

Exhibit 534

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I am writing in response to your request for medical expert evaluation of Robert E. Welch with respect to the potential relationship between his diagnosis of Parkinson's disease (PD) and his exposure to trichloroethylene (TCE) which he experienced while stationed at Camp Lejeune in North Carolina.

I. Background and Qualifications

I am a board-certified neurologist by the American Board of Psychiatry and Neurology since 1989. After completing a Neurology residency at the University of Rochester, I completed a fellowship in Movement and Inherited Neurologic Diseases (MIND) at the University of Rochester under the mentorship of Ira Shoulson MD who was one of the founders of the Parkinson's Study Group and lead investigator in the pivotal DATATOP study looking at disease modifying therapy in Parkinson's disease. As a Fellow, I was able to participate in clinical research in the DATATOP study. I also did basic research in primates looking at the impact of adrenal medullary transplant in MPTP induced parkinsonism in primates.¹ As a member of the Huntington's Study Group, I participated in clinical research experience as a site investigator for both the DOMINO study looking at the impact of minocycline on Huntington's disease and the HART trial of ACH-16 in Huntington's disease.^{2,3} I have also done clinical research in the field of Parkinson's disease looking at the reliability and value of telemedicine in Parkinson's disease.^{4,5,6} Recently, I was an investigator in a study looking at a select population in Rochester, NY exposed to high levels of TCE revealing an increased incidence of various cancers and parkinsonian features.⁷ I have also served on the Board of the American Academy of Neurology as the Chair of the Practice Committee. In that role, I supervised the development of guidelines and quality measures including both the diagnosis and treatment of Parkinson's disease. I am board certified in Headache Medicine by UCNS and have been involved in updating the American Headache Society process for guideline development.⁸ I am a topic editor for Dynamed where I review and update medical information on a variety of neurologic disorders. I have an academic appointment at the University of Rochester Medical Center as a Professor of Clinical Neurology. My practice has involved caring for patients with Parkinson's disease for over 30 years. I am informed regarding the etiology, diagnosis and management of Parkinson's disease.

My qualifications are set forth more fully in my curriculum vitae, attached as Exhibit/Appendix 1. I am being compensated \$650/hour for my time and services rendered. My compensation is in no way contingent on opinions rendered or the outcome of this litigation. The opinions expressed in this report are stated to a reasonable degree of medical and scientific certainty, and are based upon my education, training and experience, and upon the review of records, literature and material cited herein, and in my list of materials attached as Exhibit A

II. Summary of Opinions and Methodology

I have been asked by Mike Dowling of The Dowling Firm to offer an opinion on the potential causal relationship between Robert Welch's exposure to the water at Camp Lejeune and his Parkinson's Disease. My opinion is based on my education, training, research, and clinical practice, along with materials and literature that I reviewed.

In forming my opinion, I have reviewed the following records on Robert E. Welch:

Medical Records from Dr. Carolyn Neff MD 11/16/21 to 10/12/23
Medical Records from Dr. Edward Markus from 7/11/2017 to 10/13/22
Medical Records from Kevin Tabora RCP, R.T. 11/19/21
Medical Records from the VA system from 8/23/2012 to 5/23/2023
Medical Records from Dr. Thomas Dorsey from June 11, 2002
Medical Record Review from Aperio Solutions - Shiela Marie Cardona 7/4/2024
Records of deposition from Maureen Ann Welch on 5/20/24
Records of deposition from Robert E. Welch on 3/8/24
Records of depositions from Carolyn Neff MD, Jeffrey Tracy MD, Edward Markus DO
Military records bates numbered 01503_WELCH_0000000133 to 0000000135

I also conducted a Zoom interview and examination with Mr. Robert Welch on 12/19/2024 lasting 70 minutes.

In addition, I relied upon peer reviewed scientific literature relevant to the etiology of Parkinson's disease specifically as it pertains to TCE and PCE (perchloroethylene) exposure including my own research linking dry-cleaning chemicals to Parkinson's Disease.⁷ Additionally, I have considered regulatory agency documents and published studies on outcomes seen in Service people and civilians stationed at Camp Lejeune. Full citations of the scientific literature are given in this report's endnotes.

I have also reviewed the reports authored by Dr. Jason Cannon, Dr. Amelia Boehme, Dr. Lucio Costa, Dr. Briana De Miranda, Dr. Gary Miller and Kelly Reynolds, MSPH, PhD and I have reviewed the materials cited in their reports to understand and verify that their opinions were well reasoned and supported. I understand and consider these opinions on whether science supports the proposition that TCE and PCE can cause Parkinson's Disease.

From my review of the materials, I understand that the plaintiff in this case must prove causation under the Camp Lejeune Justice Act (CLJA) to a level of "at least as likely as not" which is a lower burden than the "more likely as not" causation standard. It is my professional opinion based on my education, training, and expertise as a neurologist and a movement disorder specialist, and to a reasonable degree of medical certainty, I conclude that Mr. Welch's Parkinson's disease is at least as likely as not due to his exposure to TCE at Camp Lejeune for an approximate 11-month period from November 18, 1970 to December 15, 1971.¹⁰⁰ [MOU1] [hs2]. As a result, my causation opinions in the Welch matter meets the CLJA causation standard of "at least as likely as not."

III. Parkinson's Disease

Parkinson's disease (PD) is the fastest growing neurodegenerative disease in the world and second only to Alzheimer's disease in prevalence. PD is a clinical diagnosis which can be further supported by select imaging techniques such as DAT scans. It strikes in adulthood, but age of onset and rate of progression varies. Peak incidence occurs between the ages of 70-79. Parkinson's disease is manifested as motor and non-motor symptoms, both of which are disabling. Non-motor symptoms often precede the onset of motor symptoms by as much as 20 years.^{9,10} Motor symptoms include some combination of the following: slowness of movement (bradykinesia), increased muscle tone (rigidity), unilateral or asymmetric tremor and unsteadiness of gait (postural instability).³⁸ Some of these symptoms may be responsive to medications or physical therapy but eventually become difficult to treat. These symptoms interfere with daily tasks such as writing, speaking, eating, ambulation and fine motor tasks. Non-motor symptoms can include loss of smell, depression, anxiety, hallucinations and/or delusions, cognitive decline and frank dementia, autonomic dysfunction (orthostatic hypotension, bladder control, erectile dysfunction, reduced gastrointestinal motility), sleep disorders including REM behavior disorder, swallowing and eating difficulty, restless leg syndrome, breathing difficulties, pain and fatigue.³⁹ Non-motor symptoms are more difficult to treat and often limit the ability to function outside of a supportive setting.⁴⁰

Parkinson's disease is incurable resulting inexorably progressive motor and non-motor manifestations of the disease. There are no medications that have been shown to slow the course of the disease.⁴¹ Over time, those with Parkinson's disease become less responsive to the medications used to treat the symptoms of the disease.⁵⁰ Motor fluctuations develop over time characterized by "on-off" periods associated with reduced mobility, increased tremor, involuntary movements (dyskinesias or dystonia), freezing.⁶⁷ The development of motor fluctuations increase the risk of falling. As symptoms progress, individuals often need help with basic activities such as meal preparation, managing finances, driving and eventually eating, self-care and walking. Therefore, in addition to the cost of medical care, medications and other treatments, individuals with Parkinson's disease often need costly help in the home or placement in a facility that provides a higher level of care. Clearly, these issues have a significant impact on quality of life.

The neuropathologic hallmarks of PD include: (1) loss of dopamine neurons in the substantia nigra which project to the caudate and putamen (striatum); (2) the presence of Lewy bodies which are protein inclusions in the neurons consisting of alpha synuclein and (3) neuroinflammation.^{11,12,13,14} Dopamine is a neurotransmitter produced in dopaminergic neurons in the substantia nigra which then project to the striatum. Dopamine is essential for control of motor movements.⁴² Motor manifestations of PD become apparent when at least 70-80% of dopamine neurons in the substantia nigra are lost. Loss of dopamine neurons and cell death occurs due to oxidative stress and mitochondrial toxicity as well as apoptosis mediated by alpha-synuclein which promotes the neuroinflammatory process. The mitochondria are the power plant of the cells and control the lifespan and death of the cell (apoptosis). Mitochondrial toxicity results in cell death.

Our understanding of the cause(s) of Parkinson's disease has evolved significantly over the last 30 years. In the late 1990s clusters of families with PD were identified resulting in the identification of the first gene mutation associated with PD.⁴³ The designer drug, MPTP was

associated with the development of PD in the late 1980s, but it wasn't until the early 2000s that research began to link toxin exposure to PD (See below). Parkinson's disease was once called an idiopathic disease (i.e. one without a clear cause), but current research and epidemiologic studies have provided scientific evidence, through mechanistic and animal studies, that environmental factors play a substantial role in the development of PD. Genetic susceptibility, for some, is also believed to contribute to the development of PD, as discussed below, but genetic susceptibility requires the contribution of another factor to cause PD.^{15,90} There are also potential causes for Parkinson's disease that are based on an association between these factors and the incidence of Parkinson's disease but are lacking a scientifically proven mechanism of causation. Both potential causes and scientifically proven causes will be discussed below. Age itself is not thought of as a risk factor for PD, however, increased age allows for more exposure time to environmental toxins and enough time for PD to manifest.¹⁶

A. Latency

When discussing latency in reference to the onset of Parkinson's disease vis a vis a toxic exposure, we are referring to the period between either the acute or chronic exposure to the substance and the clinical diagnosis of Parkinson's Disease, as just mentioned above. However, once Parkinson's Disease has been clinically diagnosed, we often look back in time to the onset of non-motor—called prodromal-- symptoms to determine whether they are consistent with the ongoing premotor manifestation of Parkinson's Disease. There is no scientific evidence of a timeframe where the latency period ends for the onset of Parkinson's Disease after toxic exposure. Depending on the volume of toxic exposure, the onset can take place rather quickly over months (with a concentrated exposure). However, less volume and concentration of toxic substance exposure has shown a longer period of latency, in the range of years and decades.

B. Potential Causes of Parkinson's disease¹:

- **Traumatic brain injury:** has been associated with Parkinson's disease in many epidemiologic studies.¹⁸ Some studies estimate that TBI increases the risk of PD by 56%.⁵² However, other epidemiologic studies have shown no association between TBI and PD.⁵¹ Multiple studies show that the severity of TBI directly correlates to the risk of developing Parkinson's disease. Another study suggested that TBI later in life was more likely to be associated with the development of PD.⁵³ Although there is molecular overlap between the pathology seen in TBI and PD, the mechanism of the association between TBI and PD remains speculative.¹⁸ Without a proven mechanism, it cannot be stated that TBI causes PD.
- **Particulate matter:** More recently, research shows an association between air pollution (PM2.5) and Parkinson's disease.²³ The mechanism by which particulate matter 2.5 would result in PD is purely speculative.
- **Melanoma:** Although people with Parkinson's disease generally have a lower risk of cancer, there is a well-documented association between Parkinson's disease and melanoma that is bidirectional. Epidemiologic studies document that those with a diagnosis of Parkinson's disease are 2 to 7 times more likely to develop melanoma than the general public.^{44,64} Those with melanoma are 50% more likely to develop Parkinson's disease.⁶⁵ Despite this strong association, there is no evidence of

¹ These causes are only potential causes because the science is not well developed or objectively understood.

causation meaning that there is no evidence that melanoma causes PD or vice versa. However, based on the results of many studies, patients with Parkinson's disease should consider being screened for melanoma.⁶⁶

C. Causes of Parkinson's disease²:

- **Genetics:** Between 10-30% of those with Parkinson's disease have a genetic predisposition to PD. There are over 100 genes that are associated with PD.^{15,43} Only about 2-5% of those with PD have a pure genetic cause. The most common mutation is in the LRRK2 gene but even this has incomplete penetrance.¹⁸ Most genes have limited penetrance, meaning that the gene will not cause Parkinson's disease without another factor which is likely environmental. Simply put, just having the gene is not enough to cause Parkinson's disease without some other trigger.
- **Drug Exposure:** A small cluster of cases of Parkinsonism was identified in the 1980s in Northern California, in which a contaminant of synthetic meperidine, called MPTP, was shown to be responsible for causing the same brain lesions that are found in PD¹⁹. This finding is of utmost importance as it was the first time an exogenous agent was shown to damage the dopaminergic system and cause the same symptoms as in PD. The cases were young (most in their twenties) and had in common only the fact they were drug addicts and had consumed the same batch of synthetic meperidine. They displayed typical PD symptoms and responded to pharmacological therapies used in PD. Years of subsequent experimental work has shown that MPTP is rapidly absorbed and transferred to the brain, where it is converted into the ultimate neurotoxic agent, MPP+, which accumulates in dopaminergic neurons and causes their demise by inducing oxidative stress and damaging mitochondria²⁰. This triggered research into other exogenous toxins that may cause PD.

