

Exhibit 549

Stephen M. Gollomp MD, FAAN
Past President, Philadelphia Neurological Society
The Louis and Elizabeth Papi Endowed Chair in Neurology at Lankenau Medical Center
Clinical Professor of Neurology, Thomas Jefferson University
Clinical Professor, Lankenau Institute of Medical Research
Director, Suburban Philadelphia Parkinson's Care Center

Phone: 610-642-5371
Fax: 610-642-5658
E-Mail: On Request

Suite 161, Lankenau Medical Bldg. East
100 Lancaster Avenue
Wynnewood, PA 19096

May 8, 2025

Expert Report of Stephen M. Gollomp, M.D.
ROBERT WELCH v. UNITED STATES OF AMERICA
Case No. 7:23-CV-01503

I. Qualifications

By way of background as to my qualifications, I am a graduate of the Rensselaer Polytechnic Institute/Albany Medical College six-year biomedical program during which time I earned my Bachelors of Science in Biology and my Medical Degree in six years, rather than the usual eight years, graduating in 1976. I stayed for one additional year at Albany Medical Center for my medical internship from 1976 through 1977, which was followed by a three-year neurology residency at Boston University Medical Center from 1977 through 1980. After my residency, I undertook a fellowship in movement disorders and neuropharmacology at Columbia University under the tutelage of the renowned neurologist and movement disorder pioneer, Dr. Stanley Fahn, from 1980 through 1981. During that time, I honed my clinical and scientific knowledge and skills under Dr. Fahn's supervision, along with a host of remarkable faculty and international colleagues, who participated in the programs at Columbia.

Following my time at Columbia, I entered the private practice of neurology in suburban Philadelphia at the Lankenau Medical Center, where I rose to be the neurology division chief, the vice chief of neurology for the Main Line Health System, the Papi Endowed Chair in Neurology at Lankenau and Clinical Professor of Neurology at the Lankenau Institute of Medical Research. Concurrently, I was a clinical assistant professor of neurology at Hahnemann University College of Medicine from 1981 through 1983. I then joined the fledgling University of Pennsylvania Movement Disorders Center at Graduate Hospital with Drs. Howard Hurtig and Matthew Stern from 1984 until 1996. I held the position of clinical assistant professor of neurology at the University of Pennsylvania during that time. I subsequently moved on to assume the position of clinical associate professor of neurology at Thomas Jefferson University, advancing to Clinical Professor of Neurology at Thomas Jefferson University in 2005. I was named the Papi Endowed Chair in Neurology at Lankenau in 2007 in recognition of my clinical research and medical expertise in the Parkinson's disease field.

Throughout these years, I have maintained a robust clinical practice of neurology focused upon all aspects of motor disorders, most specifically Parkinson's disease. I have cared for thousands of Parkinson's disease patients over the decades, many of them for several decades. I continue to actively practice clinical neurological medicine in my solo private practice, employing all the latest technologies and pharmaceuticals in my ministrations to my patients. I am deeply involved with all aspects of the care and management of people affected with Parkinson's disease across the entire spectrum of the course of the disorder. I have been a principal or associate investigator in over 30 clinical trials in the movement disorders space, most of them focused upon Parkinson's disease. Though I am no longer actively conducting clinical research, I continue to actively recruit patients for numerous neurotherapeutic trials and continuously interface with my colleagues engaged in these projects.

I have published extensively in the field of movement disorders and Parkinson's disease with 41 articles, 22 abstracts five book chapters and two book reviews. I was one of the founding medical editors of Practical Neurology in 2001 and remain the medical co-editor of this publication, which is well recognized and regarded in the neurological community in the United States and across the globe. I am a past president of the Philadelphia Neurological Society, the oldest continuously functioning local neurological society in the United States, founded in 1884. I remain an active member of the board of this organization in an ex-officio capacity, actively participating in its ongoing functioning and development. I am a member of the American Academy of Neurology, having served in the Movement Disorders section of this organization. I am one of the early members of the International Movement Disorders Society and remain an active member to this day.

In addition to the above, I hold an unrestricted license to practice medicine within the Commonwealth of Pennsylvania. I am Board Certified by the American Board of Psychiatry and Neurology. I actively practice neurology in private practice with an office on the campus of the Lankenau Medical Center, where I remain Chief of Neurology and hold the Louis and Elizabeth Papi Endowed Chair in Neurology. I am also an attending neurologist at the Thomas Jefferson University Hospital. As indicated above, I hold the academic appointment of Clinical Professor of Neurology at Thomas Jefferson University and at the Lankenau Institute of Medical Research.

During my career as a physician, I have diagnosed and treated many patients with Parkinson's disease such as Mr. Welch. I am familiar with the diagnosis, treatment and progression of this disorder.

My CV is attached as Appendix A.

All my opinions in this document are stated to a reasonable degree of medical certainty and are based upon my review of the records, documents, images and materials provided and my experience, training, education and authorship.

II. Compensation and Prior Testimony

I am being compensated for my rendering of opinions in this case on an hourly basis. I am being compensated at \$500 per hour.

