Exhibit 118

General Causation Expert Report of Steven B. Bird, MD

Parkinson's Disease

Prepared by:
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Contents

I. QUALIFICATIONS

I earned my Bachelor of Science degree in biology cum laude in 1991 from Yale University, where I was named a Yale University Richter Fellow. I worked in the laboratory of Professor Sidney Altman, Dean of Yale College and winner of the 1989 Nobel Prize in Chemistry. I was awarded my Doctor of Medicine degree by Northwestern University in 1995 and was also elected to the Alpha Omega Alpha national medical honor society (generally awarded to the top 10% of medical students nationally). Following medical school, I gained post-graduate training through residencies with the Naval Hospital San Diego (surgery) and the University of Massachusetts Medical School (emergency medicine). In addition, I completed a two-year fellowship in medical toxicology at the University of Massachusetts Medical School in 2004.

I began my independent clinical career in the Department of Emergency Medicine at the University of Massachusetts Medical School in 2002. I was promoted to Assistant Professor of Emergency Medicine in 2004, to Associate Professor in 2010, and to full Professor in 2016. In addition, I served as Program Director of the Emergency Medicine Residency Program and as Vice Chair of Education for the Department of Emergency Medicine at the University of Massachusetts Medical School from 2011 to 2019. I am currently the Division Chief of Medical Toxicology at the UMass Chan Medical School and UMass Memorial Health. I work as an Attending Emergency Physician at UMass Memorial Medical Center and Clinton Hospital. I am actively involved with numerous professional committees within the UMass Chan Medical School and its Department of Emergency Medicine and Division of Medical Toxicology, and in national and international scientific organizations, such as the Society for Academic Emergency Medicine, the American College of Medical Toxicology, and the American College of Emergency Physicians. I served on the Board of Directors of the Society for Academic Emergency Medicine from 2014-2020 and was President of the Society from 2018-2019. Additionally, I was formerly President of the Medical Staff of UMass Memorial Healthcare.

During my professional career, I have received several awards, including the Navy and Marine Corp Achievement Medal, the Outstanding Contribution to Medical Toxicology Research by the American College of Medical Toxicology; the Society for Academic Emergency Medicine ("SAEM") Best Resident Basic Science Presentation Award, the SAEM New England Regional Research Directors Excellence in Research Award, the teaching award (twice) from the UMass Emergency Medicine Residency, and a Young Investigator Award from the Society for Academic Emergency Medicine.

I am a reviewer for several scientific journals, including the Journal of Medical Toxicology; Clinical Toxicology; Annals of Emergency Medicine; Academic Emergency Medicine; Toxicology; the New England Journal of Medicine; and JAMA. I currently serve on the Editorial Board of Academic Emergency Medicine and was a founding editorial board member of the Journal of Medical Toxicology. I am certified by the American Board of Emergency Medicine and the American Board of Medical Toxicology. I currently hold a license to practice medicine in Massachusetts. In my practice of emergency medicine medical toxicology, I evaluate people exposed or potentially exposed to a variety of substances on a daily basis. In my review of this

case, I utilized scientifically valid and reliable methods to perform my research, followed by a differential etiology methodology and consideration of the weight of the evidence and the Bradford-Hill viewpoints.

II. METHODOLOGY

In my search of the medical and scientific literature, I conducted many searches of the PubMed database, using terms including (but not exclusive to):

> (TCE OR PCE OR benzene OR vinyl chloride OR trichloroethylene OR tetrachloroethylene OR perchloroethylene) AND Parkinson's Disease

I also performed numerous searches using Google Scholar, which gives quick access to full-text articles as well as an immediate list of citing articles for that manuscript. In order to identify even more articles, I reviewed the articles cited in the manuscripts I reviewed. I also reviewed toxicology and medical toxicology textbooks, as well as chemical toxicity databases such as the Hazardous Substances Data Bank, United States Environmental Protection Agency's (EPA) Integrated Risk Information System (IRIS), and Agency for Toxic Substances and Disease Registry (ATSDR). In addition, I reviewed the records and other materials that counsel for Plaintiffs sent to me.

My methodology for reviewing literature in this case is identical to my methodology when seeing a patient and that which I teach residents and fellows.

After exposure to a toxin, people will often present at the emergency department. In my practice of emergency medicine and medical toxicology, I evaluate people exposed or potentially exposed to a variety of substances on a daily basis. In my review of this case, I utilized scientifically valid and reliable methods to perform my research, followed by consideration of the weight of the evidence and the Bradford-Hill viewpoints.

Even though some of the epidemiological results presented in this report are not statistically significant under traditional methods, they are important and relevant information with regards to causation where the standard is equipoise because the concept of equipoise refers to genuine uncertainty within the expert medical community. Many of the results are very nearly statistically significant and are clearly *not* directed towards a decrease in occurrence or risk of the cancers. Furthermore, the use of traditional statistical significance does not capture or account for biological plausibility of cancer causation. Likewise, relying on traditional statistical significance ignores known carcinogenic properties of a substance. Lastly, biostatisticians have largely abandoned the dichotomous interpretation of statistical significance (i.e., significant vs. non-significant) and instead focus on the estimation of effect sizes.

I am being compensated at a rate of $600 per hour for review and report writing and $1,000 per hour for deposition or trial testimony.

I reserve the right to supplement this report.

III. "AS LIKELY AS NOT" STANDARD

The statute at issue in this case, the Camp Lejeune Justice Act (CLJA), states:

> (2) Standards – To meet the burden of proof described in paragraph (1), a party shall produce evidence showing that the relationship between exposure to the water at Camp Lejeune and the harm is –
>
> (A) Sufficient to conclude a causal relationship exists; or
>
> (B) sufficient to conclude a causal relationship is at least as likely as not.

This standard has significant implications for the analysis at issue in this report. The standard and its language have application in the field of toxicology, epidemiology, and other similar sciences. The determination of a causal relationship is naturally different under a standard that requires a proof "more likely than not," as compared to a standard that requires a proof "as likely as not."
To this point, ATSDR (2017) in its assessment of the evidence, utilized differing causality standards in the context of assessing the causal relationship between the toxins in the drinking water at Camp Lejeune and different diseases. Specifically, ATSDR utilized the following causality standards:

> Sufficient evidence for causation: the evidence is sufficient to conclude a causal relationship exists. This category would be met, for example, if:
>
> 1. There is sufficient evidence from human studies in which chance and biases (including confounding) can be ruled out with reasonable confidence, or
> 2. There is less than sufficient evidence from human studies but sufficient evidence in animal studies and strong evidence that the agent acts through a relevant mechanism in humans.
>
> Equipoise and above evidence for causation: The evidence is sufficient to conclude that a causal relationship is ***at least as likely as not***, but not sufficient to conclude that a causal relationship exists. This category would be met, for example, if:
>
> 1. The degree of evidence from human studies is less than sufficient but there is supplementary evidence from animal studies and/or mechanistic studies that supports causality, or
> 2. A meta-analysis does not provide convincing evidence (e.g., the summary risk estimate is close to the null value of 1.0, i.e., $\leq$ 1.1), or if the meta-analysis observes a non-monotonic exposure-response relationship) but there is at least one epidemiological study considered to be of high utility occurring after the meta-analysis has been conducted, in which an association between the exposure and increased risk of the disease of interest has been found and in which chance and bias can be ruled out with reasonable confidence.

3. A meta-analysis has not been conducted, but there is at least one epidemiological study considered to be of high utility in which an association between the exposure and increased risk of the disease of interest has been found and in which chance and biases can be ruled out with reasonable confidence.

Similar standards have been used in other areas of toxicology, epidemiology and by other governmental bodies. For example, as ATSDR notes, the classification scheme used in the 2017 assessment of the evidence is one "recommended by an IOM panel that reviewed the VA's presumptive disability decision-making process for veterans (IOM 2008).

This classification scheme is consistent with my many years of experience in these fields of science and also based on sound scientific and methodological grounds. It therefore informs an analysis of causality that is necessarily based upon toxicology, epidemiology and other similar sciences.

IV. SUMMARY OF OPINIONS

In my view, the water at Camp Lejeune, which was contaminated with significant levels of benzene, trichloroethylene (TCE), perchloroethylene (PCE), and vinyl chloride, causes Parkinson's Disease (PD).

TCE is a chemical that has been "linked to parkinsonism" since 1969. [Dorsey ER. J Parkinson Dis 2023;13:203–18] "TCE is a causative factor in a number of diseases, [including] neurotoxicity." [DeMiranda BR. Neurobiol Dis 2021;153:1053122021 at 1] "In multiple animal studies, the chemical reproduces the pathological features of PD." [Dorsey 2023 at 203] Human epidemiological studies of occupational exposure to TCE show a strong link with PD. [Gash DM. Ann Neurol 2008;63:184–192; Goldman SM. Ann Neurol 2012;71:776–84]

Although in the occupational setting, concentrations of these chemicals are often quite high, even "long-term exposures to subthreshold levels of industrial byproducts" of these chemicals can increase the risk of PD. [DeMiranda 2021 at 2] Indeed, studies of personnel at Camp Lejeune - who were exposed to TCE, PCE, and other chemicals - showed elevated risks of PD generally as well as accelerated progression of PD symptoms. [Goldman SM. JAMA Neurol 2023;80:673-81; Goldman SM. Movement Dis 2024;39:20242024] This literature demonstrates that TCE, PCE, and the water at Camp Lejeune containing those chemicals cause PD.

This literature is strong enough that peer-reviewed articles have described specific cases of personnel at Camp Lejeune who likely developed PD many years later as a result of their exposure. See Dorsey 2023 at 210 ("The roots of [NBA player Brian] Grant's PD may have been in Camp Lejeune.") (describing the case of Captain Amy Lindberg who developed PD after exposure to the water at Camp Lejeune). The Veteran's Administration (VA) "now officially recognizes PD as a service-connected disability associated with contamination from Camp Lejeune." [DeMiranda 2021 at 6]; see also Dorsey 2023 ("In 2017, the U.S. Department of Veterans Affairs established PD as having a presumptive service connection for those who served at Camp Lejeune between 1953 and 1987."). And scientists reviewing the literature have concluded that "Individuals who

lived or worked at Camp Lejeune . . . have increased risk of PD." [DeMiranda 2021 at 5]

I have been asked to evaluate the levels of exposure to these chemicals that have been identified in scientific literature to be hazardous to human beings generally and are known to cause Parkinson's Disease as likely as not. Numerous studies provide evidence of specific levels of exposure—some of which align with the contamination observed at Camp Lejeune—that are associated with increased risks for PD:

1. **Employment on base for 2.5 years**: Bove FJ, Ruckart PZ, Maslia M, Larson TC. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environ Health*. 2014;13:68.

2. **Cumulative exposure to less than 457 ppb-months of PCE**: Agency for Toxic Substances and Disease Registry (ATSDR). *Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune*. April 2018.

3. **Cumulative exposure to less than 10,868 ppb-months of TCE**: ATSDR, 2018.

4. **Cumulative exposure between 457 and 2,118 ppb-months of PCE**: ATSDR, 2018.

5. **Cumulative exposure between 10,868 and 50,563 ppb-months of TCE**: ATSDR, 2018.

6. **1-2 quarters stationed on base as a service member from 1975 to 1985**: Bove FJ, Greek A, Gatiba R, Boehm RC, Mohnsen MM. Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study. Environ Health. 2024;23(1):61.

7. **7-6 quarters stationed on base as a service member from 1975 to 1985**: Bove et al, 2024a.

8. **7-10 quarters stationed on base as a service member from 1975 to 1985**: Bove et al, 2024a.

9. **More than 10 quarters stationed on base as a service member from 1975 to 1985**: Bove et al, 2024a.

10. **5-22 quarters spent on base as a civillian worker from 1975 to 1985**: Bove et al, 2024a.

11. **More than 22 quarters spent on base as a civillian worker from 1975 to 1985**: Bove et al, 2024a.

12. **3 or more months stationed on base as a service member from 1975 to 1985 on base:** Goldman SM, Weaver FM, Stroupe KT, et al. *Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune*. JAMA Neurol. 2023;80(7):673-681.

It is also worth noting that the ATSDR, in its 2017 Assessment, stated that the "evidence from the epidemiological studies included in this assessment is not sufficeint to contradict this minimum duration," i.e., 30 days on base. My understanding of the Justice Act is that all Plaintiffs in this case were on the Camp Lejeune base for at least 30 days.

The report continued, "Moreover the results from the Camp Lejeune mortality studies suggest that a 30 day minimum duration requirement may be appropriate since elevated risks for some of the diseases evaluated were observed for exposure durations of 1-3 months. These results should not be surprising given that the levels of TCE, PCE and vinyl chloride measured or estimated in the drinking water systems at Camp Lejeune considerably exceeded their respective MCLs."

The evidence in this case mirrors one of the oldest examples of epidemiology detecting a causal association. In the mid-1800s, British physician John Snow compared the rates of cholera in people who drank water from a company who drew its water from "comparatively clean, Thames water upstream from London" and another company who drew its water from "downstream of London and therefore contaminated with sewage." [Rothman K. Modern Epidemiology, 4th Edition, 2021]. The cholera rates were higher in households who drank water from the downstream company, thereby demonstrating convincingly that contaminated water was causing the cholera.

The evidence for Camp Lejeune largely relies on similarly elegant "natural experiment" designs. The studies looked at the rates of PD in areas of high contaminant concentrations and compared those rates to areas of lower contaminant concentrations. Some of the studies did so for Camp Lejeune itself, comparing rates of PD in personnel stationed there to the PD rates in personnel stationed at bases where the water was not contaminated. Just as the differential cholera rates between the pumps demonstrated a causal relationship between cholera and the water in 1800s London, the differential PD rates between Camp Lejeune and other marine bases demonstrates a causal relationship here.

The significant evidence from the above valid and methodologically sound studies establishes that exposure to the levels of the toxins in the drinking water at Camp Lejeune were hazardous to human beings generally and are known causes of Parkinson's Disease. Much of the evidence related to Camp Lejeune stems from sophisticated natural experiment designs, particularly research comparing Parkinson's Disease incidence rates in regions with higher and lower or negligible exposure. Some studies specifically examined PD among personnel stationed at Camp Lejeune compared to those at bases with uncontaminated water supplies.

This body of evidence provides a foundation for analyzing the levels of chemical exposure generally harmful to humans. Numerous studies document elevated Parkinson's Disease hazards associated with varying levels of exposure to these chemicals. Observing increased hazards and risks at these levels demonstrates that such exposures are clearly capable of causing PD, even though it is likley that lower levels are also hazardous and pose these same risks.

A limitation in defining hazardous levels of exposure to TCE, PCE, benzene, or vinyl chloride is the absence of randomized controlled trials. Determining precise thresholds would require unethical and impractical long-term studies exposing human participants to these chemicals and

monitoring their health outcomes over decades. Instead, there is data from observational studies that estimate exposure levels and assess whether affected populations show higher-than-expected PD rates.

It is uncommon for humans to be exposed to environmental chemicals in a way that allows for a precise assessment of negative effects. Epidemiological evidence often reflects gaps in toxicological prevention or regulatory oversight. However, research into TCE, PCE, benzene, and vinyl chloride—especially federal investigations into Camp Lejeune—offers robust data on the consequences of exposure to these chemicals at various concentrations and durations. This data provides compelling evidence that the water contamination at Camp Lejeune were at levels known to cause Parkinson's Disease.

While epidemiological data often focuses on specific dose ranges where elevated risks have been observed, these should not be interpreted as definitive minimum thresholds below which no hazard exists. Rather, these ranges reflect only the levels studied. It is indeed very likely that lower concentrations also contribute to PD risk.

The available data indicates that the levels of chemical exposure at Camp Lejeune were hazardous to humans and are known to cause Parkinson's Disease. Epidemiological findings clearly demonstrate increased Parkinson's Disease hazards at these levels and provides strong support that levels lower than those found specifically in the epidemiology are also hazardous to humans generally and are known to cause Parkinson's Disease.

In my opinion, the water at Camp Lejeune more likely than not causes Parkinson's Disease — comfortably exceeding the at least as likely standard set forth by Congress. Furthermore, I believe that the quantitative risk of Parkinson's Disease from exposure to the combination of TCE, PCE, vinyl chloride, and benzene is more likely than not additive or even higher.

V. The Chemicals Found at Camp Lejeune That Can Cause Parkinson's Disease

The major drinking-water contaminants of interest at Camp Lejeune are volatile organic chemicals (VOCs): TCE and PCE, but also benzene and vinyl chloride (as well as other chemicals that I will not address in this report). All those except benzene are halogenated, short-chain aliphatic hydrocarbons (halocarbons) - benzene is an aromatic hydrocarbon.

A. CHLORINATED ETHYLENE ORGANIC SOLVENTS (TCE, PCE, VINYL CHLORIDE, AND THEIR RELATIVES)

To understand how exposure to the contaminated water at Camp Lejeune can cause PD, it's useful to consider how the underlying chemicals present can affect human health generally. TCE, PCE, benzene, and vinyl chloride are all organic solvents.

A solvent is a substance that dissolves another substance. While water is the most common solvent,

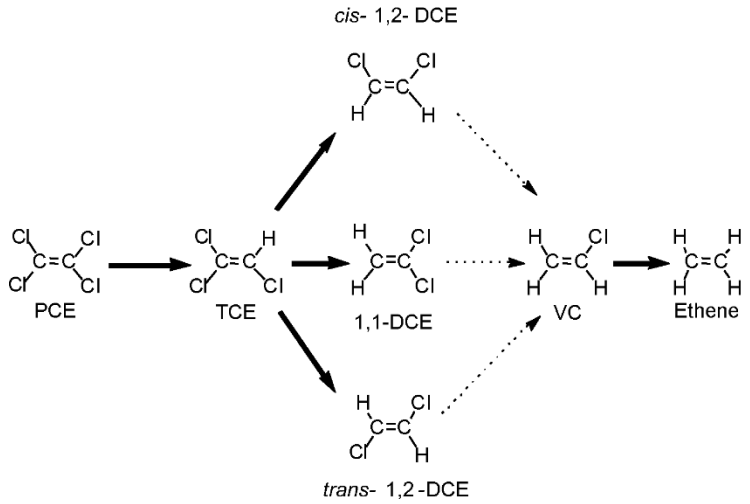
a number of substances (especially those with oils as part of their make-up), do not dissolve well in water. Organic solvents are a class of solvents made up of chemical compounds - primarily carbon and hydrogen (hence the term "hydrocarbon") - sometimes combined with other elements (e.g., chlorine), are often used to aid this process. These solvents dissolve fat and oil easily. In turn, organic solvents are also able to dissolve in fat.