Dopamine blocking agents are also associated with drug-induced parkinsonism. Specifically, medications such as metoclopramide, antipsychotics and some anticonvulsant medications (lamotrigine, levetiracetam and valproic acid) have been associated with drug induced parkinsonism.^{49,92}

- **Environmental exposures:** Environmental triggers are usually related to toxin exposure. One important fact to consider is that exposure to a neurotoxic agent may precede the appearance of PD symptoms by decades. It has been calculated that PD symptoms manifest when brain dopamine content has decreased by 70-80%. Because of the normal aging process, there is a progressive loss of dopamine, but the "threshold" for clinical signs to be manifest varies depending on a variety of factors.
 - *Exposure to pesticides:* Paraquat, rotenone and diazinon are recognized causes of Parkinson's disease. Recent research has shown there are at least 10 pesticides that are toxic to dopaminergic neurons involved in Parkinson's disease.²¹ Pesticides are thought to damage dopaminergic neurons via the

² These causes have been established through epidemiology studies, toxicology studies, and mechanistic studies.

same route as MPTP and solvents i.e. oxidative stress and mitochondrial toxicity. Oxidative stress is an imbalance between the production of harmful free radicals (reactive oxygen species) and the body's ability to counteract them with antioxidants, leading to damage to brain cells. Mitochondria are membrane-bound cell organelles that generate most of the chemical energy needed to power the cell's biochemical reactions

- Heavy metals: Heavy metals, such as iron (Fe), mercury (Hg), manganese (Mn), copper (Cu), and lead (Pb), have been linked to PD and contribute to its progression.²² The mechanism of cell death with heavy metals is also oxidative stress.
- Cleaning chemicals and solvents: A growing body of literature has demonstrated that exposure to trichloroethylene (TCE) is a risk for developing Parkinson's disease. Epidemiologic data has become more compelling over the years. The first case report of PD developing in the setting of TCE exposure (through a work exposure) was published in 1999.²⁴ In 2008, a cluster of TCE exposed workers had a higher incidence of Parkinson's disease than the general population. This study demonstrated that TCE was toxic to mitochondria with a mechanism similar to MPTP.²⁵ A twin study authored by Dr. S. Goldman published in 2011 showed that exposure to TCE increase the risk of PD over 6 fold.²⁶ Twin studies are particularly relevant since twins share nearly identical genetic markers but differ only in their exposure to TCE.

Two studies examined mortality among Marine and Navy personnel and among civilian employees at Camp Lejeune, as compared to those at Camp Pendleton.^{27,28} This is the first in a series of studies that compare Camp Lejeune (where TCE levels were well above the allowable level) and Camp Pendleton (where there is no evidence of TCE contamination). Mortality hazard ratios at Camp Lejeune were significantly higher for many causes, including PD. The study estimated levels of TCE and PCE in water in the Hadnot Point system during the period 1975-1985 of 359 µg/L and 16 µg/L, respectively.^{27,28} The Maximum Contaminant Level (MCL) set by the EPA is 5 µg/L for TCE and PCE.^{68,69}

The next major study looking specifically at those that served at Camp Lejeune between 1975 and 1985 was published in 2023.²⁹ This study evaluated records of more than 150,000 veterans that served either at Camp Lejeune or Camp Pendleton between 1975 through 1985 and had a follow up between 1997-2021. The levels of TCE at Camp Lejeune were 70 times the allowable levels (deemed by the EPA) during that time frame. Results showed that those who served at least 3 months at Camp Lejeune had a 70% greater chance of developing Parkinson's disease than those that served at Camp Pendleton. This result was highly significant (P<.001). Camp Lejeune veterans also had increased symptoms of prodromal parkinsonism

suggesting that they were more likely to develop Parkinson's disease over time. Follow up of this cohort of veterans from Camp Lejeune showed that those affected by Parkinson's disease progressed more quickly with shorter time to psychosis, fracture and falls.³⁰ These findings suggest the PD caused by TCE may progress more rapidly.

A large study from 2024 which was a follow-up to two previous studies by Bove et al. aimed at evaluating mortality of Marines, Navy personnel and civilian workers at Camp Lejeune and at Camp Pendleton. The former were exposed to TCE and to PCE over a thirty-year period (1953-1985). The findings indicate a two-fold increase of mortality due to PD in Marines and Navy personnel at Camp Lejeune, compared to Camp Pendleton.³¹ In some of my own research, we identified a cluster of attorneys with Parkinson's disease and prodromal parkinsonism who were exposed to high levels of TCE from a contaminated site near their office.⁷ Their rate of PD and prodromal parkinsonism is higher than expected for age and also higher than the control cohort. A recent series of case reports from Dorsey et al included a professional athlete who spent his early childhood at Camp Lejeune and developed PD at the age of 34.³² The above referenced studies have shown that Parkinson's disease can manifest 40-50 years after exposure to TCE but the timing of disease onset is individually variable.

In addition to epidemiologic data, animal studies have consistently shown that exposure to TCE causes damage to the dopaminergic system which mimics the changes seen in PD. The toxicity of TCE was documented in rodents of both sexes via either oral, intraperitoneal or inhalation routes of administration.^{33,34} These effects are both dose and time dependent meaning the higher or longer the exposure to TCE, the greater the damage. In fact, studies show that inhalation of TCE is more toxic than ingestion due to greater dopaminergic degradation.³⁵ Any ingestion exposure is also associated with inhalation exposure.

Animal studies show TCE results in damage to mitochondrial function, loss of dopamine containing neurons in the substantia nigra (which is the region affected in PD), increased inflammation, accumulation of alpha-synuclein, and increased activity of LRRK2 kinase, the most common mutation associated with familial PD. These findings strongly support human studies that TCE can induce brain damage consistent with that observed in Parkinson's disease. Potential mechanisms of action of TCE induced PD are related to TCE metabolites and gut microbiome changes. TaClo is a TCE metabolite which is structurally similar to MPTP and causes similar damage to dopamine containing neurons in rodents.³⁶ TaClo also stimulates LRRK2 kinase activity which as noted above is the most common genetic mutation associated with PD. TCE has been shown to change the gut microbiome in the rodent model of TCE exposure which mimics the changes in gut microbiome changes in humans with PD.³⁷ Changes in gut microbiome are

thought to alter the gut-brain axis seen in PD.

D. Dose level of TCE exposure at Camp Lejeune

TCE monthly exposure in the water supply at Camp Lejeune between 1975-1985 was modeled to reflect a mean monthly average level of 366 ug/l-M.²⁹ The Maximum Contaminant Level (MCL) set by the EPA is 5 micrograms/liter. From my review of the ATSDR water modeling data, Marines, civilians and their families who lived and or worked at the base during the approximately 3 decades of TCE contamination would have potentially been exposed to levels of TCE that far exceeded the regulatory levels set by the EPA.⁹³ More importantly, based on the previously cited literature, it has been demonstrated clearly that exposure to Camp Lejeune's water supply for 3 months or more during the decade beginning in 1975, resulted in a 70% increase in the risk of developing Parkinson's disease.²⁹

In 2017, the Agency for Toxic Substances and Disease Registry (part of the Federal government) concluded that there was "equipoise and above evidence" for causation for TCE and Parkinson's disease. Since then, epidemiologic data has made this argument even stronger. More recently, in December of 2024, the EPA banned all use of TCE and greatly restricted to use of PCE (perchloroethylene) based in part on the deleterious effects these chemicals have on the neurologic system.⁴⁸ The EPA's recent action is consistent with what has become increasingly known and recognized in the medical and scientific fields. We have recognized the causal association between TCE and PCE and Parkinson's Disease for several years., Published scientific literature proves this causal relationship. This is further explained in the reports of other experts in this case.

Based upon my review and understanding of the scientific literature on Parkinson's disease, it is my professional opinion, to a reasonable degree of medical certainty as a neurologist and movement disorder specialist, it is at least as likely as not there is a causal relationship between exposure to TCE and PD.

Included in my research and review of materials in connection with my involvement in Mr. Welch's case, I have reviewed reports from toxicologists and an epidemiologist who provided expert opinions in this case including Drs. Cannon, Costa, DeMiranda, Miller and Boehme whose summaries of the experimental data regarding TCE and PCE and PD are rigorous and thorough. While I have reviewed, understood, and considered their research, literature review and opinions, I have arrived at my own conclusions based on my own training, education and both clinical and research experience. Please refer to these documents for more detailed discussion of studies pertaining to this subject.^{59, 60, 61, 62, 63}

IV. Patient History and Presentation of Parkinson's Disease

Mr. Welch is an 80-year-old man who had an uneventful childhood and early adult life.⁷² Mr. Welch enlisted in the Marines in 1968 when he attended a Platoon leaders' class in Quantico, VA.⁷⁰ After graduating from Loyola Law School in 1969, he enlisted in the Marine Corps as a Judge Advocate.⁷¹ After basic training, he was stationed at Camp Lejeune starting in November 1970.⁷³ He attended Naval Justice Academy in Newport Rhode Island from January to March of 1971 (2.5 months) after which he returned to Camp Lejeune until December 1971.⁷⁶ He spent

approximately 11 months at Camp Lejeune between 11/18/1970 and 12/15/1971.¹⁰⁰ While he was stationed at Camp Lejeune, he lived in Tarawa Terrace and worked at Mainside.⁷⁴ The water supply at both locations was heavily contaminated with TCE which will be discussed below.