I have rendered neurological expert commentary many hundreds of times over the past 43 years for both plaintiffs and defendants. A list of prior testimony over the last four years is attached as Appendix B.

III. Expert Opinion

Based upon my experience, expertise and review of the material provided, it is my opinion to a reasonable degree of medical and scientific certainty that Mr. Welch is affected by Parkinson's disease. It is also my opinion that there is insufficient evidence to conclude that Mr. Welch's Parkinson's disease was caused by his alleged exposure to contaminated water at Camp Lejeune. I further elaborate upon the basis of these opinions later in this document.

IV. Methodology

To reach the conclusions rendered in this document, I have reviewed the records and depositions provided to me relative to Mr. Welch. All records reviewed are included in the reference sections of this report. I reviewed the current literature concerning the diagnosis, etiology, treatment and prognosis of Parkinson's disease. I have also considered my very extensive, ongoing experience in the diagnosis, care and management of people with this disorder over many decades. I also performed an independent medical evaluation of Mr. Welch on March 14, 2025, via a videoconferencing platform.

I considered Mr. Welch's alleged exposure to contaminated water at Camp Lejeune in generating my opinions as to the causation of Mr. Welch's Parkinson's disease. My opinions regarding Mr. Welch's exposure history relied on a review of the following:

- Expert Report on Robert Welch of Judy LaKind, May 8, 2025
- Expert Report on Robert Welch of Lisa Bailey, May 8, 2025

V. Background Review of Parkinson's disease

Parkinson's disease is the second most common neurodegenerative diseases to afflict man, particularly as a person ages. It is manifest by an assortment of recognizable physical manifestations, including slowness of movement, referred to as bradykinesia, stiffness of body parts, referred to as rigidity, and tremor, principally manifest in the arms when they are at rest (Tanner and Ostrem, 2024). There are other associated symptoms and signs, but the key to diagnosis are these three features. Parkinson's disease was initially described in 1817 by James Parkinson. He noted several individuals with rest tremor and weakness of that body part associated with a stooped posture and a tendency to pass from walking to running. He did not directly examine most of these individuals but rather described their manifestations as he observed them on the streets of London, England (Mulroy et al., 2024). It is very probable that

this same disorder was recognized much earlier in other parts of the world, but without the understanding of the full spectrum that James Parkinson noted.

There are two major sets of diagnostic criteria supporting the diagnosis of the disorder, namely the Movement Disorder Society (MDS) criteria and the Queens Square criteria, both very similar to each other, requiring various combinations of rigidity, rest tremor and bradykinesia with other supportive or exclusionary criteria (Gibb and Lees, 1988). Studies have shown that the MDS criteria are 92% accurate in rendering a Parkinson's disease diagnosis (Postuma et al., 2015, Virameteekul et al., 2023).

There are many symptoms of Parkinson's disease that represent non-motor features of the disorder, including autonomic nervous system dysfunction, reflected by drops of blood pressure and constipation, cognitive disturbances, depression and anxiety, sleep disorders, principally manifest by rapid eye movement, behavior disorder, painful muscle spasms, fatigue and loss of sense of smell and taste (Qamar et al., 2024; Rodriguez-Blazquez et al., 2021). When the cognitive disturbances are particularly severe a diagnosis of Parkinson's disease dementia, or dementia with Lewy bodies is rendered, which is a part of the Parkinson's disease spectrum (Borghammer et al., 2024).

The main differential diagnostic entities for Parkinson's disease are generally termed the atypical parkinsonisms. These disorders include multiple system atrophy, progressive supranuclear palsy, corticalbasal syndrome, frontotemporal dementia and drug induced parkinsonism. Various features helped to distinguish these disorders, including marked incoordination of limb movements, abnormalities of eye movements, frontotemporal behavior abnormalities, marked lower extremity involvement, administration of dopamine receptor blocking agents, absence of response to levodopa, impairment of complex motor movements termed, apraxia, and cortical sensory loss.

Laboratory testing is not generally necessary to reach a diagnosis of Parkinson's disease. However, dopamine transporter scans, which provide an index of dopamine activity within the basal ganglia correlates highly with the number of remaining dopamine neurons innervating the basal ganglion (Brucke and Brucke, 2022). In addition, various assays of abnormal alpha-synuclein can be obtained via lumbar puncture, blood, and skin biopsy for further clarification of diagnosis, but are generally not necessary for routine clinical use.

The fundamental cause of Parkinson's disease, at least the major motor manifestations, is the loss of the dopamine neurons within the substantial nigra pars compact of the brain, located in the upper brain stem. This is the critical source of dopaminergic innervation of the striatal portion of the basal ganglia, critical for complex coordinated motor function. This is recognized on the dopamine transporter scan. Generally, the microscopic hallmark of Parkinson's disease is the Lewy body which is generally seen pathologically, but in certain genetic variants of the disorder, the Lewy body is not seen (Anastassiadis et al., 2024; Morris and Lees, 2024).

There are many potential causes of Parkinson's disease, reflected by the loss of dopamine neurons in the substantia nigra pars compacta, but the vast majority of the time the cause is unknown. In addition, in the vast majority Parkinson's disease patients the cause is multifactorial. It is particularly notable that Parkinson's disease is a disorder of aging as its incidents increases with age concurrent with the age-related loss of dopaminergic neurons in the brain structures noted above (Morris and Lees, 2024; Ben-Shlomo et al., 2024; Deliz et al., 2024).

