In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 18-592V

RICHARD L. WHITE,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: August 4, 2023
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Howard Gold, Gold Law Firm, Wellesley, MA, for Petitioner.

Darryl Wishard, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On April 25, 2018, Richard White filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program")[2] alleging that he experienced Guillain-Barré syndrome ("GBS") caused by his receipt of the influenza ("flu") vaccine on October 12, 2016. Petition (ECF No. 1) at 1. Petitioner later clarified his claim, alleging that he had experienced a variant form of chronic inflammatory demyelinating polyneuropathy ("CIDP"). *See* Prehearing Memorandum, dated August 31, 2022 (ECF. No. 73) at 1.

A trial in this matter was held on September 16, 2022. Now, and for the reasons stated below, I hereby deny entitlement.

[1]Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I. Factual Background and Medical History

Pre-Vaccination Medical History

Petitioner was born on October 2, 1966, and was 50 years old when he received the vaccine at issue. Ex. 3 at 1. He had a past medical history significant for low back pain, chronic hip pain, knee pain, among other things. Ex. 5 at 25–36. Petitioner used a TENS[3] unit, and took Cyclobenzaprine and Oxycodone/Acetaminophen as needed for his low back pain. Ex. 5 at 25, 32.

On February 16, 2016, several months prior to receiving the flu vaccine, Petitioner saw his primary care provider ("PCP"), with complaints of frequent buttock pain that traveled down to his toes. Ex. 5 at 32. Petitioner reported that he used to experience that type of pain "once every couple of weeks," but now experienced it "all [of] the time." *Id.* Petitioner also reported that he felt constant numbness and tingling in his right toes, numbness in his left hand, and weakness in his right leg. *Id.* His PCP assessed Petitioner with chronic right hip pain and referred him to an orthopedic surgeon for further evaluation. *Id.* at 33.

Vaccination and Subsequent Six-Months

Petitioner received the flu vaccine on October 12, 2016. Ex. 3 at 1. At the time of this visit, he reported some symptoms comparable to prior visits, and that seemed potentially neurologic in nature. Ex. 5 at 25 (reporting that when he wakes up, "feels like someone is hanging on him there," plus numbness in toes and some weakness in the right knee). The record does not contain any evidence of an immediate reaction to this vaccination.

Twelve days later, on October 24, 2016, Petitioner saw his chiropractor, Dr. Scott Owens, complaining of low back pain, bilateral hip pain, and bilateral leg pain. Ex. 10 at 1. Petitioner was experiencing spasms in the para lumbar spine and exhibited absent reflexes at the knees and ankles. *Id.* Later that same day, Petitioner took himself to an express care clinic complaining of acute exacerbation of lower back pain. Ex. 7 at 4. He reiterated his complaints of low back pain radiating down his legs, numbness in his feet, and experiencing a feeling of heaviness in his legs. *Id.* Petitioner also reported having received a flu vaccine prior to the increase of his symptoms. *Id.*

Petitioner subsequently underwent a neurological examination which revealed intact cranial nerves and normal sensation and strength throughout. Ex. 10 at 5. A musculoskeletal examination also revealed weakness and numbness in Petitioner's bilateral lower extremities and absent pain sensation up to the mid-calf. *Id.* Petitioner was assessed with low back strain, sacroiliac

[3] "Transcutaneous Electrical Nerve Stimulation" or "TENS" involves "electrical stimulation of nerves for relief of pain, either by electrodes attached to the skin or by an apparatus manually held against the skin; the stimulus interferes with neural transmission of pain signals and thus has an analgesic effect." *Transcutaneous Electrical Nerve Stimulation*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=108464 (last visited Aug. 4, 2023).

inflammation, and GBS following vaccination—he was advised to go to the emergency department for further evaluation, and he did so that same day. *Id.*; Ex. 11 at 301.

At that time, Petitioner reiterated his complaints of low back pain, bilateral leg numbness, and difficulty walking, adding that the express care provider had proposed the symptoms might reflect GBS. Ex. 11 at 301. Petitioner was evaluated by Dr. Kristine Midcap, D.O., who upon examination noted that Petitioner exhibited no loss of patellar reflexes, and no involvement of the upper extremities. *Id.* at 316. Thus, while Dr. Midcap did not dismiss the possibility of a GBS diagnosis, she proposed that further testing and additional consultations might be required to confirm or rule it out. *Id.* Petitioner was thereafter formally admitted to the hospital on October 25, 2016. Ex. 11 at 222.

On October 26, 2016, Petitioner saw neurologist Dr. Christina Kyrtsos, M.D., who noted that Petitioner was "in his normal state of health until 10/21[,] when he noticed a subacute onset of numbness in his feet." Ex. 13 at 13. Dr. Kyrtsos also highlighted the fact that Petitioner had been experiencing an upper respiratory infection ("URI") three weeks before this time (which would have thus begun prior to vaccination), and was at the time of this encounter suffering from an ongoing residual cough. *Id*. Upon examination, Petitioner's strength was documented as 5/5 in the bilateral upper extremities, 4/5 in the bilateral iliopsoas secondary to sciatic pain, and 5/5 in the hamstrings, quadriceps, foot flexors, and foot extensors but his LP revealed elevated cerebrospinal fluid ("CSF") protein levels. *Id.* at 13–14. Dr. Kyrtsos's assessment was likely GBS, but added that it was "[u]nclear if flu shot or recent URI was trigger." *Id.* at 14. She prescribed Petitioner with IVIG[4] therapy treatment, to begin that day. *Id.* at 15.

The next day (October 27, 2016), Petitioner reported improvement in the numbness and tingling in his bilateral lower extremities, but continued to complain of worsening back pain and new numbness and tingling in his left nipple. Ex. 9 at 9. Upon examination, Petitioner exhibited normal strength and reflexes, but showed decreased pinprick in mid-thigh of his right leg and hip of his left leg. *Id.* On October 29, 2016, Petitioner was discharged home. *Id.* at 5. Dr. Kiruba Dharaneeswaran, a neurologist, noted that Petitioner's bilateral lower extremity weakness and numbness with elevated CSF protein levels were concerning for GBS but with an unclear etiology, although he also proposed the possibility that the URI Petitioner had experienced could be the cause. *Id.* at 3.

On November 10, 2016, Petitioner saw his PCP complaining of neuropathy of his feet, acute, intermittent back pain, and difficulty ambulating. Ex. 5 at 1. Upon examination, Petitioner

[4] "Intravenous Immunoglobulin" is defined as "a therapy treatment for patients with antibody deficiencies. It is prepared from a pool of immunoglobulins (antibodies) from the plasma of thousands of healthy donors." *Intravenous Immunoglobulin (IVIG)*, American College of Rheumatology, https://rheumatology.org/patients/intravenous-immunoglobulin-ivig (last visited Aug. 4, 2023).

exhibited tenderness at the lumbar spine, decreased flexion, and lateral rotation, but denied any weakness. *Id.* His PCP assessed him with GBS and degenerative disc disease ("DDD"). *Id.* at 3.

Petitioner saw neurologist Dr. Vijayalakshmi Ragnoor on December 9, 2016, at which time it was noted he had previously been diagnosed with GBS, received five days of IVIG, and showed significant improvement, allowing him to return to work the day after his hospital discharge. Ex. 6 at 1. Following examination, Dr. Ragnoor documented decreased pain, temperature, and vibratory sense below Petitioner's knees bilaterally, and decreased reflexes at the ankles and knees, although his motor exam revealed normal results. *Id.* at 3. He was assessed with GBS as well as neuropathic pain and lumbar spondylosis. *Id.*

In January and April 2017, Petitioner visited his PCP with complaints of general fatigue and weakness, lumber spine tenderness, calf tenderness, lower leg pain, and cramping and weakness in his upper and lower extremities. Ex. 5 at 7, 9. On both occasions, Petitioner was assessed with GBS and DDD, and was advised to continue taking gabapentin to help manage the pain. *Id.* at 7.

On April 18, 2017, Petitioner again saw Dr. Ragnoor, now reporting bilateral leg numbness below the calf when driving, and weakness in the hamstrings, calf muscles, and hip extensors. Ex. 6 at 6. Upon examination, Dr. Ragnoor noted that Petitioner exhibited L5-S1 retrolisthesis,[5] normal motor strength, decreased sensation below the knees bilaterally, and absent reflexes at the ankles. *Id.* at 7. He referred Petitioner to the pain clinic to receive a block for the retrolisthesis, and assessed him with GBS, neuropathic pain, and lumbar stenosis moderate to severe. *Id.*

Treatment from 2018 to 2021

Almost a year passed before Petitioner again sought treatment pertaining to the allegations in this case. In May 2018, he presented to the emergency room with a one-week history of bilateral lower extremity numbness. Ex. 12 at 110. Petitioner noted that the numbness had started abruptly, descending from the waist down. *Id.* Upon physical examination, Petitioner exhibited normal strength, except 4/5 strength in the quadriceps and iliopsoas, and a sensory examination was intact to light touch and pinprick throughout the upper extremities with decreased light touch and pinprick below T12-L1. *Id.* at 112. Based on Petitioner's abrupt onset of symptoms, there was "less concern for GBS/AIDP" and "more concern for thoracic/lumbar pathology." *Id.* Petitioner's treating physicians recommended that he undergo an MRI—the results of which indicated some stenosis and ligamentous hypertrophy at T11-T12, and significant lumbar stenosis at levels L1-L2, L3-L4, and L4-L5. Ex. 14 at 215–16.

[5] "Restrolisthesis" or "retrospondylolisthesis" is defined as "posterior displacement of one vertebral body on the subjacent body." *Retrospondylolisthesis*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=43594 (last visited Aug. 4, 2023).

On May 18, 2018, Petitioner underwent EMG[6]/NCS[7] testing, and the results were interpreted as being suggestive of S1 bilateral radiculopathies, but also deemed too mild to be significant. Ex. 14 at 424–25. In addition, there was no ongoing denervation present, and no electrodiagnostic evidence supporting the existence of a demyelinating process. *Id.* The next day, Petitioner had a consult with an infectious disease specialist for idiopathic progressive neuropathy for the past week and prurigo nodularis[8] for one month, but no identifiable infectious etiology for his symptoms was proposed. Ex. 12 at 76. Petitioner was also seen by a rheumatologist, who also found no rheumatologic cause for his symptoms. *Id.* at 82. Three days later, on May 22, 2018, neurology noted that Petitioner's "studies and exams continue to make clinical picture confusing." *Id.* at 163. Petitioner had CSF pleocytosis and elevated protein with negative HSV, and a negative EMG/NCS for peripheral demyelinating condition, leading treaters to believe that GBS was an unlikely diagnosis. *Id*. In addition, Petitioner's brain studies ruled out transverse myelitis, and his neurologic exam was normal but for some numbness from the ankles down. *Id.* at 170.

