In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: August 11, 2025

YVONNE SIMPSON,	*	
Petitioner,	*	No. 17-944V
v.	*	Special Master Young
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Leah VaSahnja Durant, Law Offices of Leah V. Durant, PLLC, Washington, DC, for Petitioner.
Felicia Langel, United States Department of Justice, Washington, DC, for Respondent.

DECISION ON ENTITLEMENT[1]

On July 13, 2017, Yvonne Simpson ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program").[2] Pet., ECF No. 1. Petitioner alleged the influenza ("flu") vaccine she received on December 14, 2010, caused her to suffer from Guillian-Barré Syndrome ("GBS"). *Id.* at 1.

After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards,[3] I find that Petitioner has failed to provide

[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755 ("the Vaccine Act" or "Act"). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

[3] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the decision will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding

preponderant evidence that the flu vaccine she received on December 14, 2010, caused her to develop GBS. Accordingly, Petitioner is not entitled to compensation.

I. Procedural History

Petitioner filed her petition on July 13, 2017. Pet. Petitioner filed medical records via compact disc on July 28, 2017. Pet'r's Exs. 1–11, ECF No. 8. Petitioner filed additional medical records and an affidavit on October 26, 2017. Pet'r's Exs. 12–15, ECF No. 11. Respondent filed his Rule 4(c) report, arguing against compensation, on June 15, 2018. ECF No. 21. That same day, Respondent filed a motion to dismiss, arguing that Petitioner's claim was time barred. ECF No. 22. Petitioner filed a response on September 5, 2018, and Respondent filed a reply on April 4, 2019. ECF Nos. 26, 31. On August 7, 2019, former Chief Special Master Dorsey denied Respondent's motion to dismiss. ECF No. 33. This case was reassigned to my chambers on October 29, 2019. ECF No. 34.

On March 6, 2020, Petitioner filed an expert report from Ahmet Hoke, M.D., Ph.D., as well as accompanying medical literature. Pet'r's Exs. 16–18, ECF No. 38. On September 14, 2020, Respondent filed an expert report from Peter Donofrio, M.D., and accompanying medical literature. Resp't's Ex. A, Resp't's Exs. A1–A5, Resp't's Ex. B, ECF No. 42. Petitioner filed a supplemental report from Dr. Hoke and medical literature on January 27, 2022. Pet'r's Exs. 19–33, ECF No. 47. Respondent filed a supplemental report from Dr. Donofrio and medical literature on March 29, 2022. Resp't's Ex. C, Resp't's Exs. C1–C6, ECF No. 48. And on September 9, 2022, Petitioner filed a final supplemental expert report from Dr. Hoke. Pet'r's Ex. 43, ECF No. 51.

On September 19, 2022, a hearing was scheduled for October 26 and 27, 2023. ECF No. 52; *see also* ECF No. 54. Petitioner filed her pre-hearing brief on June 14, 2023. Pet'r's Pre-Hearing Br., ECF No. 57. Respondent filed his pre-hearing brief on August 14, 2023. Resp't's Pre-Hearing Br., ECF No. 59. Petitioner filed a pre-hearing reply brief on September 13, 2023. Pet'r's Pre-Hearing Reply, ECF No. 62.

An entitlement hearing was held on October 26, 2023. Min. Entry, docketed Oct. 26, 2023; Tr. 1. Dr. Hoke and Dr. Donofrio testified at the hearing. Tr. 3. Petitioner filed a post-hearing brief on January 9, 2024. Pet'r's Post-Hearing Br., ECF No. 74. On February 23, 2024, Respondent filed his post-hearing brief. Resp't's Post-Hearing Br., ECF No 76. Petitioner filed a post-hearing reply brief on April 5, 2024. Pet'r's Post-Hearing Reply, ECF No. 79.

This matter is now ripe for consideration.

II. Factual Background

A. Medical Records

Petitioner was forty-two years old when she received a flu vaccine at the office of her primary care physician ("PCP") on December 14, 2010. Pet'r's Ex. 1 at 1. Her pre-vaccination

certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

medical history is significant for hypertension, diabetes, hypothyroidism, migraine headaches, chronic ear infections, insomnia, anxiety, and depression. Pet'r's Ex. 2 at 196–220.

On February 3, 2011, Petitioner presented to Greenview Regional Hospital ("GRH") reporting four days of tingling and numbness in her hands and feet. Pet'r's Ex. 2 at 198. Petitioner reported she was in her usual state of health until four days prior when she first noticed her feet and hands feeling like they were going to sleep, with progression into her mid-thighs and elbows. *Id.* She reported that she had "not noticed any weakness, ha[d] not dropped any objects or items, and [] had associated fatigue." *Id.* The day prior to her visit to GRH, Petitioner was unable to work and was "in bed [six] hours throughout the day sleeping." *Id.* On the day that she was seen, Petitioner also reported a tingling sensation in her mouth, tongue, and lips. *Id*. Petitioner was admitted for a neurological evaluation with the assessment of "ascending paresthesia for the past [four] days." *Id.* at 198–202.

Neurologist Jianhua Zhu, M.D., Ph.D. performed a neurological evaluation which showed glove stocking sensory loss with normal strength and hyperreflexia. Pet'r's Ex. 2 at 83–84. Dr. Zhu noted that Petitioner's underlying diabetes clearly interfered with objective examination such that they were not sure if the glove stocking distribution sensory loss was Petitioner's baseline sensory deficit, or it was indeed part of the neural process. *Id.* at 202. Dr. Zhu opined that Petitioner did not have a motor component to her symptoms and her symptoms "were not consistent with diabetic peripheral neuropathy." *Id.* at 196. He ordered magnetic resonance imaging ("MRI") that showed prominent disc herniations at C5-6 and C6-7 with spinal cord flattening, but no signal abnormality. *Id.* Petitioner was stable and discharged the following day, February 4, 2011, for further work-up as an outpatient. *Id.* at 197. Dr. Zhu considered cervical myelopathy and transverse myelitis. *Id.* He also noted that her condition "was thought less likely to be related to [GBS]." *Id.* Petitioner's discharge diagnosis was "progressive ascending paresthesias (unknown etiology)." *Id.* at 196. Petitioner returned to GRH on February 7 and February 10, 2011, with complaints of nausea and pain in her back and neck, and full body pain, respectively. Pet'r's Ex. 3 at 435–60. She was assessed with and treated for cervical disc disease and cervical radiculopathy. *Id.*

Four days later, on February 14, 2011, Petitioner presented to GRH in a wheelchair with complaints of progressing paresthesias and new onset of weakness. Pet'r's Ex. 2 at 216–17. Since her discharge on February 4, 2011, "she woke up with numbness in her tongue and numbness in the left side of the face [that] ha[d] subsequently disappeared." *Id.* at 216. Petitioner explained that her "neck [was] messed up," and "she ha[d] continued tingling and numbness with intermittent muscular weakness in her arms and legs, the tingling and numbness ha[d] elevated to the level of the knees on both legs[,] and [was] elevated to the level of the elbows on both arms." *Id.* at 219. Petitioner also reported that she had become weak, was unable to stand or walk on her own, and had difficulty reaching and grasping for objects due to weakened grip strength. *Id*. A repeat cervical spine MRI showed no change in the past ten days. *Id.* at 78. Dr. Zhu performed an examination where he observed significant weakness in Petitioner's bilateral upper and lower extremities. Additionally, her examination was affected by her neck and back pain. *Id.* at 79–80. Dr. Zhu doubted that Petitioner's cervical disc herniation explained all her symptoms. *Id.* Because of Petitioner's excellent reflexes, he noted that the chance of GBS was low but ordered a lumbar puncture. *Id.* at 77–80. Petitioner's spinal fluid showed elevated protein, consistent with GBS. *Id.* at 214; Pet'r's Ex. 3 at 67–68. She was given a five-day course of intravenous immunoglobulin

("IVIG") therapy "and she responded quite well." Pet'r's Ex. 2 at 214. Further, Petitioner's deep tendon reflexes were found to be "going down and almost disappearing on the left side." *Id.* Petitioner was admitted to the intensive care unit ("ICU") to watch her respiration, but she never experienced any respiratory distress. *Id.* at 213. Petitioner remained at GRH until February 22, 2011, when she was discharged for rehabilitation. *Id.* at 214. Her final diagnosis was GBS, confirmed via spinal tap, and herniated nucleus pulposus (disc) at the level of C5-C6 and C6-C7. *Id.* at 213.

Petitioner was transferred to Southern Kentucky Rehabilitation Hospital for intensive outpatient rehabilitation, including physical and occupational therapy ("PT" and "OT"). Pet'r's Ex. 2 at 213; Pet'r's Ex. 4 at 10. Petitioner requested early discharge before completing her full rehabilitation course and was discharged on March 1, 2011. Pet'r's Ex. 4 at 6–8. Her PT evaluation at discharge reflected a need for minimal assistance "for transfers and gaiting 38 feet with rolling walker. . . . She was able to propel wheelchair 60 feet [with] standby assist[ance]." *Id.* at 6. Petitioner's OT evaluation at discharge reflected "standby assist[ance] for eating and grooming, min[imal] assist[ance] for bathing and upper body dressing and lower body dressing, and max[imum] assist[ance] for toileting." *Id.* She was instructed to continue PT and OT at home and receive registered nurse and aide services. *Id.*

On March 8, 2011, Petitioner saw Dr. Zhu for a follow up. Pet'r's Ex. 2 at 75–76. He noted that Petitioner had improved from the IVIG and "continue[d] to improve," though she still had some tingling in her limbs, trace reflexes, glove stocking sensory loss, and globally decreased strength, especially in her lower extremities. *Id.* Petitioner underwent additional electromyography/nerve conduction study ("EMG/NCS") studies on March 21, 2011. *Id.* at 71–73. The indication was "numbness, tingling, weakness in all four extremities. Neck and lower back pain. Recently diagnosed and treated [for] GBS." *Id.* at 71. The study revealed severe demyelinating and axonal polyneuropathy in Petitioner's bilateral upper and lower extremities. *Id.* at 71–73. Petitioner continued with a routine follow-up examination on June 23, 2011, and a pre-employment physical examination on September 8, 2011. *Id.* 39–55. Dr. Chhabra detailed "post [GBS]- some residual tremors and weakness on the left side[, with] intentional tremors left more than right." *Id.* at 40. She also noted that Petitioner "fe[lt] she got [GBS] after the flu shot." *Id.* at 39.

