In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 22-462V

STEVEN HILLSTROM,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: November 7, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Edward Kraus, Kraus Law Group, LLC, Chicago, IL, for Petitioner.

Eleanor Hanson, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On April 22, 2022, Steven Hillstrom filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that he developed Guillain-Barré syndrome ("GBS"), or chronic inflammatory demyelinating polyneuropathy ("CIDP"), as a result of receiving a Hepatitis B ("Hep. B") vaccine on September 4, 2020. Petition (ECF No. 1) at 1.

A one-day entitlement hearing was held in Washington, D.C. on February 25, 2025. Now, having reviewed the record, all expert reports, the medical records, and associated literature, I hereby find that Petitioner is not entitled to an award of compensation. The record does not support the conclusion that Petitioner's CIDP (the more preponderantly-associated diagnosis) was caused by the Hep. B vaccine.

[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) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I. Petitioner's Medical History

Vaccination and Initial Symptoms

Petitioner's medical history is significant for Crohn's disease,[3] among other things. Ex. 1 at 530; Ex. 2 at 33–48. Petitioner had a twenty-seven year history of inflammatory bowel disease ("IBD") at the time of vaccination, and began receiving Humira (brand name for adalimumab) for its treatment on August 13, 2020. Ex. 1 at 351, 428.

In late-July 2020, Petitioner received one dose of the Hep. B vaccine at the offices of his primary care physician ("PCP"), Dr. David Kass, with a second dose administered on September 4, 2020. Ex. 4 at 2; Ex. 6 at 2. He was 55 years old at the time. There is no record evidence of any initial vaccine reaction—either between the two doses, or in the month following his receipt of the second dose. And during this timeframe, Petitioner continued to receive Humira treatments (on September 10, and 23, 2020, and then again on October 8, 2020). Ex. 3 at 39; Ex. 9 at 2.

On October 14, 2020, Petitioner sent a message to his gastroenterologist, Dr. Edward Loftus, reporting improvement of his Crohn's symptoms, but reporting a potential side effect of numbness possibly attributed to the Humira. Ex. 1 at 436. Petitioner noted specifically that after receipt of the most recent Humira dose (on October 8, 2020), he began to experience numbness in his toes within three days. *Id.* This somewhat-neurologic symptom thus occurred on October 11th—37 days after receiving the second dose of the Hep. B vaccine.

Two days later (October 16, 2020), Petitioner returned to Dr. Kass complaining of the same kind of toe numbness, which he again reported had started on October 11, 2020. Ex. 3 at 39. Petitioner recalled that he had been receiving Humira since the summer, and that he experienced recurrent diarrhea after tapering off a different anti-diarrheal medication the week prior, stating, "this seemed to be associated with the numbness involving both feet." *Id.* Dr. Kass noted that Mr. Hillstrom's numbness was "a possible side effect of Humira, but other possible factors need[ed] to be considered." *Id.*

Neurological examination revealed that Petitioner was experiencing decreased sensation in the second and third toes, but normal strength in the extremities and a normal gait. Ex. 3 at 41. Laboratory studies also yielded normal results, although lumbar spine x-rays revealed "mild degenerative disc changes." *Id.* at 42. Dr. Kass's assessment included bilateral leg paresthesia, and he advised Petitioner to follow up if the numbness continued. *Id.* at 41. Petitioner received two

[3] Crohn's Disease is defined as a "disease of the gastrointestinal tract of unknown etiology; it can involve any part of the tract, but most often is found in the terminal ileum. Characteristics include scarring and thickening of the bowel wall that frequently leads to intestinal obstruction, abscesses, and fistula formation. There is a high rate of recurrence after treatment." *Crohn Disease*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70226 (last visited Oct. 23, 2025).

other vaccines (influenza and tetanus-diphtheria vaccinations) at this visit. *Id.* at 42. Later that months (October 22nd) he received another Humira dose. Ex. 1 at 352.

Petitioner saw a chiropractor on October 28, 2020, for shoulder pain, and he reported at that time that two weeks prior, he had begun to have a sensation of having socks on while he was barefoot. Ex. 5 at 2. He also complained of pain in his left mid-back, numbness in his lips and fingertips, and no feeling in his foot from the mid-arch to his toes. *Id.* The chiropractor speculated that such pain did not have a mechanical origin, and that he should work with his gastroenterologist to "rule in/out a reaction to the [H]um[i]ra considering the timing of starting the medication 8 weeks ago." *Id.* at 3.