Mr. Welch testified that he showered twice per day for 20 minutes, drank 9 cups of coffee per day as well as 4 glasses of water per day while working at Mainside. 2 weeks a year, he was on the rifle range getting his water supply from a water buffalo (filled with TCE contaminated water), drinking the equivalent of 5-6 glasses of water per day. On the weekends he stayed on base and drank from the contaminated water supply. He also handwashed his dishes at his apartment which contributed to inhalation (and dermal) exposure to TCE.⁷⁵ After Camp Lejeune, Mr. Welch was stationed in Okinawa followed by El Toro, CA after which he entered the Marine Reserves where he retired from active duty as a Lieutenant Colonel as a Staff Judge Advocate.⁷³ After leaving active duty, Mr. Welch worked in the legal field finishing his career as a judge for the Workers Compensation Appeals Board in California where he retired in 2010.⁷⁸

Mr. Welch did sustain injuries while enlisted in the Marines.⁷⁷ In 1968, he sustained injury to head and face with pugil sticks while in leadership training. Mr. Welch denies that he had any loss of consciousness with this injury.⁵⁴ He denies any cognitive deficits after this event. In May 1970, he sustained a fall on an obstacle course landing on his back, neck and head. Again, there is no documentation of loss of consciousness or concussion.⁵⁴ Both of the above injuries led to chronic back and neck complaints. In 1985, while on reserve duty, he was beaten by a gang, knocked to the ground and kicked in the ribs, back, neck and head. Again, he denies any loss of consciousness or concussion with this injury, but he acknowledged feeling dazed for a short time after this assault.⁵⁴ He filed for VA benefits in 1996 for orthopedic injuries (back, neck, knees, hips and both feet) that were sustained during active and reserve duty. He never claimed any service-related head injury. He was eventually granted 100% service-related disability in 2002 due to these orthopedic injuries.⁷⁹

Mr. Welch began having episodes of “freezing for a moment” in 2011. He was eventually diagnosed with focal epilepsy and placed on levetiracetam (which caused irritability) and then carbamazepine with complete resolution of his symptoms since 2014.⁸⁰ An MRI of the brain in 2002 showed a 5 mm cavernous angioma in his left caudate which may be the cause of his epilepsy.⁸¹ A repeat brain MRI in 2021 showed no change in this vascular malformation. He also carries a diagnosis of chronic headaches which are thought to be cervicogenic i.e. related to the severe arthritis he has in his neck.⁸²

Mr. Welch noticed a change in his handwriting consistent with micrographia (abnormally small handwriting that becomes progressively smaller) in 2019 at the age of 75.⁵⁴ By February 2021, he noticed a rest tremor in his right hand and arm. He also noted a loss of smell, slowed walking with lack of arm swing on the right. His voice had become soft and low.⁸³ He mentioned this to his epileptologist, Dr. Markus who raised the possibility of Parkinson’s disease (PD). He was seen by Dr. Neff, a movement disorder specialist in November 2021, who confirmed the diagnosis of PD.⁸⁴ Her exam showed the hallmarks of PD including asymmetric rest tremor, cogwheel rigidity and bradykinesia. She also noted several non-motor symptoms including loss of smell, constipation, depression/anxiety and mild cognitive difficulties. He also had erectile dysfunction. At that time, she authored a letter suggesting that his PD may be related to his

exposure to water at Camp Lejeune.⁸⁵ She felt that neither his seizure disorder nor his vascular malformation had any relationship to PD.⁸⁹

Mr. Welch started on carbidopa/levodopa (standard medication for PD) and was advised to continue physical therapy.⁸⁴ Based on the diagnosis of PD, Mr. Welch applied for VA benefits because he was aware of information linking TCE in the Camp Lejeune water supply and PD. It is my understanding that the VA then evaluated Mr. Welch and confirmed that he did have PD but also diagnosed him with dementia. However, I could find no objective testing to support the VA's diagnosis of dementia. The VA then deemed him unfit to manage his VA benefits despite the fact that he did all the finances for himself and his wife.

He was seen again by Dr. Neff in March 2022 who administered the Montreal Cognitive Assessment (MoCA) which is a standardized test to assess cognition.⁸⁶ Mr. Welch scored 23/30 with deficits in short term recall and word fluency indicating that he had mild cognitive impairment but not dementia. He was placed on donepezil which is a medication used to stabilize cognitive loss in dementia. Dr. Neff felt that his cognitive difficulties were due to PD and attested to the fact that he did have capacity to manage his financial affairs.^{86,87} The last documentation I have from Dr. Neff is dated October 2023.⁸⁸ At that time, he was complaining of fatigue and poor sleep despite CPAP for his OSA. Both carbidopa/levodopa and entacapone were helping with his PD symptoms but he continued to have intermittent tremor, reduced arm swing, micrographia and difficulty with maintaining concentration. He was independent in his activities of daily living and denied falls. His exam was unchanged. No changes in his medications were recommended. He was encouraged to continue exercising.⁸⁸ In reviewing Mr. Welch's medical records, his treatment appeared reasonable and appropriate.

His past medical history was otherwise notable for hyperlipidemia, supraventricular tachycardia, paroxysmal ventricular tachycardia, benign prostatic hypertrophy, cervical and lumbar spondylosis, GERD, and obstructive sleep apnea on CPAP. He is an ex-smoker, quitting in 1973. He drinks alcohol occasionally and denies other drug use. There is no family history of Parkinson's disease.

I also had the opportunity to perform a 70-minute virtual exam with Mr. Welch on December 19, 2024. At that time, he confirmed that he drank 3 glasses of water and 8-12 cups of coffee per day while serving at Camp Lejeune. He showered 1-2 times per day for 10-15 minutes. He denied swimming on base or use of fans at work or home.⁵⁴ He clarified that none of the episodes of "head trauma" in his records (1968, 1979, 1985) were associated with loss of consciousness or with any limitation of his ability to perform his duties. Specifically, he denied any post-concussive symptoms. He clarified that his first sign of Parkinson's disease was in 2019 when he was writing holiday cards. He was 75 years old at that time. He noted tremor in his right hand and arm in 2020.⁵⁴ He reported a fall in a parking lot in March 2024 which he associates with not seeing an obstacle on the ground and not having balance to recover.

Regarding his activities of daily living (ADLs), he reports some difficulty with buttons and needs support of furniture to put his pants on. He is independent in bathing but requires some prompting from his wife for his medications. Aside from helping with the dishes, he does nothing around the house and has others do limited yardwork. He denies any difficulty with driving. He

has daytime fatigue and naps in the early afternoon. He has difficulty maintaining concentration and retaining information while reading. He experiences cramping in his legs when he first awakens in the morning which resolves after his first dose of Sinemet “kicks in”. These symptoms are consistent with “off periods”. He denies any dyskinesias.⁵⁵ His non-motor symptoms include significant constipation, nocturia (1-2 times per night), drooling at night, striking out at night while sleeping (probable REM behavior disorder), disrupted sleep on occasion (which is better with CPAP) and complete loss of smell with taste preserved.⁵⁴

His current Parkinson’s medications include: Sinemet 25/100 1 tablet 4 times per day, Sinemet CR 25/100 1 tablet at bedtime, Entacapone 100 mg 4 times per day (with Sinemet), Donepezil 10 mg at bedtime. He had seen Dr. Neff (a movement disorder specialist at Kaiser Permanente) from November 2021 through October 2023. More recently, he has been assigned to a new movement disorder specialist, but he did not recall the name.⁵⁴

During the interview, Mr. Welch was alert and fully oriented. His mood and affect were appropriate. Montreal Cognitive Assessment was performed. His score was 21/30, a decrease in his score from Dr. Neff’s assessment in 2022. In addition to having difficulty with short term recall (2/5 at 5 minutes), and a word fluency deficit (7 words in 1 minute), he also had difficulty with visual spatial tasks (trail making, copying a figure and putting the hands on a clock). These deficits were new as compared with his 2022 results.⁵⁴ On cranial nerve exam, his extraocular movements were full. He had Grade 1 hypomimia (poker face) and a low, soft voice. Motor exam revealed bradykinesia in the right>left upper extremities. Finger taps were 7/5 seconds in the right with reduced amplitude and fatiguing and 9/5 seconds on the left with reduced amplitude. Palm opening and closing was moderately slowed and dysrhythmic on the right> left side. Heel taps were mildly slowed on the right but normal on the left. During my exam, he exhibited no tremor. His gait was mildly slowed with reduced arm swing on the right>left side. He required 3-4 steps to corner.

Based on my evaluation as well as records from Dr. Neff, Mr. Welch has Parkinson’s disease with the hallmark features of asymmetric tremor, bradykinesia and gait disturbance that have progressed over time. A formal Unified Parkinson’s Disease Motor Rating Scale (UPDRS motor) cannot be performed with a virtual exam, but he has a modified Hoehn and Yahr score of at least 2 (bilateral involvement without impairment of balance) but it may be 2.5 since I was unable to perform a pull test, and he has had a fall. He does not have features suggestive of atypical parkinsonism or Parkinson plus which are neurodegenerative diseases that have similar symptoms to Parkinson disease but are not Parkinson’s disease. Specifically, he does not have early onset dementia with hallucinations, pronounced autonomic dysfunction, pronounced and early postural instability, eye movement abnormalities or alien hand syndrome which are seen in diffuse Lewy body disease, multisystem atrophy, progressive supranuclear palsy or corticobasal degeneration, respectively. Mr. Welch notes that his symptoms have progressed over the last 3-5 years. Given the nature of Parkinson’s disease, his symptoms will continue to progress with further neurodegeneration even with aggressive medical management.

In addition to his motor deficits, he has many non-motor deficits as noted above. Most notable are his cognitive deficits as demonstrated on the Montreal Cognitive Assessment. His score of 21/30 still falls in the range of mild cognitive impairment (MCI), but he is now

demonstrating some deficits in visual spatial function which is likely to impact his ability to drive. PD patients with MCI, like Mr. Welch, have a 60% chance of progressing to dementia within 4 years.⁵⁶

V. Methodology, Analysis and Conclusions

To address the etiology of Mr. Welch's Parkinson's disease, it is important to ensure a correct diagnosis. Once confirmed, known risk factors for Parkinson's disease need to be explored as they relate to Mr. Welch's history. Based on this analysis, a conclusion can be made regarding the likely etiology of Parkinson's disease for this individual.

As detailed above, based on medical records, depositions from his healthcare providers and my own virtual evaluation to a reasonable degree of medical certainty, Mr. Welch has Parkinson's disease with mild cognitive impairment. As noted previously, PD is a clinical diagnosis, and in Mr. Welch's case, the diagnosis of PD is indisputable. Although Mr. Welch sustained orthopedic injuries to his back, neck and knees, none of these injuries would predispose him to the development of Parkinson's disease. Specifically, these injuries would not cause tremor, bradykinesia, change in handwriting, autonomic dysfunction, mild cognitive impairment, etc. After examining Mr. Welch, it is my medical professional opinion to a reasonable degree of medical certainty, that it is as likely as not that his current disability is due to Parkinson's disease from water exposure at Camp Lejeune, not orthopedic injuries. Questions have been raised about whether his seizure disorder increased the likelihood of developing PD, but there is no evidence to support a link between these 2 neurologic diagnoses. As a result, I can, to a reasonable degree of medical certainty, rule out his seizure disorder as causative of his PD. Lastly, his head MRI's have shown a stable cavernous angioma. This is a congenital defect meaning it developed during early life. This vascular abnormality has no bearing on his Parkinson's disease, which Dr. Neff stated as well.

Regarding etiology, there are risk factors as I have outlined above. I would like to address each of them individually.

1. **Genetics:** Mr. Welch has no family history of Parkinson's disease. Even if he had a gene that predisposed him to PD, given the fact that these genes have incomplete penetrance, he would need another factor to manifest PD. There is no evidence that Mr. Welch has a genetic predisposition to Parkinson's disease.
2. **Head Trauma:** Mr. Welch's records document 3 episodes of head trauma but none were associated with loss of consciousness or cognitive impairment after the injury. It is important to note that "head trauma" is not synonymous with traumatic brain injury. Specifically, he denies any post-concussive symptoms. Therefore, he had no evidence of diffuse axonal damage which is the hallmark of traumatic brain injury.⁵⁸ The studies done in military personnel associating mild TBI with an increased occurrence of PD defined mild TBI as transient loss of consciousness which he did not have.⁵² As noted above, there is not a definitive causal link between TBI and Parkinson's disease. Based on this reasoning, and to a reasonable degree of medical certainty, it is my opinion to a high degree of medical certainty, the head trauma is not a causative factor for Mr. Welch's Parkinson's disease.