Petitioner was evaluated by an orthopedist for neck pain on March 21, 2012, Pet'r's Ex. 2 at 32. There were appointments with a primary care provider documented through 2014, but there are no neurology records or records that mention neurologic symptoms.

The next complaint of neurologic symptoms was on September 9, 2014. Pet'r's Ex. 3 at 41. Petitioner presented to the emergency department ("ED") with complaints of neck and right upper extremity pain. *Id.* Assessment revealed a "normal inspection, non-tender, no swelling, motor intact distally, sensory intact distally, decreased [range of motion ("ROM")] due to pain in right neck, [and] grips strong and equal." *Id.* The clinical impression was cervical radiculopathy. *Id.* at 44. Due to her GBS history, a repeat cervical spine MRI was performed outpatient on September 29, 2014. *Id.* at 29. Results showed prominent canal stenosis with cord flattening at C5-6 and C6-7. Pet'r's Ex. 2 at 448. The following month, Petitioner began seeing neurosurgeon Clark Bernard, M.D., for worsening chronic neck pain over the previous two to three months. Pet'r's Ex. 7 at 2–15. Per Dr. Bernard's recommendation, Petitioner underwent anterior cervical disc fusion

at C5-6 and C6-7 on November 24, 2014. *Id.* at 15-18. Petitioner saw Dr. Bernard post surgery on December 10, 2014. *Id.* at 6–7. An examination showed that Petitioner had a "reasonable [ROM] of her cervical spine." *Id.* at 7. During a follow-up appointment on January 29, 2015, Petitioner reported that she still had "some right shoulder pain [but n]o real significant cervical radiculopathy." *Id.* at 8. On April 3, 2015, Petitioner saw Dr. Bernard with complaints of sharp right shoulder and arm pain and reported that "her feet had been tingling ever since she had an episode of [GBS]." *Id.* at 10. EMG studies were completed on April 27, 2015, because of a complaint of "numbness of bilateral upper extremities." *Id.* at 19. The results were abnormal with "evidence for predominantly demyelinating motor polyneuropathy of [her] bilateral upper extremities," and a "[d]emyelinating and axonal sensory polyneuropathy of [her] bilateral lower extremities. *Id.* at 19–20. Dr. Bernard concluded that Petitioner's condition was consistent her GBS diagnosis and that she "may well have gone into a chronic inflammatory demyelinating polyneuropathy [("CIDP")] state." *Id*. On May 6, 2015, Petitioner was seen again and reported "right arm pain, numbness, shooting and throbbing pain." *Id.* at 12. Dr. Bernard ordered a lateral cervical x-ray and referred Petitioner to neurologist Dr. Eme Igbokwe for CIDP treatment options. *Id.*

On May 18, 2015, Petitioner presented to Dr. Igbokwe for consultation regarding "CIDP [complaints of] numbness and tingling in right arm." Pet'r's. Ex. 2 at 141. Dr. Igbokwe found weakness proximally and distally in Petitioner's arms and legs with normal reflexes. *Id.* at 142–43. He diagnosed Petitioner with CIDP and planned to initiate a five-day IVIG course, followed by monthly treatments. *Id.* at 143.

Dr. Igbokwe ordered that Petitioner begin receiving monthly IVIG for her CIDP in September 2015. Pet'r's Ex. 2 at 106. On October 12, 2015, Petitioner underwent a new patient evaluation with Dr. Chambers and reported "significant improvement" on IVIG. *Id.* at 111. She also reported that she had "lost good IV access" and had a port-a-cath placed that same month. *Id.* at 108–17. In November 2015, about six months after initiating monthly IVIG treatments, Petitioner presented with complaints of persistent hand cramping and neck and back pain. *Id.* at 101. She also described post-infusion side pain and neck pain. *Id.*

On February 11, 2016, Petitioner was prescribed Lyrica for recurring numbness in her feet and hands. Pet'r's Ex. 2 at 373. The following month, on March 21, 2016, Petitioner was hospitalized for three days with the discharge diagnoses of "ischemic colitis, GBS, abdominal pain [and] hypertension." *Id.* at 315. During that hospitalization, neurologist Dr. Zhu noted that Petitioner had developed CIDP, "since the course ha[d] evolved greater than two months." *Id.* at 325. Additionally, Dr. Zhu was "not entirely sure if [Petitioner was] not in remission." *Id.* Dr. Zhu suggested several alternatives to Petitioner current treatment course, including a higher IVIG dose administered less often or the use of immunosuppressants. *Id.* Petitioner continued to be monitored by neurology and for other complaints, including gastroenterological issues and her diabetes through June 2016 for her CIDP. *Id.* at 239–335.

Petitioner moved to Tennessee and established care with a new PCP, Kailash Bajaj, M.D., on June 8, 2016. Pet'r's Ex. 8 at 44. Petitioner reported that she had been on monthly IVIG infusions until March 2016 and that her weakness and other symptoms had remained well controlled until that time. *Id.* Petitioner reported that she was again having "some weakness and

numbness in both arms and legs (up to her knees),” and that she was very worried. *Id.* Dr. Bajaj referred Petitioner to a local neurologist. *Id.* On June 13, 2016, Petitioner called Dr. Igbokwe and requested to resume IVIG because she was having “additional numbness in all four extremities.” Pet’r’s Ex. 2 at 269. Dr. Igbokwe agreed to have her restart IVIG. *Id.*

Petitioner established care with a new neurologist in Tennessee, Michael Dew, M.D., on July 21, 2016. Pet’r’s Ex. 6 at 9. Petitioner reported that she had numbness in both “feet largely from the ankle down,” that “seem[ed] to ascend somewhat as she [got] closer to the requirement for her next IVIG therapy.” *Id.* Dr. Dew noted that Petitioner’s account did “initially sound like the possibility of [GBS] but her description of persistent complaints on EMG and waxing and waning problems raise[d] concern that this [was] more appropriately considered for CIDP.” *Id.* at 12. Dr. Dew noted that her neurologic examination was remarkable only for distal sensory loss and no motor changes with “persistent and robust reflexes[,] which would be very surprising for immune mediated polyneuropathies.” *Id.*

On August 25, 2016, Petitioner saw Dr. Dew for follow up and reported watery diarrhea following recent IVIG administration. Pet’r’s Ex. 6 at 5. Updated EMG/NCS were consistent with a sensorimotor polyneuropathy; however, they showed a mixture of axonal and demyelinating features without a specific conduction block. *Id.* Dr. Dew noted “[h]er EMG/[NCS] [was] really more in keeping with her history of diabetes than CIDP which, additionally, raise[d] concerns whether she should be maintained on immunomodulatory therapy.” *Id.* at 7–8. Dr. Dew decided to discontinue IVIG due to Petitioner’s gastrointestinal issues and history of colitis and monitor her symptoms. *Id.* at 8. He noted her diagnosis as “[t]ype 2 diabetes mellitus with diabetic neuropathy.” *Id.* at 7. When Petitioner returned to Dr. Dew on December 28, 2016, for follow up, her port was removed due to the discontinuation of her IVIG treatments. *Id.* at 1. Petitioner reported no new numbness, weakness, or falls, and Dr. Dew noted that her symptoms had not progressed as he would expect after discontinuing IVIG therapy, if she was predominately suffering from an immune mediated polyneuropathy. *Id.* at 3. Petitioner was encouraged to maintain good control of her blood sugars and return in six months. *Id.*

When Petitioner returned to Dr. Dew on June 27, 2017, she reported “increasing numbness in both of her hands.” Pet’r’s Ex. 13 at 5. The symptoms in her legs had not changed. *Id.* Dr. Dew maintained that Petitioner had “diffuse sensorimotor polyneuropathy related to her history of diabetes,” and noted that her hand symptoms “may suggest an overlapping median mononeuropathy due to entrapment.” *Id.* at 7. He ordered EMG testing to sort out “overlapping entrapment neuropathy.” *Id.* On August 31, 2017, Petitioner returned to Dr. Dew and reported that she was “dropped from her [PCP] due, apparently, to lack of resources to care for her.” *Id.* at 1. Since her last examination, Petitioner’s blood sugars had remained under 130, and she was wearing wrist splints for her hand symptoms, which she felt helped. *Id.* Petitioner reported some lower extremity numbness and Dr. Dew planned to increase her Lyrica. *Id.* at 3–14.

On October 13, 2017, Petitioner saw her new PCP, Richard K. Reed, M.D. Pet’r’s Ex. 15 at 1. Petitioner reported that she was having difficulty with her hands, numbness in her legs, and difficultly walking. *Id.* On examination, Dr. Reed found marked decreased sensation in both of her feet. *Id.* at 4. He recommended labs and an MRI of her lumbar spine as he was “concerned about

her leg weakness," and felt "[s]ome of her neurologic findings could in fact be from lumbar disc disease." *Id.*

The records indicated that Petitioner next saw Dr. Dew on June 19, 2019, for follow up for dysesthesia associated with her diabetic neuropathy. Pet'r's Ex. 37 at 288–90. She complained of numbness, tingling, burning, and balance difficulty and felt her medication, nortriptyline, wore off prior to her next dose. *Id.* She was also still taking Lyrica. *Id.* at 288.

After moving back to Kentucky, Petitioner saw Dr. Igbokwe on October 22, 2019. Pet'r's Ex. 37 at 267. She explained that she had moved to East Tennessee for a while and had not been treated for the prior two years. *Id.* at 267. Petitioner complained of numbness in her right hand and reported that she "drops things at times." *Id.* Her examination was normal. *Id.* Dr. Igbokwe's assessment included numbness and tingling of the upper right extremity and CIDP. *Id.* He ordered an EMG. *Id.*

An EMG performed on December 3, 2019, revealed findings consistent with a demyelinating motor neuropathy of the bilateral medial nerves consistent with moderately severe bilateral carpal tunnel syndrome, demyelinating motor neuropathy of the bilateral ulnar nerves, and mixed axonal and demyelinating sensory neuropathy of the bilateral upper extremities. Pet'r's Ex. 37 at 266. On December 12, 2019, Dr. Igbokwe diagnosed Petitioner with bilateral carpal tunnel syndrome and maintained his former CIDP diagnosis. *Id.* at 264. He recommended wrist splints and follow up in one to two months. *Id.* Petitioner saw Dr. Igbokwe in February and June of 2020, and his assessment remained carpal tunnel syndrome and CIDP. *Id.* at 239–47. She underwent "[b]ilateral carpal tunnel release done in January and March 2020, left and right hand respectively." *Id.* at 239.

