

Exhibit 121



December 8, 2024



David Miceli
Milberg Coleman Bryson Phillips Grossman PLLC

900 West Morgan Street
Raleigh, North Carolina 27603

Re: IN RE: CAMP LEJEUNE WATER LITIGATION
IN THE UNITED STATES DISTRICT COURT FOR THE EASTERN DISTRICT OF
NORTH CAROLINA; Case No.: 7:23-CV-897

Dear Mr. Miceli,

The purpose of this report is to analyze the role, if any, of trichloroethylene (TCE) and tetrachloroethylene (PCE) as a cause of Parkinson's disease (PD). I provide a summary of the literature on PD epidemiology, environmental risk factors for PD, TCE and PCE as causes of PD, and mechanisms behind the relationships.

Qualifications

I have a Master of Science in Public Health in Epidemiology, and a Doctor of Philosophy in Epidemiology from the University of Alabama at Birmingham, followed by a post-doctoral fellowship in neuroepidemiology at Columbia University. I have been an Assistant Professor of Epidemiology at Columbia University since completing my training. My research focuses on infections as risk factors for poor outcomes such as mortality, stroke, heart attack and cardiovascular disease as well as risk factors for stroke, stroke outcomes, and how the intersection of infections and strokes can lead to cognitive decline. I have over 200-peer reviewed, published papers including publications on neurological outcomes and risk factors for outcomes after environmental exposures. My research includes collaborations with multiple U.S. and international institutions. I have been awarded National Institutes of Health (NIH) Research Grants as a Principal Investigator investigating sepsis as a risk factor for stroke and influenza as a risk factor for cardiovascular disease. I have served as the methods expert and co-investigator on over 15 other NIH or equivalent research grants. I have served as a peer reviewer for journals including *The Lancet*, *JAMA*, *American Journal of Epidemiology*, *Epidemiology and*

Infection, and *American Public Health Association Journal*. I mentor MPH and PhD students and early career faculty members on their research projects: specifically, how to correctly apply epidemiologic methods to their projects, analyze and interpret data, and disseminate results. I have taught courses on Neurological Epidemiology and served as a mentor for the neuroepidemiology fellowship at Columbia University. I serve on the MPH and Doctoral admissions committees for the Mailman School of Public Health at Columbia University, and I was the chair of the Doctoral Qualifying Methods Exam committee. Please see my CV for additional details.

Epidemiology is a field of study that investigates the determinants and distributions of disease, and neurological epidemiology is a subset of epidemiology that studies risk factors, determinants, distributions, and outcomes of neurological outcomes. Epidemiology as a field incorporates statistics and epidemiologic methods to assess causal factors in observational studies. These methods have been developed in order to apply rigorous research methodology to observational studies of clinical questions, many of which are not appropriate to study using randomized controlled trials. Additionally, epidemiologic methods apply similar research and methodological techniques to evaluate causal factors as do randomized controlled trials and meta-analyses. Epidemiologists are trained in these methods and apply them to data and chosen content areas. This report will utilize epidemiologic and evidence-based medicine approaches to evaluate the findings reported in the literature, particularly the Bradford Hill Criteria, which are now referred to as Hill's Lists of Standards for Inference in epidemiologic texts and referred to as Bradford Hill considerations in this report, as not all of the considerations are needed to determine causality.¹ The Hill considerations will be addressed at the end of this report. These include: temporality, strength of association, consistency, specificity, biologic gradient/exposure-response, plausibility/biologic-plausibility, coherence, experiment, and analogy.¹ The measure of association, such as odds ratios or risk ratios, and 95% confidence intervals (95% CIs) will be presented for key findings in this report. The measure of association provides information on the strength of the association, and the 95% CI provides information on the level of precision around the measure of association.¹

Statistical significance will not be used in determining evidence for causality as the limitations to significance testing outweigh the benefits.¹ Statistical significance is determined by sample size

(including sample size within exposure or outcome groups), and strength of the measure of association. When a measure of association is statistically significant this means the relationship observed (or one greater) is not likely due to chance. If a result does not meet the threshold for statistical significance this can be due to bias or chance, but often times is due to either small sample sizes or a weak measure of association. A result can be statistically significant but clinically meaningless, and vice versa. If the sample size is large enough, a meaningless measure of association (e.g., odds ratio of 1.01 for a binary exposure of interest) will be statistically significant. Whereas the opposite stands such as in circumstances where a large measure of association is calculated but statistical significance is not reached due to small sample sizes, either overall or in specific groups being evaluated. The information presented in this report is based on my education, training, and experience as an epidemiologist, and the materials cited herein and reviewed in forming my opinions. I am providing my most recent curriculum vitae which outlines my qualifications, experience, and publications more fully. My opinions are all stated to a reasonable degree of scientific certainty.

Parkinson's Disease Epidemiology

Parkinson's disease (PD) is a neurodegenerative disorder that results in movement and non-movement-related symptoms. The neurodegeneration takes years to cause symptoms resulting in PD incidence typically occurring later in life, with peak incidence between the ages of 70-79.² PD is often precipitated by a prodromal phase of PD, which can last up to 20 years, when non-motor symptoms start to present prior to the onset of classic PD symptoms.³ A number of factors are associated with the risk of PD, however, PD is largely thought of as a disorder brought about due to environmental contaminants. As of the 2016 Global Burden of Disease assessment, six million people were living with Parkinson's disease (PD) globally, with disability and death rising faster than any other neurological disease in the world.⁴ PD is a progressive disease that includes both movement disorder symptoms and cognitive and behavioral elements brought about by the damage and death to nerve cells resulting in symptoms that include problems with movement, tremors, stiffness, and impaired balance, among other symptoms.^{5,6} The most common PD symptoms result from death or damage to neurons in the substantia nigra, an area near the base of the brain, resulting from the loss of 60-80% of dopamine-producing cells in this

area.⁵ Being a degenerative disease, PD disease duration can span decades with slow progression resulting in accumulating disability.⁵ There are four primary symptoms of PD: (1) tremor, (2) rigidity, (3) bradykinesia, and (4) postural instability.⁷ Additional symptoms of PD include olfactory dysfunctions, neuropsychiatric disorders, cognitive impairment, autonomic dysfunctions, and gastrointestinal disturbances, among other symptoms.⁸

Risk factors for PD include increasing age, male sex, familial history, and environmental exposures.^{5,6} The recent increases in PD incidence and prevalence were thought to be due to an aging population, however, the increasing rate of PD outpaced the expected rate of PD due to aging, indicating aging alone is not responsible for the increasing rate of PD.⁴ Age itself is not thought of as a risk factor for PD, however, increased age allows for more exposure time to environmental exposures and toxicants and enough time for PD to manifest.⁹ Genetic factors contribute to the risk of PD, however, pure genetic causes of PD only account for ~2-5% of PD cases.^{10,11} Leucine-rich repeat kinase 2 (LRRK2) activity from LRRK2 mutations are known to cause PD.¹² LRRK2 variants elevate LRRK2 kinase activity resulting in endosomal dysfunction, oxidative stress, and dysregulation in vesicular trafficking.¹³ Elevated LRRK2 kinase activity alone is not enough to cause PD in people who have the LRRK2 mutation.¹² LRRK2 mutations are the most common genetic cause of PD, but have incomplete penetrance and are only present in 2-3% of people with PD.¹⁴ The role of genetics in PD suggests factors other than inherited genes are the cause of PD.^{15,16} Additionally, genetics can facilitate the development of PD in people exposed to environmental contaminants, particularly in populations with mutations in genes that encode for metabolizing toxins.^{17,18}

The majority of PD is labeled idiopathic. Idiopathic is a term used in clinical medicine when there is no known cause of a disease or condition. However, the term can also be used when other known causes have been ruled out. It can also be used to describe a condition for which a known cause has not yet been determined.

It is widely accepted that other factors besides age and genetics are necessary for PD to develop.¹⁹ Environmental factors related to PD include exposure to pesticides, exposure to solvents, and traumatic brain injury, with the main known cause of PD being environmental toxins.²⁰ Rates of PD are highest in industrialized nations with rates of PD rising rapidly in industrializing nations.⁴

Environmental Solvent Exposures and Risk of PD

Interest in environmental exposures as a cause of PD increased following a 1983 report highlighting 1-methyl-4-phenyl-1,2,5,6-tetrahydropyridine (MPTP) and 1-methyl-4-phenyl-4-propionoxy-piperidine (MPPP) use and subsequent parkinsonian symptoms.²¹ The results of this report prompted further studies evaluating pesticide use and other environmental contaminants, including solvents, as risk factors for PD. The first report of parkinsonian symptoms induced by solvent abuse was a 1994 case report describing acute parkinsonism following heavy abuse of a solvent.²² A 1998 case-control study identified a relationship between well water use for at least 10 consecutive years and occupational exposure to chemicals, specifically organic solvents, with increased risk of PD.²³ This study built on the case report by Uitti et al.,²² further supporting the burgeoning hypothesis implicating environmental and occupational risk factors in the development of PD.²³ To explore whether exposure to solvents resulted in different PD presentations a study of patients with PD found that exposure to solvents was directly responsible for earlier PD symptom onset and more severe symptoms.²⁴ Laboratory studies were then initiated investigating the potential biological mechanisms between solvents and PD risk, and identified toxic effects on the dopaminergic pathways.²⁵ A 2003 meta-analysis evaluating pesticides and solvents on PD risk found a strong relationship between solvents and PD risk (standardized odds ratio (sOR) 1.35, 95% Confidence Interval (CI) 1.09-1.67) with the relationship being even stronger in higher quality studies (sOR 1.58, 95%CI 1.23-2.04).²⁶ These studies were foundational in establishing the link between solvent use and PD risk and provided the foundation for further investigations into types of solvents and risk of PD.

Trichloroethylene (TCE), tetrachloroethylene (PCE), and PD

Two specific solvents of interest in the role of environmental exposures on PD are trichloroethylene (TCE) and tetrachloroethylene (PCE). TCE and PCE are closely related to each other with PCE containing one additional chlorine atom compared to TCE. TCE has long been implicated as a cause of several poor health outcomes including numerous cancers, neural tube defects, and PD.²⁷ While the epidemiologic evidence on the relationship between any type of

solvent and PD is inconsistent,²⁸ the relationship between specific solvents, such as TCE, and PD is an exception to this statement. There is consistent and substantial evidence supporting the relationship between TCE exposure and PD.^{20,29} The route of exposure for TCE includes inhalation, dermal exposure, and ingestion.³⁰ All exposure routes pose health risks,²⁷ however, inhalation of TCE is a particularly potent route of exposure that results in highly powerful dopaminergic neurodegeneration.³¹ Oral consumption of TCE leads to toxic metabolites accumulating in the body resulting in dopamine neuron degeneration.³² Because PCE is more lipophilic than TCE, it has greater bioaccumulation resulting in greater toxic accumulation.³³⁻³⁵ Additionally, both TCE and PCE readily evaporate from contaminated water resulting in indoor air pollution through vapor intrusion.³⁶ Inhalation of TCE and PCE may be an even more potent route of exposure than ingestion as inhalation circumvents the liver and allows for the compounds to cross the blood-brain barrier.³⁷ The findings from epidemiologic studies on the human health risk posed by TCE and PCE has been found unreasonable by the US Environmental Protection Agency and resulted in the request to ban these substances.^{38,39}

The link between TCE exposure and PD has been hypothesized since 1969.⁴⁰ TCE as a cause of PD was first described in a 1999 case-report describing the case of a woman who developed PD after 7 years of TCE exposure.⁴¹ The authors expanded on this case by evaluating TCE exposure on neuronal death in mice finding that mice exposed to TCE presented significant dopaminergic neuronal death when compared to the control group.⁴¹ A 2008 case study built on the work evaluating solvents and PD risk by evaluating workers in a factory with TCE exposure.⁴² Of the 65 people who were included, 3 people who were directly adjacent to the TCE source with chronic inhalation and dermal exposure to TCE developed PD.⁴² There were 21 people who were stationed further away from the TCE source resulting in respiratory exposure only resulting in reported symptoms of parkinsonism.⁴²

A 2012 study evaluating the risk of PD in twins discordant for PD was the first epidemiologic study to explicitly explore the relationship between TCE and PD as well as PCE and PD.⁴³ This study found that any exposure of TCE, as evaluated through occupational exposure assessment methods, was associated with a 500% increased risk of PD, and was statistically significant (adjusted odds ratio (aOR) 6.1, 95%CI 1.2-33).⁴³ Furthermore, the risk of PD after PCE exposure was even stronger with a 950% increased risk of PD after PCE exposure, but not statistically

significant (aOR 10.5, 95%CI 0.97-113).⁴³ There were half as many people exposed to PCE compared to TCE in this study.⁴³ The low prevalence of PCE exposure is the driving force behind the association between PCE and PD not being statistically significant despite the incredibly strong association.⁴³ As described above, statistical significance is driven by sample size and magnitude of the association.

The Goldman authors also analyzed a combined category of “TCE or PCE(labeled as PERC in their study)” as a separate category of exposure on the risk of PD.⁴³ The authors combined TCE and PCE into a single category due to the shared toxic properties of TCE and PCE (labeled as PERC in their study).⁴⁴⁻⁴⁶ As shown in the table below (Figure 1),⁴³ in evaluating the combined risk of TCE and PCE (labeled as PERC in their study), they found a 790% increased risk of PD after exposure to “TCE or PCE” (aOR 8.9, 95% CI 1.7 – 47).^{Footnote 1}

Solvent	Case ⁻ / Control ⁻	Case ⁺ / Control ⁻	Case ⁻ / Control ⁺	Case ⁺ / Control ⁺	Ever/Never Exposed, OR (95% CI)	<i>p</i>
Toluene	72	11	9	7	1.3 (0.5–3.3)	>0.2
Xylene	88	6	2	3	2.2 (0.4–12)	>0.2
<i>n</i> -Hexane	85	6	7	1	1.3 (0.4–4.1)	>0.2
CCl ₄	74	14	9	2	2.3 (0.9–6.1)	0.088
PERC	93	5	1	0	10.5 (0.97–113)	0.053
TCE	87	9	2	1	6.1 (1.2–33)	0.034
TCE or PERC	85	11	2	1	8.9 (1.7–47)	0.010
Any of 6 solvents	51	19	14	15	1.7 (0.8–3.7)	0.16
Any of 4 excluding TCE and PERC	53	18	15	13	1.5 (0.7–3.1)	>0.2

^aEver exposure, adjusted for respondent type and smoking.

CCl₄ = carbon tetrachloride; CI = confidence interval; OR = odds ratio; PD = Parkinson disease; PERC = perchloroethylene (tetrachloroethylene); TCE = trichloroethylene.

Figure 1. Table 3 from Goldman et al. 2012⁴³ Solvent Exposure Frequencies and Adjusted Pairwise Odds Ratios in PD-Discordant Twins (n = 99 pairs). Note the numbers in the table reflect pairs and not

¹ The authors noted their combined category of “TCE or PERC” was included “because the compounds are thought to share toxic properties.” citing Bringmann G, God R, Feineis D, et al. The TaClo concept: 1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline (TaClo), a new toxin for dopaminergic neurons. J Neural Transm Suppl. 1995;46:235–44. [PubMed: 8821060]; Lash LH, Fisher JW, Lipscomb JC, Parker JC. Metabolism of trichloroethylene. Environ Health Perspect. May; 2000 108(Suppl 2):177–200. [PubMed: 10807551]; and, Lash LH, Parker JC. Hepatic and renal toxicities associated with perchloroethylene. Pharmacol Rev. Jun; 2001 53(2):177–208. [PubMed: 11356983].

The combined category's statistically significant finding is telling in that by combining TCE and PCE the authors were able to increase the sample size sufficiently to result in findings that are statistically significant in combination with a strong measure of effect. This further supports, or in fact demonstrates, that the number of people in the study (study power, or lack thereof) was driving whether the relationship between PCE and PD is demonstrated with statistical significance or not when PCE was evaluated on its own. Additionally, this finding illustrates the importance of the combined effects of TCE and PCE on PD.⁴³ This study further highlighted the long latency period of PD as the time from TCE or PCE exposure to PD ranged from 10-40 years.⁴³

A 2021 case-control study based in Finland used occupation as a proxy for solvent exposure as a risk factor for PD.⁴⁷ The authors found that occupations with a potential exposure to chlorinated hydrocarbon solvents, including TCE, had an increased risk of PD.⁴⁷ The measures of association found in this study are likely a conservative estimate of the association between solvent and risk of PD as the use of occupation as a proxy to solvent exposure overestimates actual exposure thereby driving the association closer to the null.

