Exhibit 533

School of Medicine and Dentistry

Department of Neurology

UNIVERSITY of ROCHESTER MEDICAL CENTER

Medicine *of* the Highest Order

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I am writing in response to your request for medical expert evaluation of Richard D. Sparks, Jr. 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 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 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.

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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 Richard Sparks' exposure to the water at Camp Lejeune and his Parkinson's Disease. My opinion is based on my education, training, research, and clinical practice.

In forming my opinion, I have reviewed the following records on Mr. Richard D. Sparks Jr.:

- Medical Records from Rocky Mountain VAMC from 2015 through July 2024
- Medical Records from the University of Colorado from 2013-2018
- Records and video from Mr. Sparks deposition from January 2024
- Records of depositions for Drs. Barrie Schmitt, Maureen Leehey, Christen Epstein NP and Sabrina Schickli PsyD
- Virtual Visit with Mr. Sparks on 12/18/2024 lasting 50 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 I have reviewed the materials cited in their reports to understand and that verify their opinions are 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. Sparks' Parkinson's disease is more likely than not due to his exposure to TCE at Camp Lejeune for approximately 15 months from March 25, 1974 to May 30, 1975.[86] [MOU1] [hs2] As a result, my causation opinions in the Sparks matter meet and exceed 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 manifest 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.[51] Motor fluctuations develop over time characterized by "on-off" periods associated with reduced mobility, increased tremor, involuntary movements (dyskinesias or dystonia), freezing.[55] 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 (collectively known as the 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 is now providing evidence through mechanistic and animal studies, for the scientific conclusion that environmental factors can be causative in the development of PD. Genetic susceptibility, for some, is also believed to contribute to the development of PD, as detailed below, but not without the contribution of another factor.[15,54] 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 documented 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 toxicants 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 pre motor 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.[54]

B. Potential Causes of Parkinson's disease[1]:

- **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%.[56] However, other epidemiologic studies have shown no association between TBI and PD.[57] 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.[58] Although there is molecular overlap between the pathology seen in TBI and PD, the mechanism of

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

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 suggests an association between air pollution (PM2.5) and Parkinson's disease.[23] However, a Finnish study showed no association between particulate air pollution and Parkinson's disease.[81] If there is an association between particulate matter and PD, research suggests that it requires long term exposure. Since 92% of the world population live in areas that exceed WHO guidelines for PM 2.5, establishing a clear link between particulate manner and PD will be challenging. Researchers have postulated mechanisms by which air pollution could increase the risk of PD but this research is in its early stages.[82] Laboratory studies in zebrafish have shown the exposure to diesel fumes (a component of air pollution) results in neurodegenerative changes, but zebrafish are not humans.[83] There is not a scientifically established causal relationship between air pollution (including diesel fume exposure) and the development of Parkinson's disease in humans.
- **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 more likely to develop melanoma than the general public with estimates ranging from 2 to 7 times more likely.[45,59] Those with melanoma are 50% more likely to develop Parkinson's disease.[53] Despite this strong association, there is no evidence of 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.[60]

C. Causes of Parkinson's disease[2]:

- **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.[17,43] 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.[17] 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 Parkinson's disese[19]. 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 transfers 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 mitochondria[20]. 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 have been associated with drug induced parkinsonism.[61]
- **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

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

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 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 increased 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 to 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.[84,85]

  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 1997-2021. The levels of TCE at Camp Lejeune were modeled to be 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 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. Activities such as showering or swimming in water contaminated with TCE would add to ingestion exposure via the inhalation pathway.

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 be 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. 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.[49] 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 more likely than 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. Sparks' case, I have reviewed reports from toxicologists and an epidemiologist who provided expert opinions in this case including Dr. Cannon, Dr. Costa, Dr. De Miranda, Dr. Miller and Dr. Boehme whose summaries of the experimental data regarding TCE and PCE and PD are rigorous and thorough. While I have reviewed 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.[62-66]