As a result of their ability to dissolve in fat (known as lipophilicity), organic solvents can permeate the human body. For example, organic solvents are uniquely able to affect the brain and nervous system by easily crossing the "blood brain barrier." This natural protective barrier separates circulating blood from the fluid from the brain, isolating the central nervous system from the rest of the body. Because of the substantial fatty component in skin, organic solvents are also easily absorbed through the skin.

Many organic solvents are volatile (easily evaporated), leading to possible exposure through inhalation. Where these solvents are present in the water supply, they can easily move into the air under conditions such as showering, dishwashing, or toilet flushing. They can also enter homes through groundwater in a process known as vapor intrusion.

Because TCE, PCE, benzene, and vinyl chloride are organic solvents, their presence in a primary water source, like at Camp Lejeune, can result in exposure through ingestion of food and water, inhalation of indoor air, and absorption through the skin. It has been estimated that exposure to occupants through the air and skin from the drinking water source alone is roughly equal to, or may even exceed, exposure from ingestion.

A subcategory of organic solvents is chlorinated volatile organic compounds (CVOCs), which can be divided into three groups based on their structures (methane, ethane, and ethylene). Of particular relevance in this case is the third subclass: chlorinated ethenes (also known as chlorinated ethylene). Chlorinated ethenes share on a common backbone: a variation on the underlying structure of the hydrocarbon known as ethylene. Ethylene is composed of two carbon atoms, connected by a double bond. Each carbon has two more places to connect other atoms. If all four connections are to hydrogen atoms, the result is ethylene (far right, figure below). Chlorinated ethenes result when a chlorine atom replaces at least one of the hydrogens.

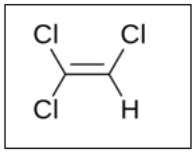


In PCE, two chlorine atoms attach to each carbon atom, displacing all of the hydrogens completely. The other chlorinated ethenes are sometimes referred to as PCE's "daughter products" because through reductive dechlorination, PCE will degrades to TCE, then to a form of dichloroethylene (DCE), to vinyl chloride, and finally to ethylene. In turn, ethylene can degrade to carbon dioxide and water.

While very little data exists on how dichloroethylene affects humans and most regulatory bodies have not evaluated its carcinogenicity "due to lack of information," [ATSDR DEC ToxFAQ 2023, at 2], the National Toxicology Program classifies DCE as "reasonably anticipated to be a human carcinogen" due to studies in experimental animals. [National Toxicology Program 15th Report on Carcinogens, 2021]. This is consistent with PCE's other daughter products: TCE and vinyl chloride. [IARC Monograph 106, 2014 at 189] ("There is sufficient evidence in humans for the carcinogenicity of [TCE]"); [IARC Monograph 97, 2008 at 425] ("There is sufficient evidence in humans for the carcinogenicity of vinyl chloride."). PCE itself is classified as "probably carcinogenic to human." [IARC Monograph 106, 2014 at 329].

The closely related chemical structure of chlorinated ethenes, specifically the three present at Camp Lejeune, does not mean they cause the same biological effects but can inform a deeper understanding of the scientific literature, especially to the degree there are limits to current research.

1. Trichloroethylene (TCE)



TCE is a human-made, colorless, volatile liquid chemical that is used as a solvent and in many other applications. TCE is used as a solvent to remove grease from metal, as a paint stripper, and in the production of other chemicals. It can also be found in some household products, such as cleaning wipes, paint removers, and adhesives. TCE is a volatile organic compound (VOC) that is highly persistent in the environment, contaminating soil and groundwater, as occurred at Camp Lejeune. Both "animal studies and human epidemiology support a causal association of TCE with PD." [Goldman 2024

at 3]

a. Animal Literature

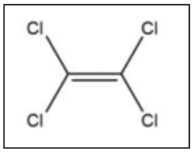
"Animal studies have supported a causal association of TCE with PD." [Goldman 2023 at 678] "TCE is a implicated as a principal risk factor for parkinsonism based [in part] on its dopaminergic neurotoxicity in animal models." [Gash 2008 at 191] Studies conducted using laboratory animals show that animals exposed to TCE developed PD-like symptoms. See Bove 2024 at 11 ("In animal studies, TCE exposure reproduces key pathological features of Parkinson disease."). Numerous studies in rodents confirm "that TCE caused the death of midbrain dopaminergic neurons, and could recapitulate other pathological hallmarks of PD, such as neuroinflammation, and a-synuclein accumulation." [DeMiranda 2021 at 4] "TCE has been found to be a mitochondrial neurotoxin in animal studies, and mitochondrial dysfunctions in substantia nigra dopamine neurons is consistent with human pathological staging of Parkinson disease." [ATSDR Morbidity Study 2018 at 54]

b. Human Epidemiology

Two human epidemiological studies confirm the link between occupational TCE exposure and PD. Gash (2008) documented a "cluster of 20 industrial coworkers with Parkinson's disease and parkinsonism subjected to long term chronic exposure to [TCE]." [Gash 2008 at 184] The three workers "with workstations adjacent to the trichloroethylene source" all developed PD. *Id*. Their colleagues who worked slightly farther away "displayed many features of parkinsonism, including significant motor slowing." The authors interpreted the results of their study as showing that TCE is "a risk factor for parkinsonism," [Gash 2008 at 184], and "demonstrate[ing] a strong potential link between chronic TCE exposure and parkinsonism." [Gash 2008 at 191]

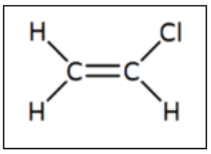
The 2012 publication by Goldman *et al*. is a study of extremely high-quality design. The authors examined a cohort of 99 pairs of twins discordant for PD, i.e., one of the twins had PD and the other did not. They then calculated each twin's TCE exposure, utilizing "occupational exposure assessment methods" that probed whether the twin had been exposed to TCE via his job or hobby. For the twin with any exposure to TCE, they found an odds ratio of 6.1 (95% CI 1.2-33.0), meaning that the twin who was exposed to TCE was more than six times as likely to develop PD than the twin who was not exposed. They observed similar results when estimating the duration and cumulative lifetime exposure to TCE, i.e., the twins with more TCE exposure showed substantially higher risks of developing PD than the twin exposed to less TCE. The authors also demonstrated a link between PCE and PD. The odds ratio for this analysis was 10.5 (95% CI 0.97-113), indicating that the twin exposed to PCE was more than 10 times as likely to develop PD than the twin who was not exposed to PCE. Again, similar results were shown for duration of PCE exposure and cumulative PCE exposure. The authors also demonstrated a latency for PD of up to 40 years, suggesting that "exposure may trigger a degenerative cascade dependent on the passage of time." [Goldman 2012 at 6] As the authors concluded, given these results, "the public health implications are considerable."

2. Perchloroethylene (PCE)



Perchloroethylene (also known as tetrachloroethylene) is widely known for its wide use in the dry-cleaning industry, but it has had other uses in industry. In the 1950s, roughly 80% of PCE was used for dry-cleaning; today, PCE use has been phased out in some states, and much less is used in dry-cleaning. Other industrial uses of PCE include as a degreaser and chemical synthesis intermediate.

3. Vinyl Chloride



Vinyl chloride is a volatile compound used almost exclusively by the plastics industry to produce polyvinyl chloride (PVC). Vinyl chloride has been detected at low concentrations in the air in the vicinity of vinyl chloride and PVC manufacturing plants and hazardous waste sites. Vinyl chloride has also contaminated groundwater from spills, landfills, and industrial sources. Vinyl chloride can also enter groundwater after being produced as a byproduct during the degradation of TCE and PCE.

B. IT IS MECHANISTICALLY PLAUSIBLE FOR THESE CHEMICALS TO CAUSE PD TOXICITY

1. TCE and Parkinson's Disease

TCE is implicated in the pathogenesis of Parkinson's Disease through several mechanisms.

Firstly, TCE exposure leads to the formation of the neurotoxin 1-trichloromethyl-1,2,3,4-tetrahydro-B- carboline (TaClo), which selectively induces dopaminergic neurodegeneration. This has been demonstrated in rodent models where TCE exposure resulted in significant loss of dopaminergic neurons in the substantia nigra and striatum, along with motor deficits. Secondly, TCE is a mitochondrial toxicant. It impairs mitochondrial complex I activity, leading to increased oxidative stress and microglial activation, which are critical factors in dopaminergic neuron death. 3-4) This mitochondrial dysfunction is similar to the mechanism of action of other known environmental dopaminergic neurotoxins like MPTP. [Gash 2008].

It has been shown that significant damage to the nigrostriatal dopamine system occurs in rats following TCE administration. Gash *et al.* found that TCE exposure resulted in a loss of dopaminergic neurons as well as a concomitant reduction in dopamine in the striatum and midbrain. While the exact mechanisms of action of TCE on these neurons is not known, they were able to show that TCE inhibits mitochondrial complex I, as seen with other parkinsonian mimetics, such as MPTP toxicity. [Gash 2008]. Another study demonstrated similar findings of loss of dopamine neurons in rats exposed to TCE, in addition to other pathological hallmarks of PD, such as accumulation of alpha-synuclein and an increase in reactive oxygen species [Liu M. J Neurochem 2010;112:773-83].

2. PCE and Parkinson's Disease

The mechanism of Parkinson's disease caused by PCE involves mitochondrial dysfunction and oxidative stress. PCE has been shown to induce mitochondrial toxicity, which is a key pathological trigger in the selective vulnerability of dopaminergic neurons. This mitochondrial dysfunction leads to elevated oxidative stress and impaired autophagic removal of damaged mitochondria, contributing to dopaminergic neurodegeneration.

The mechanism for PCE in Parkinson Disease is largely extrapolated from that of TCE based on common metabolites. "There is incomplete evidence for PCE [and DCM] with regard to injury to the SNpc. There are, however, some common findings for TCE and PCE, such as formation of the metabolite TCA and minor metabolites via conjugation with glutathione. Assuming one of these common metabolites is responsible for the neuronal injury, then PCE may produce a similar effect in rodents to that of TCE." [Lock EA. Toxicol Appl Pharmacol 2013266:345–55]

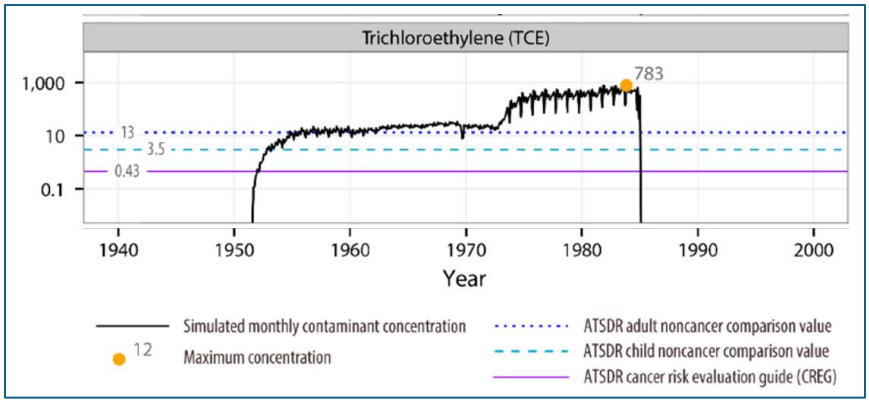
VI. CHEMICAL CONTAMINATION IN THE WATER AT CAMP LEJEUNE SPANNED DECADES

The ATSDR conducted mathematical modeling to simulate the contaminants in the water supplied to base housing and other facilities at Camp Lejeune. The modeling results revealed that water provided by the Tarawa Terrace and Hadnot Point Water Treatment Plants was contaminated with various levels of PCE, TCE 1,2-tDCE (trans-1,2-dichloroethylene), vinyl chloride, and benzene between 1953 and 1987. Detailed monthly mean contaminant concentrations over time for Tarawa Terrace, Hadnot Point, and Holcomb Boulevard are documented in the ATSDR tables and were also provided to me in Appendices H1, J, and K of the October 25, 2024, Expert Report by Morris L. Maslia.

At the Tarawa Terrace Water Treatment Plant, simulated PCE levels peaked at an average of 183 µg/L per month, with a single measured high of 215 µg/L, both far above the EPA's current limit of 5 µg/L. These levels exceeded the limit between November 1957 and February 1987. At Hadnot Point, simulated TCE levels averaged a maximum of 783 µg/L per month, with a one-time high of 1,400 µg/L, during the period from August 1953 to December 1984. Hadnot Point also supplied contaminated water to the Holcomb Boulevard housing area continuously until June 1972, when the Holcomb Boulevard Water Treatment Plant began operations. After that, Hadnot Point intermittently provided water with TCE levels peaking at 32 µg/L before June 1972 and 66 µg/L between June 1972 and February 1985.

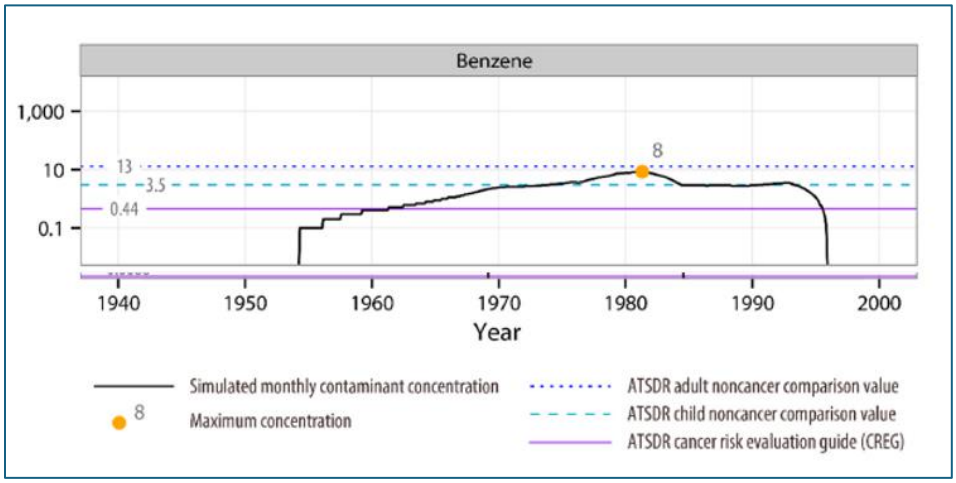
A. HADNOT POINT: TCE CONTAMINATION

At Hadnot Point, TCE concentrations ranged from 0 to 783 micrograms per liter, with a median level of 366 micrograms per liter. [Bove 2014a at 3]. The reconstructed concentrations in the water are illustrated in the graph below.



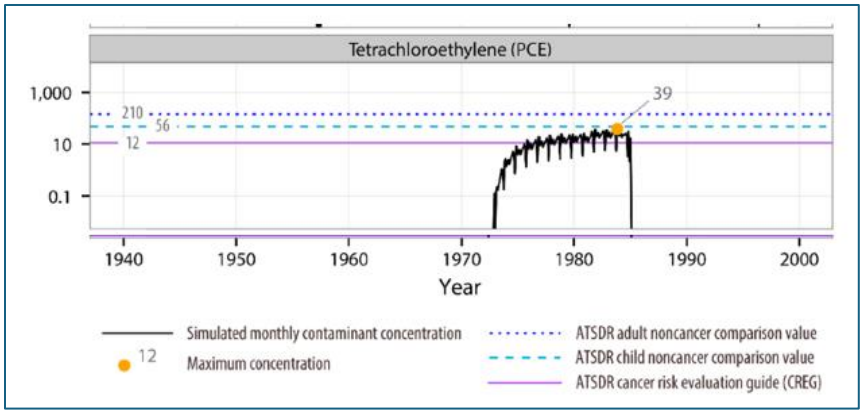
B. HADNOT POINT: BENZENE CONTAMINATION

The reconstructed benzene concentrations in the water at Hadnot Point are illustrated in the graph below.



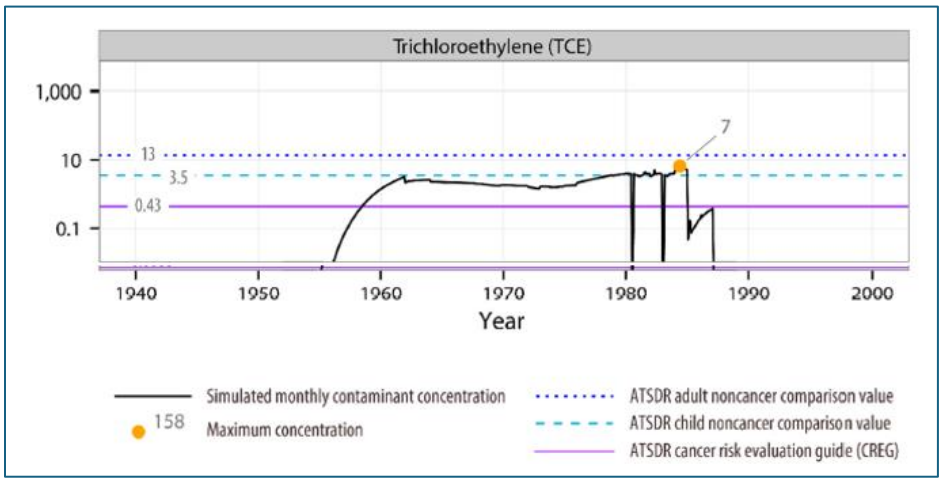
C. HADNOT POINT: PCE CONTAMINATION

At Hadnot Point, the median monthly PCE contamination was 15 micrograms per liter. [Bove 2014a at 3]. The reconstructed concentrations in the water are illustrated in the graph below.