TCE/PCE Exposure from Camp Lejeune and PD Mortality

The role of TCE and PCE on PD gained interest and received considerable attention over the last decade, particularly concerning the contaminated water at Camp Lejeune, a Marine base in North Carolina. The drinking water at Camp Lejeune was contaminated with TCE and PCE, and other volatile organic compounds (VOCs), over 30 years resulting in levels of TCE and PCE up to 3000 times the permitted safety standards.⁴⁸ This resulted in Marines and civilians living and working at Camp Lejeune being exposed to TCE and PCE through the water they used for drinking, cooking, and bathing for this 30-year period. Exposure to TCE and PCE was through ingestion, inhalation, and dermal contact.⁴⁹ For the purposes of my opinions stated in this report I will be assuming the median levels of TCE and PCE as set out Goldman, et al., 2024 (TCE median 366µg/L, PCE median 15.4µg/L) for Hadnot Point,⁵⁰ and in the Agency for Toxic Substances and Disease Registry (ATSDR) Assessment of Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases, January 13, 2017, pp. 151-152 (TCE median 3.5366µg/L, PCE median 84.9µg/L) for Tarawa Terrace.⁴⁹ The findings of Goldman, et al. 2024 will be addressed later in this report.⁵⁰

To evaluate whether people who lived or worked on Camp Lejeune were at higher risk of PD a 2014 cohort study compared civilian, full-time workers at Camp Lejeune to a similar group of people at Camp Pendleton, in California, on the risk of mortality.⁵¹ The use of civilian, full-time workers in this study provided the opportunity to evaluate the relationship between the contaminated water and risk of mortality in people who were only exposed to the contaminated water when they were at work, a lower exposure threshold than people residing on base. A prior Bove et al. 2014 study evaluated TCE/PCE and mortality in marine and naval personnel, however, there were not enough PD deaths to include PD in the analysis.⁵² Evaluating the civilian population at Camp Lejeune removed any of the concerns regarding exposures during military deployment interacting with the relationship findings between TCE/PCE exposure and PD mortality.⁵² The 2014 civilian cohort study compared populations from 2 bases, a study design approach to reduce the influence of outside factors on the evaluation of the relationship between TCE/PCE and PD mortality. The Camp Lejeune and Camp Pendleton populations used in this study were similar with regard to occupation, months employed, and education.⁵¹ The length of exposure was influenced by length of employment (median length of employment at Camp Lejeune in this study was 2.5 years), indicating that 2 years on base is sufficient to increase the risk of PD.⁵¹ The authors found an increased risk of mortality due to PD in the people who worked at Camp Lejeune compared to Camp Pendleton (adjusted hazard ratio (aHR) = 3.13, 95% CI:0.76, 12.86).⁵¹ Even though the relationship is not statistically significant, the incredibly strong measure of association indicates that it is likely that if the sample size was larger the relationship would likely be statistically significant with a strong measure of association. There was a very small number of people who died due to PD in both populations, resulting in sample size constraints and reduced power. The small number of people who died due to PD is not unexpected as despite the high morbidity associated with PD the disease has a largely normal life expectancy.⁵³ Additionally, the authors estimated cumulative exposures to TCE and PCE to evaluate the contaminant-specific effects on risk of PD mortality.⁵¹ Cumulative exposures were above the median for most of the PD deaths.⁵¹ In the Camp Lejeune population, 4 of the 5 people with PD deaths were above the median cumulative exposure for PCE and TCE.⁵¹ Cumulative TCE exposure over the median increased the risk of PD death 151% (aHR 2.51, 95%CI 0.21-30.76), while cumulative PCE exposure over the median increased the risk of PD death 168% (aHR 2.68, 95%CI 0.22-33.28)).⁵¹ Even though the relationships described in

this study were not statistically significant, the incredibly strong measure of association indicates that it is likely that if the sample size was larger the relationship would likely be statistically significant. This relates to the power of the study. A study that is underpowered will many times demonstrate a robust effect (e.g., a strong measure of association) via the adjusted hazard ratio but will also demonstrate a relatively wide confidence interval, and a lower bound below 1.0, rendering a finding that has a strong measure of association but is not statistically significant.

A 2024 cohort mortality study built on the 2014 findings by Bove et al. 2014⁵¹ by expanding the Camp Lejeune and Camp Pendleton cohorts to include military personnel and incorporating a longer follow-up.⁵⁴ The Bove et al. 2024 study found the Camp Lejeune population to have a higher risk of PD mortality than the Camp Pendleton population (Marine/Naval personnel aHR 2.05, 95%CI 0.86-4.87; Civilian personnel aHR 1.21, 95%CI 0.72-2.04), however, the total number of PD deaths remained small contributing to the non-statistically significant finding.⁵⁴ Again, this appears to demonstrate an underpowered study rather than a lack of effect. Similar to the 2014 study, the small number PD deaths contributed to the non-statistically significant findings. The strong measures of association indicates that it is reasonable to postulate that if the sample size were increased the associations would likely be statistically significant. Furthermore, the authors explored whether base duration explained some of the findings between contaminant exposure and PD mortality and found that regardless of whether the duration was low (aHR 2.07, 95%CI 0.62-6.95), medium (aHR 2.63, 95%CI 0.91-7.66), or high (aHR 1.59, 95%CI 0.51-4.96) the strong association remains between Camp Lejeune and risk of PD mortality. The authors conducted a quantitative bias analysis to explore whether exposure misclassification contributed to the findings.⁵⁴ For both the civilian and military populations exposure misclassification did not contribute to the findings.⁵⁴

TCE/PCE Exposure from Camp Lejeune and PD

There are limitations with using PD mortality as a proxy for PD since PD patients have a largely normal life expectancy. The Agency for Toxic Substances and Disease Registry (ATSDR) conducted a morbidity study on former marines, employees, and dependents exposed to contaminated drinking water at Camp Lejeune.⁵⁵ This study deployed surveys to people who had lived and worked at Camp Lejeune and as a comparison Camp Pendleton with a 31% response

rate.⁵⁵ Of note, there was a difference in age for civilians compared to the military sample in this study: 51% of the civilians were 65 or younger, while 93% of the military population were younger than 65.⁵⁵ This is an important distinction as PD is typically not identified until later in life with 90% of diagnoses occurring after age 65.² The younger military population included in this survey contributed to very low occurrences of PD within that population resulting in sparse data.⁵⁵ Among civilians, the relationship between TCE/PCE and PD was strong (aOR 3.1, 95%CI 1.2-8.3).⁵⁵ The authors found the results of this study were in support of the prior ATSDR report concluding evidence of causality for TCE and PD was equipoise and above.^{49,55}

The limitations of studying PD mortality by Bove et al. 2014⁵¹ and using a survey to assess risk of PD⁵⁵ led to a separate 2023 study evaluating the risk of PD and prodromal symptoms of PD between the Camp Lejeune and Camp Pendleton populations. The prodromal phase of PD is a phase when non-motor symptoms predate the onset of classic PD symptoms.³ Prodromal PD symptoms include constipation, hyposmia, possible REM-sleep behavior disorder (RBD), depression, anxiety disorder, and cognitive impairment.³ This phase can last up to 20 years before the onset of classic symptoms.³ This study found the Camp Lejeune population had a 70% higher risk of PD (aOR 1.70; 95%CI 1.39-2.07).⁵⁶ Additionally, the findings were similar for higher risk of prodromal PD diagnoses, or pre-PD diagnoses, including tremor (aOR 1.19, 95%CI 1.09-1.29) and higher cumulative prodromal risk scores (aOR 1.18, 95%CI 1.06-1.32) in the Camp Lejeune population compared to the Camp Pendleton population.⁵⁶ It is significant that overall PD and these prodromal pre-PD diagnoses all demonstrated clear association, and all demonstrated effect with statistical significance. The increased risk for prodromal PD identifies populations who are progressing towards PD. This is important particularly here where ATSDR was studying a disease that is typically diagnosed \geq age 65, and in an admittedly younger cohort.² Because, as stated earlier, prodromal symptoms may precede formal PD diagnosis by as much as 20 years, identifying these diagnostic markers in the younger cohort offers critical foreshadowing of which populations might be on the path to PD.

The authors conducted a subgroup analysis limiting the PD diagnosis prior to 2017 and found the risk of PD for people in Camp Lejeune remained, but was attenuated compared to the Camp Pendleton population (aOR 1.28, 95%CI 1.00-1.64).⁵⁶ Risk of probable PD was higher for the Camp Lejeune population compared to the Camp Pendleton population (aOR 1.81, 95%CI 1.46-

2.24).⁵⁶ This study found that 3 months exposure to TCE/PCE was sufficient for developing PD.⁵⁶ These robust findings and clear associations precipitated a JAMA Neurology editorial accompanying this publication stating “The research done by Goldman et al. increases the certainty that environmental exposures to TCE and PCE contribute importantly to the cause of the world’s fastest-growing brain disease” and emphasizing the acceptance of TCE and PCE as causes of PD in the field of neurology.⁵⁷

Because alternative explanations, such as genetics, could be suggested as an explanation for the example of the increased incidence at Camp Lejeune, it should be addressed. It is highly improbable the difference in the risk of PD between Camp Pendelton and Camp Lejeune, in closely matched cohorts of both military personnel and civilians is due to genetic factors. This is illustrated by calculating the excess attributable risk percent using data from this study. The attributable risk percent for developing PD due to TCE/PCE exposure in this population is 44.7%, meaning 44.7% of the PD risk is explained by exposure to TCE/PCE at Camp Lejeune, the only major difference between the two populations. This study was sufficiently powered and had sufficient sample sizes resulting in both statistically significant and clinically relevant findings, including in sub analyses, further supporting the clinically relevant findings from studies with smaller samples where findings did not meet statistical significance. The consistency in the findings across these studies with robust measures of association supports prior statements regarding sample size concerns as well as supports the consistent findings of robust associations between TCE/PCE and PD.

Following the 2023 study on PD risk in Camp Lejeune populations questions arose regarding the role of TCE/PCE exposure on PD symptoms and progression. A cohort study conducted on a population of people from Camp Lejeune exposed to volatile organic compounds and evaluated whether volatile organic compound (VOC) exposure (including TCE and PCE) is a risk factor for PD progression.⁵⁰ The authors found people who were exposed to VOCs at Camp Lejeune had faster progression to markers of PD progression including psychosis (aHR 2.19, 95%CI 0.99-4.83), falls (aHR 2.64, 95%CI 0.97-7.21), fractures (aHR 2.44, 95%CI 0.91-6.55), and death (aHR 1.12, 95%CI 0.52-2.41).⁵⁰ As the exposure to VOCs increased the risk of PD progression increased for psychosis (aHR 2.14, 95%CI 1.43-3.19), fall (aHR 2.86, 95%CI 1.77-4.61), and fracture (aHR 2.12, 95%CI 1.31-3.43).⁵⁰ The authors noted that TCE was the major

contaminant, and that a causal association was supported, and recognized that PCE was “the other predominant contaminant,” but is less studied.⁵⁰ Footnote 2

The robust findings from the above studies and laboratory studies on the role of organic solvents and PD has led to the conclusion that PD is predominantly an environmental disease.²⁰ This is particularly concerning as prevalent use and widespread contamination of TCE/PCE results in people not knowing they were exposed.^{20,58} The resulting underestimate of TCE/PCE exposure results in a significant underestimate of the influence of TCE/PCE on PD. Further, because there is evidence that Camp Pendleton is now also a Superfund site, with unknown and uncertain contamination with like VOCs during the years relevant to the Camp Lejeune contamination, any common contaminants – if there were any during relevant timeframes -- between Camp Lejeune and Camp Pendleton would only skew the comparison-driven risks toward the null, and understate the demonstrated risks.

Mechanism of Action

The epidemiologic link between TCE and PD is strongly supported by decades of research in laboratory settings. Many of the mechanisms relating TCE to PD are thought to be similar for PCE as PCE differs from TCE through an additional chlorine atom.^{59,60}

TCE and PCE are both lipophilic resulting in distribution throughout tissues in the brain and body.⁵⁹ During TCE and PCE breakdown they share a common metabolite, such as 1-trichloromethyl-1,2,3,4-tetrahydro- β -carbolin, that is implicated in toxicity resulting in mitochondrial dysfunction.^{59,60} The neurotoxicity of TCE has been well described for decades, whereas the underlying mechanisms behind TCE and subsequent neurodegeneration are a recent development with multiple potential mechanisms hypothesized behind TCE causing neurodegeneration and PD. The neurotoxicity of PCE has received less attention in the scientific literature, however, the evidence that is available implicates PCE as just as toxic, if not more so, than TCE. The structural similarities between TCE and PCE, the commonality of known

² The fact that PCE has received less attention in the scientific literature is discussed in greater detail in the report of Jason Cannon, PhD, Dr. Cannon, a neurotoxicologist, explains the structural and functional similarities of PCE and TCE, their toxicities, and the relative attention devoted to TCE and PCE in the scientific literature. I reviewed the Cannon report for context and to further understand the relative similarities and toxicities of the VOCs. I have, however, conducted my own review of the literature in forming my opinions stated in this report.

metabolites, and the co-occurrence of TCE and PCE in exposures support extrapolating the toxic effects identified in TCE studies to PCE. ^{See footnote 2}

These mechanisms include the inhibition of mitochondrial complex I activity,^{32,42,61,62} causing induction of LRRK2 activity that is similar to LRRK2 mutations known to cause PD,⁶³ disruption of α -synuclein accumulation, aggregation, and misfolding, neuroinflammation,^{32,42,64,65} microbial alterations within the gut microbiome after consumption of TCE similar to changes seen in idiopathic PD,⁶⁶ and degeneration of dopaminergic neurons.^{31,32,41,42,62,63,67} Additionally, there is an intensifying effect between the link of organic solvents, including TCE and PCE, and PD within certain populations with an increased risk of PD. This is particularly relevant for populations who are predisposed to PD through genetic predisposition to PD.⁶¹ People with a genetic predisposition to PD are particularly vulnerable to the toxic effects of TCE/PCE as the combined effects from accumulating proteins due to genetic mutations coupled with toxic effects from build-up of TCE/PCE result in the subsequent development of PD. Furthermore, people who have mutations in genes responsible for the metabolism of toxins or defense against toxins are not able to metabolize TCE/PCE resulting in the greater accumulation of toxic metabolites from TCE/PCE and subsequent development of PD.⁶⁸ These gene-environment interactions are why only some individuals exposed to TCE/PCE develop PD.²⁹

The level of exposure to TCE needed to cause disease has also been of great interest. It is widely accepted that high-dose exposure to TCE leads to PD or PD like symptoms.⁶⁸ However, exposure to lower doses of TCE has been implicated in dopaminergic neurodegeneration as well.³¹ Due to the under-capture of TCE exposure it is likely there are many people considered “unexposed” who have had low doses of exposure. A review by Dorsey et al. highlights the difficulties in assessing exposure to TCE and includes 7 specific case examples of TCE-induced PD reiterating the likely under-reporting of TCE exposure and highlighting the role of the long latency period of PD in evaluating environmental factors.²⁹ Dorsey et al. highlighted the role of TCE exposure as an invisible and highly preventable cause of PD, as well as TCE’s contribution to the global rise in PD.²⁹

Causal Assessment of TCE/PCE and PD

I have reviewed portions of the Camp Lejeune Justice Act of 2022, and specifically Sec. 2, (b), “Burdens And Standard of Proof.”

“(1) IN GENERAL.—The burden of proof shall be on the party filing the action to show one or more relationships between the water at Camp Lejeune and the harm.

(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 that a causal relationship exists; or

(B) sufficient to conclude that a causal relationship is at least as likely as not.”

Further, the ATSDR described in its 2017 Health Assessment (1/13/2017) equates the standard used in its Health Assessment of “equipoise or above” with the Camp Lejeune Justice Act’s standard of “at least as likely as not.”⁴⁹

The literature on the relationship between TCE/PCE and PD supports concluding a causal link between TCE/PCE and PD when applying these burdens and standards of proof. This is further supported by the ATSDR conclusions that the evidence for causality between TCE and PD was equipoise and above.⁴⁹ The relationship between PCE and PD that has been further refined in the literature and data reviewed above support the causal relationship between PCE and PD as “equipoise or above”.

To further evaluate the causal link between TCE/PCE and PD the following explores how the literature on TCE/PCE and PD fits within an application of the Bradford Hill considerations.⁶⁹ The considerations first explained by Sir Austin Bradford Hill are intended to be used as tools to understand, assess, and view evidence supporting causal relationships, or not.⁶⁹ As illustrated in Table 1 and the paragraphs below the majority of Bradford Hill considerations are met when evaluating the relationship between TCE/PCE and PD.

Table 1. Bradford Hill Considerations and Evidence Described in this Report

Consideration	Hill consideration met/not met
Temporality	<u>Met</u> : All studies reviewed demonstrate exposure prior diagnosis. This applies equally for TCE and PCE.
Strength of association	<u>Met</u> : The incredibly strong measure of association found in epidemiologic studies is consistently shown over and over in the above assessment.
Consistency	<u>Met</u> : The strong association in the same direction from study to study is consistently met as shown above and below.
Specificity	<u>Partially met</u> . As the ATSDR Health Assessment, 1/13/2017, demonstrates, the contaminated water at Camp Lejeune is causally related to a number of cancers and Parkinson's disease (even limiting this comment to only those for which ATSDR found "evidence sufficient to conclude a causal association exists.") Thus, specificity using this definition is not demonstrated. However, a specific population who has experienced a higher than-expected exposure has consistently demonstrated an increased risk of PD. In this sense specificity is met.
Dose-response	<u>Partially met</u> : Laboratory and case reports/studies illustrate how as the does of exposure increases the risk and severity of symptoms increase. Here are also studies that demonstrate a non-monotonic dose response.
Biological plausibility	<u>Met</u> : As illustrated throughout the report the laboratory studies consistently show varying mechanisms whereby TCE/PCE cause PD.
Coherence	<u>Met</u> : There are consistent findings between laboratory and epidemiologic literature highlighting how TCE/PCE cause PD.
Experiment	<u>Partially Met</u> : The experimental findings of laboratory studies highlight how TCE/PCE cause PD. However, there is no experimental evidence within people regarding these relationships as this is not ethical.
Analogy	<u>Met</u> : Explorations into other causes highlight how exposure to TCE/PCE at Camp Lejeune was the primary factor in development of PD.

