Exhibit 548

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May 8, 2025

Expert Report of Stephen M. Gollomp, M.D.
DIANE ROTHCHILD v. UNITED STATES OF AMERICA
Case No. 7:23-CV-00858

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 Pipi 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 Pipi 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 Pipi 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 Ms. Rothchild. 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 Ms. Rothchild is affected by Parkinson's disease. It is also my opinion that there is insufficient evidence to conclude that Ms. Rothchild's Parkinson's disease was caused by her 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 Ms. Rothchild. 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 Ms. Rothchild on March 28, 2025 via a videoconferencing platform.

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

- Expert Report on Diane Rothchild of Judy LaKind, May 8, 2025
- Expert Report on Diane Rothchild 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, corticobasal 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 is highly correlated 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 substantia nigra pars compacta 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, it is clear that in most 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 (Abbas et al., 1999; Cook et al., 2024; Coukos and Krainc, 2024; Dawson and Dawson, 2010). 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 etiologies 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 progress 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 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 drugs that treat type 2 diabetes, the GLP-1 receptor agonists, may potentially 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 Ms. Rothchild:

- Medical Records from Washington University Physicians & Barnes Jewish Hospital
  - Medical Records from Joel Perlmutter, MD; 2013-2024
  - Medical Records from Michele Mueller, FNP; 2017
  - Medical Records from Markesha Dabney, NP; 2024
  - Record from Deborah Oinzebach, OT; 2022
- Medical Records from St. Luke's Hospital
  - Medical Records from Veronica Kim, MD; 2005-2021
  - Medical Records from Stephanie Lumia, MPT; 2013-2019
  - Medical Records from Amy Hunter, PT; 2010-2019
- Additional Medical Records from:
  - Robert Kanterman, MD; 2005-2018
  - Daniel Bauwens, MD; 2005
  - Inta Berzins, MD; 2008-2014
  - Barry Highbloom, MD; 2009
  - Gabriel Desimon, MD; 2009-2015
  - Charles Gavin, MD; 2012-2014
  - Robert Ryerson, MD; 2013-2020
  - Thomas Watson, MD; 2013-2017
  - M. Fazal Majeed, MD; 2013
  - Thomas Niesen, MD; 2014
  - Mohammed Nawas, MD; 2015
  - Mehrdad Sehi, MD; 2016
  - Emad Allam, MD; 2018
  - Kishan Yalavarthi, MD; 2019-2020
  - Christine Osmon, MD; 2021
  - Megan Gau, MD; 2021

I had the opportunity to evaluate Ms. Diane Rothchild neurologically on March 28, 2025, via a remote Zoom link, commencing at 10:00 AM Eastern daylight time and completing at 11:15 AM Eastern daylight time.

Ms. Rothchild is a very pleasant 77-year-old right-handed woman, who was accompanied by her longtime friend, Gary Smith, during the evaluation. Ms. Rothchild related the following history. She first began to experience symptoms to suggest some type of motor dysfunction when she noted listing to the left side while walking in the early 1990s. By 1992, Ms. Rothchild demonstrated a significant tremor in her upper extremities and to a lesser extent in the rest of her body. She was evaluated by Dr. Perlmutter at that time, who began to see her on an annual basis and initially did not introduce any pharmacotherapy. At that time, Dr. Perlmutter indicated to Ms. Rothchild that she was suffering with a hereditary tremor. Somewhere around this time, propranolol 80 mg extended release daily was introduced. However, through the 1990s, her

symptoms progressed with increasing tremor, micrographia, and reduced ability to type on a computer. By the late 1990s, levodopa therapy was introduced with improvement of motor function. Over the ensuing years, Ms. Rothchild's tremor worsened and her ability to carry out her activities of daily living deteriorated. In more recent years, she has been noting end-of-dose effects of levodopa at approximately 3.5 hours, manifested by left foot tremor. She has been experiencing early morning dystonia in both legs, relieved after about 20 minutes by the administration of levodopa. She notes some difficulties of this sort in her legs during the night while in bed. She has not been receiving any augmenting or extended release medications to assist with end of dose. She did not report any dyskinesia. She reported increasing dysphasia for consumption of liquids. She reported that she needed to use weighted utensils to assist with motor control of her hands. She reported occasional falls. Ms. Rothchild indicated that she is no longer driving due to both her difficulties with worsening vision due to her macular degeneration, as well as slowed reaction time and left leg tremor. She reported that she is having difficulty carrying out her accounting tasks for her personal finances. Her friend, Gary Smith, provides assistance in this sphere.

Ms. Rothchild reported fragmented sleep, but thinks she probably does not have rapid eye movement behavior disorder. Her sense of smell has remained intact. She did not report any issues with constipation or other symptoms of autonomic dysfunction.

Over time, the treatment program included input from physical therapy, occupational therapy, and speech therapy.

Ms. Rothchild reported that her past medical history is remarkable for hypercholesterolemia, macular degeneration involving both eyes, worse on the right, dry eye syndrome bilaterally, migraine during childhood, essential tremor, as noted above, and a pelvic fracture sustained in 2010 during a fall.