Progression of Neurologic Symptoms and Emergency Treatment

On October 29, 2020, Dr. Kass's office advised Petitioner to go to the emergency department after experiencing facial and oral numbness that he said had been present for the prior three weeks (and thus began around the time of the October 8th Humira dose). Ex. 2 at 7. Petitioner also complained of numbness and tingling in his fingers, lips, and tongue; aching pain in both of his wrists; and an "unusual" sensation in his throat, but denied extremity weakness. *Id.*

Neurological examination at the ED revealed decreased sensation in Petitioner's hands and feet, normal strength, and normal reflexes. Ex. 2 at 9. A head CT was negative for acute intracranial pathology, and various laboratory studies and x-rays were normal. *Id.* at 11, 53–54. In the medical record's written assessment, the treating medical provider, Andrea Bouman, M.D., noted that Mr. Hillstrom had "paresthesia in a stocking-glove distribution as well as over the lips, tongue, and a patch overlying the left scapula." *Id.* at 11. Dr. Bouman further observed that Petitioner had no weakness on isolated muscle testing, making his presentation "not classic for [GBS] or neuromuscular disorder." *Id.* Petitioner was advised to follow up with his PCP to rule out multiple sclerosis or an autoimmune disorder. *Id.*

The next day, Brittany Wagunda, N.P. (primary care) examined Petitioner for worsening numbness in his feet, hands, mouth, and back. Ex. 3 at 35. Petitioner reported that he had begun to experience pain in the areas of previously-experienced numbness. *Id.* N.P. Wagunda noted that Mr. Hillstrom had begun Humira two to three months earlier. *Id.* Petitioner's neurological examination revealed no sensation in his feet in a sock-like pattern and in his hands in a glove-like pattern, but normal reflexes and strength. *Id.* at 37. N.P Wagunda's assessment was polyarthralgia and upper extremity paresthesia and pain. *Id.* Petitioner was prescribed hydrocodone for his pain and provided a neurology referral. *Id.*

Petitioner saw N.P. Wagunda again on November 5, 2020, for treatment of progressing numbness and increasing pain. Ex. 3 at 31. Petitioner now reported a vibrating sensation through

his head and body, decreasing skin sensitivity, and a shooting pain down his arms. *Id.* His last Humira dose had been two weeks prior, and he was experiencing brain fog plus increased immobility. *Id.* Petitioner's neurological examination revealed normal strength, normal reflexes, and decreased sensation in the hands and feet. *Id.* at 33. Petitioner was prescribed Dilaudid for his pain and again provided a neurology referral. *Id.* He also received an MRI the next day, and it yielded normal results, although a left shoulder x-ray showed "sclerosis involving the humeral head," suggesting the possibility of avascular necrosis, and "benign soft tissue calcification along the humeral neck." Ex. 2 at 55–56.

A visit with Dr. Kass mid-November did not shed additional light on a potential cause of his symptoms, although the view was expressed (after a cervical MRI) that disc degenerative issues did not likely explain his problems. Ex. 3 at 26, 27; Ex. 2 at 57. However, an EMG[4] performed around this time revealed evidence of a "length dependent sensorimotor peripheral neuropathy," prolonged distal latencies suggesting a peripheral demyelinating process, especially in the legs, and "no compelling evidence to support a superimposed right lumbosacral radiculopathy." Ex. 1 at 419.

That same November, Petitioner saw his regular gastroenterologist, Edward Loftus, Jr., M.D., for evaluation of "rapidly progressing paresthesia in the setting of Crohn's." Ex. 1 at 350. He reported improvement in his Crohn's symptoms after starting Humira in August 2020, but a sudden onset of feet numbness around October 11, 2020 (consistent with what he had informed previous treaters). *Id.* He had received his last dose of Humira on October 22, 2020. *Id.* Exam revealed an unsteady gait, and Dr. Loftus noted that it was not clear if Petitioner's neuropathy was related to the Humira, since the medication predated onset by two months, and that "CIDP need[ed] to be excluded." *Id.* at 352.

Mayo Clinic Evaluations

On November 16, 2020, Petitioner saw neurologist Ali Zandieh, M.D. (with Narayan Kissoon, M.D., attending), at the Mayo Clinic, for an evaluation of tingling and numbness in his extremities that Petitioner reported was worsening. Ex. 1 at 395. Petitioner reported his condition was slowly worsening. *Id.* Petitioner mentioned his Humira medication, as well as the fact that he recalled having an upper respiratory infection in September. *Id.* at 395–96. Physical examination revealed decreased sensation in all extremities, 1+ reflexes in the upper extremities, and 2+ reflexes in the lower extremities. *Id.* at 397. Dr. Zandieh assessed Petitioner with "subacute

[4] An electromyography ("EMG") is defined as "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation . . . " *Electromyography*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography (last visited Oct. 23, 2025).

progressive numbness, weakness, and paresthesia," "demyelinating sensorimotor peripheral neuropathy in the EMG," and "Crohn's disease on Humira," adding that some case reports had linked Humira to neuropathy, and that a predisposition to autoimmune disease (which could describe Petitioner) could explain his current issues. *Id.*

Petitioner was subsequently tested for autoimmune neuropathy, and a lumbar puncture was recommended to look for albumin-cytologic dissociation. Ex. 1 at 397. But a paraneoplastic autoantibody panel was negative, and other various laboratory studies were normal. *Id*. at 314, 328, 374–91. Dr. Kissoon deemed the temporal profile for Petitioner's course to be consistent with chronic inflammatory polyradiculoneuropathy, agreeing that the combination of Petitioner's history of autoimmune disease and recent Humira constituted risk factors for CIDP. *Id.* at 399.