On March 22, 2021, Petitioner was referred to rheumatologist Asad Fraser, M.D., for evaluation of possible inflammatory arthritis and polyarthralgia. Pet'r's Ex. 37 at 235–38. She complained that the pain in her hands was worse since undergoing carpal tunnel surgery four months earlier. *Id.* Dr. Fraser felt Petitioner might be suffering from "an inflammatory process, especially with the soft tissue swelling of the hands," and she expressed concern of potential rheumatoid arthritis. *Id.* at 237. Dr. Fraser also noted that Petitioner had "developed some osteoarthritis changes, especially with the loss of motion and crepitus of knees." *Id.* Petitioner began a course of steroids for osteoarthritis and arthralgias. *Id.* at 221–25.

Petitioner saw Dr. Igbokwe in April and May 2021 and had no new complaints except for chronic pain. Pet'r's Ex. 37 at 204–08, 214–18. His impression remained CIDP and bilateral carpal tunnel syndrome. *See id.* In September 2021, Petitioner was diagnosed with connective tissue disease when she presented to her rheumatologist, Dr. Fraser, unable to make a fist with her right hand. *Id.* at 185–90. She began taking methotrexate. *Id.* Petitioner continued to see Dr. Igbokwe for CIDP. *Id.* at 87–93. Petitioner also continued care with her rheumatologist with complaints of pain in her hands and neck. *See id.* at 12, 22, 36, 44, 52, 61, 69, 99-104.

B. Petitioner's Affidavit

In her affidavit filed on October 26, 2017, Petitioner described first experiencing "extreme fatigue" four days after her flu vaccination. Pet'r's Ex. 12 at ¶ 1. She explained that she remembered the timing because she celebrated Christmas early that year and traveled to Tennessee on December 18, 2010, with her family. *Id.* Following her vaccination, Petitioner stated that she was unable to dress or go to the restroom without help. *Id.* at ¶ 2. She lost her job and missed an opportunity for career advancement through training to become an EMT. *Id.* Petitioner also lost her "medical insurance due to getting sick," her home, and her car. *Id.* Due to fatigue "and shaking all the time," Petitioner was "not able to lead a normal life." *Id.*

Petitioner also described how the IVIG treatments required the insertion of a port into her chest and resulted in increased blood pressure, migraines and nausea. Pet'r's Ex. 12 at ¶ 2. She reported that she experienced uncontrollable headaches almost every day. *Id.* at ¶ 3. Instead of helping her "earn more money," Petitioner reported feeling "helpless" after her vaccination. *Id.* at ¶ 4.

III. Experts

A. Expert Qualifications

1. Petitioner's Expert, Dr. Ahmet Hoke, M.D., Ph.D.

Dr. Hoke is a board-certified neurologist. Tr. 13. He received his medical degree from Hacettepe University in Ankara, Turkey. Pet'r's Ex. 17 at 1. Thereafter, he received a Ph.D. in Neurobiology from Case Western Reserve University. *Id.* He completed an internal medicine residency, a neurology residency, and a neuromuscular fellowship. *Id.* at 1–2. Dr. Hoke is currently a Professor of Neurology and Neurosciences at Johns Hopkins University School of Medicine. Tr. 11. He teaches and sees patients, primarily with peripheral neuropathies, in a clinical setting. Tr. 12. He testified he sees close to 500 patients per year, including patients with CIDP and GBS. Tr. 12–13. Dr. Hoke has authored or co-authored numerous publications. Pet'r's Ex. 17 at 2–14, 25–28.

2. Respondent's Expert, Dr. Peter Donofrio, M.D.

Dr. Donofrio is a board-certified neurologist.[4] Tr. 101; Resp't's Ex. D at 2. He received his medical degree from Ohio State University School of Medicine. Resp't's Ex. D at 1. He completed an internal medicine residency, a neurology residency, and a neuromuscular fellowship. *Id.* at 2. He was a practicing physician for 46 years before retiring in 2021. Tr. 98. During that time, he evaluated and treated patients with GBS and CIDP. Tr. 101–02. Also prior to retiring, Dr. Donofrio was a Professor of Neurology at Vanderbilt University. Tr. 99. Currently, he is the Vice President of the Board of Directors of the GBS/CIDP Foundation. *Id.* Dr. Donofrio has authored or co-authored numerous publications. Tr. 102; Resp't's Ex. D at 13–31.

[4] Dr. Donofrio is no longer licensed to practice medicine. Tr. 101.

B. Expert Reports and Testimony

1. Expert Reports Round One

a. Petitioner's Expert

In his expert report, Dr. Hoke noted that Petitioner's medical records were "well outlined in various [e]xhibits and in the Respondent's report." Pet'r's Ex. 16 at 1. He opined that "it is more likely than not that [Petitioner] developed GBS following her [flu] vaccination. She had a partial recovery from her GBS and subsequently likely developed diabetic polyneuropathy." *Id.* at 7. He based this opinion on her presentation, including "a subacute ascending paresthesias that developed into quadriparesis with associated cyto-albuminemic dissociation and subsequent EMG/NCS findings of demyelinating polyneuropathy, all characteristics of [GBS]." *Id.* Dr. Hoke noted that there were no "other risk factors such as viral or bacterial infections in the preceding [three] months." *Id.*

According to Dr. Hoke, Petitioner "did not have CIDP, but her recovery was complicated by her diabetes and she likely developed diabetic polyneuropathy later on." Pet'r's Ex. 16 at 7. He argued that her misdiagnosis was likely because Petitioner's co-existing cervical spine disease "may [have] counter[ed] the effect of reduced reflexes" often seen in demyelinating conditions." *Id.* Dr. Hoke further noted that "10% of GBS cases may have preserved reflexes." *Id.* (citing Pet'r's Ex. 18).[5] He also referenced Petitioner's persistent paresthesias and weakness as evidence that she did not respond as expected to IVIG treatment for an immune-mediated demyelinating polyneuropathy. *Id.*

Lastly, Dr. Hoke did not believe that Petitioner suffered from entrapment neuropathy in her upper limbs, despite the results of her nerve conduction studies. Pet'r's Ex. 16 at 7. He again noted that she did not respond to the common treatment, splints, and the diagnosis would not explain the rapid onset and "her lower extremity symptoms or the diffuse weakness she developed." *Id.*

b. Respondent's Expert

In opining on Petitioner's diagnosis, Dr. Donofrio noted that from 2010 through 2017, Petitioner's medical condition was characterized by her treaters as "GBS, CIDP, diabetic neuropathy, peripheral neuropathy, possible transverse myelitis, and a cervical myelopathy." Resp't's Ex. A at 7. Throughout that time, he argued it "unlikely that [P]etitioner suffered GBS . . . except for a brief period in 2011." *Id.* The chronic nature of her illness, "and the apparent improvement she experienced from the IVIG years after vaccination and initial [symptom onset] argue against a diagnosis of GBS." *Id.* Dr. Donofrio identified GBS as "a monophasic illness from which most patients improve within [four] weeks of disease onset and require only one series of IVIG infusions." *Id.* at 8. He conceded that a second series of IVIG is needed in some GBS patients, "if there is a treatment-related fluctuation . . . within a few weeks of initial improvement after

[5] Nobuhiro Yuki et al., *Guillain-Barré Syndrome Associated with Normal or Exaggerated Tendon Reflexes*, 259 J. NEUROLOGY 1181 (2012).

receiving IVIG." *Id.* He argued that as in Petitioner's case, "[c]ontinued use of IVIG monthly for years after an initial bout of weakness would eliminate GBS as a credible diagnosis." *Id.*

There were several instances in Petitioner's record of continued symptomology and a need for additional IVIG treatments that Dr. Donofrio described as more indicative of CIDP instead of GBS, including in October 2015, and June of 2016. Resp't's Ex. A at 8. In CIDP cases, he explained it is "common practice to prescribe IVIG chronically for months to years." *Id.* He recounted how her treaters considered other conditions based on her symptom presentation. *Id.* For example, Dr. Zhu initially considered cervical myelopathy and transverse myelitis; and in 2011, Dr. Schwank considered cervical cord disease. *Id.* Dr. Donofrio further noted that Petitioner acknowledged her ultimate diagnosis of CIDP in her petition. *Id.*

Dr. Donofrio next characterized the nerve conduction study and EMG data from March 21, 2011, as "unreliable." Resp't's Ex. A at 8. He explained that the velocities were not properly calibrated for a patient with Petitioner's medical history and presentation. *Id.* He also opined that the prolonged distal latency findings "would be unlikely in a patient with a demyelinating peripheral neuropathy, the type that is seen in GBS." *Id.* The absence of a finding of reduced recruitment of motor unit action potentials was another unexpected result that caused Dr. Donofrio to question the testing. *Id.* at 9. Dr. Donofrio questioned whether the timing of Petitioner's cervical spine surgery in October 2017 suggests that "many of her symptoms in February 2011 could have been due to the cervical cord compression." *Id.* Lastly, he was unable to provide an opinion on the April 27, 2015 EMG results because, "[n]o raw data were included . . . to analyze[ ]." *Id.* Notably, he described Petitioner's August 10, 2016 EMG results, with no raw data, "as consistent with CIDP and diabetic neuropathy." *Id.*

Despite the initial diagnosis of GBS, Dr. Donofrio emphasized that CIDP was the diagnosis "mentioned in many of the outpatient notes after the year 2011." Resp't's Ex. A at 9. He explained that "[a]pproximately [five] to 15 % of cases of CIDP have an acute onset." *Id.* (citing Resp't's Ex. A5).[6] Consequently, "the initial diagnosis is almost always GBS and the diagnosis is later changed to CIDP." *Id.* Petitioner's symptom recurrence and positive response to IVIG likely led to such a change in her case. *Id.* He argued that if Petitioner recovered from GBS "and then progressed because of diabetic neuropathy, this evolution would not explain the need for IVIG infusions from 2014 to 2016 to maintain function." *Id.* Dr. Donofrio did not believe that Petitioner ever had GBS. *Id.* at 10. He reiterated that Petitioner "most likely had CIDP with an acute presentation in 2012." *Id.*