In June 2018, Petitioner was evaluated at a VA clinic by neurologist Anne Van Cott, M.D. Ex. 12 at 66. Dr. Van Cott noted that Petitioner had been admitted in May 2018 with an "unclear diagnosis at discharge." *Id.* at 67. She also noted that Petitioner's condition remained stable until then, at which point Petitioner began to re-develop progressive bilateral numbness in his lower extremities at the ankle level, but shortly thereafter began experiencing numbness which extended into his torso. *Id.* at 66–67. Following her evaluation, Dr. Van Cott was under the impression that Petitioner's presentation could be related to CIDP—adding, however, that she could not be entirely sure as Petitioner did not exhibit any weakness upon examination. *Id.* at 70. Thus, she recommended a specialized neuromuscular EMG/NCS study. *Id.* at 72. Dr. Van Cott also noted that GBS remained a consideration, although the EMG study performed the prior month was inconclusive and inconsistent with an existing demyelinating process. *Id.* at 70–71.

Petitioner had a neurosurgery consultation on July 10, 2018, at which time he complained of upper extremity numbness. Ex. 14 at 372. After examination, the provider expressed the view that Petitioner did not exhibit classic neurogenic claudication associated with lumbar spine

---

[6] "Electromyography" is defined as mesur[ing] muscle response or electrical activity in response to a nerve's stimulation of the muscle. The test is used to help detect neuromuscular abnormalities." *Electromyography*, Johns Hopkins Medicine, https://www hopkinsmedicine.org/health/treatment-tests-and-therapies/electromyography-emg (last visited Aug. 4, 2023).

[7] "Nerve Conduction Study" is defined as a "diagnostic test that evaluates the function or your peripheral nerves. An NCS can help detect the presence and extent of peripheral nerve damage." *Nerve Conduction Study*, https://my.clevelandclinic.org/health/treatments/24821-nerve-conduction-study (last visited Aug. 4, 2023).

[8] "Nodular prurigo" is defined as "a chronic, intensely pruritic form of neurodermatitis, usually in women, on the limbs, especially the anterior thighs and legs. Characteristics include single or multiple firm nodules that are red, brown, or pink and later become verrucous or fissured; scratching or rubbing of the nodules often makes the condition worse." *Nodular Prurigo*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100673&searchterm=nodular%20prurigo (last visited Aug. 4, 2023).

degenerative joint disease ("DJD"). *Id.* Sixteen days later, on July 26, 2018, an EMG/NCS was interpreted as showing electrodiagnostic evidence of right L4-S1 and left L5-S1 radiculopathies, electrically moderate, with signs of on-going denervation. The report also noted that "an inflammatory/leptomeningeal process is a consideration in this patient," and that "an anterior horn localization is also possible in conjunction with a conus medullaris syndrome given the history." Ex. 12 at 72.

Dr. Van Cott reviewed the July 2018 EMG results on August 15, 2018, and noted that they were "significantly worse in comparison to the patient's study performed in May 2018 and *are not consistent* with a diagnosis of Guillain Barre/AIDP." Ex. 14 at 371 (emphasis added). During this visit, Petitioner reported severe pain, worse since June 2018, with sensory changes at the level of the mid-thigh which ascend to the level of the umbilicus with ambulation. *Id.* It was noted that Petitioner's symptoms were relieved when lying down, although they did not completely resolve. *Id.* Moreover, Petitioner exhibited perianal numbness and difficulty ambulating. *Id.*

On August 19, 2018, Petitioner was admitted to the hospital until August 21st for worsening sensory symptoms. Ex. 14 at 79. Petitioner presented with lower extremity burning pain that became unbearable after 15 minutes of standing or walking, and he complained of numbness in the genital area, perianal region, but with anorgasmia without erectile dysfunction and without bladder or bowel incontinence. *Id.* Upon examination, Petitioner exhibited decreased temperature sensation below T10, and decreased vibratory sense in his lower extremities—it was noted that this presentation was *not* in accordance with his EMG, which showed motor pathology. *Id.* at 79. A lumber puncture was performed and showed normal cell count, but elevated protein (132). *Id.* The differential diagnoses included spinal stenosis vs. autoimmune vs. paraneoplastic vs. infectious/leptomeningeal etiology. *Id.* Petitioner's treating physicians ruled out paraneoplastic and leptomeningeal causes based on negative CT and CSF findings, and neurology recommended that his condition be treated as a sensory variant of CIDP with steroids, and to follow him as an outpatient. *Id.*

On August 28, 2018, Petitioner saw neurologist Paula Clemens, M.D., who documented that it was "difficult to put together the clinical picture with the EMG results and the prior CSF results. The EMG suggests motor involvement, while the clinical picture is almost entirely sensory." Ex. 14 at 350. Dr. Clemens also noted that "either a multiple radiculopathy syndrome due to his widespread DJD, a paraneoplastic syndrome from an occult neoplasm (though the time course back to 2016 argues against this) or an atypical autoimmune polyradiculopathy as an atypical form of CIDP" were being considered. *Id.*

During a visit with Dr. Van Cott on October 17, 2018, Petitioner exhibited a significant loss of large fibers, with absent proprioception and vibration at the ankles and toe and fingertips, as well as sensory loss to pinprick to the nipples and mid arms. Ex. 14 at 297. Petitioner also had difficulty with finger to nose and heel to shin testing and he could not rise from the exam table without losing his balance. *Id.* However, Petitioner exhibited normal strength, and his reflexes

were noted as symmetrical. *Id.* at 299. Dr. Van Cott assessed Petitioner with "[c]ontinued, progressive undefined syndrome with sensory changes/severe incapacitating pain extending from his chest to his lower extremities," and noted that he exhibited "significant [] large fiber sensory loss in the extremities with a higher thoracic sensory level." *Id.*

Petitioner continued IVIG therapy throughout 2020. Ex. 19 at 145, 179. His providers attempted to wean him off the IVIG by scheduling his treatment to once every five weeks; however, Petitioner began developing a heaviness in his legs and numbness in his hands and fingertips by the middle of week three. Ex. 20 at 73. In July 2020, Petitioner's PCP noted that he had persistent polyneuropathy and pain—an extended workup was completed thereafter which was significant for increased free kappa light chains and an abnormal EMG. *Id.* at 143. Based on Petitioner's history, examination, and testing, there was a concern for an inflammatory or leptomeningeal process and possible superimposed anterior horn involvement with a conus medullary syndrome. *Id.*

Petitioner saw neurologist Anthony Bradshaw, M.D., on June 29, 2021. Ex. 20 at 72–83. Upon examination, Petitioner exhibited normal strength except for mild weakness in the ulnar hands and great toe extensors, reduced pinprick to the proximal right shin and left knee, absent vibration in the feet and reduced vibration in the left more than right knee, intact proprioception, diffuse hyporeflexia/areflexia sparing the biceps, positive Romberg, and sensory ataxic gait. *Id.* at 82. Dr. Bradshaw noted:

> These exam findings paired with normal sensory and motor NCS on EMG in 2018 with prominent albumin/cytologic dissociation are most consistent with a diagnosis of a rare condition known as chronic inflammatory sensory polyradiculopathy (CISP) which is an atypical CIDP variant and is IVIG-responsive, similar to run-of-the-mill CIDP.

Id. Thus, Dr. Bradshaw recommended that Petitioner's IVIG treatments be increased to once every three weeks. *Id.*

Petitioner saw Dr. Bradshaw again on November 30, 2012. Ex. 21 at 59. During this visit, Dr. Bradshaw modified Petitioner's diagnosis to "CIS(M)P" (chronic inflammatory sensorimotor polyradiculopathy), a pure nerve root variant of CIDP. *Id.* EMG studies done on October 5, 2021, showed active denervation and/or increased insertional activity throughout the cervical, thoracic, and lumbosacral paraspinal muscles and all lumbosacral myotomes of the left lower extremity as well as reduced tibial, median, and ulnar F-wave persistence with intact sural and radial sensory responses. *Id.* These findings—coupled with an absence of peripheral nerve demyelination—further confirmed localization to the level of the sensory and motor nerve roots, and were therefore deemed to support a diagnosis of CIS(M)P.

II. Witness Testimony and Expert Reports

A. Petitioner's Witnesses

1. *Richard White*

Petitioner testified at hearing on his own behalf. *See generally* Tr. at 5–29. He began by discussing his pre-vaccination medical history—noting that he dealt with some sciatic issues (describing a pain "that went down from [his] buttocks down into [his] right leg"). *Id*. at 6. He had experienced difficulty walking without pain whenever these sciatic issues flared, but that he remained physically active by participating in golf and softball two to three times a week. *Id.* Petitioner also mentioned that he suffered from arthritis in his hip which had started around the same time his sciatic issues began, but eventually had a hip replacement in 2019. *Id.* at 7. Among his sciatic and hip issues, Petitioner noted that he also suffered from "dull numbness" in his left hand as a result of an incident that happened when he was younger and that it never resolved. *Id.* at 18–19.

Next, Petitioner discussed his visit to his PCP on October 12, 2016 (the date of vaccination). Tr. at 10. At this time, he needed to get a prescription renewed for his knee pain, but while there, also mentioned experiencing sciatic issues and numbness in his right toes. *Id.* at 11. Petitioner did not recall suffering from an upper respiratory infection at the time of this visit, however. At most, he was experiencing some sinus drainage, but attributed that to the change in weather. *Id.* at 13. He also noted that neither the IVIG nor the steroid treatment he had received had helped resolve the above-mentioned issues, but instead only provided him temporary relief before getting worse. *Id.* 14–16. Petitioner explained that he eventually lost his job due to his inability to perform his duties, and as a result, lost his health insurance as well. *Id.* at 16–17. This led to a gap in treatment, but he renewed his coverage in May 2018 when he became insured through the VA. *Id.*

Petitioner concluded his testimony with a discussion about when his alleged symptoms began and his subsequent treatment. Tr. at 17. He recalled starting to experience back pain on or around October 20, 2016 (eight days post-vaccination), which he maintained progressed over the next several days. *Id.* at 17–18. He went into work that Monday morning, but had noticed from the time he awoke that his feet were numb. *Id.* He then briefly described starting IVIG treatment in spring 2018—noting that he continues to receive treatment every three weeks, since its benefits only seem to last about that long. *Id.* at 19–20. Petitioner emphasized that following treatment, his numbness would gradually decrease down his thighs to his feet and ankles, but the numbness would slowly start moving up into his torso once the IVIG treatment had worn off. *Id.*

2. *Jeffrey A. Rumbaugh, M.D.*

Dr. Rumbaugh, a licensed neurologist, submitted two written expert reports and testified for the Petitioner, alleging that the flu vaccine could cause CIS(M)P and did so in this case. *See*

generally Tr. at 31–85; Report, dated Mar. 11, 2022 (ECF No. 61-1) ("Rumbaugh First Rep."); Report, dated Aug. 10, 2022 (ECF No. 72-1) ("Rumbaugh Second Rep.").