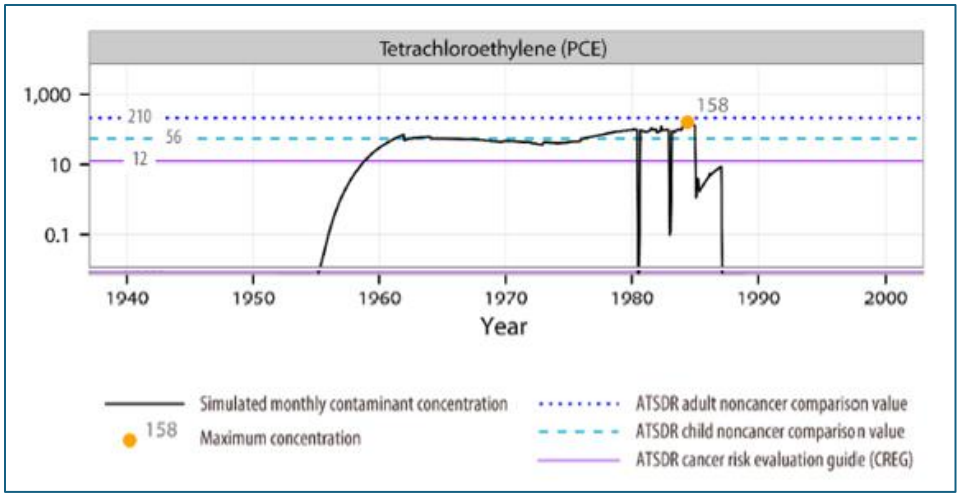
D. TARAWA TERRACE: TCE CONTAMINATION

The reconstructed TCE concentrations in the water at Tarawa Terrace are shown below.



E. TARAWA TERRACE: PCE CONTAMINATION

At Tarawa Terrace, concentrations of PCE ranged from 0 to 158 µg/liter, with a median of 85 µg/liter [Bove 2014a at 3]. The reconstructed concentrations in the water are illustrated in the graph below.



F. HOLCOMB BOULEVARD

Holcomb Boulevard was also affected by the contamination. Before 1972, its water supply came from Hadnot Point water system, meaning the exposure analysis for Holcomb Boulevard aligns with that of those in Hadnot Point. "Prior to 1972, the assessment of exposure and risk for Holcomb Boulevard Housing residents would be the same as that for Hadnot Point." [ATSDR PHA at 14]

Even after 1972, Holcomb Boulevard intermittently received water from Hadnot Point, with TCE concentrations ranging from 38 ppb to 1,148 ppb. These concentrations exceeded CREG limits of 0.43 ppb by factors ranging from over 80 to more than 2,500.

VII. ROUTES OF EXPOSURE TO THESE CHEMICALS

The topic of routes of exposure for the relevant identified chemicals naturally relates to the issue of the sources and concentrations of the subject chemicals in the potable water at the base during the statutorily delimited pertinent time period (1950s-1980s). With regard to background of the chemicals and data on their concentrations in the water, I have considered the work of the ATSDR for general background, however I do not opine on issues such as reliability of historical evidence for TCE or PCE water concentrations at the base or of ATSDR water modeling reconstruction of past contaminant levels in the water, and I defer to others with appropriate areas of expertise. With regard to historical factual background regarding the base during the time period at issue, I have in addition to ATSDR publications reviewed and considered the expert report of Dr. Kyle Longley prepared for this case. His report compiles information regarding historical facts of life and work at Camp Lejeune. I have not sought to independently verify Dr. Longley's report, however, I note that if the facts cited by Dr. Longley's report are accurate, they identify a variety of discrete VOC exposure settings at Camp Lejeune through the routes of ingestion, inhalation, and dermal exposure which are of potential significance to the present matter.

A. IN GENERAL

When evaluating the health effects of chemicals, it is important to understand how the chemicals enter and are distributed throughout the body. It is also important to understand how the body metabolizes and excretes the parent chemicals and their metabolites.

Chemicals such as PCE, TCE, benzene, and vinyl chloride are VOCs. People are exposed to VOCs in water by three major routes: inhalation, ingestion, and via dermal contact.

A number of studies have looked at the relative importance of those several routes. For example, over 25 years ago, Weisel and Jo determined estimates of internal doses of TCE due to showering [Weisel CP. and Jo WK. Environ Health Perspect 1996;104:48-51]. They concluded that inhalation and dermal exposure resulted in an internal dose of TCE comparable to the dose ingested in 2 liters of water. More recently, Gordon et al. investigated the contribution of household water use to internal doses of chloroform and other trihalomethanes [Gordon SM. Environ Health Perspect 2006;114:514-21]. They found that showering and bathing resulted in the highest blood and exhaled-breath concentrations of chloroform with both inhalation and dermal absorption being important routes of exposure.

Demonstrating the importance of water temperature, Giardino and Andelman found that the volatilization of TCE during showering was most dependent on the temperature of water [Giardino NJ and Andelman JB. J Expo Anal Environ Epidemiol 1996;6:413-23]. Adding insight to the role that all three forms of exposure play in contributing to the internal dose of TCE, Haddad et al. used assessed different home exposure scenarios and concluded that ingestion contributed less than 50% of the total absorbed dose of TCE [Haddad S. J Toxicol Environ Health A 2006;69:2095-136]. Thus, absorption from the lungs, and the gastrointestinal tract, as well as from intact and broken skin dermal contact, must be taken into account when determining the internal dose that results from use of water contaminated with VOCs.

Whatever the route of exposure to a chemical, ultimately the portion of the chemical that enters the body from the lungs, gastrointestinal tract, or skin (sometimes termed the internal dose) is the portion that exerts biological effects. Pharmacokinetics (or toxicokinetics) and physiologically based toxicokinetic ("PBTK") models are important in addressing uncertainties inherently present in health risk assessments of the water contaminants at Camp Lejeune. Toxicokinetics can be defined as the absorption, distribution, metabolism, and elimination of chemicals. The kinetic processes determine how much of an external dose is absorbed into the blood, reaches systemic circulation; binds to proteins or other sites; enters specific organs; is biotransformed (if relevant) to toxicologically active and inactive forms; interacts with target molecules, cells, and tissues; and is eliminated from the target tissue and the body [Bruckner JV. Toxic effects of solvents and vapors. In Casarett and Doull's Toxicology: The Basic Science of Poisons, 9th Ed]. One or more of those processes can vary widely from one route of exposure to another, from high to low doses, from one species to another, and from one individual to another. Furthermore, as discussed below, in a multi-chemical setting, chemicals mix. "Our knowledge of the toxicity of solvent mixtures is rudimentary relative to the toxicology of individual solvents. While the assumption is frequently

made that the toxic effects of solvents are additive, the chemicals may also interact synergistically or antagonistically." [Bruckner JV. Toxic effects of solvents and vapors. In Casarett and Doull's Toxicology: The Basic Science of Poisons, 9th Ed., Chapter 24 (Toxic Effects of Solvents and Vapors), p. 2 of 157].

Various scientific studies published regarding the relevant chemicals such as PCE and TCE have assessed exposure scenarios that involved potable water use and therefore implicitly the potable water exposure routes (ingestion, inhalation of vapor, and dermal). These have included studies reflecting comparable levels of VOC concentrations. See, e.g., Cohn P. Environ Health Perspect. 1994 Jun;102(6-7):556-61, at 557, which was a study of TCE and PCE drinking water contamination and leukemia and non-Hodgkin's lymphoma incidence in a 75-town area. "The highest assigned TCE level was 67 ppb, the highest assigned PCE level was 14 ppb…. The population-weighted concentrations of TCE and PCE in the highest categories were 23.4 ppb and 7.7 ppb, respectively. Four of the six municipalities in the highest TCE category were also in the highest PCE stratum. The population-weighted concentrations of TCE and PCE in the highest strata of the 48 municipalities added for this expanded study are 8.7 and 10.5 in 2 and 4 added towns, respectively."). Also see Fagliano J. Drinking Water Contamination and the Incidence of Leukemia: An Ecologic Study. Am J Public Health 1990;80:1209-12. That study examined the relation between the incidence of leukemias and the occurrence of VOC contamination (TCE and related solvents) of drinking water supplies within a study area. The study described the data including the mean total VOC values assigned to each town or group of towns for the analysis. TCE, PCE, TCA, and dichloroethylenes (DCE) comprised nearly all of the non-THM VOCs involved. Based on inspection of the average values for each town, three categories of total VOCs were set: 1) 37 to 72 ppb, 2) 5 to 12 ppb, and 3) down to less than 1 ppb. Among other things the authors reported that "[t]he sum concentration of all non-THM VOCs was a statistically significant predictor of total leukemia incidence, adjusted for age." Id. at p. 1211.

As another example of a study involving VOCs in drinking water, see Aschengrau A. Cancer risk and tetrachloroethylene-contaminated drinking water in Massachusetts. Arch Environ Health 1993;48:284-92, evaluating an exposure scenario involving PCE that leached from pipe liners into the public water supply. The abstract relates in part: "A population-based case-control study was used to evaluate the relationship between cases of bladder cancer (n = 61), kidney cancer (n = 35), and leukemia (n = 34) and exposure to tetrachloroethylene from public drinking water. Subjects were exposed to tetrachloroethylene when it leached from the plastic lining of drinking water distribution pipes. Relative delivered dose of tetrachloroethylene was estimated, using an algorithm that accounted for (1) residential history and duration, (2) whether lined pipe served the neighborhood, (3) distribution system flow characteristics, and (4) pipe age and dimensions. Whether or not latency was considered, an elevated relative risk of leukemia was observed among ever exposed subjects (adjusted OR = 1.96, 95% CI = 0.71-5.37, with latency; adjusted OR = 2.13, 95% CI = 0.88-5.19, without latency) that increased further among subjects whose exposure level was over the 90th percentile (adjusted OR = 5.84, 95% CI = 1.37-24.91, with latency; adjusted OR = 8.33, 95% CI = 1.53-45.29, without latency). When latency was ignored, there was also an increased relative risk of bladder cancer among subjects whose exposure level was over the 90th percentile (adjusted OR = 4.03, 95% CI = 0.65-25.10)." See id. A later publication regarding the

same site described that "[t]ypical levels [of PCE slowly leaching from vinyl pipe liners] in affected towns ranged from 1,600 to 7,750 µg/L in low-flow locations, and from 1.5 to 80 µg/L in medium- and high-flow locations." [Aschengrau A. Environ Health Perspect, 2003 Feb;111(2):167-73 at 167.]

B. ABSORPTION VIA INGESTION

1. TCE

The evidence for oral ingestion absorption of TCE from water contamination is well-documented. Studies have shown that TCE is absorbed through the gastrointestinal tract when ingested. With regard to animal studies, Liu *et al.* demonstrated that TCE exhibits linear kinetics in rats over a dosage range of 0.1 to 5.0 mg/kg, with bioavailability ranging from 12.5% to 16.4%. [Liu Y. Drug Metab Dispo: Biol Fate Chem. 2009;37:1994-8]. This indicates that a significant portion of ingested TCE from water is absorbed into the bloodstream.

In volunteer studies in humans, Weisel and Jo found that ingestion of TCE from tap water leads to its complete metabolism before entering the bloodstream, suggesting that the absorbed dose is metabolized primarily in the liver. [Weisel CP and Jo WK. Environ Health Perspect. 1996;104:48-51] This presystemic metabolism reduces the amount of TCE reaching systemic circulation, but does not negate the fact that absorption occurs. Furthermore, as discussed in the mechanism of toxicity section of this report, TCE and the other halogenated hydrocarbons undergo metabolism to more toxic chemicals that damage DNA and cause cancer. [See Bruckner JV. Toxic effects of solvents and vapors. In Casarett and Doull's Toxicology: The Basic Science of Poisons, 9th Ed., at Chapter 24 (Toxic Effects of Solvents and Vapors), p. 25 of 157, stating that "[t]he adverse effects of TCE … are generally believed to be associated with TCE's metabolites."]. Publications since Weisel and Jo 1996 provide further insight on the mechanism of TCE absorption via ingestion. [*E.g.*, Lash LH, Fisher JW, Lipscomb JC, Parker JC. Metabolism of trichloroethylene. Environ Health Perspect. 2000 May;108 Suppl 2(Suppl 2):177-200. doi: 10.1289/ehp.00108s2177. PMID: 10807551; PMCID: PMC1637769.]

Additionally, Mortuza *et al.* reported that TCE exhibits nonlinear toxicokinetics with a disproportionate increase in area under the curve and a decrease in clearance with increasing doses, in rats further supporting the absorption and systemic distribution of TCE following oral ingestion. [Mortuza T. Toxicol Appl Pharmacol 2018;360:185-92].

2. PCE

Studies of controlled dosing of PCE in humans are lacking. In one case report, PCE was detected in blood at a concentration of 21.5 µg/mL approximately 1 hour after ingestion by a 6-year-old boy who had ingested between 12 to 16 g of PCE, demonstrating that PCE is absorbed following oral exposure in humans [Koppel C. Clin Toxicol 1985;23:103-15]. The evidence for oral ingestion absorption of PCE from water contamination is also supported by several studies. In a rat study by Frantz and Watanabe, they found after drinking-water administration, the elimination kinetics of PCE were not substantially different from the disposition resulting from inhalation [Frantz SW

and Watanabe PG. Toxicol Appl Pharmacol 1983;69:66-72]. Pegg et al. also found that absorption of inhaled or oral PCE were essentially identical in rats [Pegg DG. Toxicol Appl Pharmacol 1979;51:465-74]. Similarly, PCE is nearly completely absorbed in dogs given a single dose by gavage [Dallas CE. Environ Res 1994;67:54-67].

In addition, a study by Wittlingerová *et al.* provides evidence of PCE contamination in surface water and its subsequent bioaccumulation in fish, indicating that PCE is indeed absorbed by organisms in contaminated water environments. [Wittlingerová Z. Environ Sci Poll Res Int 2016;23:5676-92].

The US EPA in 2012 completed a comprehensive toxicological review of PCE, which included an assessment of its toxicokinetics and metabolism. This review indicated that PCE is absorbed through the gastrointestinal tract when ingested, leading to systemic exposure. [Guyton KZ. Environ Health Perspect 2014;122:325-34].

3. Benzene

The evidence for oral absorption of benzene from water contamination is supported by several studies. In 1996, Beavers *et al.* assessed household exposure from gasoline-contaminated drinking water and found that ingestion of contaminated water contributed significantly to the total benzene dose, although inhalation during activities such as showering also played a major role. [Beavers JD. J Occup Environ Med 1996;38:35-8].

Santos et al. conducted a risk assessment following a gasoline station fuel leak and found that the population exposed to benzene-contaminated water had a significant intake of benzene through ingestion, with estimated benzene intake from water and food reaching up to 0.0091 µg/kg /day. [Santos M dos A. Rev Saúde Pública 2013;47(2):335-44]. The facts reflected that "the community was exposed to benzene from water consumption for 195 days and from water dermal contact and water vapor inhalation for 315 days. The mean concentration of benzene in the water estimated by the model during the oral exposure period (range of 5.1 to 235.5 µg/) was 72.6 µg/L (95%CI 40.9;104.2)."

See also generally Harrison R, Delgado Saborit JM, Dor F, et al. Benzene. In: WHO Guidelines for Indoor Air Quality: Selected Pollutants. Geneva: World Health Organization; 2010 ("Absorption of benzene is also rapid via the oral and dermal routes. Rats absorb and rapidly metabolize oral doses of benzene up to approximately 50 mg/kg.").

4. Vinyl chloride

The evidence for absorption of vinyl chloride after oral ingestion from water contamination is primarily derived from animal studies. Research indicates that vinyl chloride is absorbed and metabolized following oral administration. For instance, Green and Hathway demonstrated that after oral administration of C^{14} vinyl chloride to rats, the compound is primarily eliminated via the pulmonary route, with both unchanged vinyl chloride and its metabolites being excreted through the lungs and kidneys. This study also showed that the biotransformation of vinyl chloride involves

several metabolic pathways, leading to the formation of various metabolites, including S-(2-chloroethyl) cysteine and N-acetyl-S-(2-chloroethyl) cysteine. [Green T and Hathway DE. Chem-Bio Interact 1975;11:545-62].

Additionally, Watanabe and Gehring found that the disposition of vinyl chloride in the body is dose-dependent, with higher doses saturating metabolic or detoxifying pathways, which could correlate with its oncogenic potential. [Watanabe PG and Gehring PJ. Environ Health Perspect 1976;17:145-52] This suggests that vinyl chloride is absorbed and metabolized in a manner that is influenced by the dose ingested.

See also generally World Health Organization 2004. Vinyl Chloride in Drinking-water. WHO/SDE/WSH/03.04/119, at 4 ("Animal studies show absorption of more than 95% after oral exposure.").

C. ABSORPTION VIA INHALATION

1. TCE

Weisel and Jo's research highlighted that individuals are exposed to volatile compounds like TCE from tap water not only through ingestion but also via inhalation and dermal absorption during activities such as showering. Their study found that inhalation exposure during showering can significantly increase the body burden of TCE, indicating that inhalation is a critical route of exposure from contaminated water. [Weisel CP and Jo WK. Environ Health Perspect 1996;104:48-51]. The investigators concluded that "[t]he internal dose derived from inhalation can be calculated from the air concentration, breathing rate, duration of the shower, and adsorption efficiency across the lung barrier." Furthermore, Weisel and Jo found that "approximately equivalent amounts of volatile contaminants from water can enter the body by three different exposure routes, inhalation, dermal absorption, and ingestion, for typical daily activities of drinking and bathing. However, the exposure route affects the rates of metabolism and therefore the compound's potential toxicity. The ingested VOCs were metabolized during the first pass through the liver, thus the parent compound was not measurable in the exhaled breath and would not be present in the bloodstream. However, chloroform and trichloroethene concentrations were measurable in the breath after inhalation and dermal exposure, indicating dispersion throughout the body."

Furthermore, Liu *et al.* assessed the health risks associated with different exposure pathways of volatile chlorinated hydrocarbons, including TCE, in contaminated drinking groundwater. They found that inhalation during showering posed a higher risk compared to oral ingestion, underscoring the importance of inhalational exposure in the overall risk assessment. [Liu W. Environ Pollut 2009;255:113339].

See also ATSDR Public Health Statement for Trichloroethylene (TCE), CAS#: 79-01-6 ("When trichloroethylene is found in water, it can enter your body when you drink or touch the water or when you breathe in steam from the water. Most of the trichloroethylene that you breathe in or drink will move from your stomach or lungs into your bloodstream.").

2. PCE

The evidence for inhalational absorption of PCE from water contamination is supported by several studies that demonstrate the presence of PCE in indoor air and exhaled breath following exposure to contaminated water sources.