Dr. Donofrio filed studies that specifically address the relationship between GBS and CIDP. In Willison et al.,[7] the authors noted that in patients that are initially diagnosed with GBS based on a rapidly progressive course but then have "subsequently have further progression exceeding [four] weeks, CIDP should be considered instead of GBS." Resp't's Ex. A3 at 8. These cases are indicative of the "5% of patients initially diagnosed with [GBS that] were eventually found to have acute onset chronic inflammatory demyelinating neuropathy." *Id.* This consideration is especially prudent, according to the authors in cases wherein patients "have three or more

[6] L. Ruts et al., *Distinguishing Acute-Onset CIDP From Fluctuating Guillain-Barré Syndrome*, 74 NEUROLOGY 1680 (2010).
[7] Hugh J. Willison et al., *Guillain-Barré Syndrome*, 388 LANCET 717 (2016).

periods with clinical deterioration, or when there is a new deterioration after [eight] weeks from onset of weakness." *Id.*

The Ruts et al. study specifically aimed "to provide criteria that can help to distinguish between GBS-[treatment related fluctuations ("TRFs")] and [acute ("A")-CIDP] in the early phase of disease. Resp't's Ex. A5 at 1. Acknowledging the difficulty in differentiation because they share "many symptoms and signs in the acute phase of disease," the authors identified the time to reach nadir of symptoms as the main factor clinicians consider. *Id.* They continued that by definition, the time frame for GBS is within four weeks and more than two months for CIDP. *Id.* However, they cautioned that a significant number of patients, eight to 16%, do not fit neatly within one presentation. *Id.* at 2. Indeed, it may be very difficult to distinguish between a GBS-TRF patient's secondary deterioration and a second episode of weakness in an A-CIDP patient. *Id.* These patients would need a repeated IVIG course or "a long-term maintenance treatment with steroids, IVIG, or plasma exchange with or without immunosuppressive agents." *Id.* The authors compared 16 GBS-TRF patients to eight A-CIDP patients in a prospective longitudinal study over the course of one year. *Id.* Within the study population, "5% of the patients initially diagnosed with GBS were revealed to have A-CIDP." *Id.* at 5. All A-CIDP patients had nadir within four weeks; "however, active disease exceeded [eight] weeks." *Id.* Conversely, none of the GBS-TRF group "deteriorated after eight weeks," or had more than two TRFs. *Id.* Additionally, "[t]he median time to reach nadir, first exacerbation, and second exacerbation was significantly longer in the A-CIDP group compared to the GBS-TRF group." *Id.* The authors also noted trends that were not 100% applicable to either group. *Id.* Some A-CIPD patients were able "to walk independently at nadir of the different deteriorations," compared to the none of the GBS-TRF patients. *Id.* Also, some GBS-TRF patients "needed artificial ventilation at nadir of the different deteriorations, compared to none of the A-CIDP patients." *Id.* In summary, the authors opined:

> Patients with A-CIDP generally are less severely disabled compared to patients with GBS-TRF. Patients remaining able to walk independently at nadir of different deteriorations, having no cranial dysfunction, and showing electrophysiologic features likely to be compatible with CIDP are more likely to have A-CIDP.

Id. at 6. *See also* Resp't's Ex. A4 at 2 (In patients with prominent sensory symptoms, acute-onset CIDP is likely in a patient initially diagnosed with GBS if deterioration continues for more than two months from onset or if more than three treatment-related fluctuations occur.).[8]

2. Expert Reports Round Two

a. Petitioner's Expert

The majority of Dr. Hoke's supplemental report, beginning with the Summary of Pertinent Medical Facts on page one through the section titled Explain Relevant Disease or Diseases midway

[8] Joint Task Force of the EFNS and the PNS, *European Federation of Neurological Societies/Peripheral Nerve Society Guideline on Management of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force of the European Federation of Neurological Societies and the Peripheral Nerve Society- First Revision*, 15 J. PERIPHERAL NERVOUS SYSTEM 1 (2010).

through page eight, is a nearly verbatim resubmission of his initial expert report down to the punctuation. *See* Pet'r's Ex. 16 at 1–8; Pet'r's Ex. 19 at 1–8. Dr. Hoke added two sentences to further explain some of the aspects of Petitioner's presentation that would be considered atypical for GBS. Pet'r's Ex. 19 at 7. "In the context of someone with hyperreflexia (likely due to her cervical spine disease), it is not unusual for a patient to have a delayed reduction in reflexes and that this reduction in reflexes may not be complete (i.e. not turn into areflexia)." *Id.* Additionally, he noted that "[t]he abnormalities in her nerve conduction studies and persistent sensory complaints were misinterpreted as CIDP, when a more likely explanation – partial and slow improvement from GBS and presence of DPN – is present." *Id.* at 8.

In support of his diagnosis, Dr. Hoke filed the Sheikh[9] article that "review[ed] the clinical features, diagnosis and differential diagnosis, prognosis, pathogenesis, and current and upcoming treatments of [GBS]" Pet'r's Ex. 27 at 1. The article described GBS as a group of acute-onset disorders that are usually monophasic. *Id.* GBS is believed to be autoimmune as it is usually post-infectious with the two most common forms being acute inflammatory demyelinating polyradiculoneuropathy ("AIDP") and acute motor axonal neuropathy ("AMAN"). *Id.* The author explained that the diagnosis is "primarily clinical," but cited to several sources that identify criteria used to paralytic forms of GBS. *Id.* at 2. Two required features across multiple sources are "progressive muscle weakness that must occur in more than one limb with relative symmetry on both sides of the body and areflexia, implying loss of reflexes and/or hypoactive reflexes." *Id.* The author wrote, "[t]he majority of patients with AIDP present with sensory pain," but "AIDP is a predominantly motor polyradiculoneuropathy." *Id.* at 3. He also highlighted the monophasic course of patients in more than 95% of patients. *Id.* at 4. A third requirement identified by the Sheikh article was disease nadir within four weeks, although "a highly variable static period before the onset of recovery" can occur. *Id.* The recovery period typically begins within weeks of nadir but can be delayed, and most "patients make a complete recovery over [six] to 12 months." *Id.* Although death can occur in rare cases, "[t]he most common residual features include fatigue, pain, paresthesia, and reduced muscle strength." *Id.* The Sheikh article distinguished CIDP by a symptom progression that exceeds four weeks or "three treatment-related fluctuations in the first [eight] weeks." *Id.* at 13.

Dr. Hoke also relied on the Allen[10] article that discusses the misdiagnosis of CIDP. Pet'r's Ex. 20. The article begins with a historical summary of the misdiagnosis of CIDP and a hypothesis as to why. *Id.* at 1. The author explained that "there is no reliable biomarker by which to diagnose CIDP," and a diagnosis is determined by an aggregate interpretation of a patient's detailed health history, physical assessment data, electrophysiologic studies, and other data. *Id.* at 2. He noted that a 2015 patient review "uncovered that almost half (47%) of patients who were diagnosed" and treated with CIDP "did not actually have the condition." *Id.* Diagnostic criteria proposed by the European Federation of Neurological Societies/Peripheral Nerve Society ("EFNS/PNS") are favored by the author because they "were developed for use during daily clinical care in addition to clinical trials." *Id.* Relying on these and other guidelines, the author reviewed previously conducted studies and sought "to highlight some of the specific pitfalls that are encountered by the clinician during the diagnostic process." *Id.* at 2–3.

[9] Kazim A. Sheikh, *Guillain-Barré Syndrome*, 26 CONTINUUM 1184 (2020).
[10] Jeffrey A. Allen, *The Misdiagnosis of CIDP: A Review*, 9 NEUROLOGY & THERAPY 43 (2020).

The Allen article asserted that typical CIDP presents with "relatively symmetric proximal and distal weakness and sensory dysfunction in all extremities." Pet'r's Ex. 20 at 3. Atypical variants also involve "disruption of motor and/or sensory peripheral nerve function" but the pattern of involvement or modality affected may vary. *Id.* The author cautioned that "if the defining symptom of the disease is pain, fatigue, or a similar nebulous symptom absent the hallmarks of numbness and/or weakness . . . , then the diagnosis of CIDP is unlikely to be correct." *Id.* AIDP was identified as a demyelinating polyneuropathy differential diagnosis with characteristics that include symptom progression ending within four weeks, a peak of demyelinating features within two to three weeks, and normal or elevated cerebrospinal fluid ("CSF") protein levels. *Id.* at 7.

Dr. Hoke reiterated his position that Petitioner "never had CIDP but had a partial recovery from her GBS with a subsequent development of likely diabetic polyneuropathy." Pet'r's Ex. 19 at 8. He then turned to causation and offered the theory of vaccine-induced GBS via molecular mimicry. *Id.* Specifically, he referenced Tishler and Shoenfeld's[11] textbook explanation "in an individual who is genetically predisposed." *Id.* (citing Pet'r's Ex. 28). Molecular mimicry is a process whereby "antigenic determinants of the microorganisms are recognized by the host's immune system as similar to its own antigenic determinants and, because of the structural resemblance, antibodies and autoreactive T cells not only destroy the invading pathogen but can react with host tissues as well." *Id.* at 8–9. Tishler and Shoenfeld identified Campylobacter jejuni as the bacteria that is responsible for the development of GBS via this mechanism. *Id.* at 9; Pet'r's Ex. 28 at 3. In further explaining molecular mimicry, Dr. Hoke asserted that molecular mimicry can be amplified "when both B-cells (via antibodies they produce) and auto-reactive T cells act in concert." Pet'r's Ex. 19 at 9. He also suggested that epitope spreading and MHC binding motifs can facilitate pathological cross reactivity with vaccine components derived from infectious agents. *Id.* This mechanism is applicable to flu-caused GBS. *Id.*

Petitioner's 47-day symptom onset is outside of what Dr. Hoke described as the "arbitrary deadline of 42 days" used to define Program Table cases; however, he argued it is still within a reasonable period of time for a vaccine-induced immune response. Pet'r's Ex. 19 at 9–10. Although Dr. Zhu, the physician who diagnosed Petitioner with GBS, did not link her condition to her vaccination, Dr. Hoke noted that Dr. Chhabra recorded that "she got (GBS) after the flu shot." *Id.* at 10 (citing Pet'r's Ex. 2 at 39).