Dr. Rumbaugh attended Haverford College for his undergraduate degree in chemistry. Curriculum Vitae, filed Mar. 11, 2022 (ECF No. 62-1) ("Rumbaugh CV") at 1. He then attended the University of Rochester for his Master of Science in biochemistry, doctorate in biochemistry, and medical degree. *Id.* He completed his medicine internship and neurology residency at Johns Hopkins Hospital, where he later became the chief resident in neurology. *Id.* Dr. Rumbaugh most recently started a private practice in Tampa, Florida, where he focuses on evaluating and treating patients with multiple sclerosis. Tr. at 31; Rumbaugh First Rep. at 1. Dr. Rumbaugh is also a tele-neurologist at the National Comprehensive Neurology Services in Tampa, Florida. Rumbaugh CV at 1. He is board certified in neurology by the American Board of Psychiatry and Neurology, a member of the American Academy of Neurology and the American Neurological Association, and a licensed physician in Florida. Tr. at 31; Rumbaugh CV at 2. Dr. Rumbaugh has published articles on caring for patients with MS and neurological infections. *Id.* at 3–16. A majority of his basic science research and publications have focused on the neurological complications of human immunodeficiency virus and Lyme disease. Tr. at 56–57.

Dr. Rumbaugh began his testimony by briefly discussing Petitioner's pre-vaccination medical history. He acknowledged that Petitioner suffered from lumbar disc disease, noting that the symptoms Petitioner complained of on the day of vaccination were consistent with that disease Tr. at 32. However, Dr. Rumbaugh opined that Petitioner's symptoms from October 2016 could not be entirely explained by his prior lumbar condition. *Id.* at 35. In particular, he did not deem Petitioner's symptoms to be typical of lumbar disc disease. *Id.* at 35–36. Petitioner would have experienced "back pain, maybe sciatica into a leg or something," not recurrent numbness ascending the legs and up into the abdomen and into the hands and arms, if his subsequent problems could be explained by his prior condition. *Id.* at 35. He also questioned references in the medical records to the presence of an URI, opining that there was no objective evidence corroborating this, and that nasal drainage is not necessarily evidence of one. *Id.* at 31–32.

Dr. Rumbaugh next discussed Petitioner's initial diagnosis. Tr. at 32. He deemed it reasonable for treating physicians to have initially diagnosed Petitioner with GBS in October/November 2016, based on his presentation and the acute onset of his symptoms. *Id.* Moreover, Petitioner described numbness that was ascending his legs, and his treating physicians reported decreased sensation and decreased reflexes—all of which reflected "a fairly typical story and exam for GBS." *Id.* at 34. GBS, as explained by Dr. Rumbaugh, is "typically a self-limited condition" and oftentimes the patients get "completely better or at least nearly." *Id.*

However, Petitioner exhibited ongoing and recurrent symptoms which would later suggest he was suffering from something other than GBS. Tr. at 34. Dr. Rumbaugh in fact concluded that Petitioner's symptoms were initially *mischaracterized* as GBS, when in fact he more likely

suffered from CIS(M)P. *Id.* at 34, 36.[9] Petitioner's symptoms proved to be chronic (he had experienced symptoms since 2016), immune-mediated (noted by his responsiveness to the IVIG), and found in "multiple different nerve or nerve root distributions" (polyradiculopathy). *Id.* Moreover, Petitioner suffered from predominantly sensory symptoms (although Dr. Rumbaugh noted that it is not uncommon for some individuals who have CISP to also exhibit some motor involvement). *Id.*

Dr. Rumbaugh also referred to Petitioner's EMG/NCS studies conducted in 2021, which showed chronic denervation of the muscles and diffusion throughout all the nerve roots along the spinal cord—findings consistent with a diagnosis of CIS(M)P. Tr. at 39–40. By contrast, Petitioner's MRI findings revealed no significant abnormalities within the nerve roots. *Id.* at 40. But Dr. Rumbaugh opined that the absence of such findings did not necessarily exclude a CIS(M)P diagnosis, since the overall clinical evidence supported the diagnosis. *Id.* at 40–41. In fact, Petitioner's elevated CSF protein levels fit the "classic" CSF finding for either GBS, CIDP, CISP, CIS(M)P—"elevated protein with normal or sometimes slightly elevated white cells." *Id.* at 41.

Dr. Rumbaugh next opined that the specific mechanism for how the flu vaccine could have triggered Petitioner's condition was through molecular mimicry. Tr. at 44. As he explained, the immune system is essentially designed to recognize anything that appears foreign to it and to differentiate the foreign entity from itself, in order to respond to the external insult. *Id.* However, sometimes the immune system is unable to successfully differentiate the foreign entity, as it is very possible for foreign proteins to "look" similar to the body's own proteins, even though the immune system employs different mechanisms in order to successfully differentiate foreign antigens from self-antigens. *Id.* at 43, 47. As a result, if a vaccine component looks enough like a self-antigen, and the immune system is unable to differentiate between the two, an autoimmune response becomes possible. *Id.* at 48.

Dr. Rumbaugh proposed that virtually every autoimmune disease is "triggered by some environmental factor"—oftentimes a virus, but also potentially a vaccine—resulting in a self-attack due to this misrecognition. Tr. at 48. But because each individual is genetically different, autoimmune disease is also driven by "a component that has to do with a patient's immune system" in addition to exposure to some foreign agent (which often cannot even be identified despite its critical role as trigger). *Id.* at 44; R. Lewis, *Chronic Inflammatory Demyelinating Polyneuropathy: Etiology, Clinical Features, and Diagnosis*, UpToDate (2020), https://www.uptodate.com/contents/chronic-inflammatory-demyelinatingpolyneuropathy-etiology-clinical-features-and-diagnosis. Here, Dr. Rumbaugh opined, the precedent trigger was Petitioner's receipt of the flu vaccine, followed by an onset of symptoms eight days post-vaccination, which eventually lead to a chronic autoimmune disease. *Id.*

[9] Dr. Rumbaugh later testified that his expert opinion did not attempt to specifically identify whether the Petitioner had CISP or CIS(M)P, since he deemed the distinction between the two to be merely semantic. Tr. at 40.

Dr. Rambaugh concluded his testimony by briefly nothing that there was no evidence in the medical records to support an alternative diagnosis; that Petitioner's lack of responsiveness to steroid treatment is not an exclusionary criterion for CISP or CIS(M)P; and that the post-vaccination onset period was "a typical time course." Tr. at 50–51. On cross-examination, he discussed several points brought up in his expert reports. In particular, he addressed his heavy reliance on the diagnostic criteria for CISP set forth in one article, arguing how they were met herein. *See* S. Ong & A. Cassidy, *Chronic Immune Sensory Polyradiculopathy*, 22 Practical Neurol. 57 (2022), filed as Ex. CC Tab 3 (ECF No. 67-5) ("Ong & Cassidy"). He also addressed his contention that the medical literature associating GBS with the receipt of the flu vaccine through the mechanism of molecular mimicry could equally be applied to cases involving CIDP/CISP. Tr. at 69.

B. Petitioner's Non-Testifying Expert – Dr. Marcel Kinsbourne

Dr. Kinsbourne, a pediatric neurologist, submitted two written reports. *See generally* Report, dated Feb. 14, 2020, filed as Ex. 16 (ECF No.37-2) ("Kinsbourne First Rep."); Report, dated June 4, 2021, filed as Ex. 17 (ECF No. 54-1) ("Kinsbourne Second Rep."). Although Dr. Kinsbourne did not testify at trial, Petitioner has not formally disclaimed or abandoned his opinions, and I therefore will summarize them below.[10]

Dr. Kinsbourne received his medical degree from Oxford University in England, along with his Bachelor of Arts, and his Master of Arts. Curriculum Vitae, filed as Ex. 16 (ECF No. 37-1) ("Kinsbourne CV") at 1. He then received his M.D. from the State of North Carolina. *Id.* Thereafter, Dr. Kinsbourne did several years of different post-doctoral training in neurology, pediatrics, and chest diseases, and is a member of the American Board of Pediatrics and Royal College of Physicians. *Id.* at 1–2. Dr. Kinsbourne held several academic positions—he was previously professor of psychology, professor of pediatrics, lecturer in neurology, adjunct professor of linguistics and cognitive science, adjunct professor of occupational therapy, director of the behavioral neurology department at the Eunice Kennedy Shriver Center, and other positions related to neurologic and cognitive studies. *Id.* In addition, he also held positions on several editorial boards, professional societies, and administrative assignments. *Id.* at 3–4. Dr. Kinsbourne has conducted research into pediatric disorders, developmental delays and factors, cerebral deficiencies, learning disabilities, therapies, and epilepsy. *Id.* at 5–34. Importantly, however, Dr. Kinsbourne has not treated patients, pediatric or otherwise, for almost thirty years. *See L.M. v. Sec'y of Health & Hum. Servs.*, No. 14-714V, 2019 WL 4072130 (Fed. Cl. Spec. Mstr. July 23, 2019) (discussing Dr. Kinsbourne's more recent practice experience).

[10] I do, however, give Dr. Kinsbourne's opinion somewhat less weight overall, and do no further discuss his reports. His opinion was somewhat duplicative of Dr. Rumbaugh's (who did testify), he lacks the demonstrated immunologic expertise needed to provide an informed opinion on causation, and (as noted in other cases) his lengthy hiatus from medical practice renders him a somewhat ineffective expert overall. *See L.M. v. Sec'y of Health & Hum. Servs.*, No. 14-714V, 2019 WL 4072130 (Fed. Cl. Spec. Mstr. July 23, 2019).

