxford, England. January, 2018.

99) Parkinson's disease: environmental clues and transporter blues. Department of Pharmacology, University of Washington, Seattle, WA, February 2018.

100) Big data in toxicology: the changing landscape of publishing. Society of Toxicology, San Antonio, TX March, 2018.

101) ToxSci at 20: a tribute to John Doull. Society of Toxicology, San Antonio, TX March, 2018.

102) Parkinson's disease: environmental clues and transporter blues. University of Paris, Descartes, Paris, France April 2018

103) The exposome: measuring exposures on an –omic scale. Laboratoire d'Étude des Résidus et Contaminants dans les Aliments (LABERCA),Nantes-Atlantic National College of Veterinary Medicine, Food Science and Engineering (ONIRIS), Nantes, France April 2018.

104) The exposome: measuring exposures on an –omic scale. Toxicities Conference, University of Paris Descartes, June 2018

105) The exposome: measuring exposures on an –omic scale. Utrecht University, Utrecht, Netherlands, June 2018

106) The exposome: measuring exposures on an –omic scale. Department of Chemistry, University of Vienna, September 2018

107) Parkinson's disease: Environmental clues and transporter blues. Department of Physiology, Northwestern University, October 2018

108) The exposome and global health. The Columbia Global Center, Mumbai, India. February, 2019

109) The exposome: measuring the environment at scale. Department of Pharmacology and Toxicology, College of Pharmacy. University of Colorado, Aurora, CO, 2019

110) The exposome: an omic-scale analysis of the environment. Department of Pharmacology and Toxicology, College of Pharmacy, University of Arizona, Tucson, Arizona, 2019

111) The exposome as an example of interdisciplinary science. HESI Annual Meeting, Arlington, Virginia, 2019.

112) The exposome and toxicology. Cellular and Molecular Mechanisms of Toxicology. Gordon Conference, Andover, NH.

113) Parkinson's, dopamine, and the environment. Department of Pharmacology, University of Minnesota, Minneapolis, MN, 2019.

114) The exposome: advances and limitations. ATHLETE Exposome Kickoff Meeting, Barcelona, Spain, 2020.

115) Parkinson's disease and the environment. University of Luxembourg. Luxemburg, 2020.

116) The exposome in the future. European Commission Human Exposome Network, Brussels, 2020

117) The exposome: measuring the environmental drivers of aging. National Academy of Sciences Workshop on the Aging and the Environment.

118) The exposome and health: populations to molecules. NIA Alzheimer's Disease Summit, 2021

119) The Exposome: integrating the environment into multiomic research at NIH. NIEHS Council Meeting, Research Triangle Park, NC., 2021

120) Exposomics: from concept to bona fide approach to assess environmental contributors to health. National Exposome Conference, Paris, France, 2021.

121) The Exposome at Columbia. Columbia Precision Medicine Symposium, New York, 2021

122) The Exposome: Repackaging or Reimagining? Columbia Precision Medicine and Society Symposium, New York, 2022

123) Exposomics at Scale. Keynote at Launch of the Netherlands National Exposome Program. Utrecht, Netherlands, 2022.

124) A new paradigm for the environment and health. Keynote at Launch of Austria's National Exposome Program. Vienna, Austria, 2022.

125) Measuring the environment at scale. European Commission Conference on Research Infrastructures Brno, Czech Republic, 2022.

126) The environment, exposome, and exposomics: what's the difference? Mayo Clinic Conference on Individualized Medicine, 2022.

127) Towards exposome-driven toxicology 2.0. Society of Toxicology. Nashville, 2023.

128) Exposomic analysis of Alzheimer's disease and related dementias. Society of Toxicology. Nashville, 2023.

129) Global harmonization of exposomics. Karolinska Institute, Stockholm, Sweden, 2023.

130) Exposomics for Alzheimer's disease and Parkinson's disease. European-USA Exposome Symposium. Brescia, Italy, 2023.

131) Incorporating the environment into All of Us. All of Us Research Advisory Committee Meeting,

Mayo Clinic, Rochester, Minnesota, 2023.

Presentations to lay audiences

Rollins School of Public Health Dean's Council. October 2010
Emory Board of Visitors Meeting. November 2011
The Inquiry Club, Atlanta, GA. February 2012
Rollins School of Public Health Dean's Council. Research advances in the Rollins School of Public Health. April, 2012
Emory Board of Visitors Meeting. March 2017
Riderwood Retirement Community. Parkinson's disease research and advances. July, 2018
Columbia Global Center in Paris. The exposome and human disease. May, 2019
Columbia Global Center in Mumbai. Assessing complex exposures using exposomics. November, 2019.

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This letter documents that, except for the present case under review regarding exposures at Camp Lejeune, I have not been involved in any legal proceedings as an expert witness or consultant over the past four years.

Sincerely,

Gary W. Miller, Ph.D.