Lastly, Dr. Hoke summarized why Petitioner's presentation was inconsistent with CIDP. Pet'r's Ex. 19 at 10. He argued that "there were no documented improvements with IVIG," and that CIDP is inconsistent with a lack of progressive weakness between her improvement in early 2011 and 2014." *Id.* Dr. Hoke characterized the "majority of her complaints [as] sensory, which is typical among GBS patients with incomplete recovery." *Id.*

b. Respondent's Expert

Dr. Donofrio responded to Dr. Hoke's supplemental report by summarizing his opinion as to Petitioner's diagnosis compared to Dr. Hoke's. Resp't's Ex. C at 1. He noted that Dr. Hoke "argued two separate illnesses explain the onset and persistence of [] [P]etitioner's symptoms[:]"

[11] Moshe Tishler & Yehuda Shoenfeld, *Vaccines and Autoimmunity*, *in* THE AUTOIMMUNE DISEASES 309 (Noel R. Rose & Ian R. Mackay eds., 2006).

GBS, followed by diabetic neuropathy. *Id.* Dr. Donofrio reiterated his opinion that CIDP "is a better explanation for [] [P]etitioner's initial disease and subsequent symptoms and signs." *Id.* He acknowledged that while only a minority of CIDP patients present with acute symptoms, "most of these patients are told they have GBS before the illness fully manifests itself." *Id.* He added that in these cases, it can take "months to years" before the patient's diagnosis is retrospectively determined to be CIDP. *Id.* Furthermore, unlike A-CIDP, Dr. Donofrio argued that Petitioner's presentation was inconsistent with diabetic neuropathy. *Id.* He noted that her treaters, both pre and immediately post vaccination, did not characterize Petitioner's diabetes as uncontrolled or consider a diagnosis of neuropathy. *Id.*

Disagreeing with Dr. Dew's assessment that Petitioner suffered from diabetic neuropathy, Dr. Donofrio argued that "[n]ot all patients with CIDP respond to treatment with IVIG." Resp't's Ex. C at 3. He referred to the Dalakas[12] paper to assert that "up to 40-55% of patients with CIDP do not worsen when IVIG is stopped. *Id.* (citing Resp't's Ex. C, Tab 3). Dr. Donofrio noted that "Dr. Dew's examination [on July 21, 2016] showed clear improvement from her IVIG treatments, as compared to [P]etitioner's condition May 18, 2015." Resp't's Ex. C, Tab 3 at 3. Additionally, "[i]t is common in clinical practice that objective improvements in strength, reflexes, and sensation might not be verified even when subjective improvement is reported." *Id.* at 3–4.

The Dalakas paper described CIDP in practical terms as "the chronic counterpart of [GBS] owing to various electrophysiological, histological and immune similarities. CIDP differs from GBS, however, by its time course, mode of evolution, prognosis and responsiveness to steroids." Resp't's Ex. C, Tab 3 at 1. CIDP is chronic, but the author described it as "often monophasic with stepwise progression; . . . [and] relapsing with spontaneous remissions, necessitating periodic evaluation of the usefulness of continued immunotherapy." *Id.* at 2. The article further discussed differences between GBS and CIDP. *Id.* Most notably, that "CIDP symptoms do not usually reach [nadir] until at least [two] months from disease onset; by contrast, GBS evolves over less than [four] weeks." *Id.* The author noted however, some patients that present with "subacute onset and a monophasic course that falls between the time frame of the two diseases." *Id.* Still others "experience a more acute onset and peak symptoms within [six to eight] weeks of onset, resembling GBS." *Id.*

The cause of CIDP is still largely unknown, and the Dalakas paper noted that "given the immunopathological similarities with GBS and relapsing-remitting experimental allergic neuritis, one would expect that in CIDP, [components of the immune system would likewise] work in concert to induce an [] attack on currently unknown peripheral nerve antigens." Resp't's Ex. C, Tab 3 at 4. Further, "[n]o pathogenic autoantibody or single triggering antigen has yet been identified," and "various infections have been implicated" but not proven. *Id.* There are also reported increased incidence of CIDP following vaccination with melanoma lysates. *Id.* "Molecular mimicry could play a part in this association, as several carbohydrates epitopes are shared by myelin and melanoma cells." *Id.*

[12] Marinos C. Dalakas, *Advances in the Diagnosis, Pathogenesis and Treatment of CIDP*, 7 NATURE REVS. NEUROLOGY 507 (2011).

It is Dr. Donofrio's opinion that the Grave et al.[13] series of case studies undertaken during the period wherein Petitioner would have received her vaccine is evidence that the flu vaccines administered between 2010 and 2014 did not cause GBS. Resp't's Ex. C at 4 (citing Resp't's Ex. C, Tab 6). In the study of 3,523 cases of GBS that occurred during the 2010/2011 to 2013/2014 flu vaccination seasons, 527 patients with GBS were vaccinated against flu, and "[a] total of 140 patients developed GBS during the 42 days following [flu] vaccination." Resp't's Ex. C, Tab 6 at 1. The numbers revealed that "no association between seasonal [flu] vaccination and GBS was shown during the 42 days following vaccination." *Id.* Dr. Donofrio reasoned that "the incidence of GBS during the risk periods did not differ from the incidence of GBS during the control periods;" therefore, there was no higher incidence of GBS patients following flu vaccination. Resp't's Ex. C at 4. His opinion extends to Dr. Hoke's contention that a 47-day period between vaccination and onset is appropriate for flu vaccine induced GBS. *Id.* Dr. Donofrio stated that "[t]he science does not support a 47-day delay between [flu] vaccination and the onset of GBS or CIDP." *Id.*

Dr. Donofrio also expressed doubt that Petitioner had diabetes because her bloodwork was "normal almost every time the tests were performed." Resp't's Ex. C at 5. Accordingly, he argued that "it is difficult[] to build an argument that she had diabetic neuropathy." *Id.*

3. Petitioner's Expert's Third Report

In a final supplemental report, Dr. Hoke reiterated that Petitioner's most recent treating neurologist diagnosed her with diabetic neurology, consistent with his opinion that Petitioner suffered from GBS and later, diabetic neuropathy. Pet'r's Ex. 34 at 1. He referred to Dr. Dew's notation that Petitioner's "condition did not worsen when her IVIG was discontinued." *Id.* Next, Dr. Hoke responded to Dr. Donofrio's objection to his causation theory and relied on the Vaccine Injury Table to establish the link between the flu vaccine and GBS. *Id.* at 2. He noted that "[c]learly, the literature has conflicting data on whether there is a small increase in risk of GBS with [flu] vaccine." *Id.* However, he asserted that this issue is made moot due to the injury's presence on the Table. *Id.* Lastly, Dr. Hoke reiterated his believe that the 42-day timeframe for onset is arbitrary and five additional days did not change his analysis. *Id.*

4. Testimony

a. Petitioner's Expert

Dr. Hoke testified that Petitioner had "[GBS] at the onset of her illness back in 2011." Tr. 17:17-18. He characterized Petitioner's symptoms of ascending paresthesia, gradual muscle weakness over two to three weeks, and cytoalbuminologic dissociation as "fairly classical" and "very typical." Tr. 17:22-25, 18:5-8. For Dr. Hoke, "the most important part was really the acute onset, the ascending parethesias and paralysis and the supporting laboratory and electrophysiological evidence and her response to IVIG." Tr. 18:20-24. He identified Petitioner's symptom onset as around January 30, 2011, based on her initial presentation to her PCP on February 3, 2011, wherein she complained of a "four-day history of progressive parathesias." Tr.

[13] Clémence Grave et al., *Seasonal Influenza Vaccine and Guillain-Barré Syndrome*, 94 NEUROLOGY 2168 (2020).

19:10-11. The symptoms began as "tingling in her toes when she first woke up . . . by the evening, tingling was in her hands, and then it moved up to the elbows and up to her legs." Tr. 19:23-25, 20:1. Dr. Hoke continued that a GBS diagnosis is further supported by the abnormal results from her spinal tap. Tr. 21:17-18. He also characterized her reported facial numbness as "unusual for CIDP -- or diabetic neuropathy." Tr. 22:23-25. Furthering distinguishing CIDP patients, Dr. Hoke explained that "typically symptoms start a lot slower and slowly progress over months, whereas in GBS, typically nadir is reached within two to four weeks." Tr. 27:11-13. GBS patients also will show improvement without treatment, "whereas CIDP, unless a treatment is instituted, the patients will not show spontaneous improvement." Tr. 27:14-17.

When asked about the symptoms Petitioner was experiencing on September 8, 2011, Dr. Hoke described them as "sequelae of GBS." Tr. 28:7. He explained that the peripheral nerves damaged due to GBS undergo a two-step process to recover. Tr. 28:9-14. Damage to myelin can repair with weeks or months, but secondary "damage to the axon . . . takes a long time, and that recovery often is incomplete in GBS patients," years later. Tr. 29:18-23. According to Dr. Hoke, Petitioner was suffering from "active" GBS for "the first month, month and a half" following symptom onset. Tr. 29:2. He was unable to say when Petitioner's GBS/sequelae symptoms stopped, noting that the "presence of her diabetes could be impairing her recovery from the GBS." Tr. 29:25, 30:1. Even Petitioner's complaints in May of 2015 could be attributed to her GBS because "she clearly had distal axonal degeneration," which "these patients do no recover fully from." Tr. 30:21-24. Dr. Hoke added that her other conditions, including a herniated disc and entrapment neuropathies, could be some other "reasons for her persistent upper extremity symptoms." Tr. 32:4, 8-12.

Dr. Hoke testified that Dr. Bernard's CIDP diagnosis was based on confusion interpreting Petitioner's EMG results. Tr. 33:16-21. He explained that "electrophysiological recovery often lags," and unfamiliar treaters may misdiagnosis GBS as CIDP even if "nerve conduction studies were not really that classical." Tr. 33:16-21. Dr. Dew ordered another EMG on August 10, 2016, and Dr. Hoke opined that Petitioner's missed axonal demyelinating neuropathy was more indicative to Dr. Dew of "diabetic neuropathy rather than CIDP." Tr. 34:3-7. Petitioner's IVIG was discontinued, and her treatment was no longer consistent with a CIDP diagnosis. Tr. 34:11-12. Dr. Hoke "put aside" the CIDP diagnosis based on this change and "because [Petitioner] had no recurrence of her paralysis or weakness after the IVIG was stopped." *Id.* He agreed with Dr. Dew and opined "that the correct diagnosis at that point and her examination findings were more consistent with perhaps a combination of the sequelae of GBS but more likely that was diabetic neuropathy." Tr. 34:20-24. Dr. Hoke reiterated that based on his 20-year-plus experience, this type of misdiagnosis occurred when patients had "continued fatigue two or three years out [with] demyelinating feature on nerve conduction studies [and they were] put on IVIG with no obvious, clear documentation of an objective improvement in their symptoms." Tr. 36:13-15.