First Report

Dr. Kinsbourne began his report with a brief overview of Petitioner's onset of symptoms followed by an explanation of the similarities and differences between GBS and CIDP. Kinsbourne First Rep. at 1. GBS, Dr. Kinsbourne explained, is a "far more common polyneuropathy, [and] is monophasic, with an acute onset and a self-terminating course." *Id.* By contrast, "progression from onset to maximal deficit" in CIDP takes at least two months, if not longer. *Id.* Despite such differences, however, Dr. Kinsbourne maintained that GBS and CIDP are similar diseases—both are immune-mediated, primarily target the myelin sheaths of peripheral nerves, are comparable in terms of progression, and are responsive to immunomodulating agents. *Id.* Dr. Kinsbourne relied on several items of literature to bolster his opinion that GBS and CIDP differ only in terms of their temporal profiles. *See*, *e.g.*, R. Hughes & D. Cornblath, *Guillain-Barré Syndrome*, 366 Lancet 1653, 1655 (2005), filed as Ex. 16-L (ECF No. 37-10); M. Dalakas, *Advances in the Diagnosis, Pathogenesis and Treatment of CIDP*, 7 Nat'l Rev. Neurology 507, 507 (2011), filed as Ex. 16-H (ECF NO. 37-7) ("Dalakas I") ("CIDP can be practically viewed as the chronic counterpart of Guillain-Barré syndrome (GBS) owing to various electrophysiological, histological and immune similarities").

Next, Dr. Kinsbourne discussed the sensorimotor/sensory nature of both GBS and CIDP, although he acknowledged the rarity of either being predominantly sensory. Kinsbourne First Rep. at 2. Nevertheless, Petitioner's initial GBS diagnosis was characterized by primarily sensory symptoms, and that "motor aspect of classical GBS was little in evidence." *Id.* However, it was likely that large sensory fiber systems were affected, since muscle spasms were reported. *Id.* In fact, Dr. Kinsbourne proposed that Petitioner's motor fibers were *more* affected than the sensory fibers, despite exhibiting little to no weakness upon examination. Kinsbourne First Rep. at 3; S. Oh et al., "*Chronic Sensory Demyelinating Neuropathy": Chronic Inflammatory Demyelinating Polyneuropathy Presenting as a Pure Sensory Neuropathy*, 55 J. Neurology, Neurosurgery, & Psychiatry 677, 678 (1992), filed as Ex. 16-T (ECF No. 37-15) (studying ten patients, all of whom had a slowly progressive monophasic course, and reporting no discernable weakness upon examination, normal muscle strength in all patients, and normal muscle reflexes in four patients); R. Chin et al., *Sensory CIDP Presenting as Cryptogenic Sensory Polyneuropathy*, 9 J. Peripheral Nervous System 1 (2004), filed as Ex. 16-F (ECF NO. 37-5) (reviewing the records of eight patients with CIDP and reporting that all patients exhibited distal numbness and paresthesias and had predominantly large fiber distal sensory loss and normal muscle strength).

Overall, Dr. Kinsbourne proposed that Petitioner's "onset was monophasic as in GBS and the sensory disorder did recur in May 2018, but subsequently the course was that of progressive sensory CIDP, with late developing motor signs in addition." Kinsbourne First Rep. at 4; L. Ruts et al., *Distinguishing Acute-Onset CIDP from Fluctuating Guillain-Barré Syndrome: A Prospective Study*, 74 Neurology 1680, 1685 (2010), filed as Ex. 16-X (ECF No. 37-18) ("Ruts") (comparing cases of GBS with treatment related fluctuations with cases of acute onset CIDIP and

finding that acute onset CIDP should be considered when a patient thought to have GBS deteriorates again after 8 weeks from onset).

To support his assertion that Petitioner experienced an atypical, partial remission between onset and relapse, Dr. Kinsbourne noted that Petitioner suffered significant residual impairments after his initial attack but questioned whether such impairments were due to static damage or continued low-level inflammation. Kinsbourne First Rep. at 4. Relying on several items of literature, Dr. Kinsbourne argued that the relatively lengthy interval between Petitioner's partial recovery and the relapse of his polyneuropathy in May 2018 are largely compatible with an ongoing subclinical active adverse immune response. *Id.*; Dalakas at 509. Moreover, Petitioner's elevated CSF protein level documented at the onset of his relapse in May 2018 was consistent with a low level of continuing inflammation, and had been documented as such every time they were measured according to Dr. Kinsbourne. Kinsbourne First Rep. at 4. Thus, Petitioner's relapse "could initially be considered a 'flare' in an ongoing immune reaction against myelin and/or Schwann cells. This would explain why the sensory nature of [Petitioner's] GBS was recapitulated in the flare that greatly increased the extent of his ongoing numbness and painful paresthesia . . ." *Id.* at 5.

Dr. Kinsbourne then considered the relevance, if any, of negative electrodiagnostic test results when diagnosing CIDP. Kinsbourne Rep. at 5. Dr. Kinsbourne referenced an article which established four distinct sets of diagnostic criteria for CIDP, with sensitivity accuracies ranging between 46 to 83 percent. *Id.*; Y. Rajabally et al., *Validity of Diagnostic Criteria for Chronic Inflammatory Demyelinating Polyneuropathy: a Multicentre European Study*, 80 J. Neurology, Neurosurgery & Psychiatry 1364 (2009), filed as Ex. 16-V (ECF No. 37-16). Despite absent electrodiagnostic findings, CIDP can continue to be active—and was likely the case with Petitioner. *Id.*

Next, Dr. Kinsbourne discussed whether the influenza vaccine can cause CIDP—and what, if any, mechanism of injury is involved. Kinsbourne First Rep. at 6–7. He explained that there is medical literature suggesting that "in both GBS and CIDP[,] circulating anti-ganglioside antibodies attack gangliosides on the surface of peripheral nerve myelin and axons. The immune attack on the gangliosides is attributable to molecular mimicry of surface epitopes of infectious organisms and of vaccines." Kinsbourne First Rep. at 6; K. Kaida et al., *Antiganglioside Antibodies and Their Pathophysiological effects on Guillain-Barré syndrome and Relate Disorders—A Review*, 19 Glycobiology 676 (2009), filed as Ex. 16-M (ECF No. 37-11). However, according to Dr. Kinsbourne, the identity of the provoking organism, whether wild or a vaccine, is not a determining factor in the type of variant of polyneuropathy that results from the immune attack. Kinsbourne First Rep. at 6. Moreover, while "host susceptibility factors differ between people who manifest GBS and people with CIDP, [] similar risk (trigger) factors are involved in causing GBS and CIDP." *Id.* Thus, because it is well accepted within the medical community that the influenza vaccination can be an occasional trigger in GBS, it should logically have the same capacity with respect to CIDP. *Id.*

In so contending, Dr. Kinsbourne acknowledged that there are no controlled epidemiological study of CIDP and vaccination risks, forcing him to rely on several case reports and series. Kinsbourne First Rep. at 6.; P. McCombe et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical and Electrophysiological Study of 92 Cases*, 110 Brain 1617, 1617 (1987), filed as Ex. 16-P (ECF No. 37-13) (finding that 35 percent of 92 patients in sample had an antecedent infection). Dr. Kinsbourne further maintained that the flu vaccine is a well-recognized risk factor in individuals who already have CIDP. Kinsbourne First Rep. at 6; C. Vellozzi et al., *Safety of Trivalent Inactivated Influenza Vaccines in Adults: Background for Pandemic Influenza Vaccine Safety Monitoring,* 27 Vaccine 2114 (2009), filed as Ex. 16-CC (ECF No. 37-21) (identifying three VAERS report which describe the development of CIDP following the administration of the influenza vaccine) ("Vellozzi"). Thus, Dr. Kinsbourne maintained that such events identified in Vellozzi further supports a causal relationship between the influenza vaccine and the development of CIDP. *Id.* at 7.

Dr. Kinsbourne concluded his first report by addressing whether Petitioner's spinal stenosis could stand as an alternative explanation for some of Petitioner's symptoms. Kinsbourne First Rep. at 7–8. Dr. Kinsbourne acknowledged that Petitioner's persistent lower back pain prior to his receipt of the flu vaccine could not be completely overlooked as relevant to the proper diagnosis—but it did not, he opined, adequately explain the neurological findings. *Id.* at 7–8. He further noted that because the nerves in the cauda equina are sensorimotor and do not cause purely sensory losses, one would expect to see evidence of muscle weakness in the legs or disturbances of bowel function, but these symptoms were not observed herein. *Id.* at 8. Moreover, Dr. Kinsbourne explained, "purely sensory manifestations are referable to immune attacks, whereas any motor or sensorimotor problems could reflect neurodegenerative disc disease." *Id.*; A. Duggins et al., *Spinal Root and Plexus Hypertrophy in Chronis Demyelinating Polyneuropathy*, 122 Brain 383, 383 (1999), filed as Ex. 16-J (ECF No. 37-8) (finding that some patients with CIDP have hypertrophied cauda equina fibers, while others have symptoms that mimic spinal stenosis).

Second Report

In his second report, Dr. Kinsbourne addressed the contentions made by Dr. Chaudhry, one of Respondent's experts, that Petitioner's condition could be fully explained by his pre-existing and concurrent spinal stenosis. Kinsbourne Second Rep. at 1.

Dr. Kinsbourne proposed that the symptoms of spinal stenosis and GBS exhibit extensive overlap. Kinsbourne Second Rep. at 1; D. Xu et al., *Severe Lumbar Spinal Stenosis Combined with Guillain-Barré syndrome: A Case Report*, 9 World J. Clinical Cases 1096, 2000–01 (2021), filed as Ex. X (ECF No. 57-2) ("Xu") (relying on a single patient, and proposing that GBS should be considered in the differential diagnosis of individuals with a spinal disorder, despite MRI results consistent with lumbar spinal stenosis). And even if treaters had taken into account the possibility that stenosis explained Petitioner's symptoms, they did not attribute "the totality of Mr. White's post-immunization neurological symptoms" to it. *Id.*

Dr. Kinsbourne further noted the frequent challenge in sorting out symptoms due to autoimmune polyneuropathies from those of compression of the nerves during degenerative lumbar spinal stenosis. Kinsbourne Second Rep. at 5; J. Katz et al., *Lumbar Spinal Stenosis*, 358 New Eng. J. Med. 818, 819–20 (2008), filed as Ex. J (ECF No. 39-10) (suggesting that the most strongly associated symptoms with the diagnosis of lumbar spinal stenosis are age, severe lower extremity pain and little to no pain when seated). Thus, the "low specificity of the diagnosis of [lumbar spinal stenosis]" meant that "for clinical purposes it is easy to over diagnose [it] when it coincides with other diagnoses as it does in the present case and even to diagnose it in a healthy person." Kinsbourne Second Rep. at 9.