Epidemiologic studies demonstrate multiple risks for the development of Parkinson's disease, including toxicants such as pesticides and organic solvents, air pollution, head trauma, dairy and alcohol intake, diabetes, cardiovascular disease, infections and autoimmune conditions (Bologna et al., 2022, Rugbjerg et al., 2009). Some authorities believe that Parkinson's disease is predominantly an environmental disease, emphasizing the role of pesticides and air pollution. Consideration of all the environmental factors together is called the exposome. There is growing evidence that air pollution is particularly important (Dorsey and Bloem, 2024). Growing up on a farm is a significant risk as well, perhaps related to pesticide use for crops as has been suggested for many years (Deliz et al., 2024; Grotewold and Albin, 2024; Simon et al., 2020; Tanner and Ostrem, 2024, Cole-Hunter et al., 2023).

Relative to environmental exposures, many toxins that might lead to Parkinson's disease have been suggested, but most are quite controversial. The one proven toxin, MPTP, was inadvertently administered to a small group of heroin addicts, thereby inducing classic Parkinson's disease with Lewy body pathology at autopsy (Langston, 2017).

There are also several factors that are thought to be protective, including physical exercise, nicotine and caffeine use, the use of statin medications (cholesterol meds) and use of antihypertensive drugs (Grotewold and Albin, 2024).

There are several genes that have been identified that cause Parkinson's disease. It appears that gene mutations can have variable impact, some are likely to cause Parkinson's disease and others just elevate the risk being additive to other potential causes. Young onset Parkinson's disease patients, typically less than age 50, are much more likely to have a genetic cause for their disorder (Cook et al., 2024; Coukos and Krainc, 2024). There is controversial recent data suggesting that a different disorder, termed essential tremor, slightly elevates the risk of developing Parkinson's disease versus individuals without essential tremor (Louis et al., 2023). Interestingly, essential tremor appears to have a strong genetic underpinning, though specifically identified mutations are rare (Louis et al., 2023).

Various neurological mechanisms can cause parkinsonism (Bologna et al., 2022). These include viral or bacterial infections, particularly the post encephalitic Parkinson's disease associated with the viral epidemic of 1918 (Xing et al., 2022). There is evidence that imbalance of bacterial ecology within the gut, termed the microbiome, might be a relevant factor as well (Smeyne et al., 2021). There are recent reports that suggest upper intestinal injury as a risk factor for Parkinson's disease (Chang et al., 2024). A form of parkinsonism can be induced by multiple small strokes,

but this is a different entity pathologically, though it frequently complicates Parkinson's disease in the older adult. Head trauma with multiple concussions seems to lead to increased risk of Parkinson's disease (White et al., 2020). Interestingly, post-traumatic stress disorder in veterans also appears to be an independent risk (Weaver et al., 2024). This combined with traumatic brain injury leads to a moderate excess synergistic risk of the disorder (White et al., 2020). Medications that block or deplete the dopamine system in the brain can cause parkinsonism and occasionally are associated with increased risk of the disorder (Rajput et al. 1982; Calzetti et al., 2025).

Identification of the ideologies of the disorder are based on individual history, particularly family history, as well as various exposures and events in an individual's life. Laboratory testing will include the dopamine transporter scan, magnetic resonance imaging of the brain (Hatano et al., 2024). In research settings, cardiac metabolic imaging demonstrates abnormal catecholaminergic function (Pitton et al., 2023). The ultimate diagnosis of Parkinson's disease can only truly be fully established at autopsy, given the multiplicity of potential confounding disorders (Tanner et al., 2023).

VI. Prognosis

Parkinson's disease is a progressive disorder associated with the gradual loss of dopamine neurons within the brain. Initially, our various interventions can stabilize the outward manifestations of the disorder, but inevitably the disorder worsens at a variable rate across individuals. Past rate of progression is suggestive of ongoing progression rate. Patients often do reasonably well with our interventions for at least the first 10 to 15 years before having decline of function. For patients under 70 years of age with short disease duration, approximately 64% will be living independently 10 years after diagnosis. For those with onset after the age of 70 with the disease for at least four years, only about 5% will be living independently after 10 years (Jasinska-Myga et al., 2012). Patients who have the tremor predominant form of the Parkinson's disease generally progressed more slowly and have less disability than those who manifest initially with postural instability and gait dysfunction or those with marked rigidity and bradykinesia (Tan et al., 2024). Those with an early age of onset, seemingly mostly genetic, will also tend to progress more slowly (Abbas et al., 1999).

None of the current treatments prevent progression of disease, though there is very recent evidence that possibly drugs that treat type 2 diabetes, the GLP-1 receptor agonists, might be beneficial, but this is controversial (Meissner et al., 2024). There is also evidence that exercise is beneficial, but the mechanism of this benefit is unclear (McGinley and Nakayama, 2024).