Additionally, Dr. Hoke identified the three factors that he believes are at the center of the confusion around her diagnosis: (1) Petitioner's increased reflexes at symptom onset; (2) complaints of persistent parathesias and fatigue; and (3) failure to respond to starting and stopping IVIG. Tr. 37. He further explained that her comorbidities made each diagnosis harder to identify, noting that "in the fact that she had this GBS that didn't recover fully, she may have had -- or her recovery is impeded because of her diabetes makes it like very difficult to be 100 percent sure of

anything." Tr. 38:15-18. Dr. Hoke described Petitioner's diabetes as "not very well controlled," and opined that "it's very difficult to be sure exactly, you know, five years out,[14] exactly what she has." Tr. 38:23-25, 39:1-3. He asserted that whether Petitioner was suffering from GBS sequelae further complicated by her diabetes, or she was developing diabetic neuropathy proper, "the management would be the same." Tr. 39:7. Dr. Hoke also asserted that even patients with well controlled diabetes can develop peripheral neuropathy that affects small unmyelinated sensory fibers and presents with numbness and tingling in their feet. Tr. 39:16-20.

When asked again why he believed Petitioner had GBS, Dr. Hoke noted the acute onset, the two-to-three-week symptom nadir, and response to the IVIG treatment. Tr. 41:7-10. He also mentioned the imaging and CSF findings. Tr. 41:11-12. Responding directly to Dr. Donofrio's argument that Petitioner had an acute form of CIDP, Dr. Hoke argued that he "would have expected her to have a relapse and worsening [a] couple months after the first IVIG effects wore off, which she didn't." Tr. 41:21-23. Petitioner's four-year-period "with no treatment argues against the CIDP diagnosis or acute onset CIDP." Tr. 42:1-2.

Dr. Hoke next testified about the biological mechanism that he believed was responsible for Petitioner's vaccine-induced GBS. Tr. 43:2-3. He testified that "it's called molecular mimicry, and this has been recognized" by field experts and in the Program. *Id.* Dr. Hoke explained that molecular mimicry occurs when a foreign protein enters the body and the cells and antibodies that try to fight the protein "make a mistake and attack the patient's own peripheral nerves." Tr. 43:15-20. In Petitioner's case, the process took 47 days, which is just outside the Program's Table guidelines of 42 days for presumptive causation. Dr. Hoke cited the Schonberger et al.[15] and Langmuir et al.[16] papers which documented cases with ten-week and up to eight-week onsets, respectively. Tr. 45:23-25, 46:1 (citing Pet'r's Ex. 35; Resp't's Ex. C, Tab 5).

Lastly on direct examination, Dr. Hoke commented on more recent medical records that Petitioner filed. He noted that "she is not on any CIDP-specific treatment, apart from a very low dose of prednisone which is prescribed to her for her connective tissue disease. . . . She's not on IVIG. She's not on high-dose steroids." Tr. 47:9-14.

On cross-examination, Dr. Hoke conceded Petitioner's elevated protein levels in her CSF, her demyelination, and her positive response to IVIG are all typical indicators of CIDP. Tr. 50:10-18, 51:1. He added that her facial symptoms are unusual but not impossible. Tr. 51:2-9. Respondent's counsel asked Dr. Hoke to confirm that Dr. Igbokwe was Petitioner's most recent treater, according to the filed records, and that his diagnosis remained CIDP. Tr. 55:22, 25. Dr. Hoke testified that he was aware of Dr. Bernard's CIDP diagnosis but did not recall Dr. Zhu's opinion that Petitioner had CIDP. Tr. 57:4-5, 47:23, 25. Dr. Hoke discounted Dr. Zhu's opinion because "his note ha[d] a major error with the first sentence saying that IVIG every month ever

[14] Dr. Hoke is referring to Dr. Dew's reconsideration of Petitioner's CIDP diagnosis and consideration of diabetic neuropathy in August of 2016. Pet'r's Ex. 6 at 7.

[15] Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979).

[16] Alexander D. Langmuir et al., *An Epidemiologic and Clinical Evaluation of Guillain-Barré Syndrome Reported in Association with the Administration of Swine Influenza Vaccines*, 119 AM. J. EPIDEMIOLOGY 841 (1984).

since, which is not true." Tr. 59:15-16. He noted that Petitioner reported symptom improvements following her IVIG without corresponding objective findings. Tr. 60:8-12. After Dr. Hoke reviewed records recounting how Petitioner complained of reoccurring bilateral extremity weakness and numbness after her IVIG in 2016. Tr. 64:13. He then questioned the state of her health "from 2011 when she was in between [and] had no symptoms." *Id.*

Petitioner's records from her more recent visits with Dr. Igbokwe in May of 2022 documented normal neurology; though Dr. Hoke agreed that unlike CIDP, diabetic neuropathy is progressive and does not go through periods of remission. Tr. 75:2-14. Upon further questioning by me, Dr. Hoke opined that "the more likely explanation in all of this is that she had GBS and slowly she's recovered, and maybe she never had developed diabetic neuropathy." Tr. 76:18-20. Dr. Hoke acknowledged cases wherein patients with CIDP are treated for six months, go into remission, then relapse several years later. Tr. 81:14-17. However, Petitioner "she wasn't treated with IVIG for six months[and], her initial course did not fit into that acute onset CIDP cases." Tr. 82:1-3. He continued, "if the patient gets the IVIG, improves, and if they don't have a relapse within the first, you know, three to six months, then they can't have CIDP." Tr. 82:12-15.

Dr. Hoke noted that "there hasn't been any strong epidemiological data that links direct infections or vaccines to CIDP cases." Tr. 82:24-25, 83:1. He explained that molecular mimicry has been hypothesized to apply to CIDP. Tr. 83:3-4. That is because "the onset of an illness like GBS [] happens so abruptly and quickly, whereas typical CIDP patients often have symptoms for months before they even inquire . . . . And because of that delay, -- it's almost impossible epidemiologically to determine if there was a trigger." Tr. 83:9-15. Dr. Hoke testified that "[y]ou really can't make that -- the same type of a link between exposure to a potential trigger and the onset of CIDP, because it's so nebulous." Tr. 83:16-18.

On recall examination, Dr. Hoke was asked about his experience with CIDP patients who have long periods of remission. *See* Tr. 185. He testified that he has never seen patients with "an acute onset CIDP with no evidence of CIDP for the first four years and then [they] develop CIDP." Tr. 185:19-20. Alternatively, Dr. Hoke has seen a classical CIDP case where the patient had slow symptom onset, received IVIG therapy, came off treatment and went into remission, and then relapsed many years later. Tr. 185:22-25, 186:1-2. He also asserted that in cases where a relapse occurs and "somebody is truly dependent on IVIG and declares themselves are worsening, . . . you're not going to be able to stop that IVIG so quickly and abruptly in three months later." Tr. 186:12-16. Without a slow taper that is monitored, "they're going to have a relapse." Tr. 186:19.

I asked Dr. Hoke what happened to Petitioner in 2015, if he believed that she suffered from a classic GBS in 2011 and he answered, "to be honest, I don't know." Tr. 194:18. He testified that the later EMG/NCS results "would have been very typical of delayed electrophysiological recovery you would see in a GBS case." Tr. 189:1-3. Dr. Hoke did not believe that Petitioner suffered from diabetic neuropathy because "her exam became completely normal six years later in 2019." Tr. 189:6-8.

b. Respondent's Expert

Dr. Donofrio opined that Petitioner "had and has CIDP," and that her 2020 flu vaccination did not cause her condition. Tr. 104:5, 12-13. He asserted that GBS is a monophasic condition "that presents, evolves, and gets better, and there isn't recurrence." Tr. 106:29-21. There is an uncommon scenario that Dr. Donofrio described wherein "patients [] improve with IVIG and then worsen two or three weeks later, [] due to the continued activity of the inflammatory reaction against the peripheral nervous system. That is called the TRF." Tr. 107:17-21. It is CIDP patients that generally require more long term, IVIG maintenance therapy. Tr. 108:10-11. Dr. Donofrio noted that Petitioner's recurrence "three to four years later" is unusual, but he added "one of the clinical presentations of CIDP is a waxing and waning illness, . . . you can have an initial presentation, and then you may not see a relapse for several years." Tr. 108:15-19. Otherwise, the acute presentation "can look like GBS." Tr. 108:24. CIDP can be mild "primarily with numbness and tingling and little motor involvement." Tr. 109:17-18. There are also more severe forms that require hospitalization and even intensive care with intubation. Tr. 109:20-23. Dr. Donofrio also discussed remission, where the disease goes away, "versus a chronic stability where the patient no longer requires treatment," the latter occurring in about 30 to 40 percent of patients. Tr. 110:16-17.

Petitioner's case was also unusual because she had hyper reflexes, which are usually reduced or absent in GBS and acute CIDP. Tr. 114:18-19. Dr. Donofrio asserted that these results may have led to confusion on the part of her treating neurologist. Tr. 114:22-25. He also noted that there is a small number, about five percent of GBS patients, with hyperreflexia. Tr. 115:12-13. Petitioner's hyper reflexes are likely explained by "cervical cord pathology." Tr. 115:15-18. Dr. Donofrio walked through Petitioner's medical records including her initial hospitalization and GBS diagnosis in February of 2011. Tr. 117:19-22. He noted her tingling symptoms, and the diffuse demyelinating and axonal neuropathy revealed in her nerve conduction study in June of 2011. *Id.* These symptoms are also consistent with GBS and CIDP. *Id.*

Fast forward to 2015 and Dr. Donofrio highlighted as significant Petitioner's "mild to moderate weakness, proximally and distally, in the upper and lower extremities, except for the hands, which were even worse." Tr. 120:20-22. He believed that "she had some remission of her illness, to the point that from 2011 to 2015, at least May of 2015, she was stable, and then she [saw] a neurologist in May of 2015," after four years. Tr. 121:7-10. At this point the GBS diagnosis comes into question and CIDP is considered. Tr. 121:20-22. Dr. Donofrio noted that Dr. Igbokwe diagnosed Petitioner with CIDP and administered another round of IVIG based on her presenting symptoms and April 27, 2015 EMG results. Tr. 125:8, 16. He testified that Petitioner's treatment regimen of monthly IVIG through March of 2016 improved her strength. Tr. 127:18. This is consistent with CIDP, because "IVIG is not a treatment for diabetes or diabetic neuropathy." Tr. 128:1-2. Further, Dr. Donofrio agreed with Dr. Igbokwe that Petitioner's IVIG should not have been stopped due to concerns about ischemic colitis because of her positive response. Tr. 128:14-23.