Garnier *et al.* described the case of a boy who died in a room in which the curtains had been dry cleaned with PCE. He was asymptomatic when the door and windows were opened (thus providing ventilation), but when the door and windows were closed and he was put to bed for a nap, he died due to PCE exposure. On post-mortem examination he was found to have PCE in his blood at a concentration of 66 µ/mL [Garnier R. Clin Toxicol 1996;34:191-7].

One study conducted in Martinsville, Indiana, found that PCE was detected in all exhaled breath samples from residents living in areas with groundwater contamination, as well as in tap water samples from their homes. This indicates that PCE can volatilize from contaminated water into indoor air, leading to inhalational exposure. [Liu S. Environ Pollut 2022;297:118756]

Another study measured chlorinated hydrocarbons, including PCE, in indoor air and exhaled air samples from individuals exposed to soil contamination. The study found significant levels of PCE in both indoor air and exhaled breath, demonstrating that PCE can intrude into indoor environments from contaminated sources and be absorbed through inhalation. [Scheepers PTJ. Sci Total Environ 2019;653:223-230].

3. Benzene

The evidence for inhalational absorption of benzene from water contamination is well-documented in the literature. Because benzene is also a volatile organic compound, it can be released into the air from contaminated water, particularly during activities that increase water agitation and temperature, such as showering or bathing.

A study by Beavers *et al.* assessed household exposure to benzene from gasoline-contaminated drinking water and found that inhalation exposure during showering contributed significantly to the total benzene dose. The estimated inhaled doses of benzene were similar to the ingested doses, with over half of the inhaled dose associated with shower activities. [Beavers JD. J Occup Environ Med 1996;38:35-8].

Similarly, Santos *et al.* conducted a risk assessment following a gasoline station fuel leak and found that benzene levels in water vapor during showering reached significant concentrations, posing a potential health risk. The study highlighted that inhalation during showering was a critical route of exposure, contributing to the overall benzene intake. [Santos M dos A. Rev Saúde Pública 2013;47(2):335-44].

4. Vinyl chloride

Pleil and Lindstrom demonstrated that vinyl chloride can be absorbed through inhalation during

activities such as showering with contaminated water. They used the "single breath canister" technique to measure volatile organic compounds in exhaled breath, showing that vinyl chloride is detectable in breath samples post-exposure, indicating absorption through the respiratory route. [Pleil JD and Lindstrom AB. ClinChem 1997;43:723-30].

Additionally, studies on the pharmacokinetics of vinyl chloride in animal models, such as those by Buchter *et al.* and Hefner *et al.*, provide further evidence of rapid absorption and metabolism of vinyl chloride following inhalation. These studies showed that vinyl chloride equilibrates quickly and is extensively metabolized, supporting that inhalation is a significant route of exposure. [Buchter A. Toxicol Lett 1980;6:33-36; Hefner RE. Environ Health Perspect1975;11:85-95].

D. ABSORPTION VIA DERMAL EXPOSURE.

1. TCE

For 60 years the measurement of skin absorption of organic solvents such as TCE has been determined experimentally via their rates of decay in alveolar air [Stewart RD and Dodd HC. Am Industr Hyg Assoc J 1964;25:439-46]. However, it is unclear whether breath concentrations alone are a reliable measure of skin absorption. One reason for that is that significant differences in pharmacokinetics of chemicals can occur depending on the method of absorption. [Dollery CT. Ann NY Acad Sci 1971;179:108-14].

The evidence for dermal absorption of TCE from water contamination is supported by several studies. [Poet TS. Toxicol Sci 2000;56:61-72]. Poet *et al.* demonstrated that TCE can be absorbed through the skin in both rats and humans, with human skin showing a lower permeability coefficient (K(P) compared to rat skin. Specifically, the K(P) for TCE in a water matrix was 0.015 cm/h in humans, indicating that dermal absorption is a significant route of exposure. Nakai *et al.* also measured the permeability coefficient of TCE through human skin *in vitro*, finding a value of 0.12 cm/h, which supports the opinion that TCE can penetrate human skin from aqueous solutions. [Nakai JS. J Toxicol Environ Health 1999;58:157-70]

Weisel and Jo further corroborated these findings by showing that dermal absorption, along with inhalation, contributes to the total body burden of TCE from tap water exposure. [Weisel CP, Jo WK. Environmental Health Perspectives. 1996;104(1):48-51].

2. PCE

The evidence for dermal absorption of PCE from water contamination is supported by several studies. Nakai *et al.* also demonstrated that the permeability coefficient of PCE through human skin is 0.018 cm/h [Nakai JS. J Toxicol Environ Health 1999;58:157-70]. Dermal absorption of PCE occurs with exposure to the vapor form as well as the liquid form. When volunteers' forearms and hands were exposed to tetrachloroethylene vapor at a concentration of 6.68 mmol/L for 20 minutes, the absorption rate of PCE was 0.054 cm/h (3 times greater than the estimate of Nakai *et al.*), with a peak exhaled air concentration occurring 45 minutes after exposure began [Kezic S. Int Arch Occup Environ Health. 2000;73:415-22].

Citing a study by Bogen, the ATSDR has written that "a 70-kg human with a surface area of 18,000 cm^2, 80% immersed, would take up the [PCE] in 1L of water (of the total amount of water in which the person was immersed) in 20 minutes" [ATSDR Toxicological Profile for Tetrachloroethylene, 1997]. Studies such as these conclusively demonstrate that dermal absorption of PCE does occur and relevant and clinically important rates.

Hake and Stewart reviewed human exposure to PCE and noted that skin absorption can add to the overall exposure burden, particularly in occupational settings where both inhalation and dermal contact occur. [Hake CL and Stewart RD. Environ Health Perspect. 1977;21:231-8]. The authors stated, "Though absorption through the skin is usually not of as great consequence as through the lungs, it should not be overlooked as a contributory factor to the [PCE] body burden. . . ."

3. Benzene.

The evidence for dermal absorption of benzene from water contamination is supported by several studies that have investigated the percutaneous absorption of benzene in various settings. Williams *et al.* reviewed and analyzed data from multiple studies and found that the steady-state dermal flux for benzene-saturated aqueous solutions ranges from 0.2 to 0.4 mg/(cm^2·h). [Williams PR. Crit Rev Toxicol 2011;41:111-42] This indicates that benzene can penetrate the skin at measurable rates when present in water.

Modjtahedi and Maibach conducted an *in vivo* study on human subjects and found that the total absorption of benzene through the skin was nominal, with forearm exposure showing an average total absorption of 0.07±0.04% and palmar exposure an average total absorption of 0.13±0.04% of the applied dose. [Modjtahedi BS and Maibach HI. Food Chem Toxicol. 2008;46:1171-4]. These findings suggest that while dermal absorption of benzene from water is possible, the overall absorption rates are relatively low under controlled conditions.

4. Vinyl chloride.

Data regarding the dermal absorption of vinyl chloride are mixed. According to a review on the systemic absorption of chemical vapors, the dermal contribution ratio (DCR) for vinyl chloride is approximately 0.0002, indicating that the amount absorbed through the skin relative to total intake (skin and inhalation) is low. This suggests that vinyl chloride is primarily absorbed through inhalation rather than through the skin. The low DCR is largely explained by the chemical properties of vinyl chloride, such as its octanol:water partition coefficient and vapor pressure. [Rauma M. Adv Drug Deliv Rev 2013;65:306-14].

VIII. LITERATURE REVIEW

As detailed above, there is abundant evidence that TCE and PCE can cause PD. But this literature is unusual—and stronger—because of the human epidemiology studies that looked at people exposed to these chemicals at Camp Lejeune specifically. Often when evaluating a contamination event, epidemiology examining the specific exposure in question is lacking, requiring researchers to rely on analogies to occupational studies or similar contamination events in order to determine

whether there is likely to be an increased risk. But here, there is data on the precise exposure of interest, i.e., exposure to the water at Camp Lejeune, and what effect that exposure has on the resulting risk of PD. These studies demonstrate that exposure to the water at Camp Lejeune is indeed associated with a higher risk of PD later in life.

This is doubly beneficial. First, The Camp Lejeune studies also give insight into the cumulative effects of multiple chemical exposures. Many Camp Lejeune residents were exposed to TCE, PCE, benzene, and vinyl chloride simultaneously. Studies and scientific literature suggest that these exposures are not merely additive but could interact synergistically, creating a combined risk that is greater than the sum of individual risks. [Rosenfeld 2024 at 14] This interaction amplifies the potential harm, reinforcing the need to consider the full scope of the exposure rather than isolating each chemical's effect.

Second, the Camp Lejeune data provides conclusive evidence that the chemical concentrations present were sufficient to induce leukemia. The unique epidemiological focus on Camp Lejeune offers a rare and strong data that supports and strengthens the existing body of evidence, leaving little doubt about the harm posed to humans by these contaminants at the levels detected in the water at Camp Lejeune.

A. BOVE 2014A STUDY: *CANCER MORTALITY STUDY OF MARINES AND NAVY PERSONNEL EXPOSED TO CONTAMINATED DRINKING WATER AT CAMP LEJEUNE, NORTH CAROLINA*

The Bove 2014a study examined cancer mortality rates among military personnel stationed at Camp Lejeune compared to those at Camp Pendleton. This was a quasi-experimental design: Camp Lejeune and Camp Pendleton are both military bases and likely to be similar populations. The key difference is that people at Camp Lejeune were exposed to the chemicals of interest here (TCE, PCE, benzene, and vinyl chloride) while people at Camp Pendleton were not.[1]

Although this study showed a link between the water and several other diseases, the sample size was too small to calculate the relative risk for PD. Bove 2014a at 7 ("SMRs for male breast cancer and Parkinson's disease were not calculated because there were <5 cases in each cohort").

B. BOVE 2014B STUDY: *CANCER MORTALITY STUDY OF CIVILIAN EMPLOYEES EXPOSED TO CONTAMINATED DRINKING WATER AT CAMP LEJEUNE, NORTH CAROLINA*

The Bove 2014b study compared civilian employees at Camp Lejeune with their counterparts at

[1] Camp Pendleton was chosen in these studies as a comparison based on the assumption that the Camp Pendleton cohort was not exposed to these chemicals. But there is some suggestion in the literature that even the marines and civilians at Camp Pendleton might have been exposed. If so, that does not undermine the signal being generated from these studies. To the contrary, if the Camp Pendleton cohort was also exposed, that suggests that the results comparing Camp Lejeune to Camp Pendleton would be biased toward the null, *i.e.*, it suggests that the risk ratios in these studies are *understated*, and that if a comparison had been made to a population of Marines that was truly unexposed, the risk ratios would have been even higher.

Camp Pendleton. This retrospective cohort study sought to determine whether exposure to contaminated drinking water at Camp Lejeune increased mortality risk from cancers and other chronic diseases. [Bove 2014b at 2] Since nearly all civilian workers at Camp Lejeune lived off-base, their exposure to the contaminated water occurred primarily during work hours. This setup provides compelling evidence of the potential health effects of intermittent exposure to the contaminants.

When comparing Camp Lejeune civilians to those at Camp Pendleton, the study found a hazard ratio of 3.13 (95% CI 0.76-12.86), indicating that civilians at Camp Lejeune had a 213% higher risk of PD than those at Camp Pendleton - more than a doubling of the risk. These findings reinforce the idea that the chemicals at Camp Lejeune were present in sufficient quantities to cause PD. Given the median employment duration "was about 2.5 years", the results strongly suggest that 2.5 years of employment at the base is sufficient to cause PD. [Bove 2014b at 2]. However, this does not suggest shorter periods of exposure do not also elevate the risk—it merely reflects the median employment length in this study.

There are clear differences between civilians who only worked on base and Marines who lived on base. For instance, Marines would have had daily exposure to contaminated water through activities like showering and eating, while civilians might not all have had this additional exposure, often only being on base during their work hours. Additionally, Marines may have had more exposure to harmful vapors containing chemicals like TCE, PCE, benzene, and vinyl chloride, especially if they slept in the barracks.

This study also compared civilians at Camp Lejeune with below median exposure to those with above median exposure. For TCE, the hazard ratio was 2.51, suggesting a 151% increased risk of PD. For PCE, the hazard ratio was 2.68, suggesting a 168% increased risk of PD. For TVOC (a combination of all the chemicals), the hazard ratio was 2.52, suggesting a 152% increased risk of PD.

If anything, these results likely **understate** the true risk of PD from the water at Camp Lejeune. As the authors note, "[m]any of the diseases of interest have relatively long survival rates (e.g., … Parkinson's disease) and would require long-term follow-up of the Camp Lejeune cohort to fully evaluate the health impacts of the drinking water exposures." [Bove 2014b at 11] In other words, the study was not long enough to fully detect all of the PD cases caused by the Camp Lejeune water. If the study had been longer, the risk of PD would likely have been even greater than the (already high) risks observed in this study.

The authors went on to note that "[t]here is also evidence that Parkinson's disease is underreported on death certificates to a higher extent in the southern U.S. than in other areas of the U.S." This also would understate the true risk of PD from the Camp Lejeune water, because many cases of PD caused by the water would not have been correctly classified—and thus not picked up by the Bove 2014b analysis. Finally, "only 14% of the Camp Lejeune cohort had died by the end of the study." [Bove 2014b at 12] Since this study was a mortality study (looking at diseases that caused death) this feature of the study also would seriously underestimate the true PD risk from the water

at Camp Lejeune. Other scientists reviewing Bove 2014b have made this point as well, characterizing it is an "underpowered mortality study." [Goldman 2023 at 674] Given its lack of power, it is particularly compelling that this study showed the serious increases in PD risk that it ultimately showed.

C. ATSDR 2018 STUDY: MORBIDITY STUDY OF FORMER MARINES, EMPLOYEES, AND DEPENDENTS POTENTIALLY EXPOSED TO CONTAMINATED DRINKING WATER AT U.S. MARINE CORPS BASE CAMP LEJEUNE

The 2018 ATSDR morbidity study [ATSDR. Morbidity Study of Former Marines, Employees, and Dependents Potentially Exposed to Contaminated Drinking Water at U.S. Marine Corps Base Camp Lejeune, April 2018] was conducted by the Agency for Toxic Substances and Disease Registry and surveyed over 200,000 Marines stationed at Camp Lejeune between 1972-1985, along with a comparator group of 50,000 Marines stationed at Camp Pendleton during that same period. The survey also included more than 8,000 civilians who worked at Camp Lejeune and a comparator of 7,000 civilians who worked at Camp Pendleton. The study aimed "to evaluate whether exposure to the contaminated drinking water at Camp Lejeune was associated with medically confirmed specific diseases of interest."

Although no increased PD incidence was detected among the Marines at Camp Lejeune, the authors noted the same thing that the Bove authors noted - the cohort was likely too young to detect the risk: "few cases of Parkinson disease in Camp Lejune who are younger ages led to problems with spare data." [Morbidity Study at 54] For PD, peak incidence is between 70-79 years of age, and most cases are diagnosed in people older than 65, but more than 93% of the Marines were younger than 65. This substantially dampened any signal among the Marines.

By contrast, the civilian population - which was substantially older and thus provided higher power for a PD analysis - did show an increased risk for PD among Camp Lejeune personnel. As compared to the civilians at Camp Pendleton, the civilians at Camp LeJeune had a relative of risk of 3.1. (95% CI 1.2-8.3), corresponding to a 211% increase in PD risk. These results provide compelling evidence that exposure to the water at Camp Lejeune - and exposure to the chemicals present in the water during 1972-1985 - is sufficient to cause PD.

The study also analyzed PD risk based on specific degree of chemical exposure, comparing civilians at Camp Pendleton with those at Camp Lejeune who had varying degrees of exposure to the specific chemicals water. Using water distribution models and residential locations and periods of residence at Camp Lejeune, the authors calculated cumulative and average residential exposure to each contaminant.

For civilians with low TCE/PCE exposure combined (defined as less than 10,868 ppb-months of TCE exposure or less than 457 ppb-months of PCE exposure), the odds ratio was 2.78 (95% CI 0.87-8.94), corresponding to a 178% increased risk. For civilians with medium TCE/PCE exposure combined (defined as between 10,868 and 50,563 ppb-months of TCE exposure or between 457 and 2118 ppb-months of PCE exposure), the odds ratio was 3.47 (95% CI 1.18-10.22),

corresponding to a 247% increased risk versus civilians at Camp Pendleton. For civilians with high TCE/PCE exposure combined, the odds ratio was 2.86 (95% CI 0.67-12.13), corresponding to a increased risk of 186% versus civilians at Camp Pendleton. [2018 Morbidity Study at 86] It is clear from these results that being exposed to either 457-2,118 ppb-months of PCE or 10,868-50,563 ppb-months of TCE causes PD. In fact, even lower levels of exposure are sufficient to increases the risk of PD.

Finally, the study authors compared PD in Camp Lejeune personnel who were exposed to higher amounts of TCE and PCE to personnel who were stationed at Camp Lejeune but were exposed to lower concentrations. For civilians with medium exposure (defined as between 10,868 and 50,563 ppb-months of TCE exposure or between 457 and 2118 ppb-months of PCE exposure) the relative risk was 1.81, indicating an 81% increase in PD risk. The risk at even higher exposure climbed to 2.03 (95% CI 0.52-7.93), indicating an increased risk of 103%. [2018 Morbidity Study at 88]. These findings provide compelling evidence that exposure to the water at Camp Lejeune - and therefore, the exposure to the chemicals present in the water during 1972-1985 - is sufficient to cause PD.

D. BOVE 2024A STUDY: LONG-TERM CANCER MORTALITY AMONG MILITARY PERSONNEL EXPOSED TO CONTAMINATED DRINKING WATER AT CAMP LEJEUNE

The Bove 2024a Study investigated cancer mortality using a longer time horizon than earlier studies. This research compared Navy and Marine personnel stationed at Camp Lejeune from 1972 to 1985 with a comparator group stationed at Camp Pendleton during the same period.

Navy/Marine personnel at Camp Lejeune showed a hazard ratio for dying of Parkinson's Disease of 2.0 (95% CI 0.85-4.73) compared against those at Camp Pendleton – a true doubling of the risk. Civilian employees at Camp Lejeune showed a hazard ratio of 1.15 (95% CI 0.68-1.93), corresponding to a 15% increased risk of dying due to PD. Subgroup analyses focused on the underlying cause of death from PD also showed an increased risk of 2.05 (95% CI 0.86-4.87) and 1.21 (95% CI 0.72-2.04) for the military personnel and civilians, respectively. Consistent with earlier studies, this paper demonstrates the sufficiency of the concentrations of the chemicals present at Camp Lejeune to cause PD.