VII. Clinical Summary

Below is a list of the materials produced by the following sources delineating the care provided to Mr. Welch:

- Medical Records from Kaiser Permanente

- Medical Records from Dr. Carolyn Neff, MD; 2021 – 2023
- Medical Records from Dr. Edward Markus, DO; 2017 – 2022
- Medical Records from Dr. Jeffrey Tracy, MD; 2014 - 2024
- Medical Records from Kevin Tabora, RCP, R.T.; 2021
- Medical Records from Loma Linda VA Medical Center; 2012-2023
- Medical Records from Laguna Hills VA Medical Center; 2025
- Medical Records from Royalty Medical Group, Orthopaedic Surgery, Dr. Thomas R. Dorsey; 2002

I had the opportunity to evaluate Mr. Robert Welch neurologically on March 14, 2025, via a remote Zoom link, commencing at 12:00 PM Eastern daylight time and completing at 1:40 PM Eastern daylight time.

Mr. Welch is an 80-year-old right-handed gentlemen, who was accompanied by his wife, Maureen, during the evaluation. Mr. Welch related the following history. He first began to experience symptoms to suggest Parkinson's disease in approximately 2019 when he noted loss of sense of smell and micrographia. By 2020, he noted a slight tremor in his right hand, which he brought to the attention of his long-standing epileptologist, Dr. Markus, who referred him to Dr. Carolyn Neff, a movement disorder specialist within Dr. Markus' neurological group at Kaiser. Dr. Neff initiated carbidopa therapy in November 2021 at 300 mg per day of levodopa (25/100 tid) with nice effect.

Over time, the treatment program was altered with an increase of the carbidopa/levodopa IR to 400 mg/day (25/100 IR qid), the addition of carbidopa/levodopa 25/100 ER at bed, entacapone 100 mg with each of his daytime doses of carbidopa/levodopa IR and donepezil 10 mg at bed. The dose of extended release carbidopa/levodopa at bed was added to alleviate nocturnal right foot cramps. The donepezil was added to assist with his impaired memory. His ongoing Parkinson's care has been assumed by Dr. Besharat who has maintained this medication program. He reported that he experiences some end of dose effect over 5 hours following a medication dose. He reported that he continues to have some residual tremor with overall meaningful benefit from his medication program. For the most part, he noted no major limitations in his activities of daily living, though he needs assistance with buttoning small buttons. He indicated that he attends physical therapy classes very regularly and walks at least 45 minutes 4-5 days per week. He falls rarely with some superficial cutaneous injuries in a fall over the past year. His other daily activities include reading. He has been more apathetic with ready fatigability. He has no regular hobbies and no current exposures to any toxic/noxious materials.

Mr. Welch has had mild rapid eye movement behavior disorder over the past 5 years, attested to by his wife, Maureen and corroborated by the medical records. He is troubled by constipation, but no other reported autonomic dysfunction. He reported a decline in his concentration skills, but no other major cognitive alteration.

Mr. Welch's past medical history is remarkable for hypercholesterolemia, prostatism and possible coronary artery disease. In addition, he has been treated for obstructive sleep apnea with CPAP for the past 10-15 years. Mr. Welch has also been treated for partial seizure disorder since 2012. The seizure disorder has been well controlled with carbamazepine therapy. He also reported a history of intermittent posterior cranial and vertex headache for the past 6-7 years, occurring 1-2 times per month without any associated symptoms.

During his time in the Marines, he experienced some head trauma on at least 3 occasions without any clearly definable loss of consciousness. Testimony further delineating the details of these injuries is discussed below.

In his deposition, Mr. Welch testified that in 1968, while serving regular active duty in Quantico, Virginia, Mr. Welch was beaten with pugil sticks to his face and head (R. Welch Deposition, 2024). Then, in 1970, Mr. Welch fell approximately eight to ten feet while running through an obstacle course, sustaining injuries to his back, arms, head, and shoulders (R. Welch Deposition, 2024). In 1985, during an active training duty in Norfolk, Virginia, Mr. Welch was attacked by several men (R. Welch Deposition, 2024). During this attack, Mr. Welch was knocked down and beaten with hands and feet, sustaining injuries to the ribs, back, neck, and head during this assault without loss of consciousness (R. Welch Deposition, 2024).

Mr. Welch's current medication include the medications noted above, namely carbidopa/levodopa 25/100 qid¹, entacapone 100 mg qid, carbidopa/levodopa ER 25/100 hs² and donepezil 10 mg hs, plus terazosin 10 mg qd, aspirin 81 mg qd, atenolol 25 mg qd³, carbamazepine 300 mg bid, Metamucil, thiamine 100 mg qd, folate 1 mg qd and multivitamin. He is allergic to penicillin. He stopped atorvastatin due to intolerance.