Petitioner's normal examination on August 25, 2016, could be explained by remission caused by the IVIG or that she was "so sensitive to IVIG that it normalized all her findings on exam. And that's hard to do." Tr. 139:3-4. Dr. Donofrio believed it was the former and that

Petitioner did not have diabetic polyneuropathy. Tr. 139:8-10. He based his diagnosis on "acute presentation, acute onset CIDP 2011, then a recurrence of the illness in 2015, and a good response to IVIG thereafter." Tr. 142:4-6. Relying on the Hughes[17] and Dalakas studies, Dr. Donofrio characterized Petitioner as one of the 30 to 40 percent of patients that "go off of therapy and either go into complete remission or maintain a stable state, not requiring any treatment." Tr. 143:1-3. Hughes et al. sought to study the short- and long-term benefits of IVIG treatments for CIDP patients. Resp't's Ex. C, Tab 4 at 1. The study confirms that IVIG improved CIDP disability for at least two to six weeks compared with placebo. *Id.* at 7. Furthermore, the "extension phase of the trial showed the long-term efficacy" of IVIG, specifically, the use of caprylate-chromatography purified immune globulin intravenous ("IGIV-C"). *Id.* at 8. The Dalakas study added that notwithstanding the positive results following IVIG therapy, "the challenge remains to attempt discontinuation or reduction of immunotherapies to avoid unnecessary exposure to expensive or toxic drugs, without breakthrough of disease." Resp't's Ex. C, Tab 3 at 8.

To assess whether a treatment plan like IVIG has been successful, Dr. Donofrio focused on motor strength examination and reflex improvements. Tr. 144:16-18. He also "put stock in their complaints. Do they say they're improved? Is their numbness [and/or] pain better?" Tr. 144:20-22. Dr. Donofrio opined that "from May 2015 through March of 2016, there was documented improvement in [Petitioner's] strength." Tr. 146:5-7. He also referenced Dr. Chambers's note that "the patient remarked that there was improvement after receiving the infusions of IVIG." Tr 146:10-12. Following this improvement, Petitioner's symptoms "worsen[ed] when the IVIG was stopped because of ischemic colitis, which means that [she] had a recurrence of illness." Tr. 145:16-18. This improvement and worsening led Dr. Donofrio "to go back and call the initial presentation the acute presentation of CIDP, and the episode of worsening in 2015 as recurrence of the illness." Tr. 145:20-22.

Dr. Donofrio then compared Petitioner's presentation to A-CIDP, noting her "onset in the upper and lower extremities, followed later by weakness in the arms and legs, and difficulty with walking and loss of the use of walking." Tr. 148:24-25, 149:1-3. He ended his direct testimony by opining that Petitioner "either didn't have diabetes or she had a fantastically controlled form of diabetes." Tr. 155:3-5. Dr. Donofrio acknowledged that Petitioner was being treated for diabetes, but that her blood sugar and A1c levels were normal. Tr. 155:2-3.

On cross-examination, Dr. Donofrio criticized the treating physician's interpretation of Petitioner's March 21, 2011 EMG. Tr. 166:21. He noted that there were no limb temperatures recorded to ensure a proper reading, and he "was struck by the conduction velocities [that] were too rapid to be recorded in a human being." Tr. 167:2-3, 9-10.

IV. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the timeframe prescribed by the Vaccine Injury Table set forth at

[17] Richard A.C. Hughes et al., *Intravenous Immune Globulin (10% caprylate-chromatography purified) for the Treatment of Chronic Inflammatory Demyelinating Polyradiculoneuropathy (ICE study): A Randomised Placebo-Controlled Trial*, 7 LANCET NEUROLOGY 136 2008).

42 U.S.C. § 300aa-14, as amended by 42 C.F.R. § 100.3; or (2) that petitioner suffered an "off-Table injury," one not listed on the Table, as a result of his receiving a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Petitioner does not allege a Table injury in this case. Thus, she must prove either that her injury was caused-in-fact by a Table vaccine or that a preexisting injury was significantly aggravated by a Table vaccine.

A. Causation-in-Fact – *Althen*

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. 42 U.S.C. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of the Dept. of Health & Hum. Servs*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d at 1278–79. The *Althen* test requires petitioners to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *See id.*

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357,1361(Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. "In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Id*. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. The *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."). Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. This is because "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records,

they are not "binding on the special master or court." 42 U.S.C. § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

V. Discussion

A. Diagnosis

In cases where the diagnosis is contested, "special masters may find whether a preponderance of evidence supports any proposed diagnosis before evaluating whether a vaccine caused that illness." *Hibbard v. Sec'y of Health & Hum. Servs*, No. 07–446V, 2011 WL 1766033, at *6 (Fed. Cl. Spec. Mstr. April 12, 2011) (citing *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1345–46 (Fed. Cir. 2010)). In this case, Petitioner clarified in her post-hearing briefing that she "suffered from GBS as a result of receiving a flu vaccination and that she suffered the sequela of her GBS for longer than six months." Pet'r's Post-Hearing Reply at 1. She further contended that "[i]t is only during the damages phase of the litigation that [she] must address whether the sequela of her GBS extends to any current and ongoing neurological deficit." *Id.* at 2. Petitioner's expert, Dr. Hoke testified at the hearing that she developed GBS 47 days post vaccination and suffered from GBS sequela "most likely for a period of over five years, at least until 2015." *Id.*; *see also* Tr. 30:13. Respondent, through his expert, Dr. Donofrio, countered that Petitioner has always suffered from CIDP, that her claim is unsubstantiated, and as a procedural matter, untimely. Tr. 104:5, 12–13.

GBS is defined by Dorland's Medical Dictionary as a "rapidly progressive ascending motor neuron paralysis." *Guillain-Barré Syndrome*, DORLAND'S MED. DICTIONARY ONLINE,

https://www.dorlandsonline.com/dorland/definition?id=110689 (last visited Aug. 8, 2025). The Sheikh article filed by Petitioner identified bilateral, progressive muscle weakness, reduced reflexes, and symptom nadir within four weeks as required criteria for a GBS diagnosis. Respondent filed the Ruts et al. article that, in distinguishing GBS from CIDP, noted that none of the GBS patients studied continued to deteriorate after eight weeks. Alternatively, CIDP is defined by Dorland's as

> [a] slowly progressive, autoimmune type of demyelinating polyneuropathy characterized by progressive weakness and impaired sensory function in the limbs and enlargement of the peripheral nerves, usually with elevated protein in the cerebrospinal fluid. It occurs most commonly in young adults, particularly males, and is related to [GBS]. Presenting symptoms often include tingling or numbness of the digits, weakness of the limbs, hyporeflexia or areflexia, fatigue, and abnormal sensations.

Chronic Inflammatory Demyelinating Polyneuropathy, DORLAND'S MED. DICTIONARY ONLINE.

Petitioner's Allen article focused on a holistic, patient-specific approach to a CIDP diagnosis. The author relied on the EFNS/PNS criteria and looked for symmetric and diffuse sensory disfunction in patients. The author was wary of the diagnosis for patients with defining symptoms of pain or fatigue and also noted that symptoms should progress beyond four weeks. Dr. Donofrio filed medical literature that focused on chronic and recurrent nature of CIDP. The Ruts et al. article also noted that all A-CIDP patients studied had nadir within four weeks, but active disease continued beyond eight weeks.

It is undisputed that Petitioner suffered from a polyneuropathy, but the specific type was debated even among Petitioner's treaters over the course of several years. Fortunately, the medical providers who examined and diagnosed Petitioner provided a detailed record of the evolution of Petitioner's condition. On February 3, 2011, Petitioner's first complaints of the relevant symptoms, tingling and numbness in her hands and feet, ultimately resulted in hospital admittance and her examination by Dr. Zhu. Dr. Zhu noted her underlying diabetes as a consideration but ultimately found her symptoms to be inconsistent with diabetic neuropathy. After an initial assessment of cervical radiculopathy and a return to the hospital with continued symptom progression to weakness and mobility issues, Dr. Zhu first diagnosed Petitioner with GBS on February 14, 2011. This diagnosis occurred approximately two weeks post symptom onset. At this point, Petitioner had bilateral weakness and ascending tingling and numbness, up to both elbows and knees. She was unable to walk or stand and could not grasp objects. Petitioner also had neck and back pain but retained her reflexes. It is noteworthy that Dr. Zhu initially thought that GBS was not the most likely diagnosis because of Petitioner's maintained reflexes but ordered a lumbar puncture to be sure and confirmed the diagnosis. Petitioner completed her IVIG course, physical and occupational therapies, and was discharged home by March 1, 2011. This discharge occurred approximately two weeks following symptom nadir and four weeks following symptom onset. At discharge, Petitioner required ambulatory assistance and used a rolling walker or wheelchair depending on distance. Both Drs. Hoke and Donofrio agreed that based on Petitioner's clinical presentation, laboratory results, and the information that Dr. Zhu had at that time, GBS was the correct diagnosis.

Five weeks post symptom onset, Petitioner continued to improve but maintained symptoms to a lesser degree with EMG studies that revealed demyelination and axonal polyneuropathy. Despite these continued symptoms and Petitioner's imaging, there are no neurology records or evidence of her condition for a four-year period thereafter. Dr. Hoke initially opined that Petitioner suffered from GBS sequelae during this time and ultimately developed diabetic neuropathy to explain her later worsening symptoms. Although he later reconsidered his opinion related to the diabetic neuropathy, Dr. Hoke asserted that Petitioner's symptom onset fits squarely within the parameters of GBS and is exclusionary for CIDP. Alternatively, Dr. Donofrio argued that Petitioner suffered from a rare variant, A-CIDP, that explains her rapid onset and nadir. He described the four-year period wherein Petitioner did not seek treatment as remission prior to a relapse that occurred in 2014.