Temporality

All studies evaluating the association between TCE/PCE and PD ensured a temporal relationship, meaning the exposure happened before the outcome. This is arguably the only Bradford Hill consideration that is necessary for concluding causation, as without temporality there can be no causal relationship. The studies evaluating the relationships in people ensured the exposures occurred years prior to PD or PD mortality. In some circumstances, such as the ATSDR morbidity study,⁵⁵ the populations have not had time to develop PD yet resulting in sparse data. However, the prodromal study provided helpful early indicators of PD, with statistical, in the cohort comparing Camp Lejeune and Camp Pendleton.⁵⁰ Temporality is met. Each of the studies reviewed above clearly demonstrates that exposure to the Camp Lejeune water contaminated with TCE and PCE, preceded diagnosis, usually by years to decades. Although temporality is clearly met, it is interesting to note that Marines stationed at Camp Lejeune were diagnosed at a younger age when compared to civilian employees, and were noted to have a faster progression of disease.^{2,50} The temporality Hill consideration is met.

Strength of Association

The strength of association between TCE/PCE and PD or PD mortality was consistently very strong. Regardless of the population being assessed or how the exposure and/or outcome were being measured, the strength of association was consistently strong. While the associations were not always statistically significant, the strong effects were most likely indicating that not achieving statistical significance was due to sample size/study power. Many of the studies evaluating TCE/PCE and PD did not have enough people exposed to TCE/PCE or had small numbers of people with PD or PD mortality resulting in underpowered studies. Regardless, strong measures of association between TCE/PCE and PD were seen across different study populations and time-periods. And, recall, Goldman (2012) evaluated TCE and PCE separately and in a combined category (“TCE or PCE”), and the combined TCE/PCE group demonstrated a statistically significant effect, and with a lower bound at a higher level than TCE alone.⁴³ The strength of association Hill consideration is met.

Consistency

Consistency was an integral theme in the findings from the literature on TCE/PCE and PD. The results of the studies on TCE/PCE and PD were incredibly consistent, even with studies using proxies for TCE/PCE exposure finding strong relationships. Nonetheless, a consistently strong measure of association was found between TCE/PCE and any measure of PD outcome whether it was PD, PD mortality, or prodromal PD. The findings of these studies were always in the same direction, with a strong magnitude of association. In the study by Bove et al. 2024, concern regarding misclassification of TCE/PCE exposure was addressed through quantitative bias analysis to account for potential misclassification of TCE/PCE exposure and found an even stronger relationship with TCE/PCE exposure and PD mortality after accounting for potential exposure misclassification.⁵⁴ The consistent relationship was replicated in animal model studies further supporting the causal link (see above). The different proxies, techniques, and populations did not affect the consistent associations between TCE/PCE and PD in the literature. The consistency Hill consideration is met.

Specificity

It is widely acknowledged that exposure to TCE/PCE can lead to a wide array of poor health outcomes, including PD. The idea that a particular exposure should lead to a single outcome is rarely seen and is not necessary for drawing causal conclusions. Furthermore, the idea of specificity in causal relationships also includes whether an association is limited to a specific population. In this regard, specificity is met with the consistently elevated rates of PD in the Camp Lejeune populations. These populations should not have a higher risk for PD than the general military populations (e.g., Camp Pendleton) or the overarching United States population, however, regardless of military status people who lived or worked at Camp Lejeune were at higher risk for PD than other comparable populations. This supports the conclusion the cause is tied to a specific population at a specific location within a specific timeframe. Stated differently, living or working at Camp Lejeune results in increased diagnoses for various cancers and PD; specific to TCE/PCE and their causal link to PD, exposure to the contaminated water at Camp Lejeune does result in a higher incidence of PD – being part of the population of persons on Camp Lejeune provides specificity under this Hill consideration. The more specific an

association between an exposure and outcome, or a population exposed and outcomes, the more likely the association is causal. The specificity Hill consideration is partially met.

Dose Response

There is consistent evidence of a dose-response relationship, otherwise known as a biological gradient, whereby the findings in the literature support the idea that the higher the dose of exposure the higher the risk of the outcome. Consistently the studies found higher exposure of TCE/PCE led to higher risk of PD, as well as more severe symptoms of PD and faster progression of PD. There are studies that demonstrate a non-monotonic dose-response as well. As a result, the dose-response Hill consideration is partially met.

Biological Mechanisms

Plausible biological mechanisms are presented as to how and why TCE/PCE exposure leads to PD. Through multiple mechanisms described above TCE/PCE leads to a number of pathways that result in increased PD risk. I have set out the plausible mechanisms here, see section titled “Mechanism of Action” beginning on p.14. Additionally, the structural and functional similarities of TCE and PCE support the similarity in epidemiologic findings and toxicities. Further still, as stated above, I have reviewed the report of Jason Cannon, PhD, neurotoxicologist. Dr. Cannon explains in greater detail the biological mechanisms and metabolic pathways. Based upon my independent review of the literature cited herein, and my review and understanding of Dr. Cannon’s report, the biological plausibility consideration is met.

Coherence

Hill describes coherence as “not seriously conflict[ing] with the generally known facts” about the disease. As I described above, PD is by and large described as idiopathic or related to genetics or environmental exposures. Both the laboratory studies evaluating the association between TCE/PCE and PD in basic science and animal studies, and epidemiologic studies evaluating the relationship in humans, are entirely in line with the known mechanistic and toxicological effects of TCE and PCE. The coherence consideration is met.

Experiment

The Hill consideration of experiment is met as to biologic and mechanistic experiments that have discovered and isolated various biologic activities such as induction of LRRK2, identifying common metabolites of TCE/PCE, α -synuclein accumulation and aggregation, but the classic human experiment of de-challenge-rechallenge, or prospective controlled human studies simply are not possible. Laboratory studies have consistently shown how TCE/PCE causes PD, however, it is unethical to conduct a randomized controlled trial (RCT) or de-challenge-rechallenge to fully address the Bradford Hill Criteria of experiment. Conducting an RCT to expose people to TCE/PCE to evaluate the PD risk, particularly with the regulatory statements advising reduction of TCE/PCE exposure due to concerns for human health impacts, is not only unethical it is impractical. PD has a long latency period meaning it takes the symptoms of the disease a long time to appear after the disease process starts. This would result in an RCT with a 20–30-year follow-up, which is inefficient, costly, and not feasible. The long latency period of PD is a major reason for the use of retrospective epidemiologic studies and not prospective epidemiologic studies as following people for long periods of time results in significant loss to follow-up, resulting in biased study results. As a result, experiment is a mixture of met and unmet.

Analogy

Finally, for the reasons explained above, and explained further and in greater depth in the report of Dr. Cannon, the consideration of analogy is met. Without simply restating what has been explained above and by Dr. Cannon, the functional and structural similarities, and common and shared metabolites, coupled with the confirmation from the epidemiological evidence, demonstrates that analogy is met.

In exploring other hypotheses for the relationship between TCE/PCE and PD to date there are no confounding variables from a population-based perspective that can explain away the findings. There is no demonstrable reason populations from Camp Lejeune and Camp Pendleton should demonstrate different rates of PD, or other diseases. Their respective demographic characteristics were strikingly similar and should reflect similar event rates and risks. However, the glaring difference is the contaminated water at Camp Lejeune.

Acknowledging that the Bradford Hill considerations are a guideline for evaluating the literature on a topic when assessing causality and not a checklist, the overwhelming evidence from the literature, in addition to meeting the Bradford Hill considerations addressed above, supports the conclusion it is at least as likely as not that both TCE and PCE can cause PD. The strong, robust relationships found in all studies evaluating TCE/PCE and PD demonstrate this important causal relationship.

Conclusion

It is widely recognized that the neurodegenerative disorder PD is an environmental disease. The epidemiologic and laboratory evidence implicates TCE and PCE as important causes of PD, with increased risk in people exposed than people not exposed. The increased measure of association, whether statistically significant or not, is consistent across study designs, populations, and time. The consistent findings from the epidemiologic and laboratory studies finds an indisputable relationship between TCE and PD. There is a dose-response to TCE exposure and PD, however, laboratory results indicate even low levels of exposure to TCE increases the risk of PD. The extent of the contamination at Camp Lejeune coupled with the duration of the contamination (30 years of contamination resulting in levels of TCE and PCE up to 3000 times the permitted safety standards) resulted in a population with higher incidence and prevalence of PD and worse PD disease than other populations. Using the results from the Goldman et al. 2023 study, the attributable risk proportion found 42% of the PD risk in that population was due to TCE/PCE exposure.

TCE and PCE are widely implicated in increased risks of poor health outcomes, many of which have an onset earlier than PD. PD is a slow progressive disease with a long latency period where symptoms do not occur for years or even decades after disease onset. The competing risks of death from other outcomes due to TCE and PCE exposure in the Camp Lejeune population are not to be undersold. It is likely people with the longest exposure or highest exposure to TCE/PCE from Camp Lejeune died from other implicated health outcomes before being able to show symptoms of having PD. Even without accounting for the competing risks of death from other outcomes, the incidence and prevalence of PD in the Camp Lejeune population is much higher than the incidence and prevalence in the US. Furthermore, the populations exposed to

TCE/PCE had more severe disease and progressed faster with their PD than people who were not exposed. The increased risk, more severe disease, and faster progression of PD for people who lived or worked at Camp Lejeune emphasize the role of exposure to the contaminated water on these outcomes. The consistent findings from the epidemiologic and laboratory studies present indisputable relationships between both TCE and PD, and PCE and PD, whereby both relationships are at least as likely as not a causal relationship.

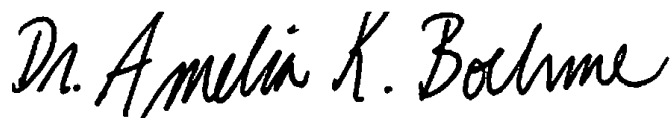
Based upon the foregoing, and upon my education, training and experience, and the data and information contained in this report, I have formed opinions to a reasonable degree of scientific certainty. These opinions are:

- That it is at least as likely as not that exposure to the water at Camp Lejeune, contaminated with TCE, can cause Parkinson's Disease.
- That it is at least as likely as not that exposure to the water at Camp Lejeune, contaminated with PCE, can cause Parkinson's Disease.
- That it is at least as likely as not that exposure to the water at Camp Lejeune, contaminated with TCE and PCE, and other volatile organic compounds, can cause Parkinson's Disease.

I am being compensated at \$350 an hour for my time devoted to researching, analyzing, and writing this report.

Dated 8 December 2024.

Sincerely,

A handwritten signature in black ink that reads "Dr. Amelia K. Boehme". The script is cursive and fluid, with the first letters of each word being capitalized and prominent.

Amelia K. Boehme, PhD, MSPH

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Additional Materials Reviewed

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CURRICULUM VITAE
Amelia Katharine Boehme, PhD, MSPH, FAHA
Date Updated: May 01, 2024

PERSONAL DATA:

Birthdate: February 26, 1984
Birthplace: Huntsville, AL
Citizenship: United States

Home Address: 245 E. 63rd Street Apt 618, New York, NY 10065
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Email: akb2188@cumc.columbia.edu
akboehme@gmail.com
amelia.boehme@aetion.com

EDUCATION:

08/2010-06/2014	University of Alabama at Birmingham School of Public Health PhD, June 2014 Major: Epidemiology Thesis Title: <i>Pharmacogenetics and Predictors of Outcomes in Patients on Ventricular Assist Devices</i> Sponsors: Gerald McGwin and Nita Limdi	Birmingham, AL
08/2008-05/2010	University of Alabama at Birmingham School of Public Health MSPH, May 2010 Major: Epidemiology Thesis Title: <i>Medication and Clinical Adherence in HIV+ Postpartum Women</i> Sponsor: Mirjam Colette-Kempf	Birmingham, AL
08/2002-05/2007	University of Alabama in Huntsville BS May 2007	Huntsville, AL

POST DOCTORAL TRAINING:

July 2014-Dec 2015 Post-Doctoral Research Fellow in Neuroepidemiology, Department of Neurology, Columbia University, New York, NY
NIH T32 NS007153 31

ACADEMIC APPOINTMENTS:

2022-present	Adjunct Assistant Professor	Columbia University
2020-present	Adjunct Associate Professor	New York University
2015-2022	Assistant Professor, Tenure Track	Columbia University
2017-2020	Adjunct Assistant Professor	New York University
2014-2015	Postdoctoral Fellow, Neurology	Columbia University
2012-2014	AHA Pre-Doctoral Fellow, Epidemiology	University of Alabama at Birmingham
2011-2013	Graduate Research Assistant, Neurology	University of Alabama at Birmingham
2010-2011	Graduate Research Assistant, Epidemiology	University of Alabama at Birmingham
2009-2010	Graduate Research Assistant, Neurosurgery	University of Alabama at Birmingham
2004-2006	Undergraduate Research Assistant, Organic Chemistry	U. of Alabama in Huntsville

NON-ACADEMIC APPOINTMENTS:**2022-present Director, Science****Aetion, Inc.****AWARDS/HONORS:**

2020 National Institute of Health Loan Repayment Ambassador
 2020 National Institute of Health Loan Repayment Award
2020 Fellow of the American Heart Association
2019 Best of Science, International Stroke Conference 2019
 2019 Early Career Reviewer Program at the Center for Scientific Review
 2019 National Institute of Health Loan Repayment Ambassador
 2018 National Institute of Health Loan Repayment Ambassador
 2018 National Institute of Health Loan Repayment Award
 2018 Reach for the First R01 Award, Columbia University CTSA
 2017 Calderone Junior Faculty Award
 2016 National Institute of Health Loan Repayment Award
 2015 National Institute of Health Loan Repayment Award
 2014 University of Alabama at Birmingham, Health Disparities Research Symposium, 2nd Place
 Charles Barkley Health Disparities Investigator
 2014 Nominated member of the research honor society Sigma XI, University of Alabama at
 Birmingham
 2014 University of Alabama at Birmingham, Graduate Student Research Day, 2nd Place
 2013 University of Alabama at Birmingham, School of Public Health Research Day, 3rd Place
 2012 American Heart Association Student Scholarship in Cardiovascular Disease and Stroke
 2012 Alabama Public Health Association 56th Annual Health Educational Conference, 3rd
 place
 2012 University of Alabama at Birmingham, Health Disparities Research Symposium, 2nd Place
 Charles Barkley Health Disparities Investigator
 2012 University of Alabama at Birmingham, School of Public Health Research Day, 1st Place
 2012 University of Alabama at Birmingham, Delta Omega Epidemiology Honor Society Poster
 Competition 3rd Place
 2010 Nominated Member of AAAS/Science Program for Excellence in Science
 2004-2007 University of Alabama in Huntsville Science Ambassador
 2003 Freshman Honor Society Alpha Lambda Delta, University of Alabama in Huntsville

PROFESSIONAL SOCIETIES AND MEMBERSHIPS:

2012-present American Heart Association
 2012-present Society for Epidemiologic Research
 2012-present American Academy of Neurology
 2012-present American College of Epidemiology
 2012-present American Statistical Association

COMMITTEES AND SERVICE:

2019-present Member of the EPI Stroke Statistics Subcommittee of the Council on Epidemiology and
 Prevention, American Heart Association
 2019-present Member of the STROKE Quality and Outcomes Committee of the Stroke Council,
 American Heart Association
 2019-present Liaison on the PVD Leadership Committee of the Council on Peripheral Vascular Disease,
 American Heart Association
 2017-2022 Member of EPIC transition team, Department of Neurology, Columbia University
 2017-present Doctoral Methods Exam Chair, Department of Epidemiology,
 Columbia University

2017-2020	Doctoral Admission Committee Member, Department of Epidemiology, Columbia University
2016-present	Doctoral Methods Exam Committee Member, Department of Epidemiology, Columbia University
2017-present	Practicum Abstract Grader, MPH Research Day, Department of Epidemiology, Columbia University
2016-2019	Membership Committee, Society for Epidemiologic Research
2015-present	American Heart Association New York Young Professional Committee, Red Ball Committee Member
2016-2019	Masters Admission Committee Member, Department of Epidemiology, Columbia University
2012-2015	Membership Committee, Student Caucus, Society for Epidemiologic Research
2012-2015	Mentor/Mentee Committee Member, Society for Epidemiologic Research

DATA SAFETY AND MONITORING BOARD:

2018-2022 DSMB Member for Alexion ECU-NMO-303

REVIEW ACTIVITIES:

Grant Review

NIH NINDS R13 Reviewer May 2023

April 2022 CUIMC CIRAD AD-RCMAR Pilot Application Review

NIH NINDS R13 Reviewer March 2022

April 2021 CUIMC CIRAD AD-RCMAR Pilot Application Review

NIH NINDS R13 Reviewer July 2020

April 2020 CUIMC CIRAD AD-RCMAR Pilot Application Review

NIH NINDS R13 Reviewer March 2020

December 2018 American Heart Association Career Development Grant Review- Population Health Sciences