Study authors also assessed the risk of PD based on exposure duration, specifically the time spent on base. For Navy and Marine personnel stationed on base, an increased risk of PD was observed even at short duration (for instance, just 1-2 quarters on base) – HR 2.07 (95% CI 0.62-6.95). [Bove 2024 Supplemental Table S6] For medium duration (2-7 quarters) the risk rose to 2.63 (95% CI 0.91-7.66). Similarly, civilians on base also showed an increased risk of PD at medium durations (e.g., 6-22 quarters on base) HR 1.19 (95% CI 0.55-2.54). [Bove 2024 Supplemental Table S8] In addition, the authors found elevated risks at high durations (e.g., 23-54 quarters) HR 1.60 (95% CI 0.88-2.90).

These findings show that even a brief exposure of 1-2 quarters on base between 1972-1985 is associated with an increased risk of PD. The same risk applies to individuals who were exposed at

different times but accumulated a dose equivalent to having spent that amount of time on base during those years. The authors ultimately concluded, "The results of this study are relevant to everyone exposed to the contaminated drinking water at Camp Lejeune" and suggested that "continued follow-up is indicated" for people who might have been exposed to these chemicals.

E. GOLDMAN 2023: *RISK OF PARKINSON DISEASE AMONG SERVICE MEMBERS AT MARINE CORPS BASE CAMP LEJEUNE*.

This study "compared the risk of PD among veterans who resided at Camp Lejeune during 1975-85 with those who resided at Camp Pendleton, a large California base that did not have contaminated water." [Goldman 2023 at 674] The study included 84,824 veterans from Camp Lejeune and 73,298 veterans from Camp Pendleton. Again, the setup was designed to be quasi-experimental, as the two populations were expected to be similar expect that those at Camp Lejeune were exposed to TCE and PCE, while those at Camp Pendleton were not (notwithstanding the potential for some water contamination at Camp Pendleton, which would push the results towards the null). Validating statistics confirmed that "populations were demographically similar." To be included in the study, Camp Lejeune personnel needed to have spent 3 months or more on base between 1975 and 1985.

In addition to looking at diagnoses of PD, the authors also looked at "prodromal PD," i.e., the prevalence of diagnoses that may precede a PD diagnosis." [Goldman 2023 at 674] This was done "because PD pathology and associated clinical features begin years before diagnosis."

The results again confirmed the increased risk of PD experienced by personnel stationed at Camp Lejeune. Overall "risk of PD was 70% higher in Camp Lejeune veterans (OR 1.70, 95% CI, 1.39-2.07). [Goldman 2023 at 676] This study again demonstrates that the water at Camp Lejeune contains sufficient levels of TCE and PCE to cause PD. It also demonstrates that three months of exposure to the water at Camp Lejeune during 1975-1985 - or exposure to similar cumulative levels of exposure during different years - is sufficient to cause PD.

F. GOLDMAN 2024: *PARKINSON'S DISEASE PROGRESSION AND EXPOSURE TO CONTAMINATED WATER AT CAMP LEJEUNE*

Building upon the results of Goldman 2023, the authors of this study sought to determine "whether PD progression is faster in individuals exposed to VOCs in water at Camp Lejeune." [Goldman 2024 at 1] The authors "found a higher risk of psychosis, falling, and fracture in a cohort of veterans with PD who were exposed to TCE and other VOCs in residential water at Camp Lejeune 40 years ago." [Goldman 2024 at 6] For veterans with above median exposure to TVOCs, the risk of psychosis, fall, and fracture was more than double the risk for those who were not exposed. [Goldman 2024 at 6] The authors ultimately concluded that "PD progression may be faster in persons exposed to [TCE] and other VOCs in water decades earlier." [Goldman 2024 at 1]

IX. OTHER RELEVANT CONSIDERATIONS

A. ADDITIVE AND SYNERGISTIC EFFECTS OF SOLVENTS AND TOXINS

Adding to the complexity of the Camp Lejeune fact set and the potential for toxicity was that the Camp Lejeune water was contaminated with at least four different chlorinated hydrocarbons--PCE, TCE, DCE, vinyl chloride – as well as by benzene. The ATSDR data reflects that when contemporaneous samples of the potable water were taken and analyzed in the early 1980s, all five chemicals were detected including at levels above today's maximum contaminant levels (MCLs).[2] Several of the chemicals were detected during the same overlapping time periods at Hadnot Point. Further, numerous individuals residing at Tarawa Terrace would have gone about their days at Hadnot Point before going home in the evening, thereby receiving exposures to mixtures of the chemicals already mixed together in the Hadnot Point water treatment system and the Tarawa Terrace water system. It is reasonably inferable that all or most individual exposures were to more than one of the relevant chemicals in the drinking water.

Further, it should be noted that these chemicals are molecules. The structural similarity between the 4 chlorinated VOCs at issue herein is striking. The differences between the molecular series proceeding from PCE to VC may be conceptualized roughly as starting with a structure with four chlorine atoms (thus the "tetra" (Latin for four) in tetrachloroethylene), then deleting one chlorine atom from the structure (leading to TCE, "tri"), then deleting one more chlorine atom (reducing to DCE, "di"), and then deleting one more (leaving vinyl chloride).

As one may intuit from the above rough conceptualization, under the right environmental conditions, PCE may degrade into TCE, and so forth, over time. Thus, in groundwater conditions, PCE initially undergoes a classical decomposition as a result of dehalogenated reduction to TCE and the Cl^- ion under aerobic conditions around Eh +100 (+50) to 0 mV. While maintaining the double bond between the carbon atoms, TCE decays under slightly reducing conditions of around Eh -50 to -100 mV to dichloroethene (DCE) and Cl^-. [Pierri D. Environ Adv 2021;5:100090]. The fact that PCE may degrade into TCE over time supports the contention that ultimately Camp Lejeune residents exposed to one of the chemicals were likely to have also been exposed to others (highlighting the question of additive effect).

PCE and TCE also upon ingestion can generate common metabolites, which can themselves by mutagenic, genotoxic, or carcinogenic.[3] "Trichloroethylene (TCE) and tetrachloroethylene (PCE) are structurally similar chemicals" and "are structurally similar chlorinated olefins." [Luo YS.

[2] *See* Figure 4 in Maslia ML. Water 2016;8:449. *See also id.* at Table 2, listing selected "measured and reconstructed (simulated) concentrations of tetrachloroethylene (PCE), trichloroethylene (TCE), *trans*-1,2-dichloroethylene (1,2-tDCE), vinyl chloride (VC), and benzene at the Hadnot Point water treatment plant."

[3] A genotoxin is a chemical or agent that can damage DNA or chromosomes in a cell, potentially causing mutations that lead to cancer or birth defects. A genotoxic agent can bind directly to DNA or indirectly damage it by affecting enzymes involved in DNA replication. Genotoxicity is a more general term than mutagenicity. A mutagen is a mutation-causing agent, such as a chemical, which results in an increased rate of mutations in an organism's genetic code. All mutagens are also genotoxins. A genotoxic carcinogen or mutagenic carcinogen can include a chemical that can damage the genetic material of a cell in a manner that can contribute to lead to cancer.

Toxicol 2018;409:33-43]. An analysis of the comparative toxicokinetics of TCE and PCE reveals that upon absorption, TCE and PCE are metabolized through oxidative and glutathione conjugation pathways. [Cichocki JA,. J Pharmacol Exp Ther 2016;359:110123]. Initial oxidation occurs on the double bond by cytochrome P450s (CYPs) to generate an epoxide, which can be further metabolized. Trichloroacetic acid (TCA) is a major oxidative metabolite of both TCE and PCE, and is a common urinary biomarker of exposure. [Forkert PG. Drug Metab Dispos 2003;31:306-11]. As of 2014, TCA was classified by the EPA as a possible human carcinogen based on evidence of carcinogenicity in experimental animals. [IARC Monograph 106, 2014]. As of 2012, TCA was considered to be a confirmed carcinogen in experimental animals.

In addition to metabolization producing TCA, TCE is also metabolized into the oxidative metabolite, trichloroethanol (TCOH), which is a TCE-specific metabolite that is formed through oxidation of TCE to chloral hydrate (CH), while PCE oxidation occurs through trichloroacetyl chloride.[4] TCOH and related chemicals have been studied for their carcinogenic potential.[5]

There are other common metabolites as between PCE and TCE. For instance, upon absorption, both TCE and PCE can enzymatically conjugate with glutathione to form dichloro- or trichloro-glutathione conjugates (DCVG or TCVG).[6] These can be further metabolized via hepatic or renal gamma-glutamyl transferase and di-peptidase to form corresponding cysteine conjugates, DCVC or TCVC, which are then n-acetylated via N-acetyltransferase to generate NAcDCVC or NAcTCVC, respectively.[7] In addition, both NAcDCVC and NAcTCVC can be deacetylated via acylase to yield DCVC or TCVC, respectively. Apart from N-acetylation, DCVC and TCVC can be further bio-activated via cysteine conjugate β lyase to generate reactive thioketenes, or flavin-containing monooxygenase to form corresponding sulfoxides.[8]

In short, the science reflects that unsurprisingly structurally similar chemicals – e.g., PCE and TCE – once absorbed into the body by ingestion, inhalation or dermal exposure routes, can be broken down or metabolized into other substances. Some of these metabolites or breakdown products are common as between the parent chemicals.

When exposure to more than one chemical occurs (as, here, to e.g. PCE and TCE), there is the potential for 3 major types of interactions: either a) the toxic effects are additive (e.g.: 1 + 1 = 2); or b), the effects are less than truly additive (e.g.: 2 + 2 = 3);[9] or c), the effects are synergistic (e.g.: 1 + 1 = 3). Any significant deviation from additivity would be classified as synergy or antagonism.

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[4] Luo 2018, citing Chiu WA. Toxicol Sci 20078;95:23-36.
[5] *See* Robert Kapp, Encyclopedia of Toxicology (Second Edition), 2005, discussing 2,2,2-Trichloroethanol and noting that acyl chlorides and free radicals that are formed from both 1,1,1-trichloroethane and 1,1,2-trichloroethane are believed to bind nucleic acids and proteins causing various cytoxic, mutagenic, and carcinogenic effects.
[6] Luo 2018, citing Lash LH. Environ Health Perspect 2000;108:177-200.
[7] *Id.*
[8] Luo 2018, citing Lash LH. Mutat Res Rev Mutat Res 2014;762:22-36.
[9] For example, assume that drinking only chemical X for a year causes a 2% chance of cancer, and drinking only chemical Y for a year causes a 3% chance of cancer; and because of some interaction between them, if one drinks both X and Y for a year, the cancer risk rises but only to 4%. Under "normal" additive conditions, using the simple math the additive effect should have been 5% instead.

Synergy can be defined as a combination effect that is greater than the additive effect expected. Synergy can also be called super additivity.

Documented evidence of various particular additive or synergistic effects of two different exposures, contaminants or stressors, include, in one study, evidence of how obesity was observed to increase the risk of arsenic-associated lung and bladder cancer by over 10-fold in individuals with elevated arsenic exposure compared to non-obese individuals. [Steinmaus C. Environ Res. 2015;142:594–601]. Studies indicate that arsenic's carcinogenicity is synergistically higher in obese individuals, smokers, and those with concurrent occupational exposures. [Steinmaus 2015; Ferreccio C. Epidemiol 2013;24:898–905]. As another specific example of apparent synergistic carcinogenic effect, a 2000 publication described a supra-additive genotoxicity of a combination of γ-irradiation and ethyl methanesulfonate in exposed mouse cells.[10]

One of the first synergistic interactions described between environmental pollutants was with a mixture of asbestos and cigarette smoke, which promotes the development of lung cancer.[11] The science of additive and synergistic interactions between multiple chemical contaminants such as, e.g., the PCE, TCE, DCE, vinyl chloride and benzene seen here, is evolving. However, the science published in the area to date is compelling and supports a qualitative conclusion (particularly under an "equipoise" or "as likely as not" standard) that Camp Lejeune Plaintiffs were exposed by multiple routes of exposure to multiple chemicals with (as likely as not) additive if not multiplicative effect.

Concepts of additive effect of multiple exposures to different carcinogens and environmental contaminants over time is related to the hypothesis of carcinogenesis as additive across various exposures and stressors. In this regard, cumulative risk assessment has been defined as the assessment of "combined risks from aggregate exposures to multiple agents or stressors, where agents or stressors may include chemical and nonchemical stressors." U.S. EPA. Framework for cumulative risk assessment. U.S Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment; Washington, DC: 2003.

X. BRADFORD HILL ANALYSIS

I generally followed the weight of the evidence approach to investigating and analyzing the data on chemical exposures and adverse health effects in the Camp Lejeune cohort, and Parkinson's Disease specifically. However, I have also evaluated adverse health effects and water through

[10] *See* Stopper H. Mutagenesis 2000;15:235-8 (from the Abstract: "While testing for genotoxicity is usually performed on single chemicals, exposure of humans always comprises a number of genotoxic agents. The investigation of potentially synergistic effects of combinations therefore is an important issue in toxicology. Combinations of 511 keV γ-radiation with the chemical alkylating agent ethyl methane-sulfonate were investigated in the in vitro micronucleus test in mouse lymphoma L5178Y cells. With combinations in the low dose linear effect range for the individual agents (0.25–2 Gy and 0.8–3.2 mM, respectively), supra-additivity by 34–86% was seen. The synergism was more pronounced at the higher dose levels. Supra-additivity was confirmed in experiments using cytochalasin B and analyzing binucleate cells only, to control for putative effects on the cell cycle. Statistical significance was shown by a 2-factor analysis of variance with interaction….").

[11] *See* Alejandro F. GeoHealth 2022;6 (so stating).

consideration of the Bradford Hill "criteria." Published as nine viewpoints in 1965, the Bradford Hill criteria are often used to determine if observed epidemiologic associations are causal. Those nine principles are: strength of association; consistency; specificity; temporality; biological gradient; plausibility; coherence; experiment; and analogy. Applying these criteria to the association between the chemicals at Camp Lejeune and Parkinson's Disease, there is substantial evidence supporting causality:

A. STRENGTH OF ASSOCIATION:

Strength of association is demonstrated by statistical significance. That is, an odds ratio for the occurrence of an adverse health effect in those exposed to the contaminated Camp Lejeune water of greater than 1.1. It should be noted that statistical significance is not itself determinative of causation; rather, it helps to explain the likelihood one would see a disease in a given population versus a control group. Therefore, studies with confidence intervals that include 1.0 do not establish that an agent does not cause a given disease, but rather that the subject disease may not be more prevalent in the exposed group than in a control group. Studies of Camp Lejeune personnel demonstrate risks of greater than 1.1 for exposure to the Camp Lejeune chemical TCE and Parkinson's Disease. For instance, Goldman *et al.* reported an odds ratio of 6.1 for PD in individuals exposed to TCE

When the body of research on TCE is considered in light of the Bradford Hill criteria, I am able to opine that exposure to TCE causes PD. However, it is also important to note that the Bradford Hill criteria were not intended to be rigid guidelines or a checklist that must be completed in order to determine causation. Rather, they are suggested guidelines to consider when determining causation.

B. CONSISTENCY:

The Bradford Hill term of consistency refers to the concept that studies done in different populations or that studies of different designs (for instance, *in vitro* tests and epidemiologic studies) yield similar results. This criterion is also met in that Gash *et al.* found that workers exposed to TCE exhibited features of parkinsonism, and animal studies confirm TCE's neurotoxicity.

C. SPECIFICITY:

Specificity in Bradford Hill's time meant that an exposure causes a single disease without any other likely explanation other than the exposure under consideration. However, we now know that a particular exposure may cause more than one disease state. For instance, it is known that the water contaminants from Camp Lejeune are known to cause several cancers and other adverse health effects, including PD. Furthermore, there are other chemical causes of PD – it is not unique to TCE. Therefore, the specificity criterion is difficult to meet with the chemical contaminants at Camp Lejeune and PD.

D. TEMPORALITY:

Temporality is the easiest of the Bradford Hill criteria to understand, and the one criterion that must be met. Simply put, the exposure must precede the development of the disease. This criterion is also met in the issue at hand with regards to the Camp Lejeune water contamination.

E. BIOLOGICAL GRADIENT:

The concept of a biological gradient is that a dose-response exists. That is, that the greater a dose (*i.e.*, exposure), the more likely a response (*i.e.*, presence of disease). However, we now know that complex dose-response relationships can occur (*e.g.*: hormesis) and that dose-response relationships are not all (or necessarily) linear. Further complicating the dose-response relationship is that amongst the exposed people at Camp Lejeune there were children as well as adults. It is unknown the degree to which children have altered absorption or kinetics of the contaminants, particularly when one considers the three different mechanisms of chemical absorption at Camp Lejeune. In animal models, Liu *et al.* observed dose-dependent loss of dopaminergic neurons in rats exposed to varying levels of TCE.

F. PLAUSIBILITY:

Biologic plausibility refers to the concept that a relationship between an exposure and an adverse health outcome can be attributed to causation based on existing biomedical and epidemiological knowledge. In the above report, some of the research into the mechanism of action of TCE and damage to dopaminergic neurons was detailed. There is now abundant evidence that TCE induces PD in animal models and that TCE causes PD in humans. It is therefore my opinion that the biologic plausibility standard has been met with regards to TCE exposure and PD.

G. COHERENCE:

The Bradford Hill criterion of coherence is very similar to biological plausibility. That is, that "the cause-and-effect interpretation of the data should not seriously conflict with the generally known facts of the natural history and biology of the disease" [Bradford Hill 1965]. There are mechanistic, animal, and human studies evaluating the effect of TCE and the development of PD. It is my opinion that the criterion of coherence has also been met.

H. EXPERIMENTAL EVIDENCE:

Bradford Hill also identified experimentation as a criterion to evaluate with regards to causation. Put simply, conduct experiments whereby you either purposely expose individuals to a toxin (such as TCE), or you eliminate such an exposure and determine the effect on adverse health outcome occurrence. Clearly one cannot ethically subject individuals for any significant length of time to TCE by any mode of exposure. However, there is experimental evidence in animals that TCE induces PD, and human epidemiology evidence which also demonstrates that TCE causes PD in humans. Therefore, it is my opinion that the experimentation criterion has also been met.