Ms. Rothchild's current medications include the following:

- propranolol 80 mg extended release once daily;
- famotidine 40 mg once daily;
- Preservision, an eye vitamin and mineral supplement, two capsules daily;
- carbidopa/levodopa 25/100 4 tablets at 8 AM, noon, 4 PM and 8 PM;
- primidone 125 mg in the morning at 250 mg in the evening;
- atorvastatin 40 mg once daily;
- cyclosporine to each eye; and
- multivitamins.

She reported no known allergies.

Her social history is remarkable for having been raised in the coal mining community of Heron, Illinois. She lived in Chesterfield, Missouri for approximately 20 years until 2006 when she moved to her current residence of Lake St. Louis, Missouri. She worked as a special education

teacher until 2009 when her increasing disabilities interfered with her ability to carry out her profession.

Ms. Rothchild does not consume alcohol or caffeine. She has not consumed tobacco products.

Family history is remarkable for her mother being affected with a unilateral tremor disorder, not otherwise specifically categorized and passing at the age of 63. Her father passed at the age of 73 of a stroke. She has one daughter, age 47, apparently in good health, though records indicate she was diagnosed with multiple sclerosis in September 2023.

A comprehensive review of systems was remarkable for intermittent mild headache, the chronic visual disturbances from her macular degeneration, a 15-pound weight loss over the past three years, a rare cough, intermittent heartburn, some nocturia, and intermittent low back pain.

Review of the outside medical records confirmed the history Ms. Rothchild provided as outlined elsewhere in this report.

The 75-minute telemedicine visit was conducted via Zoom, as already noted. This evaluation commenced approximately three hours after a dose of levodopa. 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.

Ms. Rothchild was noted to be a very pleasant, older woman in seemingly relatively good general health. She reported that she was 62 inches tall and weighed 93 lbs. There were no obvious limb deformities or edema.

Neurologically, Ms. Rothchild had seemingly normal cognitive status to social interaction. 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). More semiquantitatively, a cognitive assessment with the MoCA-VI for visual impairment, the appropriate instrument for telemedicine assessment of cognition, was conducted since proper pen and paper measures utilized with the conventional MoCA 8.1 cannot be employed remotely. Ms. Rothchild's global score was normal at 28 out of 30, with no meaningful deficits. A score between 26 and 30 means there is no cognitive deficit. Her memory index score was quite normal for age at 13 out of 15. A score above 9 is normal in all age groups.

Cranial nerve testing demonstrated normal pupillary function and normal eye movements with normal saccades and pursuits. Facial movements were symmetrical with flattening of left nasolabial fold. There was no hypomimia. Tongue protruded in the midline. Voice was intact during this evaluation.

Conventional motor exam did not demonstrate any abnormal postures or involuntary movements, save for a prominent side to side head tremor. The detailed Parkinson's exam was conducted per the Movement Disorder Society-Unified Parkinson Disease Rating Scale (MDS-UPDRS) (Postuma et al., 2015; Virameteekul et al., 2023). This scale is a well validated Parkinson's disease assessment tool, used the world over. Notably, Ms. Rothchild's stride was normal. She was sure footed with her turns, as she walked in her living room area.

The remainder of the examination consisted of a modified MDS-UPDRS, as referenced above, as the lack of an in-person exam precluded tone and postural stability assessment. Ms. Rothchild was evaluated 3.0 hours after her levodopa dose. Part III MDS-UPDRS motor score was 4 consistent with mild Parkinson's disease, as reflected by mildly impaired toe tapping bilaterally, mildly impaired finger tapping in the left hand and mildly impaired pronation-supination with the right hand. No dyskinesias were noted. Her prominent side to side head tremor is not a rating item on the MDS-UPDRS. On part I, MDS-UPDRS, reflective behavioral and nonmotor consequences of Parkinson's disease, she scored 13, consistent with mild to moderate impairment in these spheres. On part II MDS-UPDRS, which reflects the individuals reported motor impairments, she scored 22, consistent with moderate to severe Parkinson's disease. On part IV MDS-UPDRS, a measure of fluctuation of status, she scored 1, consistent with mild affectation with only mild "off" dystonia and relatively little "off" time from levodopa effect. She reported nor demonstrated any dyskinesias.

VIII. Ms. Rothchild's Relevant Medical History

Personal review of the medical records reveals reference to tremor beginning in 1993. Dr. Perlmutter diagnosed Ms. Rothchild with Parkinson's disease after treatment began in 1999, however records of the initial diagnosis have been purged so they are not directly accessible any longer. Subsequent records from September 2010 and September 2012 reference difficulties with osteoporosis, lumbar disc disease and cervical disc disease.