April 2019 CUIMC CIRAD AD-RCMAR Pilot Application Review

Editorial Board

Frontiers in Epidemiology

Frontiers in Risk Factors of Stroke

Journal Review

Circulation

Neurology

Neurology: Clinical Practice

Circulation Genetics

Circulation: Cardiovascular Quality and Outcomes

Circulation: Genomic and Precision Medicine

Circulation Research

The Lancet: Global Health
JAMA
JAMA Neurology
Stroke
Journal of the American Heart Association
American Journal of Epidemiology
American Public Health Association Journal
International Journal of Stroke
Epidemiology and Infection
Cerebrovascular Disease
Journal of Neurological Disorders and Stroke
Journal of Health Care for the Poor and Underserved
Neuroepidemiology
Psychology, Health and Medicine
AIDS Care
Clinical and Applied Thrombosis/Hemostasis

Abstract Review

American Heart Association Scientific Sessions
American Heart Association Quality of Care and Outcomes Research
American Academy of Neurology
Society for Epidemiologic Research
American College of Epidemiology

CURRENT GRANT SUPPORT:

- 1. K23HL151901** (PI: Roh) 09/01/2021-08/31/2026 NIH/NINDS
 Erythrocyte contribution to coagulopathy and cerebral oxygenation after intracerebral hemorrhage
 Role: Mentor
- 2. K23DC019678** (PI: Overdevest) 06/01/2021-05/31/2026 NIH/NIDCD
 Neurocognitive and neuropsychiatric impact of chemosensory alterations: Implications of olfactory dysfunction in COVID-19
 Role: Mentor

PAST GRANT SUPPORT:

- R01 ES028805** (PI: Kioumourtzoglou) 12/01/2017-11/30/2022 NIH/NIEHS
Principal Component Pursuit to Assess Exposure to Environmental Mixtures in Epidemiologic Studies
 The goal of this grant is to use principle component analyses to assess environmental measures as an exposure for cardiovascular disease.
 Role: Co-Investigator
- R01ES030616** (Kioumourtzoglou & Dominici) 12/01/2019 – 8/31/2024 NIH/NIEHS
 Integrating Air Pollution Prediction Models: Uncertainty Quantification and Propagation in Health Studies
 Role: Co-Investigator
- R01 NS106014** (PI: Claassen) 06/15/2019-03/31/2024 NIH/NINDS
 REcovery of CONsciousness Following Intracerebral hemorrhage
 To identify predictors of recovery of consciousness following intracerebral hemorrhage.
 Role: Co-Investigator
- R01 HD096559** (PI: Idro & Green) 09/11/2019-08/31/2024 NIH/NICHHD

Burden and Risk of Neurological and Cognitive Impairment in Pediatric Sickle Cell Anemia in Uganda (Brain Safe II)

Role: Co-Investigator

R01 AG066162-01 (PI: Gutierrez-Contreras) 09/14/2019-08/31/2024 NIH/NIA

Accelerated non-atherosclerotic brain arterial aging relationship to Alzheimer's disease

Institution: Trustees of Columbia University in the City of New York

Role: Co-Investigator

R01 NS099268 (PI: Kim) 05/15/2017-04/30/2022 NIH/NINDS

Long-Term Outcomes in Unruptured Brain Arteriovenous Malformation Patients

To identify predictors of brain hemorrhage and poor outcomes among those patients who are found to have an arteriovenous malformation in their brains and remain untreated or undergo treatment.

Role: Co-Investigator

R21 HD091836 (PI: Leavitt) 07/01/2019-06/30/2022 NIH/NICHD

Aspirin before Exercise: A Double Blind RCT of Aspirin Pretreatment to Improve Exercise Performance in MS

Role: Co-investigator

P30AG059303 (PI: Manly/Luchsinger) 09/01/2018-06/30/2023 NIH/NIA

Columbia Center for Interdisciplinary Research on Alzheimer's Disease Disparities (CIRAD)

Role: Co-Investigator

R01 NS121364 (PI: Willey) 01/01/2022-12/31/2026 NIH/NINDS

Neurovascular consequences of non-pulsatile flow from left ventricular assist devices

Role: Co-Investigator

RF1AG074608 (PI: Paulsen) 09/30/2021-08/31/2024 NIH/NIA

Unraveling the earliest phases of vascular cognitive impairment and dementia using CADASIL- a monogenic form of small vessel cerebrovascular disease

Role: Co-Investigator

R21 MD012451 (PI: Boehme) 09/24/2017-06/30/2021 NIH/NIMHD

Racial Disparities, Influenza Like Illness and the Association between Short-term Exposure to Ambient Air Pollution and Cardiovascular Outcomes

The goal of this this grant is to investigate air pollution as a trigger for influenza, and how influenza might be a mechanism whereby risk of cardiovascular is increased in areas with high air pollution, geographic disparities and racial disparities.

Role: Principal Investigator

R21 ES030093-01A1 (PI: Boehme and Hilpert) 07/01/2019-06/30/2021 NIH/NIEHS

Crowd-Sourced Traffic Data: Predicting Air Pollution and Ischemic Stroke

The purpose of this grant is to investigate the role of traffic related air pollution on the risk of ischemic stroke.

Role: Principal Investigator

R03 NS101417 (PI: Boehme) 09/01/2017-08/31/2020 NIH/NINDS

Health Disparities in Sepsis as a Risk Factor for Stroke

The goal of this cohort study is to investigate the role of racial and geographic disparities on the relationship between sepsis and stroke.

Role: Principal Investigator

Sergievsy Center Pilot Award (PI: Boehme) 01/01/2021-12/31/2021 Internal
Biomarkers of Brain Injury and Associated Neurocognitive Dysfunction in Children Treated for Sickle
Cell Anemia in NY and Uganda (pilot)
Role: Principal Investigator

Interdisciplinary Pilot Award (PI: Boehme) 05/01/2018-04/30/2019 Internal
Air Pollution, Oxidative Stress, and Cognitive Decline in a Multiethnic Cohort
The goal of this internal pilot award is to study the relationship between air pollution, oxidative stress
and cognitive decline in a multiethnic cohort and to foster interdisciplinary research within Mailman
School of Public Health.
Role: Principal Investigator

Collaborative and Multidisciplinary Pilot Research Award Internal Application Phase 1 (PI:
Boehme) 01/01/2019-04/01/2019
The Role of the Gut Microbiome in Stroke in the Young
Role: Principal Investigator

Calderone Junior Faculty Pilot Award (PI: Boehme) 01/01/2018-12/31/2018 Internal
Primary Care Utilization, Influenza Vaccination, and Stroke Risk in the Young
The goal of this internal pilot award is to develop the pilot data for an R01 submission on the primary
care utilization patterns and influenza vaccination patterns of people aged 18-45 prior to a stroke event
using administrative data.
Role: Principal Investigator

Michael J. Fox Foundation (PI: Merchant) 01/01/2019-12/31/2019 Foundation Grant
BMP Markers and their role in Parkinson's Disease development and progression
Role: Co-Investigator

Michael J. Fox Foundation (PI: Kang) 05/01/2018-10/31/2018 Foundation Grant
Seeding Assay Data Analysis of Biofind Samples
Role: Statistician

Internal Funding (PI: Choi) 10/1/2017-06/01/2018
Psychosocial Outcomes in Epilepsy Patients in CHS
Role: Statistician

P30 ES009089 (PI: Baccarelli) 06/01/1998-03/31/2018 NIH/NIEHS
Center for Environmental Health in Northern Manhattan
Role: Co-Investigator

Institutional Collaborative and Multidisciplinary Pilot Award 01/2017-06/2017
Principal Investigator: Sylvie Goldman, PhD
A Dyadic Caregiver-Child Exercise Intervention in Underserved Families at High Risk for Autism
Spectrum Disorders
Role: Co-PI

T32 NS007153 31 (PI: Elkind/Louis) 07/01/2014-11/1/2015 NIH/NINDS
Neuroepidemiology Training Program
Role: Post-doctoral fellow

Internal Pilot Grant (PI: Martin-Schild) 03/01/2013-12/31/2014 Tulane School of Medicine
Factor VIII in Acute Cerebral Ischemia
Role: Co-PI

Pre-doctoral Fellowship (PI: Boehme) 01/2012-12/31/2014 American Heart Association
Predictors of Thromboembolism and Hemorrhage in patients with Ventricular Assist Devices
Principal Investigator: Amelia K. Boehme

TEACHING EXPERIENCE:

Fall 2024	Course Instructor, Advanced Epidemiologic Methods I: Evaluation of Epidemiologic Studies, Department of Global Health, New York University
Fall 2024	Course Instructor, Epidemiology III, Department of Epidemiology, Columbia University
Spring 2024	Course Instructor, Applied Methods in Epidemiologic Research, Department of Epidemiology, Columbia University
Spring 2024	Course Instructor, Advanced Epidemiologic Methods II: Advanced Epidemiologic Applications, Department of Global Health, New York University
Spring 2024	Course Instructor, Epidemiology of Non-Communicable Diseases, Department of Global Health, New York University
Fall 2023	Course Instructor, Epidemiology III, Department of Epidemiology, Columbia University
Fall 2023	Course Instructor, Undergraduate Epidemiology, Department of Global Health, New York University
Spring 2023	Course Instructor, Applied Methods in Epidemiologic Research, Department of Epidemiology, Columbia University
Spring 2023	Course Instructor, Advanced Epidemiologic Methods I: Evaluation of Epidemiologic Studies, Department of Global Health, New York University
Spring 2023	Course Instructor, Epidemiology of Non-Communicable Diseases, Department of Global Health, New York University
Fall 2022	Course Instructor, Epidemiology I, Department of Global Health, New York University
Spring 2022	Course Instructor, Epidemiology of Non-Communicable Diseases, Department of Global Health, New York University
Spring 2022	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Spring 2022	Course Instructor, Applied Methods in Epidemiologic Research, Department of Epidemiology, Columbia University
Fall 2021	Course Instructor, Epidemiology I, Department of Global Health, New York University
Fall 2021	Course Instructor, Undergraduate Epidemiology, Department of Global Health, New York University
Spring 2021	Course Instructor, Undergraduate Epidemiology, Department of Global Health, New York University
Spring 2021	Course Instructor, Epidemiology II, Department of Global Health, New York University
Spring 2021	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Spring 2021	Course Instructor, Applied Methods in Epidemiologic Research, Department of Epidemiology, Columbia University
Fall 2020	Course Instructor, Epidemiology I, Department of Global Health, New York University
Fall 2020	Course Instructor, Epidemiology III- Design and Conduct of Epidemiologic Studies, Department of Global Health, New York University
Spring 2020	Course Instructor, Epidemiology II, Department of Global Health, New York University

Spring 2020	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Spring 2020	Course Instructor, Applied Methods in Epidemiologic Research, Department of Epidemiology, Columbia University
Fall 2019	Course Instructor, Epidemiology I, Department of Global Health, New York University
Summer 2019	Course Instructor, Epidemiology I, Department of Global Health, New York University
Spring 2019	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Spring 2019	Course Instructor, Epidemiology II, Department of Global Health, New York University
Fall 2018	Course Instructor, Epidemiology I, Department of Global Health, New York University
Summer 2018	Course Instructor, Epidemiology I, Department of Global Health, New York University
Spring 2018	Course Instructor, Epidemiology II, Department of Global Health, New York University
Spring 2018	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Fall 2017	Course Instructor, Introduction to Epidemiology, Department of Global Health, New York University
Spring 2017	Co-Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Summer 2015	Co-Course Instructor, Multilevel Modeling, EPIC Summer Program, Columbia University
Summer 2015	Co-Course Instructor, Multiple Imputation Techniques, EPIC Summer Program, Columbia University
Spring 2015	Co- Course Instructor, Neuroepidemiology, Department of Epidemiology, Columbia University
Summer 2014	Course Instructor, Multilevel Modeling, EPIC Summer Program, Columbia University
Fall 2012	Graduate Teaching Assistant, Introduction to Epidemiology
Spring 2012	Graduate Teaching Assistant, Epidemiologic Design and Analysis
Fall 2011	Graduate Teaching Assistant, Vaccinology
Spring 2006	Undergraduate Teaching Assistant, Organic Chemistry II
Fall 2005	Undergraduate Teaching Assistant, Organic Chemistry I
Summer 2004	Undergraduate Teaching Assistant, Microbiology I

INTERNATIONAL INVITED LECTURES:

2023	Infection as a Risk Factor for Stroke, Invited Symposium, World Stroke Congress, Toronto, Canada, October 2023
2022	Non-Sickle Cell Stroke in Pediatric Populations, Invited Symposium, Makerere University, April 2022

NATIONAL INVITED LECTURES:

2023	Health Outcomes Research in Stroke, Invited Symposium, International Stroke Conference, February 2023
2022	Infections and Inflammation in Post-Stroke Recovery, Department of Neurosurgery Grand Rounds, University of Alabama at Birmingham, October 2022

2022	Short Term Triggers for Stroke and other Major Cardiovascular Events, Department of Epidemiology Seminar, School of Public Health, University of Alabama at Birmingham, October 2022
2021	Epidemiology of Stroke, Invited Symposium, American Academy of Neurology Conference, April 2021
2021	Stroke Risk in Women, Invited Symposium, American Academy of Neurology Conference, April 2021
2020	Infection as a Stroke Trigger, Department of Neurology Grand Rounds, Boston University, October 2020
2020	The Role of Infections and Inflammation in Post-Stroke Recovery, Department of Neurosurgery Grand Rounds, University of Alabama at Birmingham, April 2020*
2020	Short Term Triggers for Major Cardiovascular Events, Department of Epidemiology Seminar, School of Public Health, University of Alabama at Birmingham, April 2020*
2019	Stroke Risk Factors and Triggers: The role of Infection and Inflammation, Department of Epidemiology Seminar, School of Public Health, Michigan State University, November 2019
2019	Infection and Inflammation and Stroke, Department of Epidemiology Seminar, School of Public Health, Yale University, September 2019
2019	Risk of Stroke in the Peri-menopausal Period, Invited Symposium, International Stroke Conference, Honolulu, HI February 2019
2018	Epidemiology of Cerebrovascular Disease, Invited Symposium, American Academy of Neurology Conference, April 2018, Los Angeles, CA
2018	The Infection Stroke Connection, Global Public Health Seminar, SUNY Upstate, January 2018, Syracuse, NY
2017	Health Disparities and Sex Differences in Stroke, Invited Symposium, AAN Conference, April 2017, Boston, MA

*canceled due to the COVID-19 pandemic

LOCAL INVITED LECTURES:

2021	Novel risk factors for neurologic outcomes, Department of Neurology Meet a Mentor Series, Columbia University, July 2021
2020	Infection as a Stroke Trigger in the Young, Department of Epidemiology Doctoral Seminar, Columbia University, December 2020
2020	The combined role of infection and inflammation on stroke risk, Department of Neurology Seminar, Columbia University, December 2020

2020 Infection and Inflammation as risk factors for neurologic outcomes, Department of Neurology Meet a Mentor Series, Columbia University, July 2020

2019 Stroke in the Young: Inflammation and Infections, Department of Psychology, Neuropsychology Seminar, Columbia University October 2019

2018 Novel Risk Factors for Stroke, Neurology Research Retreat, Department of Neurology, Columbia University, New York, NY

2018 Risk of Stroke in Pregnancy, Chronic Disease Epidemiology Seminar, Department of Epidemiology, Columbia University, New York, NY

2017 Systemic Inflammatory Response Syndrome and Stroke, Neurology Research Retreat, Department of Neurology, Columbia University, New York, NY

2017 Risk of Stroke after Sepsis, Chronic Disease Epidemiology Seminar, Department of Epidemiology, Columbia University, New York, NY

2017 The Relationship Between Infections and Stroke, Psychiatric and Neuroepidemiology Seminar, Department of Epidemiology, Columbia University, New York, NY

2015 Systemic Inflammatory Response Syndrome and Stroke Outcomes, Department of Epidemiology, Columbia University, New York, NY

INVITED TEACHING LECTURES:

2023 Multiple Imputation Techniques, Doctoral Seminar, Department of Epidemiology, New York University, New York, NY

2023 Propensity Score Analysis, Doctoral Seminar, Department of Epidemiology, New York University, New York, NY

2022 Administrative Claims Data for Clinical Research, Neuroepidemiology Seminar, Department of Neurology, Columbia University, New York, NY

2022 Propensity Score Analysis, Neuroepidemiology Seminar, Department of Neurology, Columbia University, New York, NY

2019 Multiple Imputation Techniques, Neuropsychology Seminar, Department of Neurology, Columbia University, New York, NY

2018 Longitudinal Data Analysis, Master's Thesis Seminar, Department of Epidemiology, Columbia University, New York, NY

2017 Introduction to Epidemiology and Biostatistics V, Neurology Resident Seminar, Department of Neurology, Columbia University, New York, NY

2017 Introduction to Epidemiology and Biostatistics IV, Neurology Resident Seminar, Department of Neurology, Columbia University, New York, NY

2017 Introduction to Epidemiology and Biostatistics III, Neurology Resident Seminar, Department of Neurology, Columbia University, New York, NY