IV. Patient History and Presentation of Parkinson's Disease

Mr. Sparks is a 71 year old man who had a medically uneventful childhood and early adult life.[67] He enlisted in the Marines in 1971. He was sent to boot camp at Camp Pendleton where he was a truck driver until June 1972. Subsequently, he was assigned as a Marine security guard in Rio de Janeiro until November 1973. From there, he was sent to Marine security guard school in Arlington, Virginia. According to the materials reviewed, none of these postings involved any exposures to known toxins. He arrived at Camp Lejeune on March 25, 1974 where he was assigned to 2nd Engineering Branch as a motor vehicle operator and then a disbursement clerk.[68, 86] Both his assignment and his living quarters were near Hadnot Point which is the water system at Camp Lejeune with the highest TCE level.[69] He moved to Beaches apartments near Camp Geiger in March 1974 which was off base. However, he continued to work at Camp Lejeune until May 30, 1975. [86] He spent approximately 15 months at Camp Lejeune. In reviewing Mr. Sparks deposition, while at Camp Lejeune, in addition to driving, he also ran 3 times per week. He drank water out of the base water fountains and drank and transported water buffaloes that were filled with contaminated water. He drank approximately 1 gallon of water a day (including coffee).[70] He occasionally showered on base but usually at his apartment. He infrequently swam in ponds and walked through swamps with contaminated water. He spent 4 years and 4 months in active duty as a Marine and was on inactive service for the remaining 6 years, joining the reserves in 1975.[68]

After leaving Camp Lejeune, he returned to Kansas City and worked as an attendant at a gas station until late 1975 when he moved to Brazil to teach English.[71] He returned to Kansas City in 1978 and attended college while working as a supervisor at the Greyhound Bus Terminal. He testified that he worked mainly inside at the ticket counter while working at the bus terminal.[44] He returned to Brazil in 1979 to teach English and remained there until 1990.

In 1990, he began working for the US Customs Service, initially in Laredo, Texas and then in Portugal where he was an attaché for Homeland Security. While in Portugal, he was seen by a neurologist for right arm and leg tremor in 2008 and was diagnosed with Parkinson's disease, at the age of 55.[72] I do not have access to this record. In 2012, he returned to the US, working for the US Customs Service at the Denver airport, examining passports, etc.[71]

The first medical record I have for Mr. Sparks is from the University of Colorado, Department of Neurology in 2014. He was seen initially by Dr. Maureen Leehey, MD who was the Chief of the Movement Disorder division of the Department of Neurology.[74] She documented that his symptoms of Parkinson's disease started in 2008 and more importantly, confirmed the diagnosis. He was also found to have mild cognitive impairment and expressed that his most bothersome symptom was tremor. He was started on trihexyphenidyl, ropinirole and alprazolam. A brain MRI was performed in 2014 which showed no structural cause, including but not limited to head trauma, for his parkinsonian symptoms.[77] At his second visit to the University of Colorado Movement Disorder Clinic in February 2014, it was noted that his tremor was improved as was his eating and hydration.[75] He was advised to

initiate regular exercise.

He continued to follow up at the University of Colorado, but his symptoms progressed despite trials of several medications including amantadine (which caused a rash), rasagiline (no benefit), ropinirole (limited benefit), trihexyphenidyl (caused memory loss). Eventually he was placed on Sinemet which he continues with some benefit, but he struggles with motor fluctuations (both wearing off and dyskinesias). Deep brain stimulation was considered because of his inadequate response to medications but the patient declined.[76] Deep brain stimulation (DBS) carries the risk of bleeding, infection, stroke and seizures. It can also cause difficulties with speech and swallowing.[80] There is no evidence that DBS slows the progression of PD. He continued to work for the US Customs Service at the Denver airport but by 2016, he sought early retirement because his symptoms of bradykinesia, rigidity and tremor made him unfit to perform his job functions.[73] He was forced to retire earlier than he had planned.

He transferred his neurologic care to Rocky Mountain VA in 2015 but was seen infrequently. The last neurologic visit available to me was with Dr. Schmitt in May 2022.[78] At that time, records document that he was having right arm and leg rest tremor, dyskinesias of his head and left leg, dystonia, bradykinesia, moderate rigidity on the right greater than left side, positive Romberg, with inability to tandem gait. He was taking a combination of Sinemet and entacapone every 4 hours (but benefit lasted only 3-4 hours) and ropinirole in the morning.