His social history is remarkable for having been raised in Menlo Park in the San Francisco Bay area. He attended junior college there, moving on to Stanford to complete his undergraduate work. In his deposition testimony, Mr. Welch further elaborated upon his military and professional career as follows. In 1968, Mr. Welch enlisted with the Marines. He began his enlistment in Quantico, Virginia, completing the platoon leaders class there. Upon graduating and becoming an officer, he attended Loyola Law school. After receiving his J.D., he went again on active duty as a Staff Judge Advocate. He arrived at Camp Lejeune on November 18, 1970, where he resided until December 15, 1971. During this 11-month period, Mr. Welch briefly left Camp Lejeune to attend Naval Justice School in Newport, Rhode Island, from January 1, 1971, through February 28, 1971 (01503_WELCH_VBA_0000002085). After leaving Camp Lejeune, he remained on active duty until September 1, 1973. He then practiced law in California before retiring in 2010. He currently lives in Southern California in San Clemente (R. Welch Deposition, 2024).

¹ Q.i.d. means four times per day.

² H.s. means before bed.

³ Q.d. means once per day.

Mr. Welch has travelled extensively in the United States, Central America and Western Europe, and Japan, but did not report any other sites of travel. He is a former cigarette smoker, having stopped on September 1, 1973. Mr. Welch testified that he began smoking at age 10 and would smoke a little less than a package a day until quitting in 1973 (R. Welch Deposition, 2024).

He moderately consumes wine on weekends. He consumes one to two cups of coffee per day. He has never consumed any recreational drugs. Save for his time at Camp Lejeune, Mr. Welch has not had any specific exposure to potentially neurotoxic materials.

Family history is remarkable for atherosclerotic cardiovascular disease in his father, mother and brother, all deceased. He has two daughters and two grandsons, all in good health. There is no family of any similar neurological problems.

The medical records and depositions confirm that Mr. Welch has a history of epilepsy, beginning around 2010. His last seizure occurred on April 16, 2014 (Dr. Markus Deposition, 2024). He was prescribed carbamazepine after this incident and his epilepsy is “well-controlled” as of 2024 (Dr. Markus, 2024). He is still taking carbamazepine for his epilepsy and has not reported any seizures since beginning this medication (R. Welch Deposition, 2024).

A comprehensive review of systems was remarkable for the intermittent headache referenced above, relieved with as needed acetaminophen. Prior to enlisting, Mr. Welch was overweight (M. Welch Deposition, 2025). However, his weight has since been stable. He has severe hearing loss bilaterally, requiring hearing aids. He reported easy bruisability, constipation, nocturia and pain in knees, back and neck.

A review of the outside medical records confirmed the history Mr. Welch provided as outlined elsewhere in this report. In addition, these materials delineated other aspects of Mr. Welch’s medical history, not relevant to his Parkinson’s disease diagnosis. Specifically, Mr. Welch sustained injuries to his knees began during his enlistment as a Marine and he would later be diagnosed with arthritis of the knees on August 27, 1998. He has also experienced issues with his ankles after a fall in April 1989 while playing volleyball (Welch Affidavit, 2000).

In addition, Mr. Welch has also experienced medical issues with his feet, beginning in 1981. He was diagnosed with plantar faciitis and heel spurs, which his treating physician attributed to the constant training while enlisted with the Marines (Welch Affidavit, 2000).

Mr. Welch’s neck issues began while serving on active duty. In 1991, while assisting with moving boxes of books and furniture, he experienced severe neck problems the following day. His physician at the time, Dr. Hill, performed an x-ray and recommended a surgery referral for the condition. Then, in 2004, a similar lifting incident further exacerbated his neck pain, resulting in limited neck movement for some time (Welch Affidavit, 2000). He was treated by CIGNA for neck and back pain in April 2000.

Mr. Welch has been treated for back-related issues since 1997. He was diagnosed with sciatica of the right leg by Dr. Hannalore Zerwick at CIGNA. He was treated for back pain again in 1998, 1999, and 2000, including an arthritis blood panel. In July of 2000, Global Hospital performed an MRI on his neck and back. Dr. Bestard, with the Garey Orthopedic Medical Group, found multiple levels of cervical and lumbar spine stenosis due to disc disease, multi spondyloarthrosis, and osteophytes. Based on these results, Mr. Welch was referred to a neurologist, Dr. Ali, and to a neurosurgeon for consultation at Loma Linda University Medical Center in Loma Linda, California.

Due to the above injuries, Mr. Welch has received epidural steroid injections, in both the neck and back, without significant improvement.

The 80-minute telemedicine visit was conducted via Zoom, as already noted. Due to the limitations of this virtual examination methodology, a comprehensive general physical and neurological examination was not possible. However, this virtual examination methodology has been well validated for evaluation and treatment of the Parkinson's disease patient. The examination that was conducted was comprehensive within those limiting parameters.

Mr. Welch was noted to be a very pleasant, though somewhat reticent, older gentleman in seemingly good general health. He had bilateral hearing aids in place. There were no obvious limb deformities or edema.

Neurologically, Mr. Welch had seemingly normal cognitive status to social interaction. More semiquantitatively, a cognitive assessment with the MoCA-VI for visual impairment, the appropriate instrument for telemedicine assessment of cognition since proper pen and paper measures utilized with the conventional MoCA 8.1 cannot be employed remotely, was conducted. Mr. Welch's global score was normal at 26 out of 30, with mild relative deficits in word fluency and short-term memory. His memory index score was normal for age at 9 out of 15. The Montreal Cognitive Assessment (MoCA) is a well validated bedside cognitive screening tool that provides an excellent window into an individual's cognitive capabilities across multiple functional domains (Nasreddine ZS, et al., 2005).