- 2017 Introduction to Epidemiology and Biostatistics II, Neurology Resident Seminar, Department of Neurology, Columbia University, New York, NY
- 2017 Introduction to Epidemiology and Biostatistics I, Neurology Resident Seminar, Department of Neurology, Columbia University, New York, NY
- 2016 Longitudinal Data Analysis, Master's Thesis Seminar, Department of Epidemiology, Columbia University, New York, NY
- 2016 How to Identify a Thesis Topic and Utilize Available Data to Answer a Question, Master's Thesis Seminar, Department of Epidemiology, Columbia University, New York, NY
- 2015 Multiple Imputation Techniques, Advanced Epidemiologic Methods, Department of Epidemiology, Columbia University, New York, NY
- 2013 Introduction to Survival Analysis, Statistical Methods II, Department of Biostatistics, University of Alabama at Birmingham, Birmingham, AL
- 2012 How to Critically Read and Evaluate a Manuscript, Department of Neurology, University of Alabama at Birmingham, Birmingham, AL
- 2012 A Brief Introduction to Multinomial and Cumulative Logit Models, Department of Sociology, University of Alabama at Birmingham, Birmingham, AL

COMMUNITY LECTURES

- 2021 Epidemiology of COVID-19, Avis Town Hall, January 2021, New York, NY
- 2020 COVID-19, Instagram Live Question and Answer Session with Suiheart Club, May 2020, New York, NY
- 2020 COVID-19, Instagram Live Question and Answer Session with Influencer Stephanie Gotlieb, May 2020, New York, NY
- 2020 COVID-19, Instagram Live Question and Answer Session with Dr. Rita Linkner, May 2020, New York, NY
- 2020 COVID-19 and Kids, Central Synagogue, April 2020, New York, NY
- 2020 COVID-19 Community Update, Central Synagogue, April 2020, New York, NY
- 2020 Epidemiology of COVID-19, Manhattan Community Health Seminar, May 2020, New York, NY
- 2020 Epidemiology of COVID-19, Manhattan Community Health Seminar, March 2020, New York, NY
- 2020 COVID-19 Scientific Update, Community Health Seminar, March 2020, New York, NY

MENTORSHIP:

Early Career Faculty

David Roh, Assistant Professor, Neurocritical Care, Department of Neurology, Columbia University
Jonathon Overdevest, Assistant Professor, ENT, Columbia University
Andrew Geneslaw, Assistant Professor, Critical Care, Department of Pediatrics, Columbia University
Erin Kulick, Assistant Professor, Department of Epidemiology, Temple University

Post-Doctoral Students

Murad Murkhurji PhD, Columbia University
Vanessa Guzman PhD, Columbia University
Soohyun Kim PhD, Columbia University

Doctoral Students

Erin Kulick, 2018 Columbia University
Danielle Crooks, 2019 Columbia University
Jorge Luna, 2020 Columbia University
Sebastian Rowland, 2020 Columbia University
Brandi Vollmer, 2022 Columbia University
Jenni Shearston, 2022 Columbia University

MPH Students:

2016

Morgan Moy, MPH 2016 Columbia University
Rebecca Hazan, MPH 2016 University of Michigan

2017

Iris Shao, MPH 2017 University of Michigan
Reese Sy, MPH 2017 Columbia University
Josephine Sarpong, MPH 2017 Columbia University
Omotooke Babalola, MPH 2017 Columbia University
Marisa Gallo, MPH 2017 Columbia University
Ashley Rodriguez, MPH 2017 Columbia University
Yi Zheng, MPH 2017 Columbia University

2018

Sonal Rastogi, MPH 2018 Columbia University
Michelle Canning, MPH 2018 Columbia University
Trevor Alvord, MPH 2018 Columbia University
Bijan Khaksari, MPH 2018 Columbia University
Yaqian (Heather) Xu, MPH 2018 Columbia University
Rita Pan, MPH 2018 Columbia University
Pooja Patel, MPH 2018 Columbia University
Adi Bothra, MPH 2018 Columbia University
Meghana Shamsunder, MPH 2018 Columbia University
Maitreyi Otai, MPH 2018 Columbia University

2019

Rebecca Passman, MPH 2019 Columbia University
Cody Young, MPH 2019 Columbia University
Elliott Rosen, MPH 2019 Columbia University
Eric Morris, MPH 2019 Columbia University

2020

Jacob Albers, MS 2020 Columbia University
Kyril Cole, MPH 2020 Columbia University
Anja Collazo, MPH 2020 Berlin University
Ekta Chaudhary, MPH 2020 Columbia University

David DeStephano, MPH 2020 Columbia University
Xing Chen, MPH 2020 Columbia University
Nicole Battaglia, MPH 2020 Columbia University
Serina Deeba, MPH 2020 Columbia University

2021

Yan Song, MPH 2021 Columbia University
Cameron Tait-Ozer, MPH 2021 Columbia University
Bree Martin, MS in Genetic Counseling 2021 Columbia University
Mary C. Thoma, MPH 2021 Columbia University

2022

Michael Toledano, MPH 2022 Columbia University

Undergraduate and High School Students:

2018-present

Hunter Jamison, BA 2020 New York University
Lucy Adjero, BA 2020 New York University
Rayan Mamoon, 2019 High School
Julia Soloway, BA 2022 Smith College

EDUCATION INITIATIVES:

NeuroCORPS Research Training Initiative

- Goal is to provide graduate students, medical students, residents, fellows and junior faculty the opportunity to conduct population outcomes in a collaborative environment

INVITED EDITORIAL AND NEUROLOGY INVITED BLOGS

1. **Boehme, AK.** Stroke in ethnic minority-serving US hospitals and Lower Carotid Revascularization Rates. *Neurology Blog*. 2019 June.
2. **Boehme, AK.** Prophylactic antibiotic use in stroke patients. *Neurology Blog*. 2018 May.
3. **Boehme, AK.** Smoking Cessation and Secondary Stroke Prevention. *Neurology*. 2017 Oct 17;89(16):1656-1657. PMID: 28887376.

PUBLISHED MANUSCRIPTS:

1. Davis, TJ; Salazar, R; Beenders, S; **Boehme, A**; LaMarca, NM; Bain, JM. A Prospective, Longitudinal Study of Caregiver-Reported Adaptive Skills and Function of Individuals with *HNRNP2*-related Neurodevelopmental Disorder. *Adv Neurodev Disord*. 2024;8(3):445-456. Epub 2023 Aug 7. PMID: 39220267.
2. Chen, MT; Vollmer, BL; Blyler, CA; Cameron, NA; Miller, EC; Huang, Y; Friedman, AM; Wright, JD; **Boehme, AK**; Bello, NA. Antihypertensive Medication Prescription Dispensation Among Pregnant Women in the United States: A Cohort Study. *Am Heart J*. 2024 Aug 21:S0002-8703(24)00205-9. Epub ahead of print. PMID: 39178979.
3. Packard, SE; Verzani, Z; Finsaas, MC; Levy, NS; Shefner, R; Planey, AM; **Boehme, AK**; Prins, SJ. Maintaining disorder: estimating the association between policing and psychiatric hospitalization among youth in New York City by neighborhood racial composition, 2006-2014. *Soc Psychiatry Psychiatr Epidemiol*. 2024 Aug 1. Epub ahead of print. PMID: 39088094.

4. Ibeh, C; Kulick, ER; **Boehme, AK**; Friedman, AM; Miller, EC; Bello, NA. Incident Stroke in Individuals with Peripartum Cardiomyopathy. *Am Heart J*. 2024 Jun 20:S0002-8703(24)00160-1. doi: 10.1016/j.ahj.2024.06.006. Epub ahead of print. PMID: 38908422.
5. Hulstaert, L; **Boehme, A**; Hood, K; Hayden, J; Jackson, C; Toyip, A; Verstraete, H; Mao, Y; Sarsour, K. Assessing ascertainment bias in atrial fibrillation across US minority groups. *PLoS One*. 2024 Apr 16;19(4):e0301991. PMID: 38626094.
6. Martin, BE; Sands, T; Bier, L; Bergner, A; **Boehme, AK**; Lippa, N. Comparing the frequency of variants of uncertain significance (VUS) between ancestry groups in a paediatric epilepsy cohort. *J Med Genet*. 2024 Mar 7:jmg-2023-109450. Epub ahead of print. PMID: 38453479.
7. Leavitt, VM; Tozlu, C; Nelson, KE; **Boehme, AK**; Donnelly, JE; Aguerre, I; Spinner, M; Riley, CS; Stein, J; Onomichi, K. A randomized controlled trial of oral antipyretic treatment to reduce overheating during exercise in adults with multiple sclerosis. *J Neurol*. 2024 Feb 28. Epub ahead of print. PMID: 38413464.
8. Albers, J; Bagos-Estevez, A; Snyder, LG; Tsalatsanis, A; **Boehme, A**; Bain, JM. Gastrointestinal symptoms have a non-temporal association with regression in a cohort with autism spectrum disorder using the simons simplex collection. *Res Autism Spectr Disord*. 2024;111:102326. doi:https://doi.org/10.1016/j.rasd.2024.102326
9. Martin SS, Aday AW, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, Baker-Smith CM, Barone Gibbs B, Beaton AZ, **Boehme AK**, Commodore-Mensah Y, Currie ME, Elkind MSV, Evenson KR, Generoso G, Heard DG, Hiremath S, Johansen MC, Kalani R, Kazi DS, Ko D, Liu J, Magnani JW, Michos ED, Mussolino ME, Navaneethan SD, Parikh NI, Perman SM, Poudel R, Rezk-Hanna M, Roth GA, Shah NS, St-Onge MP, Thacker EL, Tsao CW, Urbut SM, Van Spall HGC, Voeks JH, Wang NY, Wong ND, Wong SS, Yaffe K, Palaniappan LP; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. 2024 Heart Disease and Stroke Statistics: A Report of US and Global Data From the American Heart Association. *Circulation*. 2024 Jan 24. PMID: 38264914.
10. Mboizi V, Nabaggala C, Munube D, Ssenkusu JM, Kasirye P, Kanya S, Kawooya MG, **Boehme A**, Minja F, Mupere E, Opoka R, Rosano C, Green NS, Idro R. Hydroxyurea Therapy for Neurological and Cognitive Protection in Pediatric Sickle Cell Anemia in Uganda (BRAIN SAFE II): Protocol for a single-arm open label trial. *medRxiv [Preprint]*. 2024 Jan 13:2024.01.12.24301208. PMID: 38260320.
11. Bangirana, P; **Boehme, AK**; Birabwa, A; Opoka, RO; Munube, D; Mupere, E; Kasirye, P; Ru, G; Idro, R; Green, NS. Neurocognitive Impairment in Ugandan Children with Sickle Cell Disease Compared to Sibling Controls: A cross-sectional study. *Front Stroke*. 2024;3:1372949. doi: 10.3389/fstro.2024.1372949. Epub 2024 Apr 15. PMID: 38903696; PMCID: PMC11188974.
12. Cole, KL; **Boehme, AK**; Thacker, EL; Longstreth, WT Jr; Brown, BL; Gale, SD; Hedges, DW; Anderson, JK; Elkind, MSV. Hospital-Acquired Infection at Time of Stroke and Cognitive Decline: The Cardiovascular Health Study. *Cerebrovasc Dis*. 2023 Oct 23. Epub ahead of print. PMID: 37871579.
13. Waziry, R; Gu, Y; **Boehme, AK**; Williams, OA. Measures of Aging Biology in Saliva and Blood as Novel Biomarkers for Stroke and Heart Disease in Older Adults. *Neurology*. 2023 Oct 17:10. PMID: 37848333.

14. Green, NS; Rosano, C; Bangirana, P; Opoka, R; Munube, D; Kasirye, P; Kawooya, M; Lubowa, SK; Mupere, E; Conroy, A; Minja, FJ; **Boehme, AK**; Kang, MS; Honig, LS; Idro R. Neurofilament light chain: A potential biomarker for cerebrovascular disease in children with sickle cell anaemia. *Br J Haematol*. 2023 Aug 15. doi: 10.1111/bjh.19036. Epub ahead of print. PMID: 37581299.
15. Parks, RM; Rowland, ST; Do, V; **Boehme, AK**; Dominici, F; Hart, CL; Kioumourtzoglou, MA. The association between temperature and alcohol- and substance-related disorder hospital visits in New York State. *Commun Med (Lond)*. 2023 Sep 26;3(1): PMID: 37752306.
16. Davis, TJ; Salazar, R; Beenders, S; **Boehme, A**; LaMarca, NM; Bain, JM. A Prospective, Longitudinal Study of Caregiver-Reported Adaptive Skills and Function of Individuals with HNRNPH2-related Neurodevelopmental Disorder. *Advances in Neurodevelopmental Disorders*. Epub 2023.
17. Shinn, G; Berger, K; Roh, D; Doyle, K; **Boehme, AK**; Connolly, ES; Park, S; Agarwal, S; Claassen, J; Der-Nigoghossian, C. Concordance Between Active Partial Thromboplastin Time and Anti-Factor Xa Assays in Neurocritically Ill Patients Receiving Subcutaneous Heparin Prophylaxis. *Neurohospitalist*. 2023 Jul;13(3):221-227. Epub 2023 Apr 23. PMID: 37441213.
18. Carvalho Poyraz, F; **Boehme, A**; Cottarelli, A; Eisler, L; Elkind, MSV; Ghoshal, S; Agarwal, S; Park, S; Claassen, J; Connolly, ES; Hod, EA; Roh, DJ. Red Blood Cell Transfusions Are Not Associated With Incident Complications or Poor Outcomes in Patients With Intracerebral Hemorrhage. *J Am Heart Assoc*. 2023 Jun 6;12(11):e028816. PMID: 37232240.
19. Gurel, K; Khasiyev, F; Spagnolo-Allende, A; Rahman, S; Liu, M; Kulick, ER; **Boehme, A**; Rundek, T; Elkind, M; Marshall, RS; Bos, D; Gutierrez, J. The role of intracranial artery calcification (IAC) in stroke subtype and risk of vascular events. *J Stroke Cerebrovasc Dis*. 2023 May 13;32(8):107185. Epub ahead of print. PMID: 37186970.
20. Tao, RH; Chillrud, LG; Nunez, Y; Rowland, ST; **Boehme, AK**; Yan, J; Goldsmith, J; Wright, J; Kioumourtzoglou, MA. Applying principal component pursuit to investigate the association between source-specific fine particulate matter and myocardial infarction hospitalizations in New York City. *Environ Epidemiol*. 2023 Feb 15;7(2):e243. PMID: 37064426.
21. Roh, DJ; **Boehme, A**; Mamoon, R; Hooper, D; Cottarelli, A; Ji, R; Mao, E; Kumar, A; Carvalho Poyraz, F; Demel, SL; Spektor, V; Carmona, J; Hod, EA; Ironside, N; Gutierrez, J; Guo, J; Konofagou, E; Elkind, MSV; Woo, D. Relationships of Hemoglobin Concentration, Ischemic Lesions, and Clinical Outcomes in Patients With Intracerebral Hemorrhage. *Stroke*. 2023 Feb 13. Epub ahead of print. PMID: 36779340.
22. Tsao, CW; Aday, AW; Almarzooq, ZI; Anderson, CAM; Arora, P; Avery, CL; Baker-Smith, CM; Beaton, AZ; **Boehme, AK**; Buxton, AE; Commodore-Mensah, Y; Elkind, MSV; Evenson, KR; Eze-Nliam, C; Fugar, S; Generoso, G; Heard, DG; Hiremath, S; Ho, JE; Kalani, R; Kazi, DS; Ko, D; Levine, DA; Liu, J; Ma, J; Magnani, JW; Michos, ED; Mussolino, ME; Navaneethan, SD; Parikh, NI; Poudel, R; Rezk-Hanna, M; Roth, GA; Shah, NS; St-Onge, MP; Thacker, EL; Virani, SS; Voeks, JH; Wang, NY; Wong, ND; Wong, SS; Yaffe, K; Martin, SS; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2023 Update: A Report From the American Heart Association. *Circulation*. 2023 Jan 25. Epub ahead of print. PMID: 36695182.
23. Roh, DJ; Chang, TR; Kumar, A; Burke, D; Torres, G; Xu, K; Yang, W; Cottarelli, A; Moore, E; Sauaia, A; Hansen, K; Velazquez, A; **Boehme, A**; Vrosgou, A; Ghoshal, S; Park, S; Agarwal, S;