During my virtual visit with Mr. Sparks in his home on 12/18/2024, I obtained a detailed history from Mr. Sparks to assess his present level of functioning as well as his current complaints. I also conducted a physical exam virtually to confirm his diagnosis and assess his motor disability.[44] Mr. Sparks reported that he was now seeing a movement disorder specialist at the University of Colorado because the neurologists at the VA had not been very helpful.[44] With regard to motor symptoms, he notes significant tremor in his right greater than left upper extremity, especially when the effects of his medications wear off. He notes slowness of movement. He has had some "pretty spectacular falls". His falls are usually propulsive (forward) but recently he has had retropulsive falls (backwards). He has near-falls 2-3 times per week and is "careful with every step." He naps 1-2 times per day and feels constantly fatigued. When his medications wear off, his voice is soft and low such that others cannot understand him. He frequently drools, especially at night.

Regarding non-motor symptoms, he endorses, constipation, urinary urgency, nocturia (2-3 times per night), orthostatic lightheadedness, disrupted sleep with poor bed mobility, REM behavior disorder (frequently yelling and screaming at night and occasionally striking his wife) with vivid dreams, short term memory loss with word finding difficulties. He was an avid reader but no longer reads due to poor concentration. He notes loss of taste and smell resulting in poor appetite. He has had at least 4 melanomas removed. Functionally, Mr. Sparks needs help with putting on his socks and shoes, tying bows and buttoning while dressing. He rarely showers because of balance issues. He requires minor assistance to get in and out of the tub. The only housework he can do is putting dishes in the dishwasher and putting them away. He no longer does any yardwork. He only drives if it is a necessity and rarely leaves the house due to tremor, urinary urgency. He can no longer do woodworking or play darts. His grandson provides significant assistance to him since his wife is also quite disabled related to pulmonary disease. In fact, she is finding it difficult to help him turn over in bed since he cannot do this himself.

His medications as of 12/18/2024 were: Sinemet 25/100 2 tablets 5-6 times per day. He reports each dose lasts only 2 hours. As compared with 2016 when he started Sinemet 25/100 1 tablet 4 times per day, his dose has more than doubled and is lasting only half as long as it did in 2023. Mr. Sparks is becoming less responsive to Sinemet with more side effects. When the dose is having maximal benefit, he has mild dyskinesias which are not functionally limiting. Dyskinesias are involuntary, erratic, writhing movements of the face, arms, legs or trunk. They are often fluid and dance-like, but they may also cause rapid jerking or slow and extended muscle spasms. They are not a symptom of Parkinson's disease (PD) itself. Rather, they are a complication from some Parkinson's medications.[46] He also takes mirtazapine 30 mg in the am and sertraline 100 mg at bedtime for depression and anxiety. He is unsure whether these are helping with his mood. Systematic reviews have identified that autonomic symptoms, motor fluctuations, severity and frequency of symptoms and staging of disease are associated with the

presence of depression and anxiety in patients with PD.[87] However, personal and familial psychosocial and environmental factors play an important role in those with PD and depression influencing coping mechanisms of both patients and caregivers. This is consistent with my experience in treating patients with PD and their families. Medications cannot change the reality of day-to-day life for those with PD. Given his loss of independence due to Parkinson's disease and his inability to help his wife of over 50 years, his issues with mood are in part related to his loss of hope for the future.

Prior to our virtual visit at 1p.m. on December 18, 2024, he had taken a dose of Sinemet approximately 30 minutes before the examination and felt "on" during the testing.[44] Mild dyskinesias of his head and upper body were noted. Montreal Cognitive Assessment was performed virtually with a score of 24/30. This is consistent with mild cognitive impairment. He had 0/5 recall at 5 minutes and was disoriented to date. Cranial nerve exam was notable for reduced frequency of blink with grade 2+ hypomimia (reduced facial expression) and mild hypophonia (abnormally weak or soft voice). He had moderate bradykinesia in the UEs (Grade 3 on right and 2+ on the left). Rigidity cannot be tested remotely. He had a constant chin tremor with mild rest and sustention tremor in the right>left upper extremities. He was able to rise from the chair without the use of his arms. His gait was moderately slowed with reduced arm swing on the right>left and tremor in the right arm. He required 4-5 steps to corner which he did slowly and carefully.