Cranial nerve testing demonstrated normal pupillary function and normal eye movements with normal saccades and pursuits. Facial movements were symmetrical and somewhat hypomimic. Tongue protruded in the midline. Voice was slightly monotone and hypophonic.

Conventional motor exam did not demonstrate any abnormal postures or involuntary movements. Detailed Parkinson's exam was per the MDS-UPDRS. Notably, Mr. Welch's stride was only mildly reduced with some decrease in the right arm swing. He was sure footed with his turns, as he walked in his living room area.

The other semiquantitative measure that was conducted was the modified MDS-UPDRS, as the lack of an in-person exam precluded tone and postural stability assessment. Mr. Welch was evaluated 4.5 hours after his levodopa dose. Part III MDS-UPDRS motor score was 7, consistent

with mild to moderate Parkinson's disease, as reflected by mild right sided bradykinesia and right-hand kinetic tremor. No dyskinesias were noted. On part I, MDS-UPDRS, reflective behavioral and nonmotor consequences of Parkinson's disease, he scored 12, consistent with mild to moderate impairment. On part II MDS-UPDRS, which reflects the individuals reported motor impairments, he scored 9, again consistent with mild to moderate Parkinson's disease. On part IV MDS-UPDRS, a measure of fluctuation of status, he scored 2, consistent with mild affectation with only mild "off" dystonia as noted above and relatively little "off" time from levodopa effect. He reported no dyskinesias.

VIII. Mr. Welch's Relevant Medical History

Personal review of the available medical records concerning Mr. Welch confirms the history of cervical spinal stenosis, hyperlipidemia, gastroesophageal reflux, chronic low back pain, essential hypertension, prostatic hypertrophy, seizure disorder, sleep apnea and multiple renal cysts. These records also reference service in the US Marine Corps from 1968 to 1973, serving as an active officer from 1970 until his honorable discharge in 1973.

These records also indicate that Mister Welch sustained several traumatic injuries, none of which resulted in any trauma to suggest concussive head injury or anything more serious head injury. In 1968 while in Officer Candidate School in Quantico VA, he sustained some trauma to the face and head with pugil sticks, but the record does not suggest that he sustained any serious head injury, such as concussion. Mr. Welch was assaulted in July of 1985 during which time he sustained injuries to ribs, back neck and head, again without a story suggestive of major head injury. In May 1991, Mr. Welch was diagnosed with cervical spine arthritis. A fall in 1995 induced symptom exacerbation in his hips, neck and back.

In February 2021, Mr. Welch consulted Dr. Markus about right hand tremors. By November 16, 2021, Dr. Carolyn Neff formally diagnosed Mr. Welch with Parkinson's disease as reflected by right sided rest tremor, cogwheel rigidity and bradykinesia.

Mr. Welch continued to treat with Dr. Neff, who noted evidence of mild cognitive impairment on March 10, 2022, as reflected by a score of 23 out 30 on the Montreal Cognitive Assessment. Donepezil therapy was added to the pharmaceutical regimen. Dr. Neff noted that Mr. Welch demonstrated intact capacity for independent activities of daily living and for decision making relative to both medical and financial affairs. As of March 3, 2023, Mr. Welch continued to manifest many symptoms of Parkinson's disease, including tremor, micrographia, tiredness, loss of smell and difficulty with concentration, despite medication increase.

IX. Summary of Opinion

Formulation: Mr. Welch demonstrated findings consistent with mild to moderate, well medicated, idiopathic Parkinson's disease without evidence of any major adverse effects of the disease itself or its treatment. There was no evidence that he is suffering from an atypical form

of the disorder. Cognitively, he is intact per the MoCA VI, conducted while being treated with the central cholinesterase inhibitor, donepezil.

Turning to the potential etiology of Mr. Welch's Parkinson's disease, there is nothing in his history or medical record that is suggestive of a specific etiology of his disease. Most people do not have a specifically definable etiology, much as is the case for Mr. Welch. He has no recognized genetic or inherited risks for the disease. Structural brain disease, such as an angioma, and seizure disorders are not definable risks for the induction of Parkinson's disease. He has experienced some head trauma. He not been exposed to any specifically recognized neurotoxic drugs or metals that induce Parkinsonism. He has not had any definable pesticide exposure. He probably has had relatively higher than average exposure to air pollution, given his long-term residence in Southern California. His greatest identifiable risk factor for the development of Parkinson's disease is his age. Based on the exposure report of Dr. Bailey and the risk assessment of Dr. LaKind, any exposure Mr. Welch sustained while at Camp Lejeune was nearly 5 decades ago.

The assertion that this exposure to contaminated water at Camp Lejeune so many years ago is a significant cause of his Parkinson's disease is inherently flawed as Mr. Welch did not begin to experience any symptoms of the disease until approximately age 74, 13 years past the mean age of onset of Parkinson's disease at 61 years of age. One could just as easily postulate a relationship to his exposure to air pollution, but this would not be sufficient to conclude that such an exposure is the cause of his disease. His disease course has otherwise not been unusual. He does not demonstrate any atypical features of the disease; his medication response is typical and his non-motor symptoms of the disease are all very typical of what is manifest in an individual affected by this disease for 6 years at his age. As noted above, Mr. Welch's age is probably his greatest risk for Parkinson's disease development, but one cannot conclude that even that is the sole etiology of his disorder.