- Claassen, J; Connolly, ES; Wagener, G; Francis, RO; Hod, E. Hemoglobin Concentration Impacts Viscoelastic Hemostatic Assays in ICU Admitted Patients. *Crit Care Med.* 2023 Feb 1;51(2):267-278. Epub 2023 Nov 3. PMID: 36661453.
24. Choi, H; Elkind, MSV; Longstreth, WT Jr; **Boehme, A**; Hafen, R; Hoyt, EJ; Thacker, EL. Epilepsy, Vascular Risk Factors, and Cognitive Decline in Older Adults: The Cardiovascular Health Study. *Neurology.* 2022 Sep 2. Epub ahead of print. PMID: 36240101.
 25. Harris, J; **Boehme, AK**; Chan, L; Moats, H; Dugue, R; Izeogu, C; Pavol, MA; Naqvi, I; Williams, O; Marshall, RS. Allostatic Load Predicts Racial Disparities in Intracerebral Hemorrhage Cognitive Outcomes. *Sci Rep.* 2022 Oct 3;12(1):16556. PMID: 36192526
 26. Vollmer, BL; Solowey, J; Chen, X; Chang, BP; Williams, O; Kulick, ER; Elkind, MSV; **Boehme, AK**. Individual and joint effects of influenza-like illness and vaccinations on stroke in the young: A case-control study. *Stroke.* Epub ahead of print. PMID: 35861760.
 27. Egbebike, J; Shen, Q; Doyle, K; Der-Nigoghossian, CA; Panicker, L; Gonzales, IJ; Grobois, L; Carmona, JC; Vrosgou, A; Kaur, A; **Boehme, A**; Velazquez, A; Rohaut, B; Roh, D; Agarwal, S; Park, S; Connolly, ES; Claassen, J. Cognitive-motor dissociation and time to functional recovery in patients with acute brain injury in the USA: a prospective observational cohort study. *Lancet Neurol.* 2022 Aug;21(8):704-713. PMID: 35841909.
 28. Lao PJ, **Boehme AK**, Morales C, Laing KK, Chesebro A, Igwe K, Gutierrez J, Gu Y, Stern Y, Schupf N, Manly JJ, Mayeux R, Brickman AM. Amyloid, cerebrovascular disease, and neurodegeneration biomarkers are associated with cognitive trajectories in a racially and ethnically diverse, community-based sample. In Press. *Neurobiology of Aging.*
 29. Thakur, KT; Chu, V; Hughes, C; Kim, CY; Fleck-Derderian, S; Barrett, CE; Matthews, E; Balbi, A; Bilski, A; Chomba, M; Lieberman, O; Jacobson, SD; Agarwal, S; Roh, D; Park, S; Ssonko, V; Silver, W; Vargas, WD; Geneslaw, A; Bell, M; Waters, B; Rao, A; Claassen, J; **Boehme, A**; Willey, JZ; Elkind, MS; Sobieszczyk, M; Zucker, J; McCollum, A; Sejvar, J. Risk factors for New Neurological Diagnoses in Hospitalized COVID-19 Patients: A Case-Control Study in New York City. *Neurology: Clinical Practice.* Accepted.
 30. Nunez, Y; **Boehme, AK**; Goldsmith, J; Li, M; van Donkelaar, A; Weisskopf, MG; Re, DB; Martin, RV; Kioumourtoglou, MA. PM_{2.5} composition and disease aggravation in amyotrophic lateral sclerosis: An analysis of long-term exposure to components of fine particulate matter in New York State. *Environ Epidemiol.* 2022 Mar 30;6(2):e204. PMID: 35434459
 31. Waldrop, G; Safavynia, SA; Barra, ME; Agarwal, S; Berlin, DA; **Boehme, AK**; Brodie, D; Choi, JM; Doyle, K; Fins, JJ; Ganglberger, W; Hoffman, K; Mittel, AM; Roh, D; Mukerji, SS; Der Nigoghossian, C; Park, S; Schenck, EJ; Salazar-Schicchi, J; Shen, Q; Sholle, E; Velazquez, AG; Walline, MC; Westover, MB; Brown, EN; Victor, J; Edlow, BL; Schiff, ND; Claassen, J. Prolonged unconsciousness is common in COVID-19 and associated with hypoxemia. *Ann Neurol.* 2022 Mar 7. Epub ahead of print. PMID: 35254675.
 32. Idro, R; **Boehme, AK***; Kawooya, M; Lubowa, SK; Munube, D; Bangirana, P; Opoka, R; Mupere, E; Lignelli, A; Kasirye, P; Green, NS; Minja, FJ. Brain Magnetic Resonance Imaging and Angiography in Children with Sickle Cell Anaemia in Uganda in a Cross-Sectional Sample. *J Stroke Cerebrovasc Dis.* 2022 Feb 11;31(4):106343. Epub ahead of print. PMID: 35158150.

*corresponding author

33. Tsao, CW; Aday, AW; Almarzooq, ZI; Alonso, A; Beaton, AZ; Bittencourt, MS; **Boehme, AK**; Buxton, AE; Carson, AP; Commodore-Mensah, Y; Elkind, MSV; Evenson, KR; Eze-Nliam, C; Ferguson, JF; Generoso, G; Ho, JE; Kalani, R; Khan, SS; Kissela, BM; Knutson, KL; Levine, DA; Lewis, TT; Liu, J; Loop, MS; Ma, J; Mussolino, ME; Navaneethan, SD; Perak, AM; Poudel, R; Rezk-Hanna, M; Roth, GA; Schroeder, EB; Shah, SH; Thacker, EL; VanWagner, LB; Virani, SS; Voecks, JH; Wang, NY; Yaffe, K; Martin, SS; American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart Disease and Stroke Statistics-2022 Update: A Report From the American Heart Association. *Circulation*. 2022 Jan 26: Epub ahead of print. PMID: 35078371.
34. Angevaere, MJ; Vonk, MJ; Bertola, L; Zahodne, L; Wei-Ming Watson, C; **Boehme, A**; Schupf, N; Mayeux, R; Geerlings, MI; Manly, JJ. Predictors of Incident Mild Cognitive Impairment and Its Course in a Diverse Community-Based Population. *Neurology*. 2021 Epub ahead of print. PMID: 34853178.
35. **Boehme, AK**; Oka, M; Cohen, B; Elkind, MSV; Larson, E; Mathema, B. Readmission Rates in Stroke Patients with and without Infections: Incidence and Risk Factors. *J Stroke Cerebrovasc Dis*. 2021 Nov 15;31(1):106172. Epub ahead of print. PMID: 34798436.
36. Hunter, MD; Kulick, ER; Miller, E; Willey, J; **Boehme, AK**; Branas, C; Elkind, MS. Rural-Urban Differences in Diagnosed Cervical Artery Dissection in New York State. *CVD*. Epub ahead of print 2021. PMID: 35034032.
37. Rowland, ST; Chillrud, LG; **Boehme, AK**; Wilson, A; Rush, J; Just, AC; Kioumourtzoglou, MA. Can weather help explain 'why now?': The potential role of hourly temperature as a stroke trigger. *Environ Res*. 2021 Oct 23;112229. Epub ahead of print. PMID: 34699760.
38. Thakur, KT; Epstein, S; Bilski, A; Balbi, A; **Boehme, AK**; Brannagan, TH; Wesley, SF; Riley, C. Neurologic Safety Monitoring of COVID-19 Vaccines: Lessons From the Past to Inform the Present. *Neurology*. 2021 Sep. Epub ahead of print. PMID: 34475124.
39. Esenwa, C; Cheng, NT; Luna, J; Willey, J; **Boehme, AK**; Kirchoff-Torres, K; Labovitz, D; Liberman, AL; Mabie, P; Moncrieffe, K; Soetanto, A; Lendaris, A; Seiden, J; Goldman, I; Altschul, D; Holland, R; Benton, J; Dardick, J; Fernandez-Torres, J; Flomenbaum, D; Lu, J; Malaviya, A; Patel, N; Toma, A; Lord, A; Ishida, K; Torres, J; Snyder, T; Frontera, J; Yaghi S. Biomarkers of Coagulation and Inflammation in COVID-19-Associated Ischemic Stroke. *Stroke*. 2021 Aug 25. Epub ahead of print. PMID: 34428931.
40. **Boehme, AK**; Doyle, K; Thakur, KT; Roh, D; Park, S; Agarwal, S; Velazquez, AG; Egbebike, JA; Der Nigoghossian, C; Prust, ML; Rosenberg, J; Brodie, D; Fishkoff, KN; Hochmann, BR; Rabani, LE; Yip, NH; Panzer, O; Claassen, J. Disorders of Consciousness in Hospitalized Patients with COVID-19: The Role of the Systemic Inflammatory Response Syndrome. *Neurocrit Care*. 2021 Jun 28;1–8. dEpub ahead of print. PMID: 34184176
41. Nunez, Y; **Boehme, AK**; Li, M; Goldsmith, J; Weisskopf, MG; Re, DB; Navas-Acien, A; van Donkelaar, A; Martin, RV; Kioumourtzoglou, MA. Parkinson's disease aggravation in association with fine particle components in New York State. *Environ Res*. 2021 Jun 25;201:111554. Epub ahead of print. PMID: 34181919.
42. Melmed, KR; Carroll, E; Lord, AS; **Boehme, AK**; Ishida, K; Zhang, C; Torres, JL; Yaghi, S; Czeisler, BM; Frontera, JA; Lewis, A. Systemic Inflammatory Response Syndrome is Associated with Hematoma Expansion in Intracerebral Hemorrhage. *J Stroke Cerebrovasc Dis*. 2021 May 30;30(8):105870. PMID: 34077823

43. Pavol, MA; **Boehme, AK**; Yuzefpolskaya, M; Maurer, MS; Casida, J; Festa, JR; Ibeh, C; Willey, JZ. Cognition predicts days alive out of hospital after LVAD implantation. *Int J Artif Organs*. 2021 May 20. ePub ahead of print. PMID: 34011184
44. Kulick, ER; Alvord, T; Canning, M; Elkind, MS; Chang, BP; **Boehme, AK**. Risk of Stroke and Myocardial Infarction after Influenza-Like-Illness In New York State. *BMC Public Health*. May 2021. ePub ahead of Print. PMID: 33952233.
45. Rowland, ST; Parks, RM; **Boehme, AK**; Goldsmith, J; Rush, J; Just, AC; Kioumourtoglou, MA. The association between ambient temperature variability and myocardial infarction in a New York-State-based case-crossover study: An examination of different variability metrics. *Environ. Res*. 2021 Apr 28; 197: 111207. PMID: 33932478.
46. Thakur, KT; Miller, EH; Glendinning, MD; Al-Dalahmah, O; Banu, MA; **Boehme, AK**; Boubour, AL; Bruce, SL; Chong, AM; Claassen, J; Faust, PL; Hargus, G; Hickman, R; Jambawalikar, S; Khandji, AG; Kim, CY; Klein, RS; Lignelli-Dipple, A; Lin, CC; Liu, Y; Miller, ML; Moonis, G; Nordvig, AS; Overdevest, JB; Prust, ML; Przedborski, S; Roth, WH; Soung, A; Tanji, K; Teich, AF; Agalliu, D; Uhlemann, AC; Goldman, JE; Canoll, P. COVID-19 Neuropathology at Columbia University Irving Medical Center/New York Presbyterian Hospital. *Brain*. Accepted March 2021. PMID: 33856027.
47. Weinberger, K; Kulick, ER; **Boehme, AK**; Shengzhi, S; Dominici, F; Wellenius, G. Association between Hurricane Sandy and emergency department visits in New York City by age and cause. *AJE*. Accepted March 2021. PMID: 33910231.
48. Magid-Bernstein, J; Beaman, CB; Carvalho Poyraz, F; **Boehme, A**; Hod, E; Francis, RO; Elkind, MS; Agarwal, S; Park, S; Claassen, J; Connolly, ES; Roh, D. Impacts of ABO Incompatible Platelet Transfusions on Platelet Recovery and Outcomes after Intracerebral Hemorrhage. *Blood*. 2021 Mar 1. Epub Ahead of Print. PMID: 33649761
49. Waters, BL; Michalek, AJ; Brigham, D; Thakur, KT; **Boehme, A**; Claassen, J; Bell, M. Incidence of Electrographic Seizures in Patients with COVID-19. *Frontiers of Neurology*. 2021 Feb 4; 12:614719. PMID: 33613431.
50. Megjhani, M; Terilli, K; Weiss, M; Savarraj, J; Chen, LH; Alkhachroum, A; Roh, DJ; Agarwal, S; Connolly, ES Jr; Velazquez, A; **Boehme, A**; Claassen, J; Choi, HA; Schubert, GA; Park, S. Dynamic Detection of Delayed Cerebral Ischemia: A Study in 3 Centers. *Stroke*. ePub ahead of print. PMID: 33596676.
51. Nunez, Y; **Boehme, AK**; Weisskopf, MG; Re, DB; Navas-Acien, A; van Donkelaar, A; Martin, RV; Kioumourtoglou, MA. Fine Particle Exposure and Clinical Aggravation in Neurodegenerative Diseases in New York State. *Environ Health Perspect*. 2021 Feb; 129 (2):27003. PMID: 33555200
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9. Vanguru, HR; Mamoon, R; Lopez, CY; Masoud, H; Albright, KC; **Boehme, AK**. Outcomes of Acute Basilar Occlusion Ischemic Strokes with advent of Modern Thrombectomy Devices, a New York State Experience. Poster Presentation, American Academy of Neurology Conference 2019. Philadelphia, PA.
10. Gatollari, H; **Boehme, AK**; Connolly, ES; Friedman, AM; Elkind, MS; Willey, JZ; Miller, EC. Maternal Morbidity outcome in idiopathic moyamoya syndrome in New York State. Oral Presentation, American Academy of Neurology Conference 2019. Philadelphia, PA.
11. Alvord, T; Kulick, ER; Canning, MC; Kioumourtzoglou, MA; Elkind, MS; **Boehme, AK**. Influenza-Like Illness and risk of Stroke in New York State. Oral Presentation, International Stroke Conference 2019. Honolulu, HI.
12. Hunter, MD; Moon, YP; Kulick, ER; **Boehme, AK**; Elkind, MS. Influenza like illness may trigger Cervical Artery Dissection. Moderated Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
13. Miller, EC; Medina, J; Friedman, AM; Elkind, MS; **Boehme, AK**. Infection during Delivery Hospitalization is Associated with increased Risk of Readmission for Post-partum stroke. Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
14. Gatollari, HJ; Miller, EC; **Boehme, AK**; Connolly, ES; Elkind, MS; Willey, JZ. Is there a Distinct Phenotype for Non-Asian Moyamoya Patients? Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
15. Zambrano, MD; Friedman, AM; **Boehme, AK**; Huang, Y; Miller, EC. Racial and Ethnic Disparities in Risk for Pregnancy related Stroke: The National Inpatient Sample. Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
16. **Boehme, AK**; Oka, M; Cohen, B; Mathema, B; Larson, E. Readmission for an Infection after a Stroke Admission: The Role of Hospital Acquired Infections. Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
17. Gatollari, HJ; **Boehme, AK**; Connolly, ES; Friedman, AM; Elkind, MS; Willey, JZ; Miller, EC. Maternal Morbidity Outcomes in Moyamoya Syndrome in New York State. Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
18. Samai, AA; **Boehme, AK**; Navalkele, D; Alemayehu, C; Martin-Schild, S. Factor VIII elevation Associated with Worse Functional Outcome Among Patients with Suspected Large Vessel Occlusion. Poster Presentation, International Stroke Conference 2019. Honolulu, HI.
19. Rowland, S; Just, A; **Boehme, AK**; Rush, J; Wong, S; Frye, B; Goldsmith, J; Kloog, I; Kioumourtzoglou, MA. The Role of Temperature Variability in Risk of Stroke. Poster Presentation, International Society for Environmental Epidemiology Conference. 2018. Ottawa, Canada.
20. Wajnsztajn, F; Kim, A; **Boehme, AK**; Maurer, M; Brannagan, T. Peripheral neuropathy in wild type transthyretin amyloidosis. Poster Presentation. American Academy of Neurology Conference 2018. Los Angeles, CA.

21. Canning M; Kulick, E; Parikh, N; Elkind, MS; **Boehme, AK**. Seasonality of Influenza and Stroke – Are the Seasonal Fluctuations Related? Poster Presentation, International Stroke Conference 2018. Los Angeles, CA.
22. Xu, Y; Boshen, J; Willey, J; Parikh, N; **Boehme, AK**; Elkind, MSE. How Far is Too Far? A Decision Analysis Model of Pre-hospital Triage for Potential Large Vessel Occlusion Acute Stroke Patients. Oral Presentation, International Stroke Conference 2018. Los Angeles, CA.
23. Khaksari, B; Kulick, E; Omran, S; Elkind, MS; **Boehme, AK**. Infections increase the risk of 30-day readmissions among stroke survivors. Poster Presentation, International Stroke Conference 2018. Los Angeles, CA.
24. Rastogi, S; Canning, M; Kulick, E; Omran, S; Willey, J; Elkind, MS; **Boehme, AK**. Infections Present on Admission, Hospital Acquired Infections and Short Term Outcomes in Ischemic Stroke Patients. Poster Presentation, International Stroke Conference 2018. Los Angeles, CA.
25. Kulick, E; **Boehme, AK**; Varela, D; Sacco, R; Elkind, MS; Effect of residential proximity to high traffic roadways and modification by smoking on incident ischemic stroke in the Northern Manhattan Study. Poster Presentation, Society For Epidemiological Research, 2017. Seattle, WA
26. Kulick, E; Pan, R; **Boehme, AK**. Geographic Disparities in Risk of Acute Ischemic Stroke after Hospitalization for Influenza Like Illness. Online Abstract, Society For Epidemiological Research, 2017. Seattle, WA
27. Baker, D; Kulick, E; **Boehme, AK**; Noble, J. Effects of the New York State Lystedt Law on Concussion-Related Emergency Healthcare Visits among Adolescents, 2005-2014. Poster Presentation, American Academy of Neurology, 2017. Boston, MA.
28. Khawaja, A; **Boehme, AK**; George, A; Hays, A; Kumar, G; Alvi, M; Venkatraman, A; Miller, D; Martin-Schild, S; Mirza, M; Harrigan, M. The role of a history of coronary artery disease, the need for transfusion and outcomes in patients with intracerebral hemorrhage. Poster Presentation, American Academy of Neurology, 2017. Boston, MA.
29. Gibson, E; Albright, KC; Gupta, S; **Boehme, AK**; Shapshak, A; Lyerly, M. Foley Catheter use and infection in non-intubated intracerebral hemorrhage patients. Poster Presentation, American Academy of Neurology, 2017. Boston, MA.
30. Sundheim, K; Miller, E; Willey, J; Marshall, R; Shao, Y; **Boehme, AK**. Infection is not associated with poor outcome in young adults with hemorrhagic stroke. Poster Presentation, American Academy of Neurology, 2017. Boston, MA.
31. Miller, E; Gatollari, H; Too, G; **Boehme, AK**; Leffert, L; Elkind, MS; Willey, J. Risk Factors for Pregnancy-associated stroke in women with preeclampsia- a case-control study. Oral Presentation, American Academy of Neurology, 2017. Boston, MA.
32. Pennington, A; **Boehme, AK**; Albright, KC; Singh, M; Lyerly, M; Gropen, T; Shapshak, A. Radiographic Predictors of Prognosis In Intracerebral Hemorrhage: “If It Ain’t Broke, Don’t Fix It”. Oral Presentation, American Academy of Neurology, 2017. Boston, MA.
33. Kulick, E; **Boehme, AK**; Varela, D; Sacco, R; Elkind, MS. Residential Proximity to High-Traffic Roadways and Incident Ischemic Stroke in Northern Manhattan. Oral Presentation, New York City Epidemiology Forum, January 2017. New York, NY.