C. Respondent's Testifying Expert – Subramaniam Sriram, M.D.

Dr. Sriram testified at hearing and provided one report on behalf of Respondent. *See generally* Report, dated May 19, 2022, filed as Ex. CC (ECF No. 67-2) ("Sriram Rep."). Dr. Sriram opined that Petitioner had not likely experienced GBS or CIDP (including any of its proposed sensory variants), and that the flu vaccine he received on October 12, 2016, was not causal of Petitioner's symptoms (regardless of what they were understood to reflect). *Id*. at 23; Tr. at 91, 94.

Dr. Sriram obtained a Bachelor of Medicine and a Bachelor of Surgery from the University of Madras in Madras, India. Curriculum Vitae, filed as Ex. BB (ECF No. 67-1) ("Sriram CV") at 1. He then served as an intern and resident at Wayne State University and completed a residency in neurology at Stanford University, where he also served as chief resident and eventually completed a post-doctoral fellowship in neuroimmunology. *Id.* Dr. Sriram is board-certified in neurology and internal medicine, and he serves as director of the Vanderbilt Multiple Sclerosis Clinic. *Id*.; Tr. at 90. He also holds academic positions as a professor of experimental neurology and therapeutics as well as an associate professor in molecular biology and immunology. Sriram CV at 2. Dr. Sriram's clinical practice in the ambulatory outpatient services includes seeing patients two days a week, and in addition, he runs a basic science laboratory looking at pathways that promote neuro repair. Tr. at 90. In addition, Dr. Sriram has published numerous articles on various aspects of clinical and immune mediated diseases of the nervous system. Sriram CV at 9–21; Sriram Rep. at 1.

Dr. Sriram first discussed at length Petitioner's pre- and post-vaccination medical history. Sriram Rep. at 2–12. He noted that Petitioner had a long history of low back pain and arthritis of the back and hips, and that this pain had been present for at least a year prior to receiving the flu vaccine. *Id.* at 13. Indeed, Petitioner's medical records indicated that on the very day he received the flu vaccine, Petitioner complained of bilateral pain in the gluteus, most notable upon waking, and that it felt as though someone was hanging on him. *Id.* at 3, 13. Thus, the record suggested Petitioner's neuropathic symptoms may have predated vaccination.

Dr. Sriram then discussed whether Petitioner's symptoms could be vaccine-related. He first addressed why Petitioner's presentation did not meet the criteria for GBS, despite the initial

diagnosis upon Petitioner's hospitalization on October 26, 2016. Sriram Rep. at 13; Tr. at 93–98. To support his assertion, Dr. Sriram relied on diagnostic criteria broadly embraced by neurologists, noting that Petitioner exhibited no bilateral flaccid weakness, and had normal reflexes in the arms with reduced reflexes in the leg. Sriram Rep. at 14. In addition, Petitioner did not experience a monophasic course (and indeed alleges later related symptoms occurring more than a year after). Sriram Rep. at 14.

A CIDP diagnosis was not, in Dr. Sriram's view, supported by the record either at this time. Tr. at 94; Sriram Rep. at 15; J. Vallat et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Diagnostic and Therapeutic Challenges for a Treatable Condition*, 9 Lancet Neurology 402, 403 (2010) ("Vallat") (discussing the clinical features and patterns of CIDP). In so opining, Dr. Sriram indicated that he did not accept the view that CIDP is effectively a chronic version of GBS. Tr. at 93, 104. Rather, CIDP is an acquired disorder of peripheral nerves and nerve roots, with two possible sub-variants of its own—CISP and CIS(M)P. Sriram Rep. at 15; Tr. at 112. Unlike GBS, CIDP is slowly progressive rather than acute, and can feature a lengthy period of waxing and waning events (although as discussed below, Dr. Sriram did feel it would have an initially-predictable course before it became more chronic and persistent). Tr. at 93, 113.

There are two criteria especially important for a CISP diagnosis, Dr. Sriram maintained: (i) evidence of sensory symptoms with a polyneuropathic distribution but without weakness, and (ii) normal results from motor and sensory nerve conduction and EMG studies. Tr. at 95; Ong & Cassidy at 58. But at the time of his initial presentation on October 26, 2016, Petitioner showed no signs of polyneuropathic distribution without weakness. Tr. at 96 ("he had some numbness objectivity and subjectivity in his right foot, and that's about it"); Sriram Rep. at 18. Large nerve fiber involvement is also feature of CISP, and causes the loss of vibration and position sense in one's limbs—yet Petitioner had at most *decreased* pinprick in his mid-thigh of the right leg, as well as around his left side hip, but sensation to vibration, proprioception, and temperature were intact. Sriram Rep. at 18; Tr. at 96; Ex. 9 at 8. None of these exam responses or test results thus, in Dr. Sriram's view, supported the CIDP variant sub-diagnosis (at least in October 2016)—let alone any other form of polyneuropathy.[11] Tr. at 99–100 (deeming the form of ascending numbness Petitioner had described to be "very atypical for a polyneuropathy).

Additional diagnostic criteria relevant to a CIDP diagnosis, Dr. Sriram opined, as set forth in Ong & Cassidy were also not met by the clinical record evidence. For example, of three "secondary" criteria identified in Ong & Cassidy, the sole factor that Dr. Sriram agreed was established was evidence of an elevated protein level in CSF, with otherwise normal white blood cell count. Ong & Cassidy at 58; Tr. at 98. But even that one secondary criterion was not, in Dr. Sriram's view, fully corroborated by the record. Tr. at 98—99. Although CSF testing was

[11] EMG testing results, however, did partially support the diagnosis—*although such testing was not performed at the time of Petitioner's first claimed symptoms*. Only when such testing did occur (21 months later—in July 2018) was it, in Dr. Sriram's view, supportive. Tr. at 97.

performed both in October 2016 and then again in May 2018, evidence of elevated protein levels could have many different causes separate from demyelination (and could in fact be caused by stenosis as well). *Id.* at 99. Dr. Sriram ultimately opined it more likely that spinal stenosis explained Petitioner's elevated protein levels. *Id.* at 114.

Otherwise, Dr. Sriram disputed the contention that Petitioner's overall initial post-vaccination presentation was consistent with a chronic polyneuropathy like CIDP. In his understanding, CIDP's onset symptoms should progress from onset to nadir (maximum deficits) in eight weeks—not acutely, as the record showed had occurred here. Tr. at 108 (Petitioner's worst symptoms occurred within one week of onset); E. Ubogu, *Inflammatory Neuropathies: Pathology, Molecular Markers and Targets for Specific Therapeutic Intervention,* 130 Acta Neuropathol. 445, 455 (2015), filed as Ex. CC Tab 8 (ECF No. 67-10) ("Ubogu") (noting that CIDP's post onset "maximum severity" typically occurs within eight weeks, but that it is otherwise chronic (relapsing-remitting or progressive)).

Petitioner's presentation in May 2018 was similarly unsupportive of a CIDP diagnosis, in Dr. Sriram's estimation. He deemed it "very very atypical" for a chronic neuropathy like CIDP to present with "numbness with—below the waist or below the torso, and with the sensory distribution" Petitioner had described in his groin area. Tr. at 100–01. There was also the fact of the more than one-year gap in reported symptoms, measured from Petitioner's acute presentation in October 2016 to his alleged recurrence in May 2018. In Dr. Sriram's view, CIDP would more likely be evidenced by "some protracted worsening of his neurological deficit" in that time period, but this had not occurred. Tr. at 101. Dr. Sriram instead opined that the October 2016 and May 2018 incidents were "clearly two separate events," deeming the second far more severe than the first, but with no logical or record-demonstrated connection. *Id.* at 110.

In addition, although Petitioner had maintained that IVIG treatments led to symptom improvement (thus providing some indirect proof that Petitioner had experienced an immune-mediated injury like GBS or CIDP), response to IVIG did not, in Dr. Sriram's view, corroborate the contention that Petitioner suffered from CIDP. Sriram Rep. at 15. IVIG's efficacy could be subjective, and thus "fool" a patient into believing it was having an impact on illness course. Tr. at 101–02, 120–121; J. Allen & R. Lewis, *CIDP Diagnostic Pitfalls and Perception of Treatment Benefit*, 85 Neurology 498, 502 (2015), filed as Ex. H (ECF No. 39-8) ("Allen & Lewis") ("[e]ighty-five percent of patients without CIDP felt better with immunotherapy when benefit was broadly and subjectively defined. Even when restricted to definite subjective improvement, 66% of patients without CIDP felt better"). And Petitioner had not received IVIG treatment for much of 2017 or 2018—inconsistent with the existence of a chronic and persistent underlying condition, which would have been expected to worsen and/or reveal symptoms in that period. Tr. at 101.[12]

[12] By contrast, steroids are deemed efficacious treatment for CIDP—and yet, as Petitioner himself admitted, they had not assisted him, thus further reducing the possibility that he had experienced CIDP. *Id.* at 14–16, 105, 123

Dr. Sriram admitted on cross-examination, however, that IVIG would not be indicated to treat a condition like spinal stenosis. *Id.* at 115.

Dr. Sriram also provided his reaction to the Petitioner's theory for how the flu vaccine could cause a CIDP variant. The idea that molecular mimicry had produced CIDP in this case not only failed to account for the absence of proof Petitioner *had* CIDP in the first place, but also was insufficiently supported with medical or scientific study proof. Tr. at 103. Nothing existed of which Dr. Sriram was aware that would explain the manner in which a vaccine-induced autoimmune reaction would lead to CIDP. *Id.* at 104. He further took issue with the assumption that what had been established with respect to a flu vaccine-GBS relationship could be applied to CIDP equally, adding that more was known about even an underlying flu wild virus connection with GBS than CIDP. *Id.* at 106–07, 109.

As the above summary indicates, Dr. Sriram did at times seem to suggest that some form of spinal stenosis better explained Petitioner's likely injury. On cross-examination, however, he admitted that he could not in fact provide a diagnostic explanation for Petitioner's injury. Tr. at 128. But he resolutely denied that it could be deemed to have been CIDP or a rare sub-variant, emphasizing the extent to which the diagnostic criteria were not met consistently or completely. *Id.* at 128–30.

D. Respondent's Non-Testifying Expert – Dr. Vinay Chaudhry

Dr. Chaudhry, a neurologist, submitted two reports, although he was not called to testify at hearing.[13] *See generally* Report, dated Apr. 13, 2020, filed as Ex. A (ECF No. 39-1) ("Chaudhry First Rep."); Report, dated, Aug. 30, 2021, filed as Ex. W (ECF No. 57-1) ("Chaudhry Second Rep."). Dr. Chaudhry opined that Petitioner neither suffered from GBS, CIDP or any immune neuropathy, nor that the flu vaccine was causal of his symptoms. Chaudhry Second Rep. at 16.