While still at the Mayo Clinic, Petitioner also saw peripheral nerve specialist Kamal Shouman, M.D. Ex. 1 at 296. Petitioner provided his medical history, noting that over the last two weeks he had developed weakness in both his hands, and that he was experiencing arm and leg pain. *Id.* He also recalled his Humira course, along with the fact that he had experienced a cold in mid-September. *Id.* A lumbar puncture performed at this time revealed elevated protein at 158, with normal cell count and glucose, but blood work did not reveal the presence of any known neuromuscular disease-associated antibodies. *Id.* at 270, 284, 296. Although the differential included feet paresthesia, GBS, and CIDP, Dr. Shouman proposed "likely acute to subacute inflammatory demyelinating neuropathy," adding that symptoms seemed to have "followed an upper respiratory infection and prior to that a new treatment for his IBD with Humira." *Id.* at 295, 297. Dr. Shouman prescribed gabapentin and IVIG for five days followed by twelve more weekly follow-ups and recommended an EMG later. *Id.*

Later that month, Petitioner began to feel that his numbness and weakness was progressively expanding. Ex. 1 at 263, 267. Thereafter, between November 20–24, 2020, Petitioner received daily IVIG treatments, continuing on a weekly or tri-weekly schedule over the ensuing months. Id. at 149–240. By December 9, 2020, it was noted during a visit with Dr. Kass that Petitioner's pain and weakness were slowly improving with IVIG treatment and gabapentin, despite some ambulation limits. Ex. 3 at 22. At a PT initial evaluation in mid-December, he displayed upper and lower body weakness, paresthesia, numbness, and ambulation deficits. Ex. 2 at 173. From this point to the summer of 2021, Petitioner participated in 25 PT sessions, finding some improvement in gait issues over that timeframe. *Id.* at 68–173.

Questions re Etiology and Diagnosis During Subsequent Treatment

Petitioner filed numerous exhibits detailing his treatment over the subsequent three years (2021–24), although collectively they mainly substantiate CIDP as his likely injury.

In January 2021, for example, a pain management treater noted that Petitioner's "temporal profile [was] more consistent with CIPD rather than AIDP[5] [GBS], but a serial EMG [would] help differentiate between the two disorders." Ex. 1 at 101. A repeat EMG performed that February yielded abnormal results but did not establish what diagnostic classification was more accurate. *Id.* at 50, 62–64. But in September 2021, Dr. Kass assessed Petitioner with CIDP. Ex. 53 at 19, 21–22. This diagnosis was maintained thereafter. *See, e.g.*, *id.* at 17 (January 2022 visit to Dr. Kass); Ex. 54 at 95–96 (Dr. Loftus characterizing Petitioner's illness in March 2022 as GBS "which is now really CIDP"); Ex. 53 at 9, 11–12 (April 2022 visit with Dr. Kass); Ex. 70 at 85 (August 2024 Mayo Clinic neurology evaluation). Petitioner himself eventually seemed to accept CIDP as the proper diagnosis. *See, e.g.*, Ex. 54 at 32 (communication from Petitioner asking about "dosage and frequency of IVIG for CIDP").

Throughout (consistent with the chronic character of CIDP), Petitioner's general symptoms continued to some extent, waxing and waning over time (sometimes depending on whether he was regularly receiving IVIG treatments, his current medication course, etc.)—although he did report improvement on many fronts by April 2024 and thereafter. *See, e.g.*, Ex. 65 at 15; Ex. 70 at 177. And follow-up imaging eventually ceased to show evidence of active demyelination.

With respect to the causal role of the Hep. B vaccine, treaters often allowed for receipt of the vaccine as one of several possible triggers—but in discussing causes, also included Humira plus the fact that Petitioner had experienced a respiratory infection before onset. *See, e.g.*, January 2021 pain management visit (Ex. 1 at 97); March 2021 follow-up visit with Dr. Kass (Ex. 3 at 11); Ex. 53 at 14 (January 2022 visit to Dr. Kass). The record does not contain any definitive statement by any of Petitioner's primary treaters as to the favored etiologic explanation for his CIDP.