34. Shao, Y; Kulick, E; Kamel, H; Elkind, MS; **Boehme, AK**. Risk Factors and Composite Risk Score for Predicting Stroke after Sepsis. Moderated Poster Presentation, International Stroke Conference, 2017. Houston, TX.
35. Navalkele DD, **Boehme A**, Albright KC, Leissinger C, El Khoury R, Freeman M, Schluter L, Martin-Schild S. Feb 2017. Poster presented at the International Stroke Conference, Houston, TX.
36. Pennington AR, **Boehme AK**, Albright KC, Singh M, Lyster MJ, Gropen TI, Hays Shapshak A. Radiographic Predictors of Prognosis in Intracerebral Hemorrhage: "If It Ain't Broke, Don't Fix It". Feb 2017. Poster presented at the International Stroke Conference, Houston, TX.
37. Lazar, R; **Boehme, AK**; Marshall, R; Martin-Schild, S. Aphasia is an independent risk factor affecting acute stroke outcomes. Poster Presentation, American Academy of Neurology, 2016. Vancouver, BC, Canada.
38. Kaur, M; **Boehme, AK**; Albright, KC; Sisson, A; Lyster, M, Gropen, T. HIAT2 Predicts poor functional outcome, palliative care involvement, and In-hospital mortality in tPA treated and untreated ischemic stroke patients. Poster Presentation, American Academy of Neurology, 2016. Vancouver, BC, Canada.
39. Gropen T, **Boehme AK**, Martin-Schild S, Albright KC, Perrin B, Samai A, Pishanidar S, Janjua N, Levine SR, Brandler ES, Rosenbaum D. Comparison of Large Vessel Occlusion Prediction Scores. April 2016. Oral presented at the American Academy of Neurology 68th Annual Meeting, Vancouver, BC, Canada.
40. Kaur M, **Boehme AK**, Albright KC, Lyster M, Sisson A, Arora K, Khawaja AM, Hays Shapshak A, Gropen T. HIAT2 Predicts Poor Functional Outcome, Palliative Care Involvement, and In-hospital Mortality in tPA Treated and Untreated Ischemic Stroke Patients. April 2016. Poster presented at the American Academy of Neurology 68th Annual Meeting, Vancouver, BC, Canada.
41. **Boehme, AK**; Hazan, R; Miller, E; Yaghi, S; Rostanski, S; Willey, J; Marshall, R; Elkind, MS. Infections present on admission and stroke in the young. Oral Presentation, American Academy of Neurology, 2016. Vancouver, BC, Canada.
42. **Boehme AK**, Hays AH, Kicieliński KP, Kapoor N, Albright KC, Shiue H, Miller DW, Elkind MS, Harrigan M. Systemic Inflammatory Response Syndrome during Hospitalization for Intracerebral Hemorrhage Drives Poor Functional Outcome at Discharge. Platform Presentation. American Academy of Neurology 2015. Washington D.C.
43. **Boehme AK**, Albright KC, Sisson A, Elkind MS, Harrigan M. Outcomes in ICH Patients Should be stratified by Age with Attention to Race and ICH Score. ePoster Presentation. American Academy of Neurology 2015. Washington D.C.
44. May TL, **Boehme AK**, Svoronos A, Patel P, B, Claassen J, Park S, Riker R, Seder DB, Agarwal S. Pre-arrest Patient Characteristics Explain Most Interhospital Variation in Outcomes after Cardiac Arrest. ePoster Presentation. American Academy of Neurology 2015. Washington D.C.
45. Agarwal S, May TL, **Boehme AK**, Reynolds AS, Patel P, B, Park S, Claassen J, Riker R, Seder D. Clinical and Demographic Factors Associated with Poor Outcome Differ from those

Associated with Withdrawal of Life Support after Cardiac Arrest. Poster Presentation. American Academy of Neurology 2015. Washington D.C.

46. Reynolds AS, May TL, **Boehme AK**, Patel P, B, Park S, Claassen J, Riker R, Seder D, Agarwal S. Premorbid Status Independently Dictates Good Outcome after Status Epilepticus among Comatose Survivors of Cardiac Arrest. Poster Presentation. American Academy of Neurology 2015. Washington D.C.
47. Shiue H, **Boehme AK**, Sands K, Martin-Schild S, Hays A, Lyster MJ, Gadpaille A, Khawaja A, Sisson A, Alvi M, George, A Harrigan M. Admission Hypomagnesemia Predicts Primary Intracerebral Hemorrhage Volume. American Academy of Neurology 2015. Washington D.C.
48. Khawaja A, **Boehme AK**, Lyster MJ, Predictors and outcome of mechanical ventilation for patients with primary intracerebral hemorrhage. American Academy of Neurology 2015. Washington D.C.
49. Samai AA, **Boehme AK**, George A, Dowell L, Schluter L, El Khoury R, Martin-Schild S, *Persistent Elevation of Factor VIII and Long-term Patient Outcomes in Ischemic Stroke*, Accepted for presentation American Academy of Neurology Annual Conference 2015, Washington, D.C.
50. Samai AA, **Boehme AK**, George A, Schluter L, El Khoury R, Martin-Schild S, *Factor VIII Level is Not Modifiable by Improved Glycemic Control in Patients with Ischemic Stroke*, Accepted for presentation American Academy of Neurology Annual Conference 2015, Washington, D.C.
51. Dowell L, **Boehme AK**, Samai AA, George A, Schluter L, El Khoury R, Martin-Schild S, *Racial Variation in Association of Elevated Factor VIII and Stroke Outcome*, Accepted for presentation American Academy of Neurology Annual Conference 2015, Washington, D.C.
52. Albright KC, **Boehme AK**, Howard VJ, Howard G, Judd S, Rhodes D, Anderson A, McClure L, Safford M, Limdi N, Blackburn J. Secondary Stroke Prevention Prescribing in a National Cohort. Secondary Stroke Prevention Prescribing in a National Cohort. American Academy of Neurology 2015. Washington D.C.
53. Sands KA, **Boehme AK**, Albright KC, Howard VJ, Howard G, Rhodes D, Safford M. Intravenous Tissue Plasminogen Activator Treatment for Acute Ischemic Stroke in the REGARDS Study. American Academy of Neurology 2015. Washington D.C.
54. Miller E, Yaghi S, **Boehme AK**, Marshall R, Willey JZ. Ischemic Stroke in Pregnancy and Postpartum: A Descriptive Case Series. American Academy of Neurology 2015. Washington D.C.
55. Miller E, Yaghi S, **Boehme AK**, Marshall R, Willey JZ. Mechanisms and Outcomes of Ischemic Stroke in Pregnancy and Postpartum: A Retrospective Cross-Sectional Study. American Academy of Neurology 2015. Washington D.C.
56. Yaghi S, Willey JZ, Andrews H, **Boehme AK**, Quarles L, Marshall RS, Boden-Albala B Cortical Deficits and Prior Stroke Predict Stroke Recurrence in Patients with Mild Deficits. American Academy of Neurology 2015. Washington D.C.
57. Yaghi S, Willey JZ, Andrews H, **Boehme AK**, Quarles L, Marshall RS, Boden-Albala B Itemized NIHSS subsets predict positive MRI strokes in patients with mild deficits. American Academy of Neurology 2015. Washington D.C.

58. Gadpaille A, **Boehme AK**, Albright KC, Hays A, Harrigan M. On what type of ICH patient do neurologists order EEG on? Poster Presentation. American Academy of Neurology 2015. Washington D.C.
59. Gadpaille A, Albright KC, Boehme AK, Alvi M, Sands K, Khawaja A, Shiue H, Harrigan M. Seizure as the presenting symptom of ICH patient characteristics and EEG utilization. Poster Presentation. American Academy of Neurology 2015. Washington D.C.
60. **Boehme AK**, Kicielinski KP, Kapoor N, Arora K, Gadpaille A, Shiue H, Miller DW, Elkind MS, Harrigan M. Systemic Inflammatory Response Syndrome during Hospitalization for Intracerebral Hemorrhage Drives Poor Functional Outcome at Discharge. Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
61. **Boehme AK**, Kicielinski KP, Shiue H, Kapoor N, Alvi M, Miller DW, Elkind MS, Harrigan M. A Simple Systemic Inflammatory Response Syndrome Prediction Score for Patients with Intracerebral Hemorrhage. Moderated Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
21. Khawaja A, **Boehme AK**, Kicielinski KP, Arora K, Lyerly MJ, Alvi M, Kumar G, Harrigan M. The Influence of Low Density Lipoprotein On Intracerebral Hemorrhage Volume. Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
22. Sarraj A, **Boehme AK**, Sitton CW, Supsupin EP, Datta P, Bajgur SS, Choi JM, Bonafante-Mejia EE, Friedman E, Denny MC, Wu T, Barreto AD, Dannenbaum MJ, Chen PR, Sun CJ, Gupta R, Martin-Schild S, Grotta JC, Savitz SI. Collaterals Predicts Imaging Evolution and Patient Outcomes in Acute Ischemic Stroke Patients. Moderated Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
23. Lord AS, Brown WM, Moomaw CJ, Langefeld CD Sekar P, James ML, Osborne J, **Boehme AK**, Woo D, Elkind MS. The Impact of Fever on Presentation in Intracerebral Hemorrhage: The Ethnic/Racial Variations of Intracerebral Hemorrhage (ERICH) Study. Moderated Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
24. Sarraj A, Sitton CW, **Boehme AK**, Supsupin EP, Datta P, Bajgur SS, Sun CJ, Jia JJ, Choi JM, Bonafante-Mejia EE, Friedman E, Dannenbaum MJ, Chen PR, Wu T, Barreto AD, Gupta R, Martin-Schild S, Grotta JC, Savitz SI. Intra-Arterial Therapy in Stroke Patients Transferred from Referral Centers. Oral Presentation. *International Stroke Conference* 2015. Nashville, TN.
25. Yaghi S, Willey JZ, Andrews H, **Boehme AK**, Quarles L, Marshall RS, Boden-Albala B. Cortical Deficits and Prior Stroke Predict Stroke Recurrence in Patients with Mild Deficits. Poster Presentation. *International Stroke Conference* 2015. Nashville, TN.
26. Sisson A, Albright KC, Peck M, Nguyen LM, Lyerly MJ, Sands KA, **Boehme AK**, Harrigan M. Palliative Care Is Underutilized in Patients with Severe Intracerebral Hemorrhage. Oral Presentation. *International Stroke Conference* 2015. Nashville, TN.
27. Yaghi S, Willey JZ, Andrews H, **Boehme AK**, Quarles L, Marshall RS, Boden-Albala B. Outcome in Patients with Minor Stroke: The Effect of Itemized NIHSS Score Subsets. Oral Presentation. *International Stroke Conference* 2015. Nashville, TN.

28. Hays A, **Boehme AK**, Kicielinski KP, Arora K, Kapoor N, Lyerly MJ, Miller DW, Elkind MS, Harrigan M. The Influence Of Systemic Inflammatory Response Syndrome, In The Absence Of Infection, On The Relationship Between Intracerebral Hemorrhage Score And Outcomes. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
29. Albright KC, **Boehme AK**, Howard VJ, Howard G, Judd S, Rhodes D, Anderson A, McClure L, Safford M, Limdi N, Blackburn J. Secondary Stroke Prevention Prescribing in a National Cohort. Moderated Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
30. Sarraj A, Martin-Schild S, **Boehme AK**, Supsupin EP, Sitton CW, Bajgur SS, Choi JM, Datta P, Wu T, Bonafante-Mejia EE, Friedman E, Dannenbaum MJ, Chen PC, Sun CJ, Barretto AD, Gupta R, Grotta JC, Savitz SI. Factors Affecting Vascular Neurologists' Decisions to Pursue Endovascular Intervention of Acute Ischemic Stroke Patients. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
31. Shiue H, Albright KC, Sands KA, Gadpaille A, **Boehme AK**, Khawaja A, Sisson A, Bavarsad Shahripour R, Harrigan M. Ratio of Blood Urea Nitrogen to Serum Creatinine Predicts Primary Intracerebral Hemorrhage Volume. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
32. Samai AA, **Boehme AK**, George AJ, Monlezun DJ, Dowell L, Leissinger C, Schluter L, El Khoury R, Martin-Schild S. Von Willebrand Factor Elevation Threshold for Poor Clinical Outcomes in Patients with Ischemic Stroke. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
33. Khawaja A, **Boehme AK**, Albright KC, Bavarsad Shahripour R, Kumar G, Shiue H, Lyerly MJ, Hays A, Harrigan M. Admission Blood Pressure Predicts The Number Of Medications Upon Discharge In Patients With Primary Intracerebral Hemorrhage. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
34. Yaghi Y, Leon-Guerrero CR, Dibu J, Ali S, Noorian AR, **Boehme AK**, Keyrouz SG, Hinduja A, Bianchi NA, Marshall RS, Liebeskind DS, Schwamm L, Willey JZ. The Association Between Treatments and Hematoma Expansion in Thrombolysis Related. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
35. Alvi M, **Boehme AK**, Lyerly MJ, Bavarsad Shahripour R, Gadpaille A, Shiue H, Harrigan M. The Impact of Electrolyte Abnormalities in Primary Intracerebral Hemorrhage. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
36. Khawaja A, **Boehme AK**, George AJ, Hays A, Kumar G, Alvi M, Miller DW, Martin-Schild S, Harrigan M. The Role Of A History Of Coronary Artery Disease, The Need For Transfusion And Outcomes In Patients With Intracerebral Hemorrhage. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
37. Khawaja A, **Boehme AK**, Albright KC, Kumar G, Arora K, Lyerly MJ, Harrigan M. The Utility of Charlson Comorbidity Index in Predicting Short-term Outcomes in Patients with Primary Intracerebral Hemorrhage. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
38. Samai AA, **Boehme AK**, Monlezun DJ, George AJ, Dowell L, Leissinger C, Schluter L, El Khoury R, Martin-Schild S. How High Is Too High? An Analysis of the Upper Threshold for Serum FVIII in Ischemic Stroke. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.