The onset and progression of Petitioner's symptoms, including an extended, multi-year remission makes her case an atypical polyneuropathy, regardless of type. The Ruts et al. study effectively identified a small but significant percentage of patients that suffered from A-CIDP and reach nadir within eight weeks. Of note, in these cases, symptom progression also continued beyond eight weeks. However, the study did not address periods of remission that last for years in the interim. Dr. Hoke testified that he has never seen remission last that long, but Dr. Donofrio testified that he has. Based on the opinions of the experts and the medical literature, the time to symptom nadir and extended symptom progression period counterbalance each other as definitive criterion for Petitioner's condition. Because there are no medical records for this time, there is no treater's opinion to help settle this disagreement.

The next set of noteworthy medical records are from 2014, over three years later. Petitioner's complaints are more consistent with a cervical radiculopathy, but an MRI was done given her history of neuropathy. She underwent a cervical disc fusion to address her complaints of neck and upper extremity pain on November 24, 2014. Approximately three months later in early April of 2015, Petitioner complained of tingling in her feet "since her GBS episode." Pet'r's 7 at 10. Her complaints also included bilateral numbness in her upper extremities. Petitioner's treater at this time, Dr. Bernard, suspected CIDP. Petitioner's imaging also showed evidence of demyelinating motor and axonal sensory polyneuropathy of her upper and lower extremities, respectively. On May 18, 2015, Petitioner was diagnosed by Dr. Igbokwe with CIDP. Dr. Igbokwe initiated another five-day IVIG course followed by monthly treatments. In October 2015, Petitioner reported symptom improvement but developed side effects and was hospitalized. Petitioner's treating neurologist during her hospitalization, Dr. Zhu, also believed that Petitioner had developed CIDP. Dr. Zhu's diagnosis is of particular importance because he is the treater that initially diagnosed Petitioner with GBS on February 14, 2011.

The next neurologist to examine Petitioner, Dr. Dew, was concerned with possible diabetic neuropathy given that Petitioner's 2016 imaging results revealed axonal and demyelinating features. Dr. Dew was also concerned about entrapment neuropathies. In Dr. Hoke's expert reports, he agreed with Dr. Dew's opinion that Petitioner had developed diabetic neuropathy. However, additional, more current medical records filed subsequent to Dr. Hoke's reports caused him to retract that opinion during the hearing. When asked directly to characterize Petitioner's condition in 2015, he stated, "to be honest, I don't know." Tr. 194:18. Dr. Hoke explained the imaging results as typical of someone recovering from GBS that also suffered from diabetes, but he also said that

her normal 2019 examination would rule out a diabetic neuropathy diagnosis at any point prior. Tr. 189:6–8. Dr. Donofrio agreed that Petitioner did not suffer from diabetic neuropathy and questioned if she had diabetes at all. Petitioner returned to Dr. Igbokwe who continued treating her for CIDP through 2021. With the exception of Dr. Dew, the medical providers all believed that Petitioner suffered from CIDP with intervening comorbidities, including cervical radiculopathy and carpal tunnel syndrome. Dr. Dew's opinion, which included a diabetic neuropathy diagnosis, was discounted by Petitioner's own expert and is not persuasive to override the other treaters in this case.

The agreement between Petitioner's neurologists, specifically Dr. Zhu, her first treater, and Dr. Igbokwe, her most recent treater (as per the record), is the most persuasive evidence that Petitioner suffered from CIDP. Dr. Bernard added even more credibility to an already fairly settled issue within Petitioner's medical record by being the first to change Petitioner's diagnosis before Drs. Zhu and Igbokwe. Dr. Hoke offered medical literature and testimony that explained how and why CIDP is misdiagnosed, but the factors that he described as exclusionary were not so in the eyes of Petitioner's treaters. Furthermore, they can be explained as rare exceptions to the rule of CIDP.

For example, Dr. Hoke effectively highlighted the unlikelihood of Petitioner going into remission for four years. However, as Petitioner's expert, his role is not just to identify potential weaknesses in Respondent's argument, but to rebut them. Dr. Donofrio responded to Dr. Hoke's critique with medical literature documenting A-CIDP. While the literature does describe remission that can last years, it did not identify a maximum time period. Dr. Hoke testified that he had never seen a four-year remission period, but he was unable to assert that was not possible, and he did not present medical literature that said such. Dr. Donofrio, conversely, testified that a patient "can have an initial presentation, and then [] may not see a relapse for several years. [He] ha[d] personally seen that many times." Tr. 108:18–20.

Dr. Hoke also argued that Petitioner's response to the IVIG courses was not consistent with CIDP. He argued that if Petitioner did suffer from CIDP, then her relapse would have occurred within months of her first IVIG, after the effects wore off. *See* Tr. 41:21–23. He also argued that when she restarted her IVIG, there was no "obvious, clear documentation of an objective improvement." Tr. 36:13–15. This contention is addressed in the medical record by documentation that Petitioner reported symptom improvement following IVIG, and she specifically requested additional treatments from Dr. Igbokwe in 2016 after her 2015 relapse.

After consideration of all of the evidence, including but not limited to Petitioner's medical history, filed medical literature, expert reports and testimony, I find that Petitioner has not presented preponderant evidence that she suffered from GBS. The record contains preponderant evidence that Petitioner suffered from A-CIDP.

B. Statute of Limitations

In the Chief Special Master's August 7, 2019 Order denying Respondent's motion to dismiss, she held that "§16(b) applies to [P]etitioner's non-table GBS claim as alleged in the petition." ECF No. 33 at 9. The Order explained that "the volume of causation-in-fact flu-GBS cases compensated

in the program, followed by the March 2017 Table revision adding GBS to the Table, . . . significantly increase[d] [P]etitioner's likelihood of obtaining compensation for her specific causation-in-fact claim." *Id.* While the claim was allowed to proceed as a GBS causation-in-fact claim, the Order did not consider the merits of Petitioner's assertion that her injury was GBS, nor does it determine the applicability of the lookback provision to CIDP cases under the significantly increased likelihood of success standard. Throughout this litigation and again in his post-hearing Brief[18], Respondent noted that Petitioner's case could only proceed due to the Act's §16(b) lookback provision for GBS cases. Because Respondent maintains that Petitioner suffered from CIDP, not GBS, he reiterated his argument that Petitioner's claim is time-barred.

Petitioner did not address the applicability of the lookback provision to CIDP. Instead, she asserted that the only diagnosis to consider here is GBS. Petitioner also focused on the similarities between GBS and CIDP and an earlier decision that described CIDP as GBS sequelae. That decision, filed in 2017, was issued prior to some of the more recently published medical literature that endeavors to define these conditions. *See* Pet'r's Ex. 24;[19] Pet'r's Ex. 20. Furthermore, there are older articles that were also filed and seek to differentiate the diseases so they can be properly diagnosed and treated. *See* Resp't's Ex. C1; Resp't's Ex. C3. Dorland's describes the diseases as closely related, but neither it, nor any of the medical literature in the record describe CIDP as an evolution of GBS. At best, CIDP is described by the Dalakas article as "the chronic counterpart of [GBS] owing to various electrophysiological, histological and immune similarities." Resp't's Ex. C3 at 1. However, the author continued that "CIDP differs from GBS, [] by its time course, mode of evolution, prognosis, and responsiveness to steroids." *Id.* The chronic nature of CIDP and the difference in symptom progression over time has contributed to Respondent's hesitance to treat it as comparable to GBS for case resolution purposes. Indeed, the presence of GBS on the Table has not reduced litigation involving CIDP. Respondent has consistently argued that the biological mechanism used to explain vaccine-caused GBS, an acute monophasic condition, is not applicable to a chronic progressive disease like CIDP. I agree. There is no volume of CIDP claims previously compensated in the program that increase the likelihood that a CIDP claim will be successful for §16(b) to apply. As the Chief's Order was specific to a non-Table GBS claim, I find it inapplicable to Petitioner's CIDP, as diagnosed by multiple treaters. Petitioner's claim was able to proceed as a GBS claim, but the evidence does not establish a GBS injury. Petitioner did not provide any evidence to support an alternative CIDP claim. That is just as well because the GBS lookback provision is inapplicable, and a CIDP case is time-barred. Petitioner is not entitled to compensation.

C. Causation-in-Fact

I have thoroughly reviewed the entire record, including all of the evidence that Petitioner has presented in support of her general causation theory, logical sequence of cause and effect, and appropriate temporal relationship for flu vaccine-caused GBS. However, I have determined that a detailed analysis of the *Althen* prongs is not necessary in this case because Petitioner has failed to present preponderant evidence that her December 14, 2010 flu vaccination caused her to suffer

[18] Respondent did not seek to relitigate this issue following the Chief Special Master's Order but sought to preserve the argument pending a final decision on diagnosis. *See* Resp't's Post-Hearing Br. at 18 n.13.
[19] Kelly Gwathmey, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy and Its Variants*, 26 CONTINUUM 1205 (2020).

from the injury that she has alleged. For the sake of completeness, I will briefly note that Petitioner presented preponderant evidence of a sound and reliable medical theory that could connect the flu vaccination to GBS pursuant to *Althen* prong one. Furthermore, Petitioner's 47-day onset is appropriate based on off-Table GBS claims that have been successful in the program. Petitioner has not alleged that this causation theory is applicable to CIDP, and she has not alleged a CIDP injury with a different causation theory given the differences between the conditions that have been detailed in this decision. Therefore, Petitioner has failed to present preponderant evidence that her causation theory for flu vaccine-caused GBS is applicable or analogous to her CIDP diagnosis. She has failed to meet her burden under *Althen* prong two.

VI. Conclusion

After a careful review of the record, Petitioner has failed to prove by preponderant evidence that she suffered from GBS that was caused-in-fact by her December 14, 2010 flu vaccination. Accordingly, I **DENY** Petitioner's claim and **DISMISS** her petition.[20]

IT IS SO ORDERED.

s/Herbrina D. S Young
Herbrina D. S. Young
Special Master

[20] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.