3. **Drug exposure:** Mr. Welch was never exposed to MPTP. Dopamine blocking agents such as metoclopramide can cause a drug induced parkinsonism, but based upon my reviews of Mr. Welch's medical records he has never been exposed to any of these drugs.⁵⁷ Because Mr. Welch has never been exposed to dopamine blocking agents, I can rule out drug exposure as a possible cause of his Parkinson's disease.
4. **Environmental exposure:** Mr. Welch denies any exposure to pesticides or organophosphates. He has lived most of his life in urban settings and has never lived on a farm where pesticides or organophosphates were used. He has had no exposure to heavy metals. Pollution and specifically, particulate matter (PM 2.5) may be associated with an increased risk of Parkinson's disease, but the mechanism remains uncertain.
5. **TCE/PCE Exposure:** Mr. Welch was exposed to toxic levels of TCE, and other volatile organic compounds, during the time that he was stationed at Camp Lejeune. More specifically, Mr. Welch was stationed at Camp Lejeune between November, 18, 1970 to December 31, 1970 and March 12, 1971 to December 15, 1971 where he was exposed to TCE and tetrachloroethylene (PCE) both at work (Hadnot Point, HP) and at home (Tarawa Terrace, TT). During that time, he had an estimated exposure of 280 microgram/liter month of TCE, 524 microgram/liter month of PCE. The following chart indicates the specific concentrations in the water when Mr. Welch was at Camp Lejeune:⁹⁹

Exposure Dates	HP TCE (ug/l-M)	TT TCE (ug/l-M)	HP PCE (ug/l-M)	TT PCE (ug/l-M)	HP VC (ug/l-M)	TT VC (ug/l-M)	HP BZ (ug/l-M)	TT BZ (ug/l-M)
11/18/1970 - 11/30/1970	25	2	0	45	0	2	3	0
12/1/1970 - 12/31/1970	22	2	0	44	0	2	2	0
1/1/1971 - 1/31/1971	0	0	0	0	0	0	0	0
2/1/1971 - 2/28/1971	0	0	0	0	0	0	0	0
3/1/1971 - 3/11/1971	0	0	0	0	0	0	0	0
3/12/1971 - 3/31/1971	17	2	0	44	0	2	2	0
4/1/1971 - 4/30/1971	24	2	0	44	0	2	3	0
5/1/1971 - 5/31/1971	19	2	0	44	0	2	2	0
6/1/1971 - 6/30/1971	19	2	0	44	0	2	2	0
7/1/1971 - 7/31/1971	19	2	0	44	0	2	2	0
8/1/1971 - 8/31/1971	24	2	0	43	0	2	3	0
9/1/1971 - 9/30/1971	21	2	0	43	0	2	2	0
10/1/1971 - 10/31/1971	22	2	0	43	0	2	2	0
11/1/1971 - 11/30/1971	25	2	0	43	0	2	3	0
12/1/1971 - 12/15/1971	22	2	0	43	0	2	3	0
	259	21	0	524	0	28	29	0

Based on Mr. Welch's documented exposure to TCE, his diagnosis of Parkinson's disease is as least as likely as not to be related exposure to toxic levels of TCE at Camp Lejeune.

Mr. Welch was exposed to the chemicals at Camp Lejeune via consumption/ingestion for approximately 313 days, although we know he was also exposed via dermal and inhalation routes. He undoubtedly had inhalation exposure to TCE as well which is felt to be even more toxic.

This constitutes a substantial exposure. This opinion is based on the (1) amount of exposure (the levels of the chemicals in the water and how often Mr. Welch was consuming the

water), (2) the duration of exposure, (3) the intensity of the exposure (as shown by the ATSDR water modeling data and other data as to the levels of the chemicals in the water) and (4) the frequency Mr. Welch was exposed in his day to day life at Camp Lejeune.

I was able to determine that Mr. Welch had substantial exposure just based upon the records at issue, Mr. Welch’s deposition and the ATSDR water modeling reports. However, I additionally reviewed exposure charts provided to me from Plaintiff’s expert Dr. Kelly Reynolds. Dr. Reynolds put together charts that detail a reasonable estimated dose of ingestion exposure for Mr. Welch. These charts support my opinion that Mr. Welch sustained a substantial exposure that was causally related to his Parkinson’s disease. For example, Dr. Reynolds charts indicate that Mr. Welch would have likely ingested the following amounts of the toxins at issue in this case:⁹⁹

		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition	Chart 4 Deposition/FM
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Hadnot Point					
TCE	259	6,951	8,248	10,644	13,524
PCE	0	0	0	0	0
VC	0	0	0	0	0
BZ	29	765	908	1,171	1,488
Terawa Terrace					
TCE	21	543	1,123	1,522	1,888
PCE	524	13,660	28,245	38,262	47,469
VC	28	732	1,514	2,051	2,544
BZ	0	0	0	0	0
Totals HP & TT					
TCE	280	7,494	9,371	12,166	15,412
PCE	524	13,660	28,245	38,262	47,469
VC	28	732	1,514	2,051	2,544
BZ	29	765	908	1,171	1,488

As with many diseases associated with toxin exposure, manifestation of symptoms comes years after the exposure. Mr. Welch had an approximate 11-month exposure to toxic levels of TCE while serving at Camp Lejeune between November 18, 1970 and December 15, 1971.¹⁰⁰ In fact, his work area was adjacent to the most highly contaminated water supply on the base (Hadnot Point) and his base apartment was supplied with contaminated base water. Epidemiologic studies outlined above have shown a statistically significant association between toxic TCE exposure and Parkinson’s disease. This is further supported by extensive animal research that documents TCE exposure causing the same pathology and mechanism of cell death that occurs in humans with PD. Using the Bradford Hill framework applied by the general causation experts like Dr. Cannon, Dr. De Miranda, Dr. Miller, Dr. Costa and Dr. Boehme (strength of association, consistency, temporality, biologic gradient, plausibility, coherence experimental evidence and analogy), the

evidence strongly supports a causal relationship between TCE exposure and development of PD.

VI. Opinion on Causation

Based on my education, training, and expertise as a neurologist and a movement disorder specialist, and my review of the materials addressed and referenced in this report, and the materials listed on my reliance list as Exhibit A, to a reasonable degree of medical certainty, I conclude that Mr. Welch's Parkinson's disease is as least as likely as not due to his exposure to TCE at Camp Lejeune for an approximate 11 month period from November 18, 1970 to December 15, 1971.¹⁰⁰ [MOU1] [hs2]

A. Future Care Considerations

His symptoms of Parkinson's disease continue to progress. He has required more medications to control his motor symptoms. Despite donepezil, he has had some progression of his cognitive deficits which is likely to progress to dementia in the future. He currently has limitations with short term memory, word fluency and visual spatial skills. He remains independent with his ADLs but requires some prompting for medication compliance. These factors will undoubtedly progress over time necessitating increased care and cost. To a reasonable degree of certainty based on my experience caring for patients with Parkinson's disease, it is likely he will need:

1. Transportation assistance as he will likely be unable to drive within a year
2. Assistance with dressing and bathing within 2-3 years
3. Skilled nursing care within 4-5 years.

These considerations are based on anticipated decline from PD but not on any potential acute events such as a fall or aspiration pneumonia which would likely accelerate his need for skilled nursing care. Progression of his cognitive decline to dementia will require constant supervision reducing Mr. Welch's sense of agency and quality of life while making care decisions more difficult for the family.

B. Life Expectancy Considerations

Life expectancy for those with Parkinson's disease is dependent on many factors including age of onset, progression of disease, comorbidities, etc. Studies have clarified those patients with Parkinson's disease live fewer years than age and sex matched population comparators. Men with Parkinson's disease at age 75 live on average 5 more years while those without Parkinson's disease live 10 more years.⁹¹

February 5, 2025

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EXHIBIT A

1. Gash DM; Bohn MC; Fiandaca MS; Okawara SH; Kordower JH; Notter MFD; Snyder J; **Schwarz HB**; Shoulson I. "Adrenal Medullary Implantation Promotes Tyrosine Hydroxylase Immunoreactivity in Host Striatum of MPTP Animal Models of Parkinsonism". *Archives of Neurology*. 1988; 45(7): 810-811
2. Cudkovicz, M. and (2010), A futility study of minocycline in Huntington's disease[‡]. *Mov. Disord.*, 25: 2219-2224. <https://doi.org/10.1002/mds.23236>
3. Huntington Study Group HART Investigators. A randomized, double-blind, placebo-controlled trial of pridopidine in Huntington's disease. *Mov Disord*. 2013 Sep;28(10):1407-15. doi: 10.1002/mds.25362. Epub 2013 Feb 28. PMID: 23450660.
4. Korn RE, Wagle Shukla A, Katz M, Keenan HT, Goldenthal S, Auinger P, Zhu W, Dodge M, Rizer K, Achey MA, Byrd E, Barbano R, Richard I, Andrzejewski KL, **Schwarz HB**, Dorsey ER, Biglan KM, Kang G, Kanchana S, Rodriguez R, Tanner CM, Galifianakis NB. Virtual visits for Parkinson disease: a multi-center noncontrolled cohort. *Neurol Clin Pract*. 2017 Aug;7(4):283-295. doi: 10.1212/CPJ.0000000000000371.
5. Dorsey, ER et al. "National Randomized Controlled Trial of Virtual House Calls for People with Parkinson's Disease: Interest and Barriers". *Telemedicine and e-Health*. 2016;22 (7):590-598.
6. Dorsey, ER;: Wagner, JD; Bull, MT; Rizzieri, A; Grischkan, J; Achey, MA; Sherer, T; Chowdhury S; Meunier, C; Cappelletti, L; Rocher, C; Richard, IH; **Schwarz, H**; Kang, G; Ahmad, SH; Biemiller, RA; Biglan, KM. "Feasibility of Virtual Research Visits in Fox Trial Finder". *Journal of Parkinson's Disease*. 2015; 5: 505-515
7. Dorsey, E.R., Kinel, D., Pawlik, M.E., Zafar, M., Lettenberger, S.E., Coffey, M., Auinger, P., Hylton, K.L., Shaw, C.W., Adams, J.L., Barbano, R., Braun, M.K., **Schwarz, H.B.**, Lawrence, B.P., Kieburtz, K., Tanner, C.M., de Miranda, B.R. and Goldman, S.M. (2024), Dry-Cleaning Chemicals and a Cluster of Parkinson's Disease and Cancer: A Retrospective Investigation. *Mov Disord*, 39: 606-613. <https://doi.org/10.1002/mds.29723>
8. Hershey AD, Armand CE, Berk T, Burch R, Buse DC, Dougherty C, Marmura MJ, Minen MT, Robblee J, **Schwarz HB**. Updated process for American Headache Society Guidelines. *Headache*. 2021 Apr;61(4):565-566. doi: 10.1111/head.14093. PMID: 33891346
9. Hirsch L, Jette N, Frolkis A, Steeves T, Pringsheim T. The Incidence of Parkinson's Disease: A Systematic Review and Meta-Analysis. *Neuroepidemiology*. 2016;46(4):292-300. doi:10.1159/000445751
10. Roos DS, Klein M, Deeg DJH, Doty RL, Berendse HW. Prevalence of Prodromal Symptoms of Parkinson's Disease in the Late Middle-Aged Population. *J Park Dis*. 2022;12(3):967-974. doi:10.3233/JPD-213007
11. Antony, P. M., Diederich, N. J., Krüger, R. & Balling, R. The hallmarks of Parkinson's disease. *Febs j*280, 5981-5993 (2013). <https://doi.org:10.1111/febs.12335>