Dr. Chaudhry obtained his Bachelor of Medicine and Bachelor of Surgery degrees from the All India Institute of Medical Sciences in New Delhi, India. Curriculum Vitae, filed as Ex. DD (ECF No. 71-1) ("Chaudhry CV") at 1. He later completed residency training in neurology at the University of Tennessee Center for the Health Sciences and the University of Alabama at Birmingham School of Medicine. *Id.* at 2. He then completed a fellowship in neuromuscular disease at Johns Hopkins University School of Medicine. *Id.* Following the completion of his fellowship, Dr. Chaudhry became an instructor at Johns Hopkins University school of Medicine, where he later became a Professor of Neurology. *Id.* at 2–3. Currently, Dr. Chaudhry is a Distinguished Professor at the University of North Carolina Chapel Hill School of Medicine. *Id.* at 2. In addition to his several academic appointments, Dr. Chaudhry is involved in clinical

[13] Consistent with my treatment of Dr. Kinsbourne, I also give Dr. Chaudhry's written reports somewhat less emphasis in determining the outcome herein than Dr. Sriram's in-person testimony (especially to the extent it duplicates what was presented at hearing)—although some of Dr. Chaudhry's diagnostic views warranted more weight than Dr. Kinsbourne's, given his comparatively greater expertise with respect to the study and treatment of peripheral neuropathies.

research and has published over 120 publications, which include peer-reviewed articles, reviews, and book chapters. *Id.* 4–23; Chaudhry First Rep. at 1. Dr. Chaudhry has an active clinical practice and estimates that he evaluates over 2,000 patients a year with some form of peripheral neuropathy. Chaudhry First Rep. at 1.

First Report

Dr. Chaudhry began his first report by providing a brief overview of Petitioner's medical history. Chaudhry First Rep. at 1–14. He then evaluated the diagnoses that Petitioner's treating physicians considered over the course of his treatment, focusing on GBS and CIDP. *Id.* at 16. GBS, Dr. Chaudhry noted, is known as "an acquired disease of the peripheral nerves that is characterized by rapidly progressing (with peak of <4 weeks) ascending paralysis with paresthesias." Chaudhry First Rep. at 16; Vallat at 404. It is often accompanied by areflexia, involvement of the cranial nerves, diaphragm, and autonomic nervous system, generally exhibited by a demyelinating electrophysiology on nerve conduction studies, and monophasic in nature *Id.*

Based on such factors, Dr. Chaudhry opined that Petitioner's immediate post-vaccination presentation was inconsistent with GBS. Petitioner had actually exhibited sensory and motor symptoms *prior* to receiving the flu vaccine, presented with acute exacerbation of his back pain and sensory loss up to his abdomen, showed no signs of weakness, had no respiratory, cranial, or autonomic nervous system involvement, and did not have a monophasic course. Chaudhry First Rep. at 16. In addition, the persistence of his symptoms, the saddle anesthesia, the ongoing T11 sensory level problems, the relapses, and the negative EMG also did not match GBS. *Id.* Dr. Chaudhry further deemed it significant that Petitioner's first diagnoses of GBS occurred after a reported URI, with several of his treating physicians identifying it as a possible explanation for his illness. *Id.*

Dr. Chaudhry next contested the propriety of a CIDP diagnosis. Chaudhry First Rep. at 17. As he explained, CIDP often presents with "chronically progressive, stepwise, or recurrent symmetric proximal and distal weakness and sensory dysfunction of all extremities, developing over at least 2 months; and absent or reduced tendon reflexes in all extremities." *Id.*; Vallat at 403; P. Van den Bergh et al., *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*, 17 Eur. J. Neurol. 356 (2010), filed as Ex. F (ECF No. 39-6). But as evidenced by Petitioner's clinical findings at various points throughout the course of his treatment, physicians never documented Petitioner as having subjective or objective weakness, with his strength consistently noted at 5/5 in all extremities. *Id.* at 17–18; *See also* Ex. 9 at 6; Ex. 14 at 155, 200–18; Ex. 10 at 67. Petitioner also did not have areflexia, and his EMG studies were not indicative of a primarily demyelinating process. Chaudhry First rep. at 18.

Despite this, Dr. Chaudhry acknowledged, Petitioner was given a diagnosis of immune neuropathy because of an elevation of CSF. But Dr. Chaudhry argued that while a CSF profile (along with nerve biopsy or imaging findings) can *support* a diagnosis of CIDP, no single such abnormality can be diagnostic of the illness by itself. A. Breiner et al., *Updated Cerebrospinal Fluid Total Protein Reference Values Improve Chronic Inflammatory Demyelinating Polyneuropathy Diagnosis*, 60 Muscle Nerve 180, 182 (2019), filed as Ex. G (ECF No. 39-7) ("Breiner"); J. Allen et al., *Challenges in the Diagnosis of Chronic Inflammatory Demyelinating Polyneuropathy*, 8 Brain Behavior (2018), filed as Ex. I (ECF No. 39-9) ("Allen") (reporting that an elevation of CSF protein over 60 mg/dl might be due to a process other than CIDP). Indeed, Dr. Chaudhry maintained that severe cervical and lumbar spondylosis was a misdiagnosis as well, given the exam and test results. First Chaudhry Rep. at 19.

Dr. Chaudhry also proposed a more likely alternative diagnosis in his reading of the medical record: spinal stenosis. First Chaudhry Rep. at 19. The most common symptom associated with lumbar spinal stenosis, he noted, is neurogenic claudication[14]—something that all of Petitioner's treating physicians agreed Petitioner displayed. *Id.* at 20. Petitioner had experienced back pain with periodic exacerbations (most notable when walking or standing), he suffered from saddle anesthesia, and his imaging showed severe lumbar stenosis, thoracic cord compromise and lumbosacral radiculopathy. *Id.*; *See also* D. Binder et al., *Lumbar Spinal Stenosis*, 22 Seminars Neurology 157, 159–60 (2002), filed as Ex. K (ECF No. 39-11) ("Binder") (emphasizing that postural aggravation of radiculopathy or spinal claudication is a common characteristic of lumbar stenosis, and involves the immediate onset of back and leg pain when standing in an erect but is immediately relieved by sitting); P. Storm et al., *Lumbar spinal Stenosis, Cauda Equine Syndrome, and Multiple Lumbosacral Radiculopathies*, 13 Physical Med. Rehabilitation Clinics N. Am. 713, 719 (2002), filed as Ex. L (ECF No. 40-1) (walking or standing exacerbates an individual's symptoms, but sitting can substantially alleviate the pain).

Relying on his alternative diagnostic opinion, Dr. Chaudhry maintained that the flu vaccine could not have caused Petitioner's symptoms. First Chaudhry Rep. at 22. On the day of vaccination, Petitioner noted pain in his gluteus muscles, numbness in his toes, and weakness in the right knee. But the subsequent recurrence of symptoms in May and August 2018 seemed to have a *positional* quality, was associated with saddle anesthesia, and was not corroborated by the kind of EMG findings needed to confirm the presence of a neuropathy. He also observed that "in contrast to GBS where molecular mimicry with bacterial or viral antigens triggers the disease in some patient subsets, there is no convincing evidence that viral infections are antecedent events in

[14] "Neurogenic Claudication" is defined as "claudication accompanied by pain and paresthesias in the back, buttocks, and lower limbs, relieved by stooping or sitting; it is usually caused by lumbar spinal stenosis that may be a mechanical disturbance due to posture, and less often by ischemia or the cauda equina." *Neurogenic Claudication*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=65508&searchterm=neurogenic+claudication (last visited Aug. 4, 2023).

CIDP." M. Dalakas, *Pathogenesis of Immune-Mediated Neuropathies*, 1852 Biochimica et Biophysica Acta 658, 662 (2015), filed as Ex. Q (ECF No. 40-6) ("Dalakas II"); E. Mathey et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Pathology to Phenotype*, J. Neurology, Neurosurgery & Psychiatry 1, 3 (2015), filed as Ex. R (ECF No. 40-7) ("Mathey"). This, plus other explanations for Petitioner's symptoms, made it highly unlikely the flu vaccine could have harmed Petitioner as alleged.

Second Report

In his second report, Dr. Chaudhry responded to the contentions made by Dr. Kinsbourne regarding Petitioner's overall diagnoses, reiterating his prior conclusion that Petitioner neither had GBS nor CIDP. Chaudhry Second Rep. at 16.

Dr. Kinsbourne had asserted that the symptoms of spinal stenosis and those of GBS mimic one another and essentially overlap, but Dr. Chaudhry disagreed. Chaudhry Second Rep. at 8. Unlike the patient discussed in Xu, Petitioner never exhibited *ascending* weakness, he only experienced variable improvement from IVIG, both his EMG study results were unremarkable for abnormalities of demyelination or of sensory nerve involvement, and his clinical course did not progress in a monophasic manner. *Id.* at 9. Dr. Chaudhry further reiterated the observation that Petitioner not only failed to meet any of the criteria for GBS, but that several of Petitioner's treating physicians had deemed Petitioner's course clinically inconsistent with a diagnosis of GBS or any other typical autoimmune/inflammatory peripheral nerve disease. *Id.* at 10; *See also* Ex. 14 at 144, 157, 299, 371 402.

Dr. Chaudhry agreed with Dr. Kinsbourne that Petitioner exhibited clear evidence of lumbosacral spinal stenosis *prior* to his vaccination and the subsequent years. Chaudhry Second Rep. at 11. He noted that Petitioner had intermittent neurologic deficits, MRI findings consistent with severe spinal stenosis as defined by the medical literature, EMG studies showing bilateral multi-radicular abnormalities, and that all of his treating physicians agreed on the diagnosis of lumbar stenosis with neurogenic claudication. *Id.* at 12; Binder at 160. Dr. Chaudhry deemed Petitioner's response to the IVIG treatment to have a placebo quality, rejecting Dr. Kinsbourne's view that this confirmed the presence of an autoimmune condition. Chaudhry Second Rep. at 13–14; Allen & Lewis at 502; T. Levine et al., *Review Process for IVIG Treatment* 8 Neurology: Clinical Practice 429, 431 (2018), filed as Ex. AA (ECF No. 57-5) ("Levine") (suggesting that IVIG is overused, and finding that approximately "only 32.3% of 248 patients had an immune neuropathy and were appropriate candidates for IVIg therapy, whereas 46.4% had neuropathies that were not immune mediated").