I. ANALOGY:

With analogy, Bradford Hill meant to say that when there is strong evidence of an exposure-disease dyad, one should be more inclined to accept causation with a similar exposure and/or disease. Other environmental toxins, such as pesticides, have also been linked to PD, supporting the analogy that TCE, a similar neurotoxicant, could also contribute to PD [Gash 2008]. It is my opinion that the analogy criterion has also been met.

XI. CONCLUSION

The water at Camp Lejeune was contaminated for decades with TCE, PCE, vinyl chloride, and benzene. It is my opinion that these water contaminants have been shown to cause adverse health effects, including Parkinson's Disease, in occupational studies, environmental studies outside of Camp Lejeune, and specifically in Marines and civilians who were based at Camp Lejeune, especially given the reduced standard at issue in this litigation, an as likely or not standard, or equipoise.

It is also my opinion that the levels of exposure to these chemicals at Camp Lejeune are hazardous to humans, and specifically as likely as not cause Parkinson's Disease. Epidemiologic studies of occupational exposure to these chemicals, as well as environmental contamination by these chemicals, provide evidence that the level of exposure to these chemicals on Camp Lejeune were sufficient to cause PD.

Respectfully,

Steven B. Bird, MD

Appendix A

Nov 2024

Steven B. Bird, M.D.

PERSONAL INFORMATION

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EDUCATION

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Northwestern University
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POST-GRADUATE TRAINING

Fellow in Toxicology 2002 – 2004
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Resident in Emergency Medicine 1999 – 2002
University of Massachusetts Medical School
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US Naval Flight Surgeon 1996 – 1999
Marine Corps Air Station Futenma
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Resident in Surgery 1995 – 1996
Naval Hospital San Diego
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LICENSURE AND BOARD CERTIFICATION

American Board of Emergency Medicine, 2003 and 2013

American Board of Toxicology, 2004 and 2014

Massachusetts Physician License # 205932

ACADEMIC APPOINTMENTS

Professor of Emergency Medicine — 3/2016 - current
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Associate Professor of Emergency Medicine — 1/2010 – 3/2016
Department of Emergency Medicine
University of Massachusetts Medical School
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Assistant Professor of Emergency Medicine — 9/2004 – 1/2010
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

Instructor of Emergency Medicine — 8/2002 – 8/2004
Department of Emergency Medicine
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DEPARTMENTAL, SCHOOL, and HOSPITAL APPOINTMENTS

Chief of Medical Toxicology — 3/2024 – current
UMassMemorial Health
Worcester, MA

IT Steering Council — 2/2020 – 11/2024
UMassMemorial Health
Worcester, MA

Space Allocation and Utilization Committee — 2/2020 – 2022
UMass Medical School & UMassMemorial Health
Worcester, MA

Clinician Experience Officer (CXO) — 9/2019 – 11/2024
UMassMemorial Health
Worcester, MA

Medical Center/Medical Group Leadership Team — 9/2019 – 11/2024
UMassMemorial Health
Worcester, MA

Joint Leadership Team — 9/2019 – 11/2024
UMass Medical School & UMassMemorial Health

Worcester, MA

Chair, Division Director of EMS Search Committee 3/2017 - 1/2018
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

Comprehensive Stroke Center Director Search Comm. 8/2016 - 12/2016
University of Massachusetts Medical School
Worcester, MA

Clinician Health and Well-Being Committee Co-Chair 8/2015 – 11/2024
University of Massachusetts Medical School
Worcester, MA

Dept of Neurosurgery Chair Search Committee 9/2014 - 6/2016
University of Massachusetts Medical School
Worcester, MA

Dept of EM Clinical Quality Review Committee 7/2013 - current
UMassMemorial Health
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Medical Staff President 7/2013 - 6/2015
UmassMemorial Health
Worcester, MA

Chief Medical Officer Search Committee 6/2013 - 9/2013
UMassMemorial Health
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Vice Chair of Education 3/2012 - 9/2019
Department of Emergency Medicine
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Chair of Hospital Credentials Committee 7/2011 - 6/2013
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Medical Staff President-Elect 7/2011 - 6/2013
UMassMemorial Health
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Assistant Director of Clinical Operations 3/2011 - 11/2013
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Program Director for Emergency Medicine Residency 3/2011 - 8/2019
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Medical Staff Executive Committee 6/2010 – 11/2024
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Attending Emergency Physician University of Massachusetts Medical Center Worcester, MA	7/2002 - current
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Attending Emergency Physician Clinton Hospital Clinton, MA	7/2002 - current

MEMBERSHIPS AND SOCIETIES

Council of Residency Directors for Emergency Medicine	2011- 2019
American College of Medical Toxicology	2001 - current
Massachusetts College of Emergency Physicians	1999 - current
Society for Academic Emergency Medicine	1998 - current
American College of Emergency Physicians	1998 - current

HONORS AND AWARDS

Outstanding Contribution to Medical Toxicology Research American College of Medical Toxicology	2021
National Leadership Award UMass Department of Emergency Medicine	2019
Emergency Medicine Residency Teaching Award UMass Emergency Medicine Residency	2018
Emergency Medicine Residency Teaching Award UMass Emergency Medicine Residency	2016
Lean Yellow Belt	2015
Best Scientific Presentation American College of Medical Toxicology Annual Meeting	2014
Team Award for Quality Care UMassMemorial Healthcare	2012
Lean White Belt	2012
Best New Speaker Award American College of American Physicians Annual Meeting Perfect audience evaluation score of 100%.	2012

Young Investigator Award Society for Academic Emergency Medicine	2007
Best Resident Basic Science Presentation Society for Academic Emergency Medicine	2002
Excellence in Research Award New England Regional Research Directors	2002
Navy and Marine Corps Achievement Medal	1999
Alpha Omega Alpha	1994
Yale University Richter Fellow	1990

PROFESSIONAL ACTIVITIES

Departmental/Institutional

Division Chief of Medical Toxicology 3/2024 – current
UMassMemorial Health
Worcester, MA

- Responsible for executive direction and execution of 9-person Division of Medical Toxicology and its 4 fellows.

Claims Committee 4/2021 - current
UMassMemorial Health
Worcester, MA

- Member of the Claims Committee of our self-insured captive
- Review all claims and lawsuits brought against UMassMemorial Health and covered individuals
- Evaluate each claim and lawsuit and give recommendations to the Director of Risk Management and the CEO with regards to defense, settlement, or trial, as well as recommend financial limits on any settlement

Clinician Experience Officer (CXO) 9/2019 – 11/2024
UMassMemorial Health, Medical Group, and Medical School
Worcester, MA

- CXO for jointly funded position of the health system, group practice, and medical school. Responsible for all wellness and engagement activities for all physicians, advanced practice providers, residents, and fellows.
- Led efforts that saw our Press Ganey physician engagement at the University Campus climb from the 1st percentile to the 18th percentile.
- Reports directly to hospital president, Dean, and group practice president.
- Member of Medical Center/Medical Group Leadership Team as well as Joint Leadership Team (involving medical school).
- Successfully led to UMass joining the Stanford Physician Wellness Academic Consortium in June 2020.
- Created a cadre of wellness coaches to allow for free wellness coaching for all faculty, residents, and fellows.
- Jointly-led the Caring for the Caregiver efforts during COVID-19 pandemic.

Peer Support Program 6/2016 – 11/2024
UMassMemorial Health
Worcester, MA

- Creator, with the assistance of a competitive grant from risk management, of a peer support network at UMassMemorial Healthcare. The peer support network is a group of 25 physicians trained in providing assistance to physicians facing difficulties related to poor patient outcomes, litigation, and other stressors. The Peer Support Program receives a new referral roughly once every 2 weeks.

Clinician Health and Well-Being Committee 8/2015 – 11/2024
UMassMemorial Health
Worcester, MA

- Selected by System CMO to co-chair the Clinician Health and Well-Being Committee (CHWC). The mission of this committee is to proactively identify, counsel, and refer physicians before an adverse event occurs.

Wellness Committee Chair 4/2015 - current
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

- Created a wellness committee for our residency and department. Invited national speakers on the topic and facilitated a “Notes Day” (modeled on the process improvement structure of Pixar) to help identify local, institutional, and departmental factors associated with physician burnout. Instituted wellness initiatives within the residency, including a wellness and empathy curriculum.

Vice Chair of Education 3/2012 - 9/2019
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

- Responsible for all aspects of education within the Department of Emergency Medicine, including undergraduate, graduate, and allied health professional education. The Department has 40 residents in a PGY1-3 residency; 70+ faculty members; 911fellows; and UMass and visiting medical students.

- Oversaw the development and implementation of mandatory UMass medical student class “Emergency Clinical Problem Solving”. This class began in May, 2013, and is required for all 125 4th year medical students. Responsible for 4th-year medical student elective in Emergency Medicine. Direct report for 5 physicians and oversees staff of 4 administrative assistants.

President of the Medical Staff 7/2013 - 6/2015
UMassMemorial Health

- Served a two-year term as president of the medical staff. Responsible for review of all new and renewal applications to the medical staff. Coordinated with Chief Medical Officer all institution peer reviews, including the Chief Physician Officer, hospital general counsel, applicable department chairs, and the individual physician in question. As president of the medical staff I also presided over quarterly medical staff meetings, participated in Joint Commission preparation focus groups, and assisted the Chief Medical Officer and Group Practice President as needed.

Clinical Competency Committee 3/2012 – 7/2024
Emergency Medicine Residency

University of Massachusetts Medical School
Worcester, MA

- Responsible for determining the competency and promotion for 36 emergency medicine residents and coordinates decisions with the Graduate Medical Education office.

Chair, Medical Staff Credentialing Committee 7/2011 - 6/2013
UMassMemorial Health

- Served two years as Chair of the Medical Center's credentialing. Reviewed all new and renewal applications to the medical staff. Coordinated with Chief Medical Officer and Department Chairs or Division Chiefs for candidates that are conditionally approved or not recommended for approval.

Assistant Director of Clinical Operations 3/2011 - 12/2013
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

- Worked closely with the Director of Clinical Operations to strategize long-term vision and processes for the Department's clinical activities. Interviewed all candidates for faculty positions and fellowships. Responsible for the yearly performance evaluations for 5 faculty members.

- Instrumental in the initiation of the Departmental Peer Review Process, a nationally recognized model of peer review and process improvement.

Emergency Medicine Residency Curriculum Committee 2011 - current
University of Massachusetts Medical School
Worcester, MA

- Responsible for overhaul of entire 18-month emergency medicine residency curriculum.

Peer Review Committee 2011 - current
Department of Emergency Medicine
University of Massachusetts Medical School

- A nationally recognized peer review process whose monthly meeting of approximately 12 individuals confidentially and anonymously reviews concerns of care. Feedback delivered to individual practitioners and findings presented at weekly departmental Morbidity and Mortality conference.

Medical Staff Executive Committee 2010 – 11/2024
UMass Memorial Medical Center
Worcester, MA

- Executive committee of the medical staff. Reviews and approves all hospital policies. Responsible for approval of medical staff privileges and recommending/monitoring physicians' compliance with Physician Health Services as needed.

Research Committee 2003 - current
Department of Emergency Medicine
University of Massachusetts Medical School
Worcester, MA

- Committee charged with providing guidance and vision for Departmental research; reviewing internal and extramural proposals; and awarding internal funding.

Emergency Medicine Residency Selection Committee 2002 - 2023
University of Massachusetts Medical School
Worcester, MA

- 21 years of service on committee that reviews, interviews, and ranks medical students applying to UMass for emergency medicine through the NRMP Match. Chair of this committee from 2011-2019.

Chair of Physician Incentive Compensation Committee 2006 - 2011
Department of Emergency Medicine
UMassMemorial Health
Worcester, MA

- Responsible for the development and growth of the emergency medicine physician incentive compensation plan. Grew this plan from a total of $70,000 per year in 2005 to over $1.1 million in 2011 (and now up to nearly $2 million). Responsible for determination of incentive plan metrics, monitoring performance of those metrics across 70+ faculty, and yearly reporting of the metrics.

Procedural Sedation Committee 2004 - 2010
UMassMemorial Health
Worcester, MA

- Committee responsible for writing institutional policies regarding procedural sedation. Also responsible for reviewing quality data and any adverse events related to procedural sedation for the hospital and clinics.

National

Board of Directors – Immediate-Past President 5/2019 – 5/2020
Society for Academic Emergency Medicine

- Immediate-Past President of the 6700-member Society for Academic Emergency Medicine. Responsible for guiding the 9-member Board of Directors on overall strategic plan for the organization, as well as guiding a $4.5 million budget.

Board of Directors – President 5/2018 - 5/2019
Society for Academic Emergency Medicine

- President of the 6700-member Society for Academic Emergency Medicine. Responsible for guiding the 9-member Board of Directors on overall strategic plan for the organization, as well as guiding a $4.5 million budget.

Board of Directors – President-Elect 5/2017 - 5/2018
Society for Academic Emergency Medicine

- Elected to the President-Elect role of SAEM in March 2017. Will assume role of President in May 2018.

AAMC Standardized Video Interview Workgroup 1/2017 - 8/2019

- Member of workgroup convened by the AAMC to define rubric for scoring of the Standardized Video Interview (SVI) project. Furthermore, we analyzed interim data from a trial of the SVI and have

informed the AAMC on methods to improve the SVI, as well as creating a research agenda around the SVI.

National Academy of Medicine (NAM) 1/2017 – 8/2024
Clinician Well-Being Action Collaborative

- I represent the field of emergency medicine on this national collaborative involving the entire house of medicine. The mission of the NAM Clinician Well-Being Action Collaborative (chaired by Drs. Victor Zhou, Thomas Nasca, and Darrel Kirch) is to create a body of knowledge, research agenda, and implementation science to mitigate burnout amongst physicians, promote wellness, and return joy to the practice of medicine. I am one of just 3 emergency physicians involved in this national effort.

Board of Directors – Secretary-Treasurer 5/2016 – 5/2017
Society for Academic Emergency Medicine

- Member of the Board of Directors. Responsible for financial oversight of the largest academic emergency medicine society in the U.S., with annual budget of over $4 million. Participate in the strategic direction of the Society.

Board of Directors 2011 - 2013 & 2016 - 2019
Emergency Medicine Foundation

- Member of the Board of Directors of the EMF, a 501c3 research funding organization affiliated with the American College of Emergency Physicians. Responsible for directing areas of research focus as well as fund raising and approving grant funding of approximately $1 million per year.

Board of Directors 5/2014 – 5/2020
Society for Academic Emergency Medicine Foundation

- Member of the Board of Directors of the SAEM Foundation, a 501c3 research funding organization with a corpus of over $11 million. Responsible for directing areas of research focus as well as fund raising.

Board of Directors (member-at-large) 5/2014 - 5/2016
Society for Academic Emergency Medicine

- Member of the Board of Directors. Responsible for oversight and providing strategic direction for the largest academic emergency medicine society in the U.S.

Search Committee 10/2014 - 5/2015
CEO of the Society for Academic Emergency Medicine

- Member of 10-person search committee for new CEO of the Society for Academic Emergency Medicine. Resulted in the hiring of CEO Megan Schagrin.

Search Committee 9/2014 - 7/2015
Academic Emergency Medicine Editor-in-Chief

- Member of 6-person search committee for new Editor-in-Chief of Academic Emergency Medicine.

NIH Special Emphasis Panel Review Member 2012 - 2016

- Serves as review for NIH panel ZRG1 MDCN-B

Finance Committee 2011 - 2013

American College of Emergency Physicians

- Committee responsible for generating and approving ACEP's yearly budget of roughly $22 million. Interacted directly with ACEP's Executive Director, CFO, and Board of Directors.

Annual Meeting Program Committee 2008 - 2014
Society for Academic Emergency Medicine

- Co-chair of scientific subcommittee. Responsible for coordinating reviewers and reviewing more than 1,200 abstracts to the SAEM annual meeting. Responsible for organization of the entire scientific aspects of the meeting (determining oral presentations, poster presentations, assigning moderators, meeting room assignments at host hotels, etc.

Grants Committee 2004 - 2014
Society for Academic Emergency Medicine

- Responsible for reviewing grant applications to SAEM. Served as chair of the Institutional Research Training Grant category in 2010, the Education Research Grant in 2011, and the Spadafora Medical Toxicology Grant in 2012. Made recommendations for funding to the SAEM Board of Directors for grants totaling approximately $400,000/year.

Scientific Review Committee 2003 - 2011
American College of Emergency Physicians

- Responsible for reviewing grant applications to Emergency Medicine Foundation. Made recommendations for funding to the EMF Board of Directors for grants totaling more than $1,000,000/year.

Research Committee 2003 - 2004
Society for Academic Emergency Medicine

International

Southeast Asia Toxicology Research Consortium 2004 – 2019

Scholarly

Editorial Board 2019 - current
The Journal of Wellness

Editorial Board 2009 - current
Academic Emergency Medicine

Editorial Board 2009 - 2013
The Open Toxinology Journal

Editorial Board – founding member 2005 - 2011
Journal of Medical Toxicology

Manuscript reviewer for *JAMA; Academic Emergency Medicine; Annals of Emergency Medicine; Pediatrics; Journal of Emergency Medicine; Journal of Medical Toxicology; Clinical Toxicology; The Open Toxinology Journal; PLoS One*

Invited Attendance

Extracorporeal Removal of Toxins in Poisoning (ExTRIP) working group
Montreal, Canada, October 2019

American College of Medical Toxicology Chemical Agents of Opportunity symposium
Nashville, TN, May 2019

12th International Symposium on Protection Against Chemical Warfare Agents
Munich, Germany, April 2019

Western Regional SAEM Conference, Napa, CA, March 2019

NINDS CounterACT meeting, Boston, MA, June 2017

10th International Symposium on Protection Against Chemical Warfare Agents
Munich, Germany, April 2017

NINDS CounterACT meeting, Davis, CA, June 2016

NINDS CounterACT meeting, New York, NY, June 2015

NINDS CounterACT meeting, Denver, CO, June 2014

13th Congress of APAMT, Shenyang, China, September 2014

NIH Workshop on Neurologic Effects of Nerve Agents, Bethesda, MD, February 2014

NY Chapter of the American College of Emergency Physicians, Lake George, NY, July 2013

NINDS CounterACT meeting, Bethesda, MD June 2013

NINDS CounterACT meeting, San Francisco, CA June 2012

11th National Congress of the Iranian Society of Toxicology, Mashad, Iran, August 2011

5th Congress of APAMT, Colombo, Sri Lanka, August 2006

8th International Symposium on Protection Against Chemical Warfare Agents
Munich, Germany, May 2004

SIGNIFICANT MENTORING

Sneha Shah, MD	AMA Women's Section Award	2014
John Haran, MD	SAEM Research Training Grant	2014-2015
Chad Darling, MD	K23 from NHLBI	2010-2015

Romolo Gaspari, MD K08 from NINDS 2007-2012

COMMUNITY ACTIVITIES

St. John's High School Gala – Planning committee 2018 - 2020

- Assisted in securing sponsorships and auction items, selling tables, and planning the annual St. John's High School Galal. This event raised more than $250,000.