In conclusion, it is my opinion to a reasonable degree of medical certainty that Mr. Welch's Parkinson's disease is not the result of his alleged exposure to contaminated water at Camp Lejeune in the early 1970s. He is suffering with relatively later onset, tremor predominant Parkinson's disease of idiopathic origin. We cannot assign any specific etiology of his disorder, save for possibly his advancing age.

X. Rebuttal of Dr. Schwarz's Opinion

The Plaintiffs' expert, Dr. Heidi Schwarz, has opined that Mr. Welch's exposure was at least as likely as not to be the sole etiology of Mr. Welch's Parkinson's disease. As indicated above, this is an inherently flawed conclusion as it clear that in the vast majority of these patients, one cannot assign a singular, specific cause for their Parkinson's disease, given the multiplicity of potential risk factors for disease induction. In addition, in this particular patient, as already indicated above, the significantly later onset of his disorder than the mean age of onset is utterly inconsistent with disease induction by any exposure five decades previously, which would certainly have accelerated dopaminergic cell death and resulted in a much earlier age of onset.

RE: ROBERT WELCH

PAGE - 13

I have stated all the above within a reasonable degree of medical certainty. I reserve the right to supplement the opinions expressed in this report, subject to the availability of additional materials related to this case.

Sincerely yours,

A handwritten signature in black ink, appearing to read "Stephen M. Gollomp", written in a cursive style.

Stephen M. Gollomp M.D.

References Cited

Abbas N, Lücking CB, Ricard S, Dürr A, Bonifati V, De Michele G, Bouley S, Vaughan JR, Gasser T, Marconi R, Broussolle E, Brefel-Courbon C, Harhangi BS, Oostra BA, Fabrizio E, Böhme GA, Pradier L, Wood NW, Filla A, Meo G, Deneffe P, Agid Y, Brice A. A wide variety of mutations in the parkin gene are responsible for autosomal recessive parkinsonism in Europe. French Parkinson's Disease Genetics Study Group and the European Consortium on Genetic Susceptibility in Parkinson's Disease. *Hum Mol Genet.* 1999 Apr;8(4):567-74.

Abaza A, Jamil A, Gutlapalli SD, Ali M, Oble MJ, Sonia SN, et al. Parkinson's Neuropathology Puzzle: A Systematic Review Uncovering the Pathological Culprits Behind the Neurological Disease. *Cureus* 2023;15(8):e44353

Adams JW, Kirsch D, Calderazzo SM, Tuz-Zahra F, Tripodis Y, Mez J, et al. Substantia Nigra Pathology, Contact Sports Play, and Parkinsonism in Chronic Traumatic Encephalopathy. *JAMA Neurol* 2024.

Ben-Shlomo Y, Darweesh S, Libre-Guerra J, Marras C, San Luciano M, Tanner C. The epidemiology of Parkinson's disease. *Lancet* 2024;403(10423):283-92.

Berg D, Postuma RB, Adler CH, Bloem BR, Chan P, Dubois B, et al. MDS research criteria for prodromal Parkinson's disease. *Mov Disord* 2015;30(12):1600-11.

Bäckström D, Granåsen G, Domellöf ME, Linder J, Jakobson Mo S, Riklund K, Zetterberg H, Blennow K, Forsgren L. Early predictors of mortality in parkinsonism and Parkinson's disease: A population-based study. *Neurology.* 2018 Nov 27;91(22):e2045-e2056.

Bologna M, Truong D, Jankovic J. The etiopathogenetic and pathophysiological spectrum of parkinsonism. *J Neurol Sci* 2022;433:120012.

Borghammer P, Okkels N, Weintraub D. Parkinson's Disease and Dementia with Lewy Bodies: One and the Same. *J Parkinsons Dis* 2024;14(3):383-97.

Brucke T, Brucke C. Dopamine transporter (DAT) imaging in Parkinson's disease and related disorders. *J Neural Transm (Vienna)* 2022;129(5-6):581-94.

Calzetti S, Negrotti A. Outcome of Drug-Induced Parkinsonism in the Elderly: A Permanent Nonprogressive Parkinsonian Syndrome May Occur Following Discontinuation of Cinnarizine and Flunarizine. *Ann Pharmacother.* 2025 Mar; 59(3):289-293.

Chang JJ, Kulkarni S, Pasricha TS. Upper Gastrointestinal Mucosal Damage and Subsequent Risk of Parkinson's disease. *JAMA Netw Open* 2024;7(9):e2431949.

Cook L, Verbrugge J, Schwantes-An TH, Schulze J, Foroud T, Hall A, et al. Parkinson's disease variant detection and disclosure: PD GENERation, a North American Study. *Brain* 2024;147(8):2668-79.

Coukos R, Krainc D. Key genes and convergent pathogenic mechanisms in Parkinson's disease. *Nat Rev Neurosci* 2024;25(6):393-413.