Based on my evaluation, to a reasonable degree of medical certainty, I can confirm clinically, that Mr. Sparks has Parkinson's disease with the hallmark features of asymmetric tremor, bradykinesia and gait disturbance with postural instability. 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 3 indicating mild to moderate bilateral disease; some postural instability; physically independent.[47] 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 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. Sparks 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. These are functionally limiting to him regarding dressing, bathing, sleep and ability to function outside of his home. A cognitive assessment tool used during my virtual visit also revealed that he has mild cognitive impairment (MCI) as demonstrated on the Montreal Cognitive Assessment with significant short term memory loss. PD patients like Mr. Sparks with MCI have a 60% chance of progressing to dementia within 4 years.[48]

Mr. Sparks past medical history is notable for hyperlipidemia, vitamin B12 deficiency (on adequate oral replacement), fatty liver and pulmonary, liver and spleen granulomas likely due to a prior infection. None of these diagnoses would predispose him to Parkinson's disease. He is an ex-smoker and drinks beer infrequently. He occasionally uses marijuana to help with sleep. His prior hobby of woodworking did not expose him to toxins. He has no family history of Parkinson's disease and denies prior head trauma.[44]

V. Methodology, Analysis and Conclusions

To address the etiology of Mr. Sparks' Parkinson's disease, it is important to ensure a correct diagnosis. Once confirmed, risk factors for Parkinson's disease need to be explored as they relate to Mr. Sparks' 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

examination that to a reasonable degree of medical certainty, Mr. Sparks has Parkinson's disease with mild cognitive impairment. As noted previously, PD is a clinical diagnosis. There is nothing in Mr. Sparks' history or exam that suggests another diagnosis.

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

1. **Genetics**: Mr. Sparks 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. Sparks has a genetic predisposition to Parkinson's disease, and therefore I can rule out this risk factor in my differential etiology.
2. **Head Trauma**: Mr. Sparks has no history of head trauma and denies head injury. In addition, the MRI of his brain showed no evidence of prior brain trauma allowing me to rule out this risk factor as a potential etiology.
3. **Drug exposure**: Mr. Sparks was never exposed to MPTP. Dopamine blocking agents such as metoclopramide can cause a drug induced parkinsonism, but based on my review of Mr. Sparks' medical records, he has never been exposed to any of these drugs.[50] Because Mr. Sparks 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. Sparks 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 increase the risk of Parkinson's disease, but Mr. Sparks has never lived in a region with a high PM 2.5 level. A question was raised about his exposure to diesel fumes while working for a short time at the bus terminal. However, Mr. Sparks testified that he spent little time outside near the buses but was at the indoor ticket counter. There was also a question of toxin exposure while working for US Customs at Laredo, TX. Again, Mr. Sparks testimony outlined only one incident treated as a hazardous material exposure, but this turned out to be plastic pellets which are not hazardous. In order to include an exposure as a potential contributing cause, I must first satisfy myself that there is reasonable evidence that an exposure actually occurred, and that the chemical agent can be identified with some level of certainty, beyond mere speculation. Lastly, there was concern raised about radiation exposure while working for Homeland Security in Portugal. Mr. Sparks testified that he worked in a totally isolated cab while testing for radiation. He never encountered a shipment with a high level of radiation. In addition, radiation is not a known cause of PD. There is no evidence that pesticide exposure or pollution has increased Mr. Spark's risk of Parkinson's disease.
5. **TCE/PCE exposure:** Mr. Sparks was exposed to toxic levels of TCE and PCE, and other volatile organic compounds, during the time he was working at Camp Lejeune. Mr. Sparks was stationed at Camp LeJeune/Hadnot Point between March 25, 1974 to May 30, 1975 where he was exposed to TCE and tetrachloroethylene (PCE). During that time, he had an estimated exposure of 3195 microgram/liter of TCE, 63 microgram/liter of PCE.[88] The following chart indicates the specific concentrations in the water when Mr. Sparks was at Camp Lejeune:

Exposure Dates	HP TCE (ug/l-M)	HP PCE (ug/l-M)	HP VC (ug/l-M)	HP BZ (ug/l-M)
3/25/1974 - 3/31/74	163	3	7	2
4/1/1974 - 4/30/1974	116	2	5	3
5/1/1974 - 5/31/74	142	2	6	2
6/1/1974 - 6/30/1974	179	3	8	2
7/1/1974 - 7/31/1974	209	4	9	2