12. Henderson, M. X., Trojanowski, J. Q. & Lee, V. M. α -Synuclein pathology in Parkinson's disease and related α -synucleinopathies. *Neurosci Lett* 709, 134316 (2019). <https://doi.org/10.1016/j.neulet.2019.134316>
13. Lee, J. K., Tran, T. & Tansey, M. G. Neuroinflammation in Parkinson's disease. *J Neuroimmune Pharmacol* 4, 419-429 (2009). <https://doi.org/10.1007/s11481-009-9176-0>
14. Tansey, M. G. et al. Inflammation and immune dysfunction in Parkinson disease. *Nat Rev Immunol* 22,657-673 (2022). <https://doi.org/10.1038/s41577-022-00684-6>
15. Ye H, Robak LA, Yu M, Cykowski M, Shulman JM. Genetics and Pathogenesis of Parkinson's Syndrome. *Annu Rev Pathol.* 2023 Jan 24;18:95-121. doi: 10.1146/annurev-pathmechdis-031521-034145. Epub 2022 Sep 13. PMID: 36100231; PMCID: PMC10290758.
16. Pang SY, Ho PW, Liu HF, et al. The interplay of aging, genetics and environmental factors in the pathogenesis of Parkinson's disease. *Transl Neurodegener.* 2019;8(1):23. doi:10.1186/s40035-019-0165-9.
17. Lee AJ, Wang Y, Alcalay RN, et al. Penetrance estimate of LRRK2 p.G2019S mutation in individuals of non-Ashkenazi Jewish ancestry. *Mov Disord Off J Mov Disord Soc.* 2017;32(10):1432-1438. doi:10.1002/mds.27059
18. Padmakumar S, Kulkarni P, Ferris CF, Bleier BS, Amiji MM. Traumatic brain injury and the development of parkinsonism: Understanding pathophysiology, animal models, and therapeutic targets. *Biomed Pharmacother.* 2022 May;149:112812. doi: 10.1016/j.biopha.2022.112812. Epub 2022 Mar 12. PMID: 35290887; PMCID: PMC9050934.
19. Langston JW et al. Chronic Parkinsonism in humans due to a product of meperidine analog synthesis. *Science* 219, 979-980, 1983.
20. Langston JW. The MPTP story. *J. Parkinsons Dis.* 7 (S1), S11-S19, 2017.
21. Paul, K.C., Krolewski, R.C., Lucumi Moreno, E. et al. A pesticide and iPSC dopaminergic neuron screen identifies and classifies Parkinson-relevant pesticides. *Nat Commun* 14, 2803 (2023). <https://doi.org/10.1038/s41467-023-38215-z>
22. Pyatha S, Kim H, Lee D, Kim K. Association between Heavy Metal Exposure and Parkinson's Disease: A Review of the Mechanisms Related to Oxidative Stress. *Antioxidants.* 2022; 11(12):2467. <https://doi.org/10.3390/antiox11122467>
23. Krzyzanowski B, Searles Nielsen S, Turner JR, Racette BA. Fine Particulate Matter and Parkinson Disease Risk Among Medicare Beneficiaries. *Neurology.* 2023 Nov 21;101(21):e2058-e2067. doi: 10.1212/WNL.0000000000207871. Epub 2023 Oct 30. Erratum in: *Neurology.* 2024 Jul 9;103(1):e209596. doi: 10.1212/WNL.0000000000209596. PMID: 37903644; PMCID: PMC10663024.
24. Guehl D et al. Trichloroethylene and parkinsonism: a human and experimental observation. *Eur. J. Neurol.* 6, 609-611, 1999.
25. Gash, D.M., Rutland, K., Hudson, N.L., Sullivan, P.G., Bing, G., Cass, W.A., Pandya, J.D., Liu, M., Choi, D.-Y., Hunter, R.L., Gerhardt, G.A., Smith, C.D., Slevin, J.T. and Prince, T.S. (2008), Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. *Ann Neurol.*, 63: 184-192. <https://doi.org/10.1002/ana.21288>
26. Goldman, S.M., Quinlan, P.J., Ross, G.W., Marras, C., Meng, C., Bhudhikanok, G.S., Comyns, K., Korell, M., Chade, A.R., Kasten, M., Priestley, B., Chou, K.L., Fernandez, H.H., Cambi, F., Langston, J.W. and Tanner, C.M. (2012), Solvent exposures and

- parkinson disease risk in twins. *Ann Neurol.*, 71: 776-784. <https://doi.org/10.1002/ana.22629>
27. Bove FJ et al. Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study. *Environ. Health* 13, 10, 2014a
 28. Bove FJ et al. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. *Environ. Health* 13, 68, 2014b
 29. Goldman SM, Weaver FM, Stroupe KT, et al. Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune. *JAMA Neurol.* 2023;80(7):673–681. doi:10.1001/jamaneurol.2023.1168
 30. Goldman, S.M., Weaver, F.M., Gonzalez, B., Stroupe, K.T., Cao, L., Colletta, K., Brown, E.G. and Tanner, C.M. (2024), Parkinson's Disease Progression and Exposure to Contaminated Water at Camp Lejeune. *Mov Disord*, 39: 1732-1739. <https://doi.org/10.1002/mds.29922>
 31. Bove FJ et al. Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study. *Environ. Health* 23, 61, 2024
 32. Dorsey ER et al. Trichloroethylene: an invisible cause of Parkinson's disease? *J. Park. Dis.* 13, 203-2018, 2023
 33. Keane PC et al. Trichloroethylene and its metabolite TaClo lead to neurodegeneration of substantia nigra dopaminergic neurons; effects of wild type and human A30P mutant alpha-synuclein mice. *Neurosci. Lett.* 711, 134437, 2019.
 34. De Miranda BR et al. The industrial solvent trichloroethylene induces LRRK2 kinase activity and dopaminergic neurodegeneration in a rat model of Parkinson's disease. *Neurobiol. Dis.* 153, 105312, 2021
 35. Adamson A et al. Low-dose inhalation exposure to trichloroethylene induces dopaminergic neurodegeneration in rodents. *Toxicol. Sci.* 196, 218-228, 2023
 36. Liu M et al. Trichloroethylene and Parkinson's disease: risk assessment. *Mol. Neurobiol.* 55, 6201-6214, 2018
 37. Chen H et al. Environmental triggers of Parkinson's disease- Implications of the Braak and dual-hit hypotheses. *Neurobiol. Dis.* 163, 105601, 2022
 38. Zafar S, Yaddanapudi SS. Parkinson Disease. [Updated 2023 Aug 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK470193/>
 39. Kumaresan M, Khan S. Spectrum of Non-Motor Symptoms in Parkinson's Disease. *Cureus.* 2021 Feb 11;13(2):e13275. doi: 10.7759/cureus.13275. PMID: 33728210; PMCID: PMC7949722.
 40. Radad K, Moldzio R, Krewenka C, Kranner B, Rausch WD. Pathophysiology of non-motor signs in Parkinson's disease: some recent updating with brief presentation. *Explor Neuroprot Ther.* 2023;3:24–46. <https://doi.org/10.37349/ent.2023.00036>
 41. Zahoor I, Shafi A, Haq E. Pharmacological Treatment of Parkinson's Disease. In: Stoker TB, Greenland JC, editors. *Parkinson's Disease: Pathogenesis and Clinical Aspects* [Internet]. Brisbane (AU): Codon Publications; 2018 Dec 21. Chapter 7. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK536726/> doi: 10.15586/codonpublications.parkinsonsdisease.2018.ch7

42. Zhou, Z.D., Yi, L.X., Wang, D.Q. *et al.* Role of dopamine in the pathophysiology of Parkinson's disease. *Transl Neurodegener* **12**, 44 (2023).
<https://doi.org/10.1186/s40035-023-00378-6>
43. Klein C, Westenberger A. Genetics of Parkinson's disease. *Cold Spring Harb Perspect Med.* 2012 Jan;2(1):a008888. doi: 10.1101/cshperspect.a008888. PMID: 22315721; PMCID: PMC3253033.
44. Ye Q, Wen Y, Al-Kuwari N, Chen X. Association Between Parkinson's Disease and Melanoma: Putting the Pieces Together. *Front Aging Neurosci.* 2020 Mar 10;12:60. doi: 10.3389/fnagi.2020.00060. PMID: 32210791; PMCID: PMC7076116.
45. <https://www.parkinson.org/understanding-parkinsons/movement-symptoms/dyskinesia>
46. https://www.physio-pedia.com/Hoehn_and_Yahr_Scale
47. Janvin CC, Larsen JP, Aarsland D, Hugdahl K (2006) Subtypes of mild cognitive impairment in Parkinson's disease: progression to dementia. *Mov Disord* 21:1343–1349.
48. <https://www.epa.gov/newsreleases/biden-harris-administration-announces-latest-actions-under-nations-chemical-safety-law#:~:text=EPA%20is%20finalizing%20its%20prohibition,commercial%20and%20a1%20consumer%20products.>
49. Shin HW, Chung SJ. Drug-induced parkinsonism. *J Clin Neurol.* 2012 Mar;8(1):15-21. doi: 10.3988/jcn.2012.8.1.15. Epub 2012 Mar 31. PMID: 22523509; PMCID: PMC3325428.
50. Beckers, M., Bloem, B.R. & Verbeek, M.M. Mechanisms of peripheral levodopa resistance in Parkinson's disease. *npj Parkinsons Dis.* **8**, 56 (2022).
<https://doi.org/10.1038/s41531-022-00321-y>
51. Kenborg L, Rugbjerg K, Lee PC, Ravnskjaer L, Christensen J, Ritz B, Lassen CF. Head injury and risk for Parkinson disease: results from a Danish case-control study. *Neurology.* 2015 Mar 17;84(11):1098-103. doi: 10.1212/WNL.0000000000001362. Epub 2015 Feb 13. PMID: 25681453; PMCID: PMC4371406.
52. Gardner RC, Byers AL, Barnes DE, Li Y, Boscardin J, Yaffe K (2018) Mild TBI and risk of Parkinson disease: A Chronic Effects of Neurotrauma Consortium Study. *Neurology* 90:e1771–e1779.
53. Gardner RC, Burke JF, Nettiksimmons J, Goldman S, Tanner CM, Yaffe K. Traumatic brain injury in later life increases risk for Parkinson disease. *Ann Neurol.* 2015 Jun;77(6):987-95. doi: 10.1002/ana.24396. Epub 2015 Mar 28. PMID: 25726936; PMCID: PMC4447556.
54. Virtual visit with Mr. Robert Welch on December 19, 2024
55. https://www.physio-pedia.com/Hoehn_and_Yahr_Scale
56. Janvin CC, Larsen JP, Aarsland D, Hugdahl K (2006) Subtypes of mild cognitive impairment in Parkinson's disease: progression to dementia. *Mov Disord* 21:1343–1349
57. Shin HW, Chung SJ. Drug-induced parkinsonism. *J Clin Neurol.* 2012 Mar;8(1):15-21. doi: 10.3988/jcn.2012.8.1.15. Epub 2012 Mar 31. PMID: 22523509; PMCID: PMC3325428
58. <https://www.ninds.nih.gov/health-information/disorders/traumatic-brain-injury-tbi>
59. General Causation Report by Briana Di Miranda, PhD 12/8/2024
60. General Causation Report by Lucio Costa, PhD 12/6/2024
61. General Causation Report by Gary W. Miller, PhD 12/7/2024