Deliz JR, Tanner CM, Gonzalez-Latapi P. Epidemiology of Parkinson's Disease: An Update. *Curr Neurol Neurosci Rep* 2024;24(6):163-79.

Fereshtehnejad SM, Zeighami Y, Dagher A, Postuma RB. Clinical criteria for subtyping Parkinson's disease: biomarkers and longitudinal progression. *Brain* 2017;140(7):1959-76.

Gibb WR, Lees AJ. The relevance of the Lewy body to the pathogenesis of idiopathic Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1988;51(6):745-52.

Gibbons CH, Levine T, Adler C, Bellaire B, Wang N, Stohl J, et al. Skin Biopsy Detection of Phosphorylated alpha-Synuclein in Patients With Synucleinopathies. *JAMA* 2024;331(15):1298-306.

Grotewold N, Albin RL. Update: Protective and risk factors for Parkinson's disease. *Parkinsonism Relat Disord* 2024;125:107026

Hatano T, Okuzumi A, Matsumoto G, Tsunemi T, Hattori N. alpha-Synuclein: A promising Biomarker for Parkinson's Disease and Related Disorders. *J Mov Disord* 2024;17(2):127-37.

Langston JW. The MPTP Story. *J Parkinsons Dis* 2017;7(s1):S11-S9.

Louis ED, Berry D, Ghanem A, Cosentino SA. Conversion Rate of Essential Tremor to Essential Tremor Parkinson's disease: Data From a Prospective Longitudinal Study. *Neurology Clinical practice* 2023;13(3):e200162.

Morris HR, Lees AJ. Limitations of the alpha-Synuclein Seed Amplification Assay in Clinical Practice: Understanding the Pathological Diversity of Parkinson Syndrome. *JAMA Neurol* 2024.

Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I, Cummings JL, Chertkow H. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *J Am Geriatr Soc.* 2005 Apr;53(4):695-9.

Pitton Rissardo J, Caprara ALF. Neuroimaging Techniques in Differentiating Parkinson's Disease from Drug-Induced Parkinsonism: A Comprehensive Review. *Clin Pract* 2023;13(6):1427-48.

Postuma RB, Berg D, Stern M, Poewe W, Olanow CW, Oertel W, et al. MDS clinical diagnostic criteria for Parkinson's disease. *Mov Disord* 2015;30(12):1591-601.

Postuma RB, Poewe W, Litvan I, Lewis S, Lang AE, Halliday G, et al. Validation of the MDS clinical diagnostic criteria for Parkinson's disease. *Mov Disord* 2018;33(10):1601-8.

Qamar MA, Tall P, van Wamelen D, Wan YM, Rukavina K, Fieldwalker A, et al. Setting the clinical context to non-motor symptoms reflected by Park-pain, Park-sleep, and Park-autonomic subtypes of Parkinson's disease. *International review of neurobiology* 2024;174:1-58.

Rajput AH, Rozdilsky B, Hornykiewicz O, Shannak K, Lee T, Seeman P. Reversible drug-induced parkinsonism. Clinicopathologic study of two cases. *Arch Neurol*. 1982 Oct;39(10):644-6.

Rodriguez-Blazquez C, Schrag A, Rizos A, Chaudhuri KR, Martinez-Martin P, Weintraub D. Prevalence of Non-Motor Symptoms and Non-Motor Fluctuations in Parkinson's Disease Using the MDS-NMS. *Mov Disord Clin Pract* 2021;8(2):231-9.

Simon DK, Tanner CM, Brundin P. Parkinson's disease Epidemiology, Pathology, Genetics, and Pathophysiology. *Clin Geriatr Med* 2020;36(1):1-12.

Tanner CM, Ostrem JL. Parkinson's Disease. *N Engl J Med* 2024;391(5):442-52.

Virameteekul S, Revesz T, Jaunmuktane Z, Warner TT, De Pablo-Fernandez E. Clinical Diagnostic Accuracy of Parkinson's Disease: Where Do We Stand? *Mov Disord* 2023;38(4):558-66.

Weaver FM, Cao L, Stroupe KT, Gonzalez B, Brown E, Colletta K, et al. Post-Traumatic Stress Disorder and Risk of Parkinson's Disease in a Veteran Cohort. *J Parkinsons Dis* 2024.

White DL, Kunik ME, Yu H, Lin HL, Richardson PA, Moore S, et al. Post-Traumatic Stress Disorder is Associated with further Increased Parkinson's Disease Risk in Veterans with Traumatic Brain Injury. *Ann Neurol* 2020;88(1):33-41.

Xing F, Marsili L, Truong DD. Parkinsonism in viral, paraneoplastic, and autoimmune diseases. *J Neurol Sci* 2022;433:120014.

Zetuský WJ, Jankovic J, Pirozzolo FJ. The heterogeneity of Parkinson's disease: clinical and prognostic implications. *Neurol* 1985;35(4):522-6.

Appendix B

MaryAnn Cottrell v. Holtzberg, et al, January 20, 2023, Superior Court of New Jersey, Las Division, Middlesex County, Docket #MID-L-5557-16