Yale Alumni Schools Committee – Central Mass 2009-2012

- Responsible for coordinating, assigning, and reviewing approximately 40 Yale alumni interviews of applicants to Yale University.

Spring Street School Chess Club 2009-2014

- Organized, coached, and facilitated the chess club for Spring Street School in Shrewsbury, Massachusetts, for grades 1-4. Increased participation in this chess club to nearly 40% of students in the school, creating the largest elementary chess club in New England.

Central Mass Heart Ball – Planning committee 2010 & 2011

- Responsible for securing sponsorships and auction items, selling tables, and planning the annual American Heart Association Ball. This event raises more than $300,000 annually.

TEACHING RESPONSIBILITIES

Grand Rounds/Invited Lectures
University of Vermont Grand Rounds. "Chest Pain Testing in the ED" December 11, 2017, Burlington, VT.

University of West Virginia Grand Rounds. "Rationale Testing in the ED" August 24, 2017, Morgantown, WV.

Society for Academic Emergency Medicine Annual Meeting, "Before Taking Care of Others You Must Take Care of Yourself" May 2017, Orlando, FL

Society for Academic Emergency Medicine Annual Meeting, "Accepting Risk and the Myth of Zero" May 2017, Orlando, FL

Falmouth Hospital Emergency Care Conference, "Emerging Drugs of Abuse and Testing Conundrums" March 2017, Falmouth, MA

University of Vermont Larner School of Medicine, Emergency Medicine Update, "Visual Toxicology". February 2017, Stowe, VT

University of Vermont Larner School of Medicine, Emergency Medicine Update, "Pattern Recognition in Toxicology". February 2017, Stowe, VT

North American Congress of Clinical Toxicology, "Neurotoxicology of Organophosphorus Pesticides". October 2016, Boston, MA

Controversies and Consensus in Emergency Medicine conference, "Safely Decreasing Stress Testing from the Emergency Department". September 2016, Northampton, MA

Society for Academic Emergency Medicine Annual Meeting, “Accepting Risk and the Myth of Zero” May 2016, New Orleans, LA

Society for Academic Emergency Medicine Annual Meeting, “Metacognition: How Physicians Think” May 2016, New Orleans, LA

Boston Medical Center faculty retreat, “Wellness, Resiliency, and Empathy”, April 2016, Newport, RI

American College of Emergency Physicians Annual Meeting, “Beyond the Bends” October 2015, Boston, MA

American College of Emergency Physicians Annual Meeting, “Dangerous Drug Interactions” October 2015, Boston, MA

American College of Emergency Physicians Annual Meeting, “Nature’s Deadliest Creatures” October 2015, Boston, MA

ACEP Toxicology Interest Group, “From Benchtop to Sri Lanka: One Toxicologists Journey” October 2015, Boston MA

Society for Academic Emergency Medicine Annual Meeting, “Do Your Patients Know You Care? Methods to Convey Empathy” May 2015, San Diego, CA

American College of Emergency Physicians Annual Meeting, “Dangerous Drug Interactions” October 2014, Chicago, IL

American College of Emergency Physicians Annual Meeting, “Environmental Emergencies” October 2014, Chicago, IL

Rhode Island Hospital/Brown University. “How Physicians Think” September 2014, Providence, RI.

Asia Pacific Association of Medical Toxicology, “Translational Therapies for Acute Organophosphorus Inhibitor Poisoning” September 2014, Shenyang, China.

Sapporo Medical University, “Novel Therapies for Acetylcholinesterase Inhibitor Poisoning” September 2014, Sapporo, Japan.

Society for Academic Emergency Medicine Annual Meeting, “Metacognition: Thinking About How You Think” May 2014, Dallas, TX

American College of Emergency Physicians Annual Meeting, “Dangerous Drug Interactions That Can Kill Your Patients” October 2013, Seattle, WA

American College of Emergency Physicians Annual Meeting, “Cutting-Edge Ideas in Toxicology” October 2013, Seattle, WA

Albany Medical College Department of Emergency Medicine, “How to Give a Presentation” August 2013, Albany, NY

Boston Medical Center Department of Emergency Medicine. “Metacognition” August 2013, Boston, MA

New York chapter of the American College of Emergency Physicians: “New and Emerging Drugs of Abuse” July 2013, Lake George, NY

New York chapter of the American College of Emergency Physicians: “Drug-Drug Interactions in the Emergency Department” July 2013, Lake George, NY

American College of Emergency Physicians Annual Meeting: “What Goes Down, Must Come Up: Diving Medical Emergencies” October 2011, San Francisco, CA

American College of Emergency Physicians Annual Meeting: “Marine Envenomations” October 2011, San Francisco, CA

North Country Hospital: “Pattern Recognition in Adverse Drug Events” February 2011, Newport, Vermont

Washington University School of Medicine: ““Translational Research in Emergency” September 2010, St. Louis, MO

University of Massachusetts Medical School: “Translational Research in Emergency Medicine and Building an Academic Career” July 2009 Worcester, MA

Children’s Hospital Boston - Pediatric Emergency Medicine and Massachusetts Poison Control Center; “Acetylcholinesterase Inhibitors” May 2008, Boston, MA

University of Iowa, Department of Emergency Medicine. “Organophosphates and Chemical Nerve Agents.” November 2005, Iowa City, IA

University of Iowa, Department of Emergency Medicine. “Antidepressant Poisoning.” April 2006, Iowa City, IA

University of Iowa, Department of Emergency Medicine. “Pattern Recognition in Toxicology.” April 2006, Iowa City, IA

University of Massachusetts Medical School: “Translational Research in Emergency Medicine: from Benchtop to Sri Lanka” June 2007 Worcester, MA

Brigham and Women’s Hospital, Division of Emergency Medicine: “Cardiovascular Poisonings” May 2006, Boston, MA

Center for Disease Control and Prevention. Agency for Toxic Substances and Disease Registry. “Agents of Opportunity: Toxic Gases” March 2005, Hartford, CT

Brigham and Women’s Hospital, Division of Emergency Medicine: “Procedures in Toxicology” February 2005, Boston, MA

Baystate Medicine Center, Department of Emergency Medicine “Poison Control Center Functions” March 2004, Springfield, MA

Portsmouth Naval Medical Center: “Pattern Recognition in Toxicology” March 2003, Portsmouth, VA

Harvard School of Public Health: “Neurotoxicology” October 2003, Boston, MA

Classroom Lectures (selected)
University of Massachusetts Emergency Medicine Residency: “Toxicology In-Service Review” February 2016, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “How to Give a Presentation”

August 2015, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Toxicology In-Service Review” February 2015, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Metacognition”
June 2013, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Toxicology In-Service Review” February 2013, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Dysbarism”
Sept 2012, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Impact Factor and Bibliometric Indices”
July 2012, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “How to Give a Presentation”
Sept 2011, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Pattern Recognition in Toxicology”
July 2011, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Ethanol Forensics”
Apr 2011, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Acetaminophen Toxicity”
Aug 2007, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Endocrine Emergencies”
Feb 2007, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Pattern Recognition in Toxicology”
July 2006, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Toxic Alcohols”
April 2004, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Central Venous Access”
August 2003, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Introduction to the Poisoned Patient”
July 2003, Worcester, MA

Massachusetts College of Pharmacy: “Summertime Poisonings”
July 2003, Worcester, MA

Massachusetts College of Pharmacy: “Introduction to the Poisoned Patient”
May 2003, Worcester, MA

Emergency Medical Services: “A Trip Through the Medicine Cabinet”
December 2002, Williamstown, MA

University of Massachusetts Emergency Medicine Residency: “Acetaminophen”
August 2002, Worcester, MA

University of Massachusetts Emergency Medicine Residency: “Anti-hypertensive Poisonings”
January 2002, Worcester, MA

Clinical Teaching and Supervision
Responsible for all aspects of training for 36 emergency medicine residents

Oversees residents and medical students approximately 50 hours/month in the emergency department

Oversees 3 medical toxicology fellows and one emergency medicine resident per month on the toxicology consultation service

Participates in weekly toxicology conference for residents, fellows, and pharmacists

PAPERS IN PEER-REVIEWED JOURNALS

1. S Howard-Wilson, J Ching, S Gentile, M Ho . . . **Bird SB** et al. Efficacy of a Multimodal Digital Behavior Change Intervention on Lifestyle Behavior, Cardiometabolic Biomarkers, and Medical Expenditure: Protocol for a Randomized Controlled Trial. JMIR Research Protocols 13 (1), e50378

2. Ligibel JA, Goularte N, Berliner JI, **Bird SB**, Brazeau CMLR, Rowe SG, Stewart MT, Trockel MT. Well-being parameters and intention to leave current institution among academic physicians. *JAMA Open Network* 2023; 6: e2347894-e2347894

3. Ghannoum G, Gosselin S, Hoffman RS et al. Extracorporeal treatment for ethylene glycol poisoning: systematic review and recommendations from the EXTRIP workgroup. *Critical Care* 2023;27:56.

4. Lu D, Lee J, Alvarez A, Sakamoto J, **Bird SB**, Vandana S, Laa M, Nordenholz M, Manfredi R, Blomkalns Factors Driving Burnout and Professional Fulfillment Among Emergency Medicine Residents: A National Wellness Survey. *Acad Emerg Med Ed Training* 2022; 6:S5-S12.

5. Bouchard J, Yates C, Calello DP et al. Extracorporeal Treatment for Gabapentin and Pregabalin Poisoning: Systematic Review and Recommendations From the EXTRIP Workgroup. *Am J Kid Dis*. 2022;79: 88-104.

6. Lu D, Lee J, Alvarez A, Sakamoto J, **Bird SB**, Vandana S, Laa M, Nordenholz M, Manfredi R, Blomkalns A. Drivers of Professional Fulfillment and Burnout Among Emergency Medicine Faculty: A National Wellness Survey by the Society for Academic Emergency Medicine. *Acad Emerg Med* 2022; published online March 19, 2022. ttps://doi.org/10.1111/acem.14487

7. Ghannoum G, Berling I, Lavergne V et al. Recommendations from the EXTRIP workgroup on extracorporeal treatment for baclofen poisoning. *Kid Interl* 2021;100:720-36.

8. Brower KJ, Brazeau CMLR, Kiely SC, et al. The Evolving Role of the Chief Wellness Officer in the Management of Crises by Health Care Systems: Lessons from the Covid-19 Pandemic. *NEJM Catalyst*. 2021; 5. DOI:https://doi.org/10.1056/CAT.20.0612.

9. Bouchard J, Shepherd G, Hoffman RS, et al. Extracorporeal treatment for poisoning to beta-adrenergic antagonists: systematic review and recommendations from the EXTRIP workgroup. *Crit Care* 2021;25**:** 201-. https://doi.org/10.1186/s13054-021-03585-7.

10. Wong A. Hoffman RS, Walsh SJ, et al. Extracorporeal treatment for calcium channel blocker poisoning: systematic review and recommendations from the EXTRIP workgroup. *Clin Toxicol* 2021;59: 361-375.

11. Mowry JB, Shepherd G, Hoffman RS, et al. Extracorporeal Treatments for Isoniazid Poisoning: Systematic Review and Recommendations from the EXTRIP Workgroup. *Pharmacotherapy* 2021; 00:1-16.

12. Berling I, King JD, Shepherd G, et al. Extracorporeal Treatment for Chloroquine, Hydroxychloroquine, and Quinine Poisoning: Systematic Review and Recommendations from the EXTRIP Workgroup. *J Am Soc Nephrol* 2020 Oct;31(10):2475-2489. doi: 10.1681/ASN.2020050564

13. Nordenholz KE, Alvarez A, Lall MD, **Bird S**, Blomkalns AL. Optimizing Wellness in Academic Emergency Medicine. *J Wellness* 2020. DOI: 10.18297/jwellness/vol2/iss2/8

14. Gallahue FE, Deiorio NM, Blomkalns A, **Bird SB**, et al. The AAMC Standardized Video Interview – Lessons Learned from the Residency Selection Process. *Acad Med* 2020. doi: 10.1097/ACM.000000000000357

15. Melnyk BM, Kelly SA, Stephens J . . . **Bird SB**. Interventions to Improve Mental Health, Well-Being, Physical Health, and Lifestyle Behaviors in Physicians and Nurses: A Systematic Review. *Am J Health Prom*, 2020 Nov;34(8):929-941.

16. Greenberger SM, Finnell JT, Chang BP, Garg N, Quinn SM, **Bird SB,** et al. Changes to the ACGME Common Program Requirements and Their Potential Impact on Emergency Medicine Core Faculty Protected Time. *Acad Emerg Med Ed & Training*. Nov 23, 2019. ttps://doi.org/10.1002/aet2.10421

17. **Bird SB**, Hern HG, Blomkalns A et al. Innovation in Residency Selection: The AAMC Standardized Video Interview. *Acad Med*. 2019;94:1489-97. doi:10.1097/ACM.0000000000002705

18. Gallahue FE, Hiller KM, **Bird SB** et al. The AAMC Standardized Video Interview: Reactions and Use by Residency Programs During the 2018 Application Cycle. *Acad Med* 2019;94:1506-12. doi: 10.1097/ACM.0000000000002714

19. Dyrbye LN, Meyers D, Ripp J, Dalal N, **Bird SB**, Sen S. A pragmatic approach for organizations to measure health care professional well-being. *National Acad Medicine*, Oct 2018, pp 1-11.Doi.org/10.31478/201809g

20. Deiorio NM, Jarou ZJ, Alker A, **Bird SB**, et al. Applicant Reactions to the AAMC Standardized Video Interview During the 2018 Application Cycle. *Acad Med* 2019 Oct;94(10):1498-1505. doi: 10.1097/ACM.0000000000002842.

21. Jarou Z, Karl E, Alker A, **Bird SB**, et al. Factors Affecting Standardized Video Interview Performance: Preparation Elements and the Testing Environment. *EM Resident*, April 17, 2018.

22. **Bird SB**, Blaomkalns A, Deiorio NM, Gallague FE. Beyond test scores and medical knowledge: the standardized video interview, an innovative and ethical approach for holistic assessment of applicants. *Acad Med* 2018;93:151.

23. **Bird S**, Blomkalns A, Deiorio NM, Gallague FE et al. Stepping up to the plate: emergency medicine takes a swing at enhancing the residency selection process. *AEM Ed & Training* 2017;2: 61-5. Doi: 10.1002/aet2.10068.

24. Shah S, Church R, Butler M, **Bird SB**. Assessment of emergency medicine faculty milestone competencies. *Intl J Ed Res Tech* 2017; 8 (2): 1-7.

25. **Bird SB**. Neurologic and pregnancy effects of carbon monoxide exposure. *Toxicol Open Access*. 2017, 3:4. Doi: 10.4172/2476-2067.

26. Bunya N, Sawamoto K, Benoit H, **Bird SB**. The Effect of Parathion on Red Blood Cell Acetylcholinesterase in the Wistar Rat. *J Toxicol* 2016. doi.org/10.1155/2016/4576952

27. **Bird SB**, Krajacic P, Sawamoto K, Bunya N, Loro E, Khurana TS. Pharmacotherapy to protect the neuromuscular junction after acute organophosphate poisoning. *Proc Ann NY Acad Sci* 2016; 1674:86-93.

28. Marin JR, Lewiss RE, Shook JE, et al. Point-of-care ultrasonography by Pediatric Emergency medicine physicians. *Pediatrics* 2015;135:e1113-22.

29. Temple C, Gaspari R, **Bird S**. Caffeine reduces organophosphate induced respiratory failure; effect of caffeine on dichlorvos induced central respiratory failure in a rat model *Curr Topic Toxicol* 2015; 11, 15 – 21.

30. Reznek MA, Kotkowski KA, Arce MW, Jepson ZK, **Bird SB**, Darling CE. Patient safety incident capture resulting from incident reports: a comparative observational analysis. *BMC Emergency Medicine* 2015, **15**:6 doi:10.1186/s12873-015-0032-7.

31. Broach J, Krupa R, **Bird SB**, Manuell M. Regional preparedness for mass acetylcholinesterase inhibitor poisoning through plans for stockpiling and interhospital sharing of pralidoxime. *Am J Disaster Med*. 2014:9;4, 1-9. Doi:10.5055/ajdm.2014.0000

32. Jepson ZK,Darling CE, Kotkowski KA, **Bird SB**, Arce MA, Volturo GA, Reznek MA. Emergency department patient safety incident characterization: an observational analysis of the findings of a standardized peer review process. *BMC Emergency Medicine* 2014,14:20-27.DOI:101186/1471-227X-14-20

33. Neavyn MJ, Blohm E, Babu KM, **Bird SB**. Medical marijuana and driving. *J Med Toxicol*. 2014; available online March 2014. DOI 10.1007/s13181-014-0393-4.

34. Jackson CJ, Carville A, Ward J, Mansfield K, Ollis DL, Khurana T, **Bird SB**. Use of OpdA, an Organophosphorus (OP) hydrolase, prevents lethalilty in an african green monkey model of acute OP poisoning. *Toxicol* 2014;317:1-5. doi: 10.1016/j.tox.2014.01.003

35. Sawamoto K, **Bird SB**, Katayama Y, Uemura S, Tanno K, Narimatsu E. Outcome from severe accidental hypothermia with cardiac arrest resuscitated with extracorporeal cardiopulmonary resuscitation. *Am J Emerg Med* 2014; 32(4):320-4. doi: 10.1016/j.ajem.2013.12.023.

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TEXTBOOK EDITOR

Irwin and Rippe's Intensive Care Medicine. "Pharmacology, Overdoses and Poisonings" Toxicology section editor. Lippincott. 9th Ed.