62. General Causation Report by Amelia Boehme, PhD, MSPH 12/8/2024
63. General Causation Report by Jason Cannon, PhD 12/12/2024
64. Bose A, Petsko GA, Eliezer D. Parkinson's Disease and Melanoma: Co-Occurrence and Mechanisms. *Journal of Parkinson's Disease*. 2018;8(3):385-398. doi:10.3233/JPD-171263
65. Olsen JH, Friis S, Frederiksen K (2006) Malignant melanoma and othertypes of cancer preceding Parkinson disease. *Epidemiology* 17, 582-587.
66. Bertoni JM, Arlette JP, Fernandez HH, et al. Increased Melanoma Risk in Parkinson Disease: A Prospective Clinicopathological Study. *Arch Neurol*. 2010;67(3):347-352. doi:10.1001/archneurol.2010.1
67. <https://www.webmd.com/parkinsons-disease/motor-fluctuations>
68. [https://wwwn.cdc.gov/TSP/PHS/PHS.aspx?phsid=171&toxid=30#:~:text=EPA%20set%20a%20maximum%20contaminant,%2FL%3B%205%20ppb\).](https://wwwn.cdc.gov/TSP/PHS/PHS.aspx?phsid=171&toxid=30#:~:text=EPA%20set%20a%20maximum%20contaminant,%2FL%3B%205%20ppb).)
69. <https://matracking.ehs.state.ma.us/Environmental-Data/Water-Quality/pce.html#:~:text=Exposure%20to%20PCE%20at%20levels,long%2Dterm%20exposures%20to%20PCE.>
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86. Medical Records from Dr. C Neff, 0153_WELCH_0000000263, pg 33-40
87. Medical Records from Dr. C Neff, 0153_WELCH_0000000263, pg 51
88. Medical Records from Dr. C Neff, 0153_WELCH_0000000263, pg103-108
89. Deposition of Dr. C Neff MD on April 26, 2024, pg 37.
90. Dorsey ER et al. [Journal of Parkinson's Disease](#), vol. 14, no. 3, pp. 363-381, 2024 DOI: 10.3233/JPD-240019
91. Dommershuijsen, L.J., Darweesh, S.K.L., Ben-Shlomo, Y. *et al.* The elephant in the room: critical reflections on mortality rates among individuals with Parkinson's disease. *npj Parkinsons Dis*. 9, 145 (2023). <https://doi.org/10.1038/s41531-023-00588-9>
92. Sarkis RA. Anti-Seizure Medications on Trial Again: Accused of Parkinson's Disease! *Epilepsy Curr*. 2023 Jun 13;23(5):277-279. doi: 10.1177/15357597231180068. PMID: 37901782; PMCID: PMC10601026.

93. ATSDR, CAMP LEJEUNE DRINKING WATER, U.S. MARINE CORPS BASE
CAMP LEJEUNE, NORTH CAROLINA JANUARY 20, 2017
94. General Causation Report of Dr. Jason Cannon
95. General Causation Report of Dr. Briana Di Miranda
96. General Causation Report of Dr. Amelia Boehme
97. General Causation Report of Dr. Luigi Costa
98. General Causation Report of Dr. Gary Miller
99. Report of Kelly Reynolds, MSPH, PhD
100. 01503_WELCH_0000000133 to 01503_WELCH_0000000135

Heidi Beck Schwarz, M.D., FAAN

University of Rochester Medical Center | School of Medicine and Dentistry |

Email: heidi_schwarz@urmc.rochester.edu

EDUCATION

MD Medicine | Univ Rochester Sch Med/Dent | 1983

BA Chemistry | Mount Holyoke College | 1979 Magna Cum Laude

POST-DOCTORAL TRAINING AND RESIDENCY

Fellow and Instructor in Movement and Inherited Neurologic Disease, Department of Neurology, University of Rochester | *June 1988 - June 1989*

Chief Resident in Neurology, University of Rochester | *July 1987 - June 1988*

Assistant and Associate Resident in Neurology, University of Rochester | *July 1985 - June 1987*

Resident and Assistant Resident in Medicine University of Rochester, Strong Memorial Hospital | *June 1983 - June 1985*

FELLOWSHIP AWARDS

POST-DOCTORAL

Fellow and Instructor in Movement and Inherited Neurologic Disease, Department of Neurology, University of Rochester | *07/01/1988 - 06/30/1989*

CERTIFICATIONS

BOARD

Headache Medicine | *October 2012 - October 2024*

American Board of Psychiatry and Neurology (Neurology Certificate), certificate number 32287 | *November 1989*

National Board of Medical Examiners, Certificate number 279704 | *1983*

OTHER

NIH Stroke Scale Certification | *September 2005 - Present*

LICENSURE

Iowa Medical License | *2015 - 2018*

Missouri Medical License | *2015 - 2018t*

Pennsylvania Medical License | *2015 - 2018*

West Virginia Medical License | *2015 - 2018*

New York State Medical License, 164731 | *1984 - Present*

FACULTY APPOINTMENTS

Professor of Clinical Neurology (Part-Time) | Neurology | Headache Medicine | SMD | *2014 - present*

Clinical Associate Professor | Neurology | SMD | *2010 - 2014*

Associate Professor | Neurology | Highland Neurology | SMD | *2008 - 2010*

Assistant Professor | Neurology | Mind | SMD | *2003 - 2008*

APPOINTMENTS

ACADEMIC - INTERNAL

Associate Chair for Community Affairs of the Department of Neurology, University of Rochester | *October 2006 - December 2009*

Director of the Stroke Center, Highland Hospital | *01/30/2005 - 03/15/2010*

Chair, Department of Neurology at Highland Hospital | *August 2004 - January 2010*

Attending Neurologist at Strong Memorial Hospital | *January 2003 - Present*

OTHER EMPLOYMENT

Legal consultation on malpractice and worker's compensation cases, *2000-present*

Topic Editor for General Neurology, Dynamed, *2018-present*

MEMBERSHIPS

CORPORATE

Board member of Midlakes Management Corporation | *January 1995 - January 2005*

PROFESSIONAL

Active member American Headache Society | *2011 - 2024*

American Stroke Association | *June 2008 - 2010*

Fellow of American Academy of Neurology | *2008 - Present*

Canandaigua Medical Society (Past President, Current Secretary/Treasurer) | *1995 - Present*

New York Medical Society | *1990 - 2011*

Ontario and Monroe County Medical Society | *1990 - 2011*

Active member of the American Academy of Neurology | 1988 – 2008

LECTURESHIPS

LOCAL

Arnot Ogden Health System Grand Rounds: Implicit Bias: What is it and why is it important? April 2019
Canandaigua Medical Society: Implicit Bias, April 2019
Accountable Health Partners Clinical Grand Rounds: Best Practice Guidance on Outpatient Treatment of Migraine, March 2019
Batavia Regional Neuroscience Conference: Update on Migraine and Memory Disorders, October 2018
URMC Neuroscience APP Symposium lectures on Clinical Neuroanatomy, Neurologic Exam, Headache, Neuroanatomy of Coma | *November 2015 - December 2015*
Headache Lectures to Neurology residents and ED residents | *2015 - Present*
Women in Neurology Series, The Journey of a Wayward Neurologist | *2014*
NYS Neurologic Society Annual Meeting, PQRS for Neurologists | *2013*
URMC Grand Rounds, Update in Headache | *2013*
Unity Health System Grand Rounds: Update in Movement Disorders, Part 1 | *2011*
Highland Hospital Primary Care Grand Rounds: Neurologic Complications of Psychiatric Drugs | *02/23/2010 - 02/23/2010*
Regional Conference of the Flying Physicians Association: Gender Differences in Stroke | *2010*
Annual Lifecare Seminar, Waterloo, NY Gender Differences in Stroke | *11/05/2009 - 11/05/2009*
Clinical Challenges of Women's Health: Stroke Therapy in Women (lecture for providers) | *09/15/2009 - 09/15/2009*
Upstate NY Stroke Care Symposium: Emergency evaluation and management of patients with suspected stroke | *06/05/2009*
What Every Woman Should Know About Stroke | *09/19/2008 - 09/19/2008*
"Gender Differences in Stroke" presented at the Upstate NY Stroke Care Symposium. | *05/30/2008 - 05/30/2008*
Lecture for the Neurology for Primary Care Symposium: "Gender Differences in Stroke" | *12/06/2007 - 12/06/2007*
Presentation at the Alessi Health Fair: What Every Woman Should Know About Stroke | *10/04/2007*
Monroe Community Hospital Geriatric Grand Rounds: Management of Acute Stroke in the Geriatric Population | *01/17/2007 - 01/17/2007*
Developing a Stroke Center: Upstate NY Stroke Care Symposium in Rochester, NY | *05/25/2006*
Hypothermic Treatment for Cardiac Arrest; Upstate NY Stroke Care Symposium | *05/25/2006*
Treatment of Acute Stroke: Highland Hospital Grand Rounds | *10/11/2005*
Update in Headache" presented at Noyes Memorial Hospital Grand Rounds | *May 2005*
Therapeutic Hypothermia: To Freeze or Not To Freeze; Highland Hospital Grand Rounds | *03/15/2005*
Migraine Update 2004: A New Chronic Disease; Neurology for Primary Care Provider XI | *12/02/2004*
Monthly Migraines in Women" presented at the Canandaigua VA | *December 2004*
Regional Conference of the Flying Physicians Association: Update in Movement Disorders, Bedford Springs, PA

NATIONAL

Thriving as Medicine Evolves: AHS Scottsdale Meeting November 2021
Not Dead Yet: Late Career Options for Neurology: AAN annual meeting, May 2019
Neuromodulation Therapy for Headache: AAN annual meeting, May 2019
Industry Round Table presentation: AAN Commitment to Wellness, April 2018
The Ripple Effect of Positive Psychology: AAN annual meeting 2018 and 2019
Live Well, Lead Well AAN Leadership Program, Co-director, 2017-2018
Panelist on Second Opinion: Mystery Case, Public Television 2016
What in the World is Positive Psychology and How Can It Help Me?. AAN annual meeting 2016
Resident Burnout and Wellness, AAN annual meeting 2016
APP symposium: Update in Headache, AAN annual meeting | *2015*
iTalk on APP's in Neurology Practice, AAN annual meeting | *2015*
Telemedicine: Emerging Business and Practice Models. AAN Breakthrough Meeting, Phoenix | *2015*
AAN Webinar on APPs in Neurologic Practice | *2014*

Practice Colloquium Director, AAN Annual Meeting | 2014
Resident and Fellow Early Career Breakfast, AAN Annual Meeting: The State of Neurology | 2014
The State Resident and Fellow Early Career Luncheon, AAN Annual Meeting of Neurology, The State of Neurology | 2013
Practice Colloquium Director, AAN Annual Meeting | 2012
18th Annual Educational Conference: Association of Administrative Law Judges, Buffalo NY Multiple Sclerosis, CVA and Other Neurologic Disorders | 10/07/2009 - 10/07/2009