8/1/1974 - 8/31/1974	274	5	12	3
9/1/1974 - 9/30/1974	217	4	9	3
10/1/1974 - 10/31/1974	50	1	2	3
11/1/1974 - 11/30/1974	399	8	17	3
12/1/1974 - 12/31/1974	369	8	15	3
1/1/1975 - 1/20/1975	179	4	7	3
1/21/1975 - 1/31/1975	0	0	0	0
2/1/1975 - 2/5/1975	0	0	0	0
2/6/1975 - 2/28/1975	252	6	11	3
3/1/1975 - 3/31/1975	261	6	11	2
4/1/1975 - 4/30/1975	174	4	7	3
5/1/1975 - 5/31/1975	211	5	9	3
	3,195	**65**	**135**	**40**

Mr. Sparks was exposed to the chemicals at Camp Lejeune via consumption/ingestion for approximately 450 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. Sparks 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. Sparks was exposed in his day to day life at Camp Lejeune.

I was able to determine that Mr. Sparks had substantial exposure just based upon the records at issue, Mr. Sparks' 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. Sparks. These charts support my opinion that Mr. Sparks sustained a substantial exposure that was causally related to his Parkinson's disease.

For example, Dr. Reynolds charts indicate that Mr. Sparks would have likely ingested the following amounts of the toxins at issue in this case:[88]

		Chart 1: 1L	Chart 2: ATSDR marine in training	Chart 3: Deposition informed activity days and ATSDR 6L & 3L exposures
	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)
Hadnot Point				
TCE	3,195	90,063	390,333	281,649
PCE	65	1,830	7,931	5,723
VC	135	3,806	16,495	11,902
BZ	40	1,121	4,858	3,506

Exposure to amounts of TCE of 281,649 micrograms is clearly a substantial exposure. The compounding part for Mr. Sparks is that in addition to this very significant and substantial ingestion exposure, Mr. Sparks was also exposed to 5,723 of ug (micrograms) of PCE. TCE is a chemical that is causally related to Parkinson's disease. Mr. Sparks' TCE exposure put him at dangerously higher risk for the development of Parkinson's disease.

As with many diseases associated with toxin exposure, manifestation of symptoms comes years after the exposure. Mr. Sparks had an approximate 15-month exposure to toxic levels of TCE while serving at Camp Lejeune between March 25, 1974 and May 30, 1975.[86] In fact, his work area was adjacent to the most highly contaminated water supply on the base (Hadnot Point). 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 overwhelming 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 to a reasonable degree of medical certainty, based on the standard of causation under the Camp Lejeune Justice Act defined as "at least as likely as not," I conclude that Mr. Sparks Parkinson's disease is more likely than not, which exceeds the standard of is "at least as likely as not", due to his exposure to the water at Camp Lejeune containing TCE from March 25, 1974 to May 30, 1975.[86] [MOU1] [hs2]

A. Future Care Considerations

Mr. Sparks' parkinsonian symptoms continue to progress and his responsiveness to medications is diminishing. He has developed motor fluctuations ("off" periods with poor mobility and "on" periods with dyskinesias) which will limit benefit from medications. He currently requires assistance with many activities

of daily living. He also has mild cognitive impairment which has a high likelihood of progressing to dementia. His disability will undoubtedly progress over time necessitating increased care and cost. I defer considerations of neuropsychiatric nature to a specialist in the field. However, from a movement disorder perspective regarding medical care and treatment, 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 due to inability to drive within the next year
2. Assistance with dressing and bathing within 3 years
3. Skilled nursing care within 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. Sparks' 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 that 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.[79]

February 5, 2025

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EXHIBIT A

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88. Modeling and exposure data from Dr. Kelly Reynolds.

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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 Epilespy | *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 Omeag 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 URMC 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 URMC 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 URMC in outpatient neurology | *2010 - present*
Morning report for the Neurology Service at URMC | *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 Highlnad 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
URMC 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 techiniques 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'Suilleaubhain 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, URMC 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 Medicne 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, Kieburtz 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. doi10.1212/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; CascinoTL. 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**; Schecter 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, URMC June 2019

DOMINO abstract poster presentation at the MDS 13th International Cogress 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*