Dr. Chaudhry concluded his final report by maintaining that Petitioner had exhibited a clear history of lower back pain and numbness prior to his receipt of the flu vaccine, that his MRI results supported a diagnosis of lumbar spinal stenosis, and that his EMG studies were consistent with

what is seen in spine disease. Chaudhry Rep. at 15. All of his symptoms could thus be explained by his lumbar spinal stenosis. *Id.*

III. Procedural History

As noted above, this claim was initiated in 2018, and the matter was originally designated as a "Special Processing Unit" (the "SPU") case, based on the initial supposition that the claim was likely to settle. ECF Nos. 1, 4. All medical records with the Statement of Completion were filed by May 3, 2018, and Petitioner's affidavit was subsequently filed on May 9, 2018. ECF Nos. 8–9. Additional medical records were filed thereafter with one amended Statement of Completion filed on November 5, 2018, and another on February 8, 2019. ECF Nos. 19, 27. Respondent filed a Rule 4(c) Report contesting Petitioner's right to compensation on March 21, 2019 (ECF No. 29), and thereafter the case was transferred out of SPU. Expert reports were subsequently filed over a two-year period, and after the matter was assigned to my docket from another special master, a one-day entitlement hearing was held on September 16, 2022. ECF No. 65. After the filing of post-hearing briefs in December 2022, the matter was ripe for resolution.

IV. Applicable Law

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also 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, 1320 (Fed. Cir. 2006).[15] In this case, Petitioner was unable to advance a Table claim because of his CIDP diagnosis (as I observed in dismissing that version of the claim). *See* ECF No. 42.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard).

[15] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(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 proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). 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." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245 ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a

'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." (citing *Moberly*, 592 F.3d at 1322)); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *appeal docketed,* No. 23-1816 (Fed. Cir. Apr. 28, 2023). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs*., 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as 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'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs*., 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the

phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd sub nom. Rickett v. Sec'y of Health & Hum. Servs.*, 468 F. Appx. 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993

F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie*, 2005 WL 6117475, at *20. Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy*, 23 Cl. Ct. at 733 (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

There are, however, situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("'[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent'") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *Lalonde v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these 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) ("uniquely 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"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, 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*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters

must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

Expert opinions based on unsupported facts may be given relatively little weight. *See Dobrydnev v. Sec'y of Health & Hum. Servs.*, 556 F. Appx. 976, 992–93 (Fed. Cir. 2014) ("[a] doctor's conclusion is only as good as the facts upon which it is based") (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993) ("[w]hen an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert's opinion")). Expert opinions that fail to address or are at odds with contemporaneous medical records may therefore be less persuasive than those which correspond to such records. *See Gerami v. Sec'y of Health & Hum. Servs.*, No. 12-442V, 2013 WL 5998109, at *4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013), *aff'd*, 127 Fed. Cl. 299 (2014).

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("[w]e 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. Appx. 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. An Overview of Relevant Medical Terms and Prior Decisions

A. *CIDP as a Demyelinating Disease*

As noted above, CIDP has been defined as a progressive, immune-mediated peripheral neuropathy that occurs due to an autoimmune attack. Dalakas I at 557; Mathey at 1. It results in numbness, paresthesia, and sensory ataxia that presents as relapsing-remitting, stepwise progressive, or gradually progressive, and more often than not involves motor and sensory nerve dysfunction. Ubogu at 455; Mathey at 1. These symptoms tend to be symmetrical and involve lower and upper limbs, although variants can involve different phenotypic presentations. Dalakas

I at 508; Mathey at 1 (clinical presentations of variants depend on differing "immunogenetic variations").

There is no doubt that GBS and CIDP overlap, and that the latter has often been viewed as the "chronic counterpart" of the former as a result. Dalakas II at 659. However, reliable science supports the conclusion that "those conceptions may be oversimplistic." Ubogu at 455. In particular, GBS has an acute onset, is monophasic, and is not steroid-responsive in comparison to CIDP. Vallat at 402. CIDP can *present* acutely, but has a relapsing and meandering course—and it can be difficult to diagnose in part due to its somewhat insidious and smoldering character. *Id.* In addition, far less is known about the autoantibodies that might drive CIDP, what their human nerve tissue targets would be, and even whether those targets are shared with GBS. Mathey at 6; Ubogu at 457. Indeed, some CIDP-focused research seems to have identified nerve "nodal regions" as likely targets for attack—and (importantly) that damage to the node of Ranvier or paranode might explain some of CIDP's presentation—whereas GBS is more generally thought to involve attacks on nerve surface myelin. Mathey at 6–7, 9 ("disruption of nodal function is likely to interfere with normal nerve excitability and membrane potentials, contributing to conduction failure"); Dalakas at 659 (GBS represents several syndromes based on the degree of involvement of the motor or sensory nerve fibers and the myelin sheath or axon).

Because of the above, for purposes of deciding entitlement in Program cases it is simplistic to characterize CIDP as merely "long GBS." *Houston v. Sec'y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *17 (Fed. Cl. Spec. Mstr. Aug. 19, 2021) (noting CIDP versus GBS distinctions). More contemporaneous evidence persuasively rejects such an easy equivalence. The two diseases are distinguishable not only in their course and treatment, but also in the inciting events that cause them—even if both are *mediated* by autoimmune processes.

In underscoring the distinction between GBS and CIDP, however, I do not maintain that medical science regarding GBS (or demyelinating neuropathies more generally) has *no* application whatsoever in this case. But the overlap between GBS and CIDP cannot be employed as a shortcut to entitlement, simply because certain principles that have been *preponderantly* shown bearing on the flu vaccine-GBS connection (like the mechanism of molecular mimicry) could *plausibly* be extended to this context. Instead, it is reasonable to ask for evidence *specific* to CIDP itself—as the Program requires—before determining that it can be vaccine-caused. Evidence that strongly supports a GBS-flu vaccine causal relationship rings weaker when applied to CIDP. And this is especially so where, as here, the claimant seeks to prove the existence of a particular sub-variant, with characteristics not completely congruent with the usual features of CIDP.

B. *Prior Program Decisions on Flu Vaccine-CIDP Association*

Admittedly, there are many prior cases in which Petitioners alleging the flu vaccine caused CIDP have obtained favorable results.[16] *See, e.g.*, *Jastisan v. Sec'y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950, at *1–3 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). I have myself acknowledged their existence in my own prior decisions, and the fact that such determinations should be given consideration as persuasive guidance (although *settled* cases certainly lack precedential value, in comparison to reasoned decisions). *Nieves v. Sec'y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148 (Fed. Cl. Spec. Mstr. May 22, 2023), *mot. for review docketed on other grounds,* May 15, 2023 (Fed. Cl.); *Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (finding that the flu vaccine can cause CIDP); *Daily v. Sec'y of Health & Hum. Servs.*, No. 07-173V, 2011 WL 2174535, at *8 (Fed. Cl. Spec. Mstr. May 11, 2011).

However, there are few persuasive *reasoned* decisions in which a special master *explained* with any specificity why a causal theory associating the flu vaccine with CIDP was persuasive. Rather, special masters have tended to lump CIDP and GBS together as virtually-interchangeable peripheral neuropathies—leading them to assume that the extensive science supporting causation for GBS after vaccination applies to CIDP, but without close consideration of the actual persuasiveness of a claimant's prong one showing, based on expert opinions or relevant literature *specific* to the injury. *See, e.g.*, *Tomsky v. Sec'y of Health & Hum. Servs.*, No. 17-1132V, 2020 WL 5587365, at *15 (Fed. Cl. Spec. Mstr. Aug. 24, 2020) ("for purposes of this decision I merely assume but do not decide that petitioner has established a medical theory causally linking the flu vaccine to CIDP"); *Strong*, 2018 WL 1125666, at *22.

As I have recently noted, this presumption is worthy of more careful analysis, if not full reconsideration. *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V. 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (finding a flu vaccine-CIDP causal relationship was established—but noting that "the fact that reliable science establishes an association between GBS and the flu vaccine does not inerrantly lead to the conclusion that CIDP can also be deemed to be similarly-associated").[17] I have, however, been reluctant in prior cases to disregard the fact that CIDP has often been assumed to be flu-vaccine associated. *Nieves*, 2023 WL 3580148, at *36.

[16] Even though prior decisions from different cases do not control the outcome herein, special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39 (2007) ("[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim") (emphasis added). They would therefore be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions.

[17] Some decisions from the past ten years have at least started that reconsideration process. In a 2014 case, for example, a petitioner was unsuccessful in claiming her ongoing neurological condition was aggravated by two influenza vaccinations. *Jacunksi v. Sec'y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422, at *14 (Fed. Cl. Spec.

II. Petitioner Has Not Carried His Burden of Proof

A. The Record Does not Preponderantly Support the CIS(M)P Proposed Diagnosis

Despite Petitioner's initial presentation, GBS is not a proper diagnosis for his illness—meaning the case could not have succeeded as a Table claim. Not only has Petitioner *acknowledged* in filings that this is not the injury he alleges, but the medical history alone demonstrates that he did not experience a single, monophasic event in the fall of 2016,[18] but went on to experience a second event more than a year later that has some factual similarity to the first, and which he alleges is connected. Such a history would not be consistent with GBS as it is classically understood.

Petitioner instead seeks to advance a causation-in-fact claim that he suffered from a CIDP variant: CIS(M)P. It is often proper in Program cases to evaluate whether a claimant has established the alleged injury—especially where, as here, the causation theory relies on establishing that injury. *Broekelschen*, 618 F.3d at 1346; *see Lombardi*, 656 F.3d 1343, 1352-1353 (Fed. Cir. 2011) ("the statute places the burden on the petitioner to make a showing of at least one defined and recognized injury"). Therefore—and also because Petitioner hopes to rely on existing decisions and science associating the flu vaccine with CIDP—it makes sense to evaluate whether and to what extent Petitioner proved the existence of the alleged CIDP variant injury.

Here, the evidence preponderates against the finding that Petitioner experienced any form of CIDP, including a sensory subvariant. Only one treater, Dr. Bradshaw (whom Petitioner saw, it bears noting, in the summer of 2021—more than three years after the claim's filing, and five years after vaccination) has so opined, at least formally. But although a treater view deserves some weight, it is not sacrosanct (especially given the circumstances in which it was obtained). Otherwise, while CIDP was often included in the differential diagnosis (as reflected by Dr. Van Cott's workup of Petitioner in 2018, it was not fully embraced until Dr. Bradshaw entered the picture.