Irwin and Rippe's Intensive Care Medicine. "Pharmacology, Overdoses and Poisonings" Toxicology section editor. Lippincott. 8th Ed.

Emergency Medicine Research Handbook for Residents and Medical Students. Emergency Medicine Residents' Association. 1st Ed.

Irwin and Rippe's Intensive Care Medicine. "Pharmacology, Overdoses and Poisonings" Toxicology section editor. Lippincott. 7th Ed.

Aghababian's Emergency Medicine: The Core Curriculum. Section editor of 25 chapters. Jones and Bartlett, 2nd Ed.

Irwin and Rippe's Intensive Care Medicine. "Pharmacology, Overdoses and Poisonings" Toxicology section editor. Lippincott. 6th Ed.

Aghababian's Emergency Medicine: The Core Curriculum. Section editor of 25 chapters. Jones and Bartlett, 1st Ed.

TEXTBOOK CHAPTERS

Bird SB. "Acetaminophen Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 9th edition.

Bird SB. "Anticonvulsant Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 9th edition.

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Bird SB. "Antiarrhythmic Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 9th edition.

Bird SB. "Chromium" Goldfrank L. et al., editors. *Goldfrank's Toxicologic Emergencies,* McGraw Hill. 10th edition.

Bird SB. "Acetaminophen Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 8th edition.

Bird SB. "Anticonvulsant Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 8th edition.

Bird SB. "Antipsychotic Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 8th edition.

Bird SB. "Antiarrhythmic Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 8th edition.

Bird SB. "Chromium" Goldfrank L. et al., editors. *Goldfrank's Toxicologic Emergencies,* McGraw Hill. 9th edition.

Bird SB. "Organophosphates and Carbamates" Aghababian R. editor. *Emergency Medicine: The Core Curriculum.* Jones & Bartlett, 2nd edition.

Bird SB. "Acetaminophen Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 7th edition.

Bird SB. "Antipsychotic Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 7th edition.

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Bird SB. "Beta Blockers" Shannon M. et al., editors. *Clinical Management of Poisoning and Drug Overdose*, WB Saunders. 4th Ed.

Bird SB. "Organophosphates and Carbamates" Aghababian R. editor. *Emergency Medicine: The Core Curriculum.* Jones & Bartlett, 1st edition.

Bird SB. "Chromium" Goldfrank L. et al., editors. *Goldfrank's Toxicologic Emergencies,* McGraw Hill. 8th edition.

Bird SB. *Organophosphates and Carbamates*. UpToDate, 2004-present.

Bird SB. *Manual of Overdoses and Poisonings*. Linden, Rippe, and Irwin, Eds. 1st edition. Lipipincott Williams & Wilkins, Philadelphia, 2005. **Author of 12 chapters.**

Bird SB. "Acetaminophen Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 6th edition.

Bird SB. "Anticonvulsant Poisoning" *Intensive Care Medicine*. Rippe, and Irwin, Eds. 6th edition.

ABSTRACTS (selected presentations at national or international meetings)

1. Bunya N, Benoit H, Krajacic P, Loro E, Gaspari R, Khurana T, **Bird SB**. Pancuronium Improves Survival in a Rat Model of Acute Parathion Poisoning. May 2016, SAEM Annual Meeting, New Orleans, LA

2. Shah SH, **Bird SB**. Do Your Patients Know You Care? SAEM Annual Meeting 2015, San Diego, CA.

3. Bunya N, Benoit H, Krajacic P, Loro E, Gaspari R, Khurana T, **Bird SB**. Development of a rodent model of the Intermediate Syndrome. June 2015, NY Academy of Sciences, NY.

4. Bunya N, Sawamoto K, Benoit H, Gaspari R, Khurana T, **Bird SB**. Novel Neuromuscular Protection to CounterACT Organophosphorus (OP) Poisoning. CounterACT meeting 2014, Denver, CO.

5. Shah SH, Heitmann D, Mangolds V, Zgurzynski P, **Bird SB**. Evaluating the Implementation of an Interprofessional Team STEPPS Curriculum for Medical Students Using High Fidelity Simulation. CORD Annual Meeting 2014, New Orleans, LA.

6. Shah SH, Church R, **Bird SB**. Evaluating Faculty Milestone Competencies. CORD Annual Meeting 2014, New Orleans, LA.

7. Sawamoto K, Krajacic P, McCall J, DeLa Puente R, Ford-Webb T, Gaspari R, Khurana T, **Bird SB**. Novel Neuromuscular Protection to CounterACT Organophosphorus (OP) Poisoning. CounterACT meeting 2012, San Francisco, CA

8. **Bird SB**, Carville A, Mansfield K, Ollis D. Use Of An Organophosphorus Hydrolase Prevents Lethality In An African Green Monkey Model Of Acute Organophosphorus Poisoning. SAEM 2011 Annual Meeting, Boston, MA

9. Sharma K, Nelson L, Kurt K, **Bird S**, Brent J, Wax P. The Practice of Medical Toxicology in the US. NACCT Annual Meeting 2010, Denver, CO.

10. Weibrecht K, Dayno M, Darling C, **Bird S**. Liver aminotransferases are elevated with rhabdomyolysis in the absence of liver injury. NACCT Annual Meeting 2009, San Antonio, TX.

11. Rosenbaum C, **Bird S**. Timing and Frequency of Physostigmine for Anticholinergic Toxicity. NACCT Annual Meeting 2008, Toronto, Canada.

12. Acute Hepatotoxicity Associated with Amiodarone Administration Courtney J, Ganetsky M, **Bird SB**, Boyer EW. Clin Toxicol 2006; 44.

13. Isoniazid-Induced Psychosis in an Adolescent Male Gresham HW, Babu KM, Ali F, **Bird SB**, Boyer EW. Clin Toxicol 2006; 44.

14. Non – Fatal Cardiac Dysrhythimias Associated with Severe Salicylate Toxicity Kent KJ, Cohen JE, Ganetsky M, **Bird SB.** Clin Toxicol 2006; 44.

15. **Bird SB**, Schmidt K, Kulkarni P, Ferris C. M1 Receptor Activation in the rat: a phMRI Analysis. Society for Neuroscience Annual Meeting 2005, Miami, FL.

16. **Bird SB**, Lo JCY, Dickson EW, Sivilotti MLA. Multiple centrally-acting antidotes protect against severe organophosphate toxicity.,. J Toxicol Clin Toxicol 2005; 43.

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20. **Bird SB**, Mazzola JL, Brush DE, Boyer EW, Aaron CK. A Prospective Evaluation of Abbreviated Oral N-acetylcysteine (NAC) Therapy for Acetaminophen Poisoning. Acad Emerg Med 2003;10:521.

21. **Bird SB**, Gaspari RF, Barnett KA, Dickson EW. Diazepam Attenuates Acute Central Respiratory Depression from Acute Organophosphate Poisoning. Acad Emerg Med 2003;10:520-521.

22. Lovesky D, **Bird S**, Restuccia M, Mangolds G, Dickson EW. Effect of a Paramedic Pain Management Training Program on Pre-hospital Analgesic Use. Acad Emerg Med 2003;10:450.

23. Lane DR, **Bird SB**, Zarum RS. Documentation of Emergency Medicine Resident Procedures Using a Personal Digital Assistant. Acad Emerg Med 2003;10:537-538.

24. **Bird SB**, Eddleston M, Sutherland TD, Ollis D. Pharmacokinetics of an Organophosphorus Hydrolase in the African Green Monkey. SAEM 2008 Annual Meeting, New Orleans, LA.

25. **Bird SB**, Gresham H, Sutherland T, Eddleston M. Use of a Recombinant Bacterial Hydrolase for Acute Dichlorovos Poisoning. NACCT 2006 Annual Meeting, San Francisco, CA

26. **Bird SB**, Gresham H, Sutherland T, Eddleston M, Eyer P. Use of a Recombinant Bacterial Hydrolase for Acute Parathion Poisoning. SAEM 2006 Annual Meeting, San Francisco, CA.

27. **Bird SB**, Gaspari RJ, Aaron CK, Boyer EW, Dickson EW. Synergistic Effects of Glycopyrrolate, Ipratropium, and Diazepam on Mortality in a Rat Model of Lethal Organophosphate Poisoning. European Association of Poison Control Centres and Toxicologists 2003 annual meeting, Rome, Italy.

28. **Bird SB**, Mazzola JL, Boyer EW, Brush DE, Aaron CK. A Prospective Evaluation Of Abbreviated Oral N-Acetylcysteine (NAC) Therapy For Acetaminophen (Paracetamol) Poisoning. European Association of Poison Control Centres and Toxicologists 2003 annual meeting, Rome, Italy.

29. **Bird SB**, Gaspari RJ, Aaron CK, Boyer EW, Dickson EW. Nebulized Ipratropium Bromide Offers No Protection Against Severe Organophosphate Poisoning. J Tox Clin Tox 2002;40:695.

30. Sivilotti MLA, **Bird SB**, Montalvo M, Aaron CK, Brison RJ, Linden CH, "Serum a-Glutathione S-transferase (aGST) Becomes Elevated Shortly After Subtoxic Acetaminophen Overdose" Acad Emerg Med 2002;9:534-535.

31. **Bird SB**. Critical Care Toxicology: Organophosphate Poisoning. 2002 North American Congress of Clinical Toxicology, Palm Springs, CA.

32. **Bird SB**, Gaspari RJ, Lee WJ, Dickson EW. Early Death due to Acute, Severe Organophosphate Poisoning is a Centrally Mediated Process. Acad Emerg Med 2002;9:485.

33. **Bird SB**, Gaspari RJ, Lee WJ, Dickson EW. Diphenhydramine as a Protective Agent in Severe Organophosphate Poisoning. Acad Emerg Med 2002;9:357.

34. **Bird SB**. Case Presentation Competition, 2002 Society for Academic Emergency Medicine annual meeting, St. Louis, Missouri.

35. **Bird SB**, Zarum RS. Emergency Medicine Resident Procedure Documentation is Not Increased Using a Handheld Computerized Device. 2002 Society for Academic Emergency Medicine annual meeting, San Francisco, CA.

36. **Bird SB**, Ni Y. Comparison of a Numeric Rating Scale and the Visual Analog Scale in Extremity Pain. American College of Emergency Physicians 2001 Annual Meeting, Las Vegas, NV.

37. **Bird SB**, Sullivan J, Mangolds G, Schmidt E, Nichols C, Dickson EW. Clinically Significant Changes in Pain Along the Entire Visual Analog Scale. American College of Medical Toxicology 2001 annual meeting, San Francisco, CA.

FUNDING (completed)

"A Fitbit Digital Health Intervention in the UMass ACO" $ 100,000
Jan 2021-Dec 2021
Massachusetts Digital Health Right Care 4 You Grant Program

"RCT of Wellness Coaches to Decrease Burnout" $ 12,000
Jan 2020-Dec 2020
Carl Atkins Risk Management Grant
UMassMemorial Healthcare

"Development of a Peer Support Network" $ 10,000
June 2016-May 2017
Carl Atkins Risk Management Grant
UMassMemorial Healthcare

"Pharmacotherapy to counterACT parathion-induced NMJ dysfunction"
Principal Investigator: **Steven B. Bird, MD**
U01 NIH/NINDS $3,082,749
Sept 2013 – Aug 2016

"Novel Neuromuscular Protection to CounterACT Acute Organophosphate Poisoning"
Principal Investigator: **Steven B. Bird, MD**
R21 NIH/NINDS $ 823,588
Oct 2011 – Sept 2013

"Use of a bacterial OP hydrolase antidote for parathion poisoning"
Principal Investigator: **Steven B. Bird, MD**
R21 NIH/NIEHS $ 446,875
Aug 2007 – Aug 2009

"Functional MRI Assessment of Acute Organophosphate Poisoning"
Principal Investigator: **Steven B. Bird, MD**
K08 NIH/NIEHS
Dec 2004 - Dec 2008 $ 580,669

"Recombinant Organophosphate Hydrolase for Acute Parathion Poisoning"
Principal Investigator: **Steven B. Bird, MD**
American College of Medical Toxicology
July 2005 – June 2006 $ 7,500

"Recombinant Organophosphate Hydrolase for Acute Dichlorvos Poisoning"
Principal Investigator: **Steven B. Bird, MD**
Emergency Medicine Foundation
July 2005 – June 2006 $ 5,000

"Ipratropium bromide as a treatment of organophosphate toxicity"

Principal Investigator: **Steven B. Bird, MD**
Emergency Medicine Foundation Resident Research Grant Award
2001 – 2002 $ 5,000

Appendix B

Steven B. Bird, MD

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Shrewsbury, Massachusetts 01545

List of all cases in which, during the previous 4 years, I have testified as an expert at trial or by deposition.

Howe v. Tiffany Warren and Ascension Medical Group; Case No. 2022-CV-944
In the 18th Judicial District Court of Sedgwick Co, Kansas
Deposition: November 2024

Garcia v. Webb County; Case No. 5-23-CV-00137
In the Southern District of Texas
Deposition: November 2024

Hodys v. Barnes; Case No. PC2017-5776
In the Superior Court of Providence, Rhode Island
Deposition: October 2024

Kimbrow v. Walgreens; Case No. 2023-L-0005405
In the Circuit Court of Cook County, Illinois
Trial: September 2024

Gross v. Walgreens; Case No. 2023-L-000469
In the Circuit Court of Cook County, Illinois
Trial: July 2024

Joiner v. Walgreens; Case 2023-L-004568
In the Circuit Court of Cook County, Illinois
Trial: July 2024

Valadez v. GSK; Case No. 2023-L-000483
In the Circuit Court of Cook County, Illinois
Trial: May 2024

Patrick Feindt, Jr. v. United States of America; Case No. 1:22-cv-397-LEK-KJM
In the District Court of Hawaii
Trial: May 2024

Mejia v. Stanford Hospital; Case No. FST-CV20-6046034S
In the Superior Court for Judicial District of Stamford/Norwalk of Connecticut
Trial: March 2024.

Hankins v. Jenkins; Case No. 2:22-CV-01590.
In the United States District Court for the Northern District of Alabama

Trial: March 2024

Kimbrow v. Walgreens; Case No. 2023-L-0005405
In the Circuit Court of Cook County, Illinois
Deposition: March 2023

Valadez v. GSK; Case No. 2023-L-000483
In the Circuit Court of Cook County, Illinois
Deposition: January 2023

Kasza v. Walgreens et al; Case No. 2023-L-005404
In the Circuit Court of Cook County, Illinois
Deposition: December 2023

Valdes v. GSK; Case No. 2021-021945-CA-01
In the 11th Judicial Circuit for Miami-Dade County Florida
Deposition: December 2023

Williams v. Walgreens et al.; Case No. 2023-L-004599
In the Circuit Court of Cook County, Illinois
Deposition: December 2023

Feindt v. United States of America: Case No. 22-cv-297lLEK-KJM
In the United States District Court of Hawaii
Deposition: November 2023

Wilson v. GSK; Case No. 22-CA-000284
In the 13th Judicial Circuit for Hillsborough County Florida
Deposition: November 2023

Hall v. Baptist Easley; Case No. 2018-CP-23—01576
In the Circuit Court for Greenville County of South Carolina
Trial: October 2023

Reinhart v. Short Mountain Trucking; Case No. 3:21-CV-03122
In the United States District Court for the Central District of Illinois, Springfield Division
Deposition: August 2023

Pagan v. Saranita; Case No. 12-CA-424 2015CA00424
In the Fifth Judicial Circuit Court for Lake County of Florida
Trial: July 2023

Heinrich v. Serens; Case No. 2904978/2018
In the Supreme Court for Onondaga County of New York
Trial: July 2023

Hall v. Baptist Easley; Case No. 2018-CP-23—01576
In the Circuit Court for Greenville County of South Carolina
Deposition: July 2023

Cooper v. Advocate Christ; Case No. 2019L004866
In the Circuit Court for Cook County of Illinois
Deposition: May 2023

Richey v. CSX Transportation; Case No. 19-CI-007780
In the Jefferson Circuit Court of Kentucky, Division Five
Deposition: April 2023

Bowditch v. MedStar; Case No. 2021 CA 003778 M
In the Superior Court in Washington D.C.
Deposition: April 2023

Pimentel v. HUMC; Case No. BER-L-93-20.
In the Superior Court for Bergen County of New Jersey
Deposition: January 2023

Devani v. Honor Health; Case No. CV2021-050489
In the Superior Court for Maricopa County of Arizona
Deposition: November 2022

Ruepke v. BNSF Railroad; Case No. 2019-L-007730
In the Circuit Court for Cook County of Illinois
Deposition: August 2022

Hartman v. Illinois Central Railroad; Case No. 2:20-cv-1633
In the United States District Court for the Eastern District of Louisiana
Deposition: June 2022

Hankins v. Jenkins; Case No. 2:22-CV-01590.
In the United States District Court for the Northern District of Alabama
Deposition: June 2022

Fravel v. Herard; Case No. 2021 L 32
In the Circuit Court of the 21st Judicial Circuit for Kankakee County of Illinois
Deposition: June 2022

Lloyd v. Memorial Hospital; Case No. 16-2019-CA-000961
In the Fourth Judicial Circuit for Duval County of Florida.
Deposition: June 2022

Shephard v. Mease; Case No. 17004700CI
In the Sixth Judicial Circuit for Pinellas County of Florida

Deposition: April 2022

State of Florida v. Baldie;
In the Ninth Judicial Circuit for Orange County of Florida
Trial: April 2022

Mejia v. Stanford Hospital; Case No. FST-CV20-6046034S
In the Superior Court for Judicial District of Stamford/Norwalk of Connecticut
Deposition: December 2021.

Florida v. Baldie; Case No. 2020-CF-004830-AO
In the Ninth Judicial Circuit for Orange County of Florida
Deposition: November 2021.

U.S. v. Carvajal; Case No. 1:20-CR-10023-GAO-1
In the United States District Court of Massachusetts
Trial: November 2021

Bacon v. AnMed Health Cannon; Case No. 2019-CP-39-00937
In the Circuit Court for Pickens County of South Carolina
Deposition: June 2021

Gordanier v. Waldo; Case No. 19AE-CC00286
In the Sixth Judicial Circuit for Platte County of Missouri
Deposition: May 2021

Rybar v. DePuy; Case No. 4:16-cv-01579-CEJ
In the Circuit Court for the City of St. Louis of Missouri
Deposition: April 2021

Steven B. Bird, MD