39. Samai AA, **Boehme AK**, Shaban A, George AJ, Monlezun DJ, Dowell L, Leissinger C, Schluter L, El Khoury R, Martin-Schild S. A Model for Predicting Persistent Elevation of Factor VIII Among Patients with Acute Ischemic Stroke (AIS). Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
40. Sarraj A, Martin-Schild S, **Boehme AK**, Sun CJ, Sitton CW, Supsupin EP, Jagolino AL, Bajgur SS, Datta P, Wu T, Friedman E, Barretto AD, Dannenbaum MJ, Chen PC, Gupta R, Grotta JC, Savitz SI. Examining the Effects of Intra-Arterial Therapy in Acute Posterior Circulation Occlusions. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
41. Shaban A, **Boehme AK**, Ryan M, Albright KC, El Khoury R, Martin-Schild S. The Vascular Distribution of Stroke Among Patients with Hypercoagulable State. Poster Presentation. *International Stroke Conference 2015*. Nashville, TN.
42. Albright KC, **Boehme AK**, Mullen MT, Martin-Schild S, Gonzales N, Sen B, Savitz SI. Stroke Severity in Men and Women: What Proportion of the Disparity Can Be Explained by Differences in Cardiovascular Risk Factors? Oral Presentation. American Academy of Neurology 2014. Philadelphia, PA.
43. Wolff C, **Boehme AK**, Wu T, Mullen M, Branas C, Grotta JC, Savtiz SI, Carr B. Sex Disparities in Access to Stand Alone Primary Stroke Centers: Can Telemedicine Mitigate This Effect? Poster Presentation. American Academy of Neurology 2014. Philadelphia, PA.
44. Mullen MT, Albright KC, **Boehme AK**, Kasner S, Kallan M, Branas C, Carr B. Gender Differences in Primary Stroke Center Evaluation and Utilization of rt-PA. Poster Presentation. American Academy of Neurology 2014. Philadelphia, PA.
45. Sarraj A, **Boehme AK**, Olowu A, Chung-Huan J. S, Bibars W, Sitton C, Supsupin E, Martin-Schild S, Gupta R, Grotta J and Savitz S. Examining the Effect of Intra-Arterial Therapy in IV tPA Ineligible Ischemic Stroke patients. Accepted for platform presentation, *International Stroke Conference 2014*, San Diego, CA.
46. Sarraj A, Sitton C, Supsupin E, **Boehme AK**, Olowu A, Bibars W, Martin-Schild S, Grotta J and Savitz S. Refining the CT Angiography Collaterals Scoring System to Better Predict Outcome in Acute Ischemic Stroke. Accepted for platform presentation, *International Stroke Conference 2014*, San Diego, CA.
47. Sarraj A, Bibars W, **Boehme AK**, Martin-Schild S, Grotta J and Savitz S. The Global Impact of Recent Intra-Arterial Therapy (IAT) Randomized Controlled Trials on Current and Future Stroke Practice. Accepted for a moderated poster presentation, *International Stroke Conference 2014*, San Diego, CA.
48. Friedant, A; Gouse, B; **Boehme, AK**; Siegler, JE; Albright, KC; Monlezun, DJ; George, AJ; Beasley, TM; Martin-Schild, S. A Simple Prediction Score for Hospital Acquired Infections. *International Stroke Conference*. San Diego, CA. February 2014. Poster Presentation.
49. **Boehme, AK**; Siegler, JE; Albright, KC; George, AJ; Monlezun, DJ; Friedant, A; Gouse, B; Beasley, TM; Martin-Schild, S. The Relationship between Leukocytosis and Time to Neurodeterioration. *International Stroke Conference*. San Diego, CA. February 2014. Poster Presentation.

50. **Boehme, AK**; Dillon, C; McGwin, G; Pamboukian, SV; Limdi, NA. Lactate Dehydrogenase Levels are Associated with Acute Ischemic Stroke in Ventricular Assist Device Patients. *International Stroke Conference*. San Diego, CA. February 2014. Poster Presentation.
51. Albright, KC; **Boehme, AK**; Sen, B; Aswani, M; Mullen, MT; Gonzales, N; Savitz, SI; Martin-Schild, S. Stroke Severity in Men and Women: What Proportion of the Disparity Can Be Explained by Differences in Cardiovascular Risk Factors? *International Stroke Conference*. San Diego, CA. February 2014. Poster Presentation.
52. Pineda, DA; Dawson, E; Albright, KC; Siegler, JE; Kimar, AD; Gillette, MA; **Boehme, AK**; Beasley, TM; Martin-Schild, S. Isolated National Institute of Health Stroke Scale Subscore Worsening is Predictive of Poor Outcomes in Acute Ischemic Stroke Patients. *International Stroke Conference*. San Diego, CA. February 2014. Poster Presentation.
53. Monlezun DJ, **Boehme AK**, Gouse BM, Siegler JE, Brag K, George AJ, Albright KC, Beasley TM, Martin-Schild S. The Role of Elevated Factor VIII in Renal Dysfunction in Ischemic Stroke Patients. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
54. Gouse BM, **Boehme AK**, Siegler JE, Brag K, George AJ, Monlezun D, Albright KC, Beasley TM, Martin-Schild S. New Thrombotic events in Ischemic Stroke Patients with Elevated Factor VIII. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
55. Herman M, Albright KC, Monlezun DJ, Scullen T, Siegler JE, Munshi N, George AJ, **Boehme AK**, Beasley TM, Martin-Schild S. Factors Associated with EMS Mode of Arrival versus Private Vehicle Among Patients with Acute Ischemic Stroke. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
56. Herman MP, Albright KC, Monlezun DJ, Scullen T, Siegler JE, Munshi N, George AJ, **Boehme AK**, Beasley TM, Martin-Schild S. For Stroke, EMS Trumps Private Vehicle for Pre-hospital Transport. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
57. Herman M, Albright KC, Monlezun DJ, Scullen T, Siegler JE, Munshi N, George AJ, **Boehme AK**, Beasley TM, Martin-Schild S. EMS and Hospital Stroke Care Coordination Not Associated with Racial Disparities in Pre-hospital Stroke Activation and Transportation. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
58. Monlezun DJ, Albright KC, Herman M, Sullen T, Siegler JE, Munshi N, George AJ, **Boehme AK**, Beasley TM, Martin-Schild S. Stroke Mimics May Delay Hospital Arrival Compared to Stroke Patients. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
59. Monlezun DJ, Albright KC, Herman M, Sullen T, Siegler JE, Munshi N, George AJ, **Boehme AK**, Beasley TM, Martin-Schild S. Hospital Arrival Mode Does Not Influence Short-Term Outcome in Ischemic Stroke. American Neurological Association's (ANA) 2013 Annual Meeting, New Orleans, LA. Poster Presentation.
60. **Boehme, AK**, George, AJ, Beasley, TM, Dunn, C, Siegler, JE, Behring, M, Albright, KC, Martin-Schild, S. Back to the basics: Neurologic Exam trumps 24-hour follow-up computed tomography scans in determining Symptomatic ICH. 65th Annual American Academy of Neurology Meeting, San Diego, CA. 2013. Oral Presentation.

61. Crisan, D, **Boehme, AK**, Shaban, A, Dubin, P, Sudkamp, J, Schulter, L, Siegler, JE, Albright, KC, Beasley, TM, Martin-Schild, S. Predictors of Recovery of Functional Swallow After Gastrostomy Tube Placement for Dysphagia in Stroke Patients After Inpatient Rehabilitation. 65th Annual American Academy of Neurology Meeting, San Diego, CA. 2013. Poster Presentation.
62. George, AJ, **Boehme, AK**, Dunn, C, Beasley, TM, Siegler, JE, Behring, M, Albright, KC, Martin-Schild, S. Utility of a Routine 24-Hour Follow-Up Computed Tomography Scan in Patients with Acute Ischemic Stroke. 65th Annual American Academy of Neurology Meeting, San Diego, CA. 2013. Poster Presentation.
63. Kumar, AD, **Boehme, AK**, Siegler, JE, Gillette, M, Albright, KC, Martin-Schild, S. Admission Leukocyte Levels Differ According to Stroke Etiology in Patients with Neurological Deterioration Following Acute Ischemic Stroke. 65th Annual American Academy of Neurology Meeting, San Diego, CA. 2013. Poster Presentation.
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77. **Boehme, AK**; Kumar, AD; Dorsey, AM; Siegler, JE; Lyerly, MJ; Monlezun, DJ; Albright, KC; Beasley, TM; Martin-Schild, S. The impact of infection on Neurological Deterioration and Outcome in Acute Ischemic Stroke. International Stroke Conference. Honolulu, HA, January 2013. Poster Presentation.
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Stay. International Stroke Conference. Honolulu, HA, January 2013. Poster Presentation.

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87. Lysterly, MJ; Albright, KC; **Boehme, AK**; Shahripour, RB; Houston, JT; Rawal, PV; Kapoor, N; Alvi, M; Sisson, A; Houston, JT; Alexandrov, AW; Alexandrov, AV. The Potential Impact of Maintaining a 3-Hour IV tPA Window. International Stroke Conference. Honolulu, HA, January 2013. Poster Presentation.
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92. **Boehme, AK**; Ogawaro, KM; Weiner, H; Shrestha, S; Kaslow, RA; Aissani, B. The MHC Determinants of AIDS-related Kaposi's sarcoma. 11th International Conference on Molecular Epidemiology and Evolutionary Genetics of Infectious Diseases. 2012, New Orleans, LA. Oral Presentation.
93. Aissani, B; Zhang, K; Weiner, H; Wu, J; **Boehme, AK**; Ogwaro, KM; Shrestha, S; Kaslow, RA. A

Novel Approach to identify host susceptibility genes in virally induced cancer: Application to HIV-related Kaposi's sarcoma. 11th International Conference on Molecular Epidemiology and Evolutionary Genetics of Infectious Diseases. 2012, New Orleans, LA. Oral Presentation.

94. Aysenne A, Mathias T, **Boehme AK**, Chang T, Albright K, Beasley TM, Martin-Schild S. 24-Hour ICH Score is a Better Predictor of Mortality than Admission ICH Score. Poster presented at the 64th Annual American Academy of Neurology Meeting, 2012, New Orleans, LA.
95. Siegler J, **Boehme AK**, Gillette M, Kumar A, Albright K, Martin-Schild S. Does Admission Leukocytosis or Neutrophilia Negatively Impact Outcome In Acute Ischemic Stroke? Poster presented at the 64th Annual American Academy of Neurology Meeting, 2012, New Orleans, LA.
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103. **Boehme AK**, Kumar A, Gillette M, Siegler J, Albright K, Martin-Schild S. Persistent Leukocytosis - Is this a Persistent Problem for Patients with Acute Ischemic Stroke? Poster presented at the 64th Annual American Academy of Neurology Meeting, 2012, New Orleans, LA.
104. Gillette M, **Boehme AK**, Siegler J, Kumar A, Albright K, Martin-Schild S. Admission

Leukocytosis is Not Associated with Early Response to Tissue Plasminogen Activator in Patients with Acute Ischemic Stroke. Poster presented at the 64th Annual American Academy of Neurology Meeting, 2012, New Orleans, LA.

105. Chang T, **Boehme AK**, Aysenne A, Albright K, Burns C, Beasley TM, Martin-Schild S. Packed Red Blood Cell Transfusion is Associated with Adverse Outcomes in ICH Patients. Poster presented at the 64th Annual American Academy of Neurology Meeting, 2012, New Orleans, LA.
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112. Jones, EM, **Boehme, AK**, Albright, KC, Burns, C, Beasley TM, Martin-Schild S. Emergency Department Nursing Shift Change and Adverse Outcomes in Patients with Intracranial Hemorrhage. International Stroke Conference, New Orleans, LA, January 2012. Poster Presentation.
113. Chang, TR, Albright KC, Kruse-Jarres, R, Lessinger, C, **Boehme, AK**, Beasley, TM, Martin-Schild, S. Relationship between tPA and Factor VIII Levels in Patients with Acute Ischemic Stroke. International Stroke Conference, New Orleans, LA, January 2012. Poster Presentation.
114. Chang, TR, **Boehme, AK**, Aysenne AA, Albright KC, Burns C, Beasley, TM, Martin-Schild, S. Nadir Hemoglobin Predicts Poor Functional Outcome and Death in ICH Patients. International Stroke Conference, New Orleans, LA, January 2012. Poster Presentation.
115. Kepplinger J, Barlinn K, **Boehme AK**, Pallesen L, Schrempf W, Albright K, Alexandrov

AV, Bodechtel U. Does Newly Diagnosed Sleep Apnea Favour Sleep-Related Ischemic Stroke? International Stroke Conference, New Orleans, LA. January 2012. Poster Presentation.

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117. **Boehme, A.K.**, Davies, S., Shrestha, S., Schumacher, J., Moneyham, L., Kempf, M.K. Barriers and Facilitators to ART Medication and Clinic Visit Adherence in HIV Postpartum Women. Poster Presentation. In the 5th annual Minority Health Research Symposium, Birmingham, AL, April 2011.
118. Kempf, M.K., McLeod, J., **Boehme, AK.**, Walcott, M., Wright, L., Seal, P., Norton, W., Mugavero, M., Moneyham, L. A Qualitative Study of the Barriers and Facilitators to Retention-in-Care among HIV-positive Women in the Rural Southeastern U.S.: Implications for Targeted Interventions. Oral Presentation. In the 5th International Conference on HIV Treatment Adherence, Miami, FL, May 2010.
119. **Boehme, A.K.**, Davies, S., Shrestha, S., Schumacher, J., Moneyham, L., Kempf, M.K. ART non-adherence in HIV+ postpartum women: Barriers and Facilitators. Poster Presentation. In the 5th International Conference on HIV Treatment Adherence, Miami, FL, May 2010.
120. McBride, S. A.; **Boehme, A. K.**; Shannon, C. N.; Riva-Cambrin, J.; Rozzelle, C. J.; Blount, J. P.; Oakes, W. J.; Carlo, W. A.; Wellons, J. C., Imaging Characteristics Were Not Predictive Of Ventriculosubgaleal Shunt Conversion In VLBW Infants With Post-hemorrhagic Hydrocephalus. Oral Presentation. In American Association of Neurological Surgeons Section of Pediatric Neurosurgery, Boston, MA, 2009.

List of Cases

2020

Case: *Jola Adegboyo v. Howard County General Hospital, Inc. et al*

Attorneys: Pessin Katz Law, P.A.

Party: Defendant

Expert Area: Epidemiology, Neuroepidemiology

Settled

Case: *Osagioduwa Idahor v. Arbor East Cobb, LLC* (COVID-19 Transmission & Exposure Risk)

Attorneys: Freeman Mathis & Gary, LLP

Party: Defendant

Expert Area: Epidemiology, Infectious disease epidemiology

Settled

Case: *Betty Owens v. FLAGLER HOSPITAL, INC., GEORGE P. JONES, IV, PA-C, and FLAGLER CARE CENTER*

Attorneys: Terrell Hogan

Party: Defendant

Expert Area: Epidemiology, Stroke epidemiology, Neuroepidemiology

Settled

Case: *LORRAINE D'ALESSIO, et al., v. DONG CHUL PARK, M.D., et al.*

Insurance Company: Medical Mutual Liability Insurance Society of Maryland

Attorneys: Shaw and Morrow, PA

Party: Defendant

Expert Area: Epidemiology, Stroke epidemiology, Neuroepidemiology

Deposed 13 May 2021

Settled

2021

Case: *Mary M. Parker et al. vs. Whitney Rothschild Matz, et al.*

Attorneys: Shaw and Morrow, PA

Party: Defendant

Expert Area: Epidemiology, Infectious disease epidemiology

Deposed 18 March 2021

Settled

Case: *Meining Jin vs. Greater Baltimore Medical Center*

Attorneys: Goodell, DeVries, Leech & Dann, LLP

Party: Defendant

Expert Area: Epidemiology, Neuroepidemiology

Deposed 14 February 2022

Settled

2022

Case: *Jacqueline Burks vs. Pamela Munster, et al.*

Attorneys: Bostwick and Peterson LLC

Party: Plaintiff

Expert Area: Epidemiology

Deposed 21 June 2022

Settled

Case: *Stephen Waldman MD vs. Traveler's Insurance*

Attorneys: Brach/Eichler LLC

Party: Plaintiff

Expert Area: Epidemiology, Neuroepidemiology

Settled

Case: *Maxwell Berger vs. JUUL; Khan vs. JUUL*

Attorneys: Lief, Cabraser, Heimann & Bernstein LLP

Party: Plaintiff

Expert Area: Epidemiology, Stroke epidemiology, Neuroepidemiology

Ongoing

2023

Case: *Tori Henson vs. George Washington University Hospital*

Attorneys: Goodell, DeVries, Leech & Dann, LLP

Party: Defendant

Expert Area: Epidemiology, Neuroepidemiology

Settled

Case: *Megan McLenithan vs. Greater Baltimore Medical Center*

Attorneys: Shaw and Morrow, PA

Party: Defendant

Expert Area: Epidemiology, Stroke epidemiology, Neuroepidemiology

Settled

Case: Camp Lejeune

Attorneys: Plaintiff's Leadership Group

Party: Plaintiff

Expert Area: Epidemiology, Neuroepidemiology

Ongoing

2024

Case: *Stephen Markowitz v. Akintayo Akinwunmi PNG*

Attorneys: Swartz Culleton PC

Party: Plaintiff

Expert Area: Epidemiology, Neuroepidemiology

Settled

Case: *Ning Pan, et al. v. Sutter Health, et al.*
Attorneys: Moseley Collins
Party: Plaintiff
Expert Area: Epidemiology, Neuroepidemiology
Settled

Case: *Roman Landeros v. Valley Children's Hospital, et al.*
Attorneys: Moseley Collins
Party: Plaintiff
Expert Area: Epidemiology, Neuroepidemiology
Ongoing

List of Depositions

2020

Case: *LORRAINE D'ALESSIO, et al., v. DONG CHUL PARK, M.D., et al.*
Insurance Company: Medical Mutual Liability Insurance Society of Maryland
Attorneys: Shaw and Morrow, PA
Party: Defendant
Expert Area: Epidemiology, Stroke epidemiology, Neuroepidemiology
Deposed 13 May 2021
Settled

2021

Case: *Mary M. Parker et al. vs. Whitney Rothschild Matz, et al.*
Attorneys: Shaw and Morrow, PA
Party: Defendant
Expert Area: Epidemiology, Infectious disease epidemiology
Deposed 18 March 2021
Settled

Case: *Meining Jin vs. Greater Baltimore Medical Center*
Insurance Company:
Attorneys: Goodell, DeVries, Leech & Dann, LLP
Party: Defendant
Expert Area: Epidemiology, Neuroepidemiology
Deposed 14 February 2022
Settled

2022

Case: *Jacqueline Burks vs. Pamela Munster, et al.*

Insurance Company:

Attorneys: Bostwick and Peterson LLC

Party: Plaintiff

Expert Area: Epidemiology

Deposed 21 June 2022

Settled