COMMUNITY SERVICE

Brain Health Workshops, Ongoing community based resources and presentations in Rochester, NY underserved communities, 2022-present
Real Talk with Martha Hope:Headache Feb 2021, Rochester Free Radio
Community Engagement Committee for URMC: 2021-2024
AAN Brain Health Fair volunteer: 2016-2018
Neurology Preceptor, URWell Clinic | 2015 - present
Speaking of Women's Health: What Every Woman Should Know About Stroke | 09/17/2009 - 09/17/2009
Presentation at Speaking of Women's Health: What Every Woman Should Know About Stroke | 09/19/2008 - 09/19/2008
Presented "Gender Differences in Stroke" to public through the Preferred Care Outreach Program on two occasions. | April 2008 - May 2008
Presentation for the general public at the Jim Alessi Health Fair: " What Every Woman Should Know About Stroke" | 10/03/2007 - 10/03/2007
Participant on the "Ask the Expert" Panel for the Epilepsy Foundation conference, "Faces of Epilepsy | 11/04/2006 - 11/04/2006
Two community lectures on the warning signs and treatment of stroke sponsored by American Stroke Association | April 2006 - May 2006
Board Member for the Dennis Morga Memorial Scholarship Fund | 2006
Emergency Medicine Service lectures in the Rochester area on Acute Mangement of Stroke for Emergency Personnel sponsored by American Stroke Association | 2005 - 2024
Panel member for the Epilepsy Foundation's annual community conference | November 2003 - November 2003

AWARDS AND HONORS

Senior Faculty Award, URMC Neurology, presented at AAN annual meeting 2016
Fellow, American Academy of Neurology | American Academy of Neurology, Minneapolis, MN | May 2008 - Present
Leadership Development Fellowship | American Academy of Neurology, Washington, D. C. | 03/01/2008 - 03/04/2008
A. B. Baker Teacher Recognition Award | American Academy of Neurology, Chicago, IL | March 2008 - Present
Chief Resident Teaching Award | Department of Neurology URMC, Rochester, NY | June 2004
Alpha Omeg Alpha Awardee | AOA | 1983 - Present
Citation for Fourth Year Medical Students | American Medical Women's Association, Rochester, NY | 1983
Mary P. Dole Fellowship | Mount Holyoke College Alumnae Association, Rochester, NY | 1981
Phi Beta Kappa Inductee | Phi Beta Kappa Society, South Hadley, MA | 1979 - Present
American Chemical Society Award | American Chemical Society, South Hadley, MA | 1979
Sigma Xi Inductee | Sigma Xi Society, South Hadley, MA | 1979
Mary Lyons Scholar | Mount Holyoke College, South Hadley, MA | 1979
Magna Cum Laude in Chemistry | Mount Holyoke College, South Hadley, MA | 1979
Sarah Williston Scholar | Mount Holyoke College, South Hadley, MA | 1977

CONTRIBUTIONS

EDUCATIONAL

Phase 2 and 3 evaluations for 2nd and 3rd year medical students 2022
Bedside Skills Exam, Neurology 3rd year Med Students: 2019-present
Annual Lectures to the Headache Fellows and Headache Division on Epidemiology, TAC, High and Low Pressure Headache, Neuromodulation and Emergency Management
Professor Rounds for Neurology Service at URM 2018-present
Development of Best Practice Guidance for Migraine Treatment for AHP, 2019
Precepting neurology resident firm (half day per week) | *March 2015 - Present*
Precept Neurology residents in URM Headache Center 2014 to Present
Bedside Skills Examinations for 3rd year Medical Students during Neurology Clerkship, 2010-present
Annual Lectures to Medical Students and Neurology Residents on Emergency Treatment of Migraine, TAC and Healthcare Policy 2014- present
Biannual lectures to Internal Medicine Resident at Unity Health: Migraines and Movement disorders | *2010 - 2013*
Medical Student Preceptor for URM in outpatient neurology | *2010 - present*
Morning report for the Neurology Service at URM | *January 2009 - 2010*
Precept Internal Medicine, Med Peds and Family Practice residents on a weekly basis in my office | *2005 - Present*
Attending at Morning Report (once/month) for the Internal Medicine Residents at Highland Hospital | *2005 - 2010*
Career Mentor to many of the neurology residents and medical students at the University of Rochester | *2003 - Present*
Provide lectures regularly to the medical students and the internal medicine residents on a variety of topics | *2003 - 2010*

HOSPITAL

Member of the Ad Hoc committee for Determination of Brain Death for Highland Hospital | *2006*
Member of the Strategic Planning Committee at Highland Hospital | *2006*
Chairwoman of the Stroke Committee at Highland Hospital and Director and Founder of the Stroke Center at Highland Hospital. | *January 2005 - 2010*

PROFESSIONAL

AAN Foundations for Success: AAN Annual Meeting 2017-2019
AAN Presentation to Medical Students on Headache Careers: AAN Annual Meeting 2019
Neurology on the Hill, 2016-present
Member of the Rochester Area Stroke Task Force as part of the American Stroke Association | *2005 - 2010*

COURSES

TAUGHT

Being a Resilient Leader, Course Co-Director, AAN Annual Meeting 2017 and 2018
URM Neuroscience APP Symposium lectures on Clinical Neuroanatomy, Neurologic Exam, Headache, Neuroanatomy of Coma | *November 2015 - December 2015*
Mindfulness in Medicine taught several times per year to both Neurology Residents and Third Year Medical Students. Various modules including: Professionalism, How Doctors Think, Healing and Suffering, Self Care and Burnout, Errors and Bad Outcomes. | *08/01/2007 - Present*

TAKEN

Mindfulness for Physician Educators which included exposure to various techniques such as narratives, story telling, reflection, stress reduction, observational skills which will be used as tools for teaching Mindfulness in Medicine to residents and students. | *February 2007 - June 2007*

PROFESSIONAL ASSIGNMENTS

ADMINISTRATIVE

Director of the Highland Hospital EEG lab | *August 2004 - February 2010*

RESEARCH GRANTS

PI: Dorsey, ER, Regional Multi-Disciplinary Care Network for Parkinson Disease, Sponsor: Greater Rochester Health Foundation and Safra Philanthropic Foundation, 2017

PI: Dorsey, ER | Investigators: Boyd, C; Schmidt, P; Willis, A; Biglan, K; Beck, C | Title: Using technology to deliver multi-disciplinary care to individuals with Parkinson disease in their homes | Sponsor: PCORI | Grant Type: non-NIH | Grant ID: AD-12-11-4701 | Awarded: 09/23/2013 | 2014 – 2016

PI: Dorsey, ER | Investigators: Biglan, K | Title: Using Telemedicine to Deliver Patient-Centered Care to Patient with Parkinson's Disease Anywhere | Sponsor: Davis Phinney Foundation for Parkinson's | Grant Type: non-NIH | Awarded: 08/01/2013 | 2014 – 2016

PI: Karl Kieburz MD, MPH | Investigators: Anderson K, Bordelon Y, Chouinard S, Corey-Bloom J, Dure L, Guttman M, Hyson C, Kostyk S, Leavitt B, Kumar R, Mendis T, O'Suilleabhain P, Paulsen J, Revilla F, Rosenblatt A, Schwarz H, Shannon K, Wieler M, Wojcieszek J, Wright Willis A (HSG) | Title: A multi-center, North American, randomized, double blind, parallel group study comparing three doses of ACR16 versus placebo for symptomatic treatment of Huntington Disease | Sponsor: NeuroSearch Sweden AB | Grant Type: non-NIH | Grant ID: 77,419 | Awarded: 06/24/2008 | 10/15/2008 – 2010

PI: Cudkowicz, Merit E. | Investigators: Schwarz, Heidi; Mazzoni, Pietro; Rosas, H. Diana; Sanchez-Ramos, Juan; Paulson, Henry; Perlmutter, Joel; Furtado, Sarah; Higgins, Donald; Leavitt, Blair; Seeberger, Lauren; Dure, Leon; Ashizawa, Tetsuo | Title: A Multicenter Double-Blind, Pilot Study Of Minocycline in Huntington's Disease (DOMINO) | Sponsor: FDA | Grant Type: non-NIH | Grant ID: IND No: 60,943 | Awarded: October 2005 | 01/01/2006 - 09/30/2008

PRESENTATIONS

Poster presentation: Community-Driven Brain Health Workshop Series: A Novel Approach to Brain Health Promotion in Rochester's Historically Marginalized Communities; Schwarz, HB et al; AAN Annual Meeting, April 2024

Community-Driven Brain Health Workshop Series: A Novel Approach to Brain Health Promotion in Rochester's Historically Marginalized Communities; April 2024, AAN Annual Meeting

Wellness at Any Career Stage: Preparing for Retirement; April 2024, AAN Annual Meeting

Poster presentation: Community-Driven Brain Health Workshop Series in Historically Marginalized Communities: A Novel Approach to Brain Health Promotion and Education; Zizzi C et al, ANA Annual Meeting 2023

Neurology Grand Rounds at University of Minnesota, Wellbeing: The Newest Vital Sign in Health Care, 2/2020

Neurology Junior Faculty Presentation: Finding Eudamonia, URM 4/2017

Teaching the Neurologic Exam to Medicine Residents, Annually, Unity Health System | 2014

Parkinson's Disease Lecture to the Medical Residents, Unity Health System | 2011

Highland Hospital Grand Rounds: Stroke: The New Frontier-Opening the therapeutic window beyond 3 hours.

| 05/12/2009

"Gender Differences in Stroke" presented at Unity Grand Rounds | 12/23/2008 - 12/23/2008

Lecture to the Neurology Residents: "Gender Differences in Stroke" | 01/10/2008 - 01/10/2008

Brain Death Determination (using the new SMH criteria) for the Critical Care Fellows and Attendings, annually.

| 06/05/2007 - Present

Lecture to the Neurology Residents on Determination of Brain Death | 03/30/2007 - 03/30/2007

An Update in Headaches for the Medicine and FP residents at HH, annually. | 01/03/2007 - Present

Mindfulness teaching with third year medical students, "How Doctors Think", 4 times per year | 2007 - Present

Clinical Case Presentation: Gluten Sensitivity presenting as Neurological Disease | 09/28/2006

Practice Options presented to the Neurology Residents, University of Rochester | 06/16/2006

Stroke Presentation for Clinical Grand Rounds at Highland Hospital | 05/19/2006

Highland Neurology Quality Improvement Data presented at Neurology Clinical Steering Committee Meeting | May 2006

Hypothermic Treatment for Cardiac Arrest, presented to the Internal Medicine residents at HH | *March 2006*
Overview of the Highland Stroke Management Program presented to the NYS DOH for site visit for Stroke Center Designation | *01/17/2006*
Highland Hospital Neurology presented at the Neurology Staff Meeting | *11/10/2005*
Evaluation and Management of Acute Stroke for the FF Thompson medical staff | *October 2005*
General Neurology at URMC" presented at Neurology Department Retreat | *September 2005*
Evaluation and Management of Acute Stroke presented on multiple occasions to the Internal Medicine residents at Highland Hospital | *August 2005 - Present*
Overview of the Highland Stroke Center presented for Clinical Grand Rounds at Highland Hospital | *May 2005*
Update in Headache presented to the 3rd year medical students | *May 2005*
Wernicke's Encephalopathy and Korsakoff Syndrome" presented to Internal Medicine Residents at Highland Hospital | *October 2004*
Practice and Life Choices" presented to Neurology Residents, University of Rochester | *June 2003*
Neck and Back Pain: Quarterly lecture for 3rd year medical students | *2003 - Present*

EDITORIAL BOARDS

Ad Hoc Reviewer for Neurology and Neurology Clinical Practice | *2004 – present*
Reviewer for NAM Clinician Wellbeing and Resilience

COMMITTEES

DEPARTMENTAL

Neurology Department Faculty Development:Leadership Circle: 2019
Junior Faculty Mentor for Wellness for Residents 2019
Member of URMC Neurology Diversity Council 2017-present
Chief of Highland Neurology | *August 2004 - January 2010*
Clinical Steering Committee, Department of Neurology | *August 2004 - January 2010*
Executive Committee of the Department of Neurology | *August 2004 - January 2010*