Per the available medical records, Dr. Joseph Perlmutter assessed Ms. Rothchild on May 15, 2013 at which time he thought that she was demonstrating the clinical findings of essential tremor. He could not detect any clinical evidence of Parkinson's disease. This led him to recommend a reduction of high dose carbidopa/levodopa therapy. By May 28, 2014, Dr. Perlmutter noted that the reduction in levodopa dose had led to much more tremor and acknowledged that her clinical picture was most likely that of slowly progressive, very dopa responsive Parkinson's disease. By July 22, 2015, Dr. Perlmutter was more convinced of the diagnosis of Parkinson's disease based upon subtle signs of the disorder on examination. This led him to adjust the medication program with reduction of propranolol dosage and an increase of the carbidopa/levodopa dosage. Dr. Perlmutter continued to see Ms. Rothchild on roughly an annual basis thereafter, noting repeatedly that she was demonstrating signs of mild stage 2 Parkinson's disease with excellent response to carbidopa/levodopa. As late as September 13, 2023, Dr. Perlmutter concluded that Ms. Rothchild continued to demonstrate stage 2 Parkinson's

disease along with long standing action and postural tremor, reasonably well controlled with propranolol, primidone and carbidopa/levodopa, though he thought that the propranolol dosage required upward titration. He also noted the development of leg dyskinesias, worsening balance and some dysphagia along with worsening kinetic tremor.

In September 19, 2024 Dr. Perlmutter noted: "Her PD is likely tremor-predominant subtype which is associated with better prognosis overall which explains her long-term course… She has mild cognitive complaints but no dementia (no change in her ability to do ADLs)."

IX. Summary of Opinion

Formulation: Ms. Rothchild 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. However, the disease is causing some meaningful limitations of her ability to conduct activities of daily living as noted above. There was no specific evidence that she is suffering from an atypical form of the disorder. Cognitively, she is intact per the MoCA VI. In addition, Ms. Rothchild has a kinetic tremor syndrome, consistent with hereditary essential tremor, which has also responded well to medication therapy.

Turning to the potential etiology of Ms. Rothchild's Parkinson's disease, there is considerable evidence that there is a hereditary/genetic etiology for the induction of her disease, as reflected by the very early age of onset of the disorder, the extremely slow progression of the disorder, the ready responsiveness to levodopa, the lack of any cognitive impairment, the lack of rapid eye movement behavior disorder, the lack of major autonomic dysfunction and the presence of leg dystonia (Abbasi et al., 1999; Cook et al., 2024, Dawson and Dawson, 2010). Though the family history is a bit obscure, her mother's affectation with some type of tremor disorder is an additional piece of evidence that is very suggestive of a hereditary origin of Ms. Rothchild's Parkinson's disease (Grotewold and Albin, 2024; Louis et al., 2023). Putting all of these clinical clues together, it is most likely that Ms. Rothchild caries the PARK2 mutation (Parkin gene) as the etiology of her genetically modulated Parkinson's disease. In the alternative, though far less likely, she could be carrying the PINK1 mutation (PINK1 gene) (Dawson and Dawson, 2010).

Ms. Rothchild has not reported exposure to any specifically recognized neurotoxic drugs or metals that induce Parkinsonism. She has not had any definable pesticide exposure. Based on the reports of Dr. LaKind and Dr. Bailey, any exposure that Ms. Rothchild sustained while at Camp Lejeune nearly 5 decades ago did not cause an increased risk.

Further, the development of Ms. Rothchild's Parkinson's disease indicates a genetic component. Early onset and slow progression suggest a genetic cause. Ms. Rothchild was diagnosed with Parkinsons at 45 and, as demonstrated by her IME and Dr. Perlmutter's notes, a slow progression.

The assertion that this exposure to contaminated water at Camp Lejeune so many years ago is a significant cause of her Parkinson's disease is inherently flawed as Ms. Rothchild has a compelling clinical picture pointing to a genetic etiology for her disorder. Her disease course has been extremely slow and relatively benign, again very atypical for Parkinson's disease associated with toxic injury to the dopaminergic neurons in the substantia nigra or significant depletion of dopaminergic neurons in this structure as seen in more typical idiopathic Parkinson's disease.

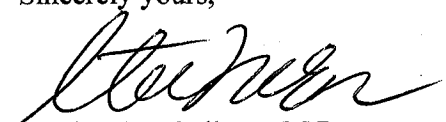
In conclusion, it is my opinion to a reasonable degree of medical certainty that Ms. Rothchild's Parkinson's disease is not the result of her alleged exposure to contaminated water at Camp Lejeune in the early 1970s. She is suffering with relatively early onset, tremor predominant Parkinson's disease of probable genetic origin, most likely PARK2. I cannot assign any other specific etiology of her disorder.

X. Rebuttal of Dr. Andruska's Opinion

The Plaintiffs' expert, Dr. Kristin Andruska, has opined that Ms. Rothchild's exposure was at least as likely as not to be the sole etiology of her 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. More specifically in Ms. Rothchild's case, we have very compelling clinical evidence that is very suggestive of a genetic etiology of her Parkinson's disease, rather than being the result of any neurotoxic exposure.

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,



Stephen M. Gollomp M.D.

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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