Mstr. Sept. 23, 2014) (flu vaccine did not significantly aggravate CIDP; noting distinction between strength of evidence supporting flu vaccine-GBS association and flu vaccine-CIDP connection).

In addition, special masters (including me) have been more definitive in rejecting theories that vaccines *other* than the flu vaccine cause CIDP. *See, e.g.*, *Howard v. Sec'y of Health & Hum. Servs.*, No. 16-1592V, 2022 WL 4869354 (Fed. Cl. Spec. Mstr. Aug. 31, 2022) (Tdap vaccine not causal of CIDP), *mot for review den'd*, slip op. (Fed. Cl. Feb. 27, 2023); *Sanchez v. Sec'y of Health & Hum. Servs.,* No. 18-1012V, 2022 WL 1013264 (Fed. Cl. Spec. Mstr. Mar. 11, 2022) (Tdap vaccine not causal of CIDP).

[18] Petitioner in fact actively seeks to connect the two events. I note for the record, however, that even if his claim only focused on the October 2016 event, ignoring wholly the second event, the petition would still be properly dismissed. Petitioner cannot show that the first event was GBS, which would be expected to present acutely. And Respondent has effectively rebutted the conclusion that this event reflects some kind of acute-onset CIDP, given the fact that the record does not at this time support the diagnosis.

By contrast, both of Respondent's experts, after a careful review of Petitioner's overall history, disputed the diagnosis—and they did so by reference to the medical record and testing evidence, considered against the backdrop of the diagnostic criteria set in Ong & Cassidy. Ong & Cassidy at 58. Of these, one of two primary criteria (sensory symptoms with a polyneuropathic distribution without weakness) was *never* met (in October 2016 or May 2018), and Petitioner could only point to a secondary symptom (elevated CSF protein levels), which even Dr. Rumbaugh agreed was "kind of a nonspecific finding." Tr. at 68, 88. Respondent's experts also cast doubt on the weight that should be given to Petitioner's reported positive response to IVIG (although this factor sways me less than the record evidence pertaining to diagnostic criteria). Tr. at 101–02, 120–121; Allen & Lewis; Levine at 431.

In addition, the significant gap from when Petitioner ceased treatment in late spring 2017, and when he again complained of severe symptoms in May 2018, cannot be explained fully by Petitioner's financial circumstances or lack of medical insurance coverage. The record of the May 2018 incident reveals a sudden and acute emergence of seemingly-neurologic symptoms, and belies contentions of ongoing symptoms in that treatment hiatus period. It is unlikely, based on what is known about CIDP generally, that the condition would go into remission for such a period of time.

Because Petitioner did not preponderantly demonstrate he has suffered from CIS(M)P, I find that he has not alleged a vaccine injury that could be a basis for a claim consistent with the theory presented.[19] This alone is grounds for dismissal, given the extent to which Petitioner's causation theory borrows what is known about the flu vaccine-GBS relationship (and in turn its applicability to CIDP).

B. Petitioner Has Not Demonstrated the Flu Vaccine Can Cause CIS(M)P

Even had I found that CIS(M)P was preponderantly supported as the likely diagnostic explanation for Petitioner's symptoms, I would not also be able to find that the flu vaccine can cause CIS(M)P. Reaching this conclusion would not give me the pause that I have expressed in the context of other cases, where CIDP is alleged to be the injury without identification of a more specific variant.

This aspect of Petitioner's evidentiary showing was notably thin, and not just in

[19] I cannot on the basis of the present record determine whether a *more likely* explanation for Petitioner's symptoms has been established. Indeed, as noted above, Dr. Sriram could not either—and although Dr. Chaudhry opined that Petitioner suffered from spinal stenosis, I give that view somewhat less weight than what Respondent's testifying expert, Dr. Sriram, stated. But it is not required in Program cases, under *Broekelschen* or any other authority, that *some* name must be given to a claimant's injury in all cases. Rather, petitioners bear the ultimate burden of proving an injury—and they can fail that burden even when no other counter-explanation can be preponderantly identified.

comparison to the evidence and testimony offered on diagnosis. Dr. Rumbaugh was the sole testifying expert for Petitioner—and although his neurologic focus rendered him generally qualified to offer an opinion in this case on possible vaccine-related causes for an immune-mediated neurologic disease, he lacked specific knowledge of the topic gleaned from his own study or practice that would imbue his opinion with the same persuasiveness and reliability that might attach to the views of a professional immunologist or medical professional with a demonstrated focus on peripheral neuropathies like CIDP and its variants. Otherwise, the actual evidence relied upon was mostly the fact that in numerous prior Program cases (a) it has been established that the flu vaccine is associated with GBS, and (b) that association in turn has been invoked when CIDP is alleged, based on received assumptions about the CIDP-GBS relationship.

In other cases, when presented with this issue I have tended to err on the side of finding a bare preponderant showing for the first *Althen* prong is met, since I do not wish to undo so many years of Program case law without a robust and up-to-date showing from Respondent undercutting the idea that the same thinking about a demyelinating peripheral neuropathy applies to a variant form of the injury. *See, e.g.*, *Nieves*, 2023 WL 3580148, at *36. But here, Petitioner is proposing a very specific, *sub-variant* of CIDP, more sensory in nature, further attenuating the assumed GBS-CIDP connections that often carry the day. In such a case, more evidence specific to the form of disease is reasonably called for. *Haubner v. Sec'y of Health & Hum. Servs.*, No. 16-1426V, 2021 WL 5614942, at *32 (Fed. Cl. Spec. Mstr. Oct. 22, 2021).

I thus will not in this circumstance do as I have done in other contexts, and deem the "can cause" prong to have been met, based in part of the fact that in many decisions the difference between GBS and CIDP has been elided without much analysis. And in fact, in this case Petitioner has not demonstrated a good reason to find that the flu vaccine can likely cause CIS(M)P. Dr. Rumbaugh (even coupled with the more conclusory, non-testifying opinion provided by Dr. Kinsbourne) has simply not offered enough evidence for me to conclude that the flu vaccine could spark an autoimmune process leading to an acute presentation, evolving (after a lengthy quiescent period exceeding one year). Little in the way of persuasive and reliable literature has been offered explaining how the flu vaccine would start such a process, or even analogizing it to a comparable wild infection (and as Respondent observed there is not even an understood viral or bacterial infection analog with CIDP, let alone CIS(M)P). Far more literature filed by Petitioner (if not nearly all of it) involves the issue of diagnosis than causation in the narrow context of an uncommon CIDP variant. Thus, the "can cause" prong is also unmet.

C. The Evidence Does not Preponderantly Support the Conclusion that the October 2016 Flu Vaccine Did Cause Petitioner's Alleged CIS(M)P[20]

Even more significant to my disposition of this case than my *Althen* prong one finding is my determination that Petitioner's October 2016 receipt of the flu vaccine did not likely cause his CIS(M)P (assuming that diagnosis had been preponderantly substantiated). Even if the flu vaccine-CIDP association accepted (without much analysis) in other cases were simply applied here, such that *Althen* prong one was deemed satisfied, Petitioner's medical history does not support the conclusion that *his* injury was vaccine-caused.

The medical record herein contains at best equivocal treater evidence *contemporaneous* with the October 2016 vaccination suggesting it was causal of Petitioner's GBS-like symptoms, as opposed to a URI, which was also included in that causal differential. *See, e.g.*, Ex. 13 at 14. Other treaters later seemed to favor the URI.[21] Ex. 9 at 3. Thus, this is not a case where initial treater views on causality are helpful in determining the "did cause" prong. Further, as Dr. Sriram persuasively established, CIDP once initiated would likely progress within a month or two to nadir, separately from its otherwise remitting/relapsing course. Tr. at 108. But Petitioner's actual experience involved a much more rapid descent to nadir, with improvement occurring progressively that fall even though it was incomplete. In addition, his overall treatment from October 2016 to through the spring of 2017, with other diagnostic explanations (DDD, retrolisthesis) entering the picture, further reduced the likelihood of a CIDP diagnosis.

In addition, there is significant evidence of other explanations for Petitioner's post-vaccination symptoms—explanations relating to lower back, hip, or limb pain that predate vaccination. *See, e.g.*, Ex. 5 at 25–36. His initial post-vaccination complaints echo these earlier symptoms. Ex. 10 at 1; Ex. 7 at 4. Indeed, Petitioner mentioned the existence of such concerns at the time he received the flu vaccine, and does not otherwise deny they existed and predated vaccination. Tr. at 10–11; Ex. 5 at 25. While I am not on the basis of this record able to identify an alternative cause for Petitioner's symptoms, his argument that the vaccine explains his injuries is greatly complicated by this medical history, and he did not adequately differentiate these symptoms from what he experienced post-vaccination.

[20] Because all three *Althen* prongs must be established to obtain entitlement to damages, I do not discuss the third prong herein.

[21] The fact that the precise nature of the URI is not confirmed by evidence does not undercut it as an intercurrent factor that is unhelpful to Petitioner's case. Sufficient record evidence supports the conclusion that Petitioner not only reported to initial treaters that he had recently experienced a URI, but still was suffering from some residual symptoms. Ex. 13 at 13. I agree with Petitioner that it has not been exclusively established as an alternative cause on this record—but I reasonably evaluate it as secondary evidence, within the context of determining if Petitioner has met his initial burden of preponderantly supporting all three *Althen* prongs.

Then, the record shows a significant gap in treatment of over a year's time, further attenuating the relationship between the October 2016 event and what drove Petitioner to seek professional assistance in May 2018. While I give some weight to Petitioner's assertion that insurance coverage was an issue for him that resulted in fewer treater visits, the records overall support the conclusion that he sought treatment promptly when faced with acute events—as he did so in the fall of 2016 and then in May 2018. Ex 12 at 110 (May 2018 record reporting one-week onset).

This treatment gap is thus not wholly explained by financial concerns, but instead "more likely than not" reflects the fact that Petitioner was *not* experiencing the kind of remitting but chronic and persistent symptoms that a CIDP patient would likely experience in that timeframe. Rather, the gap is, as Dr. Sriram testified, inconsistent with CIDP *of any kind*, which would not feature such a total symptoms cessation. And Petitioner did not successfully establish that IVIG treatment explains the gap—in part due to Respondent's questions about its assumed efficacy in all situations involving demyelinating neuropathies, but also given the lack of record evidence of IVIG treatment *during this interval*. At bottom, this is too long a period of time to connect a putative, remote vaccine event with subsequent relapses.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such as showing. Petitioner is therefore not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[22]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[22] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.