HOSPITAL

Member of the Wellness Strategic Planning Committee for URMC 2017-present
Chairwoman of the Stroke Committee at Highland Hospital | *2005 - 2010*
Clinical Council at Highland Hospital | *August 2004 - January 2010*
Medical Executive Committee at Highland Hospital | *August 2004 - January 2010*

NATIONAL

Mentor in the Women in Neurology Program at AAN 2019-present
Co Chair of the AAN Joint Coordinating Council on Wellness 2019-present
Member AAN Conference Subcommittee | *2015 - 2019*
Member AAN Meeting Management Committee | *2015 – 2018*
Drug Pricing Task Force, AAN 2017-2019
Chair AAN Practice Committee | *2015 – 2019*
Chair person of the Child Neurology Topic Work Group, 2015-2019
Member Board of Directors, AANI | *2015 - 2019*
Co Chair AAN Task Force on Burnout | *2015 -2017*
Nominating Committee AAN | *2014*
Co Chair APP Work Group AAN | *2013 - 2015*
Value of Neurology Work Group AAN | *2012 - 2015*
Vice Chair AAN Practice Committee | *2011 - 2014*
AOA councilor for University of Rochester Medical School | *06/30/2009 - 2015*
Practice Committee Member of the American Academy of Neurology | *05/03/2009 - 04/17/2011*
Neurology on the Hill-AAN members advocate with legislators regarding issues involving our patients and the practice of neurology. | *03/04/2008, 03/04/2016, 2/27/2018*
American Academy of Neurology subcommittee on Stroke and Vascular Disease | *2007 - 2012*

INTERNATIONAL

Huntington's Study Group | *2004 - 2010*

OTHER

Member of the Rochester Area Stroke Task Force Subcommittee on Rehabilitation | *2007 - 2010*
Member of the Rochester Area Stroke Task Force which operates under the auspices of the American Heart Association/American Stroke Association | *2006 - 2010*
Committee member of the Neurology subcommittee of Rochester Individual Practice Association | *January 2000 – 2007*

JOURNAL ARTICLES

Dorsey ER, Kinel D, Pawlik ME, Zafar M, Lettenberger SE, Coffey M, Auinger P, Hylton KL, Shaw CW, Adams JL, Barbano R, Braun MK, **Schwarz HB**, Lawrence BP, Kiebertz K, Tanner CM, de Miranda BR, Goldman SM. Dry-Cleaning Chemicals and a Cluster of Parkinson's Disease and Cancer: A Retrospective Investigation. *Mov Disord*. 2024 Mar;39(3):606-613. doi: 10.1002/mds.29723. Epub 2024 Feb 23. PMID: 38389433.

Hershey AD, Armand CE, Berk T, Burch R, Buse DC, Dougherty C, Marmura MJ, Minen MT, Robblee J, **Schwarz HB**. Updated process for American Headache Society Guidelines. *Headache*. 2021 Apr;61(4):565-566. doi: 10.1111/head.14093. PMID: 33891346.

Calabresi P, Nigro P, **Schwarz HB**. A nurse-led model increases quality of care in Parkinson disease. *Neurology*. 2019 Apr 16;92(16):739-740. doi: 10.1212/WNL.0000000000007295. Epub 2019 Mar 22. PMID: 30902906.

LaFaver k, Miyasaki JM, Keran CM, Rheaume C, Gulya L, Levin KH, Jones EC, **Schwarz HB**, Molano JR, Hessler A, Singhal D, Shanafelt TD, Sloan JA, Novotny PJ, Cascino TL, Busis NA. Age and sex differences in burnout, Career satisfaction, and well-being in US neurologists. *Neurology*. 2018 Nov 13;91(20):e1928-e1941.

Levin KH, Shanafelt TD, Keran CM, Busis NA, Foster LA, Molano JRV, O'Donovan CA, Ratliff JB, **Schwarz HB**, Sloan JA, Cascino TL Author response: Burnout, career satisfaction, and well-being among US neurology residents and fellows in 2016. *Neurology*. 2018 Jan 30;90(5):248. doi: 10.1212/WNL.0000000000004882

Busis NA, Shanafelt TD, Keran CM, Levin KH, **Schwarz HB**, Molano JR, Vidic TR, Kass JS, Miyasaki JM, Sloan JA, Cascino TL Author response: Burnout, career satisfaction, and well-being among US neurologists in 2016. *Neurology*. 2017 Oct 10;89(15):1650-1651. doi: 10.1212/WNL.0000000000004484. Epub 2017 Oct 9.

Miyasaki JM, Rheaume C, Gulya L, Ellenstein A, **Schwarz HB**, Vidic TR, Shanafelt TD, Cascino TL, Keran CM, Busis NA. Qualitative study of burnout, career satisfaction, and well-being among US neurologists in 2016. *Neurology*. 2017 Oct 17;89(16):1730-1738. doi:10.1212/WNL.0000000000004526. Epub 2017 Sep 20.

Kerry H. Levin, MD; Tait D. Shanafelt, MD; Christopher M. Keran, BA; Neil A. Busis, MD; Laura A. Foster, MD; Jennifer Rose V. Molano, MD; Cormac O'Donovan, MD; Jeffery B. Ratliff, MD; **Heidi B. Schwarz, MD**; Jeff A. Sloan, PhD; Terrence L. Cascino, MD. Burnout, Career Satisfaction, and Well-Being Among U.S. Neurology Residents and Fellows in 2016. *Neurology*. 2017 Feb 21;88(8):797-808. doi: 10.1212/WNL.0000000000003640. Epub 2017 Jan 25

Korn RE, Wagle Shukla A, Katz M, Keenan HT, Goldenthal S, Auinger P, Zhu W, Dodge M, Rizer K, Achey MA, Byrd E, Barbano R, Richard I, Andrzejewski KL, **Schwarz HB**, Dorsey ER, Biglan KM, Kang G, Kanchana S, Rodriguez R, Tanner CM, Galifianakis NB. Virtual visits for Parkinson disease: a multi-center noncontrolled cohort. *Neurol Clin Pract*. 2017 Aug;7(4):283-295. doi: 10.1212/CPJ.0000000000000371.

Busis NA; Shanafelt TD; Keran CM; Levin KH; **Schwarz HB**; Molano JR; Vidic TR; Kass JS; Miyasaki JM; Sloan JA; Cascino TL. Burnout, career satisfaction, and well-being among US neurologists in 2016. *Neurology*; 2017; 88: 797-808.

Dorsey, ER et al. "National Randomized Contolled Trial of Virtual House Calls for People with Parkinson's Disease: Interest and Barriers". *Telemedicine and e-Health*. 2016;22 (7):590-598.

Dorsey, ER; Wagner, JD; Bull, MT; Rizzieri, A; Grischkan, J; Achey, MA; Sherer, T; Chowdhury S; Meunier, C; Cappelletti, L; Rocher, C; Richard, **IH**; **Schwarz**, H; Kang, G; Ahmad, SH; Biemiller, RA; Biglan, KM. "Feasibility of Virtual Research Visits in Fox Trial Finder". *Journal of Parkinson's Disease*. 2015; 5: 505-515.

Schwarz, HB; Frtiz, JV; Govindarajan, R; Murray, RP; Boyle, KB; Getchius, TSD; Freimer, M. "Neurology advanced practice providers: A position paper of the American Academy of Neurology". Neurology Clinical Practice. 2015; 5(1): 333-337

Parchi P; Capellari S; Chin S; **Schwarz HB;** Schechter NP; Butts JD; Hudkins P; Burns DK; Powers JM; Gambetti P. "A subtype of sporadic prion disease mimicking fatal familial insomnia." Neurology. 1999;52(9):1757-63.

O'Brien CF; **Schwarz HB;** Kurlan R. "Neuroacanthocytosis without Acanthocytes". Movement Disorders. 1990; 5(Supplement 1): 98.

Kang UJ; Fahn S; **Schwarz H;** Shoulson I; Vallejos H; Goldman J. "Case 1, 1989: juvenile-onset parkinsonism, dystonia, and pyramidal tract signs." Movement Disorders 1989;4(4):363-70

Gash DM; Bohn MC; Fiandaca MS; Okawara SH; Kordower JH; Notter MFD; Snyder J; **Schwarz HB;** Shoulson I. "Adrenal Medullary Implantation Promotes Tyrosine Hydroxylase Immunoreactivity in Host Striatum of MPTP Animal Models of Parkinsonism". Archives of Neurology. 1988; 45(7): 810-811

BOOKS AND CHAPTERS

Schwarz K, Schwarz H, Meltzer R. "Neurologic Complications of Cardiac Surgery". Heart and Brain: Interaction of Cardiac and Neurologic Disease. Weintraub M, Fass A. New York: PMA Publishing Corporation, 1991. 233-251.

EDITORIALS

Editorial: Could Exercise Be the Answer?

Disease Modification With Long-term Regular Physical Activity in Parkinson Disease

Margaret K. Y. Mak, Heidi Beck Schwarz

Neurology Feb 2022, 98 (8) 303-304; DOI: 10.1212/WNL.0000000000013208

Editorial: Cook CL, Schwarz HB. Advanced Practice Clinicians-Neurology's Underused Resource. JAMA Neurol. 2021 Aug 1;78(8):903-904. doi: 10.1001/jamaneurol.2021.1416. PMID: 34028498.

Editorial: Schwarz HB, Robbins MS. Are Two Head(ache)s Better Than One: Consequences of Diagnosing Migraine and Occipital Neuralgia. Neurol Clin Pract. 2021 Feb;11(1):1-2. doi: 10.1212/CPJ.0000000000000801. PMID: 33968465; PMCID: PMC8101313.

Editorial: Jones LK Jr, Schwarz HB. Elasticity in Health Care: How Much Can We Stretch the System, and Our Patients? Neurology. 2021 Jan 19;96(3):87-88. doi: 10.1212/WNL.0000000000011313. Epub 2020 Dec 23. PMID: 33361252.

Editorial: A nurse-led model increases quality of care in Parkinson disease, Calabresi P, Nigro P, Schwarz HB; Neurology 2019, April 16;92(16):739-740.

Commentary: Wernicke encephalopathy after bariatric surgery: Losing more than just weight. Neurology | 12/27/2005

MISC. PUBLICATIONS

The AAN Live Well, Lead Well Program: Promoting Wellness through Leadership, Heidi B. Schwarz MD FAAN (URMC), Neil A. Busis MD FAAN (UPMC), Jennifer R. Molano MD FAAN (University of Cincinnati), Poster Presentation at the AAN 2019 Annual Meeting, May 2019

Post marketing experiences with erenumab (Aimovig) for the treatment of chronic migraine in a real-life clinical setting, Carolyn E. Zyloney, M.D., Heidi Schwarz M.D., Raissa Villanueva, M.D.: Poster Presentation at the Schwid Symposium, URM June 2019

DOMINO abstract poster presentation at the MDS 13th International Congress of Parkinson's Disease and Movement Disorders Meeting in Paris, France. Lead Author: M Cudkowicz MD | 06/08/2009

Book Review in Neurology: Therapeutic Hypothermia by SA Meyer and D Sessler. | 12/26/2006 - 12/26/2006

ABSTRACTS

Ramchandani C, Schwarz HB, Amusia: Unique Presentation of Stroke, 66th Annual Meeting of AAN | 05/01/2014
Schwarz HB, Eskin T, Brumback R, Caine E, Coleman P, Flood D, Haber S, McNeill T, Hamill RW, Late
Onset Hallervorden-Spatz Disease Presenting as Cortical Dementia, Neurology (suppl) | 1987