II. Hearing Witnesses

A. *Petitioner's Witnesses*

1. Steven Hillstrom – Petitioner was the sole fact witness to testify at hearing. *See generally* Tr. 7–40. He began his testimony by recounting his pre-vaccination medical history. *Id.* at 8. The only significant issue relevant to the claim that he had previously experienced was ulcerative colitis that developed into Crohn's disease. *Id.* Petitioner had been managing his condition with gastroenterologist Dr. Loftus at the Mayo Clinic in Rochester, Minnesota. *Id.* Petitioner would go to the Mayo Clinic once a year for treatment, and would have phone consults with Dr. Loftus between visits. *Id.* at 9.

[5] "Acute demyelinating polyradiculoneuropathy (AIDP) is the most common variant [of GBS] but accounts for a higher percentage of cases in North America and Europe." Its clinical features include relatively symmetric weakness in two or more limbs, either with or without sensory symptoms, and hypoactive or absent reflexes. E. Monohan & T. Brannagan III, *Immune-Mediated Neuropathies: Top 10 Clinical Pearls*, 45 Semin. Neurol. 122, 122–23 tbl. 1 (2024), filed as Ex. C-3 (ECF No. 47-4) ("Monohan & Brannagan III")

During one of these visits, Petitioner recalls discussing changing his medication with Dr. Loftus when Petitioner's receipt of the Hep. B vaccine was brought up. Tr. at 9. Petitioner believed Dr. Loftus had communicated that Petitioner's insurance required him to have the vaccine, and that the clinic would test Petitioner for antibodies before choosing to administer it as a preventative measure. *Id.*

Petitioner received his first Hep. B vaccine dose on July 31, 2020, and the second on September 4, 2020—roughly five weeks later. Tr. at 10. Petitioner did not recall any adverse symptoms from the vaccines other than a minor rash, but considered that a normal vaccine reaction for him. *Id.* Petitioner began to undergo treatment with Humira on August 12, 2020, and received two other doses on September 23rd and October 8th of that same year. Ex. 3 at 39; Ex. 9 at 2. It was only on October 10, 2020, that Petitioner started to experience numbness in his toes. Tr. at 11. Later that week, Petitioner contacted the Humira representative who felt like this could be a possible side effect of the medication, and instructed Petitioner to follow up with his PCP. *See id.* at 13. Petitioner also remembers reaching out to Drs. Loftus and Kass (Petitioner's PCP) regarding the toe numbness around the same time. *Id.* at 12–13. The doctor notes from Petitioner's October 16, 2020, visit with Dr. Kass detailed that Petitioner had started Humira and was able to taper off from an anti-diarrheal medication during the "prior week," but had noticed recurrent diarrhea on Sunday, October 11, 2020, which "seemed to be associated with onset of numbness involving both feet." *Id.* at 12, 13:4–12. Dr. Kass at that same appointment administered a flu shot to the Petitioner's surprise. *Id.* at 14.

A week or two after that appointment, Petitioner remembered the numbness growing to his legs and into his upper body. Tr. at 14. He also claimed that he started to experience a lot of back and arm pain for which he sought out a chiropractor in case he had pinched nerves, but the chiropractor did not find anything. *Id.* It was after this, on October 29, 2020, that Petitioner went to the emergency room complaining of numbness in his feet and hands. *Id.*

During his stay at the hospital, Petitioner recalled drooling and having difficulty swallowing. Tr. at 15. He remembers receiving an x-ray of his lungs and being sent home by hospital staff because he was able to breathe well enough. *Id.* at 15–16.

Petitioner's pain persisted, and Petitioner recalled that the pain medications prescribed to him by Dr. Kass had not been helping. Tr. at 16. Petitioner remembered that he was unable to get upstairs on his own and he had to sleep downstairs in is home. *Id.* at 17. Walking was also difficult for the Petitioner and his wife had to drive him around. *Id.* at 18. On cross, Petitioner testified that he started using a wheelchair during his visit to the ER, and was given a wheelchair by his family to use around their house when needed. Tr. at 39–40.

Petitioner was eventually able to see a Mayo clinic neurologist on November 16, 2020, where a spinal tap and EMG showed the Petitioner had GBS. *Id.* at 19, 22. Petitioner was subsequently referred to underwent IVIg treatments which Petitioner believed helped stop the pain from progressing and improved his condition. *Id*. at 24–26. Petitioner testified that during this time he asked both Dr. Kass and his neurologist, Dr. Shouman, whether he should receive his final Hep. B vaccination. *Id.* at 26. To which Petitioner recalls that they both advised the Petitioner not to take it. *Id.*

Dr. Kass retired and Petitioner switched providers to Dr. Wright who had a discussion with Petitioner about his diagnosis of GBS versus CDIP. Tr. at 32. Petitioner testified that Dr. Wright refused to "do anything" unless Dr. Shouman was guiding treatment directly, and Petitioner saw Dr. Wright once a year. *Id.*

With IVIg treatment and PT, Petitioner testified that he was able to slowly begin walking again, and then progressively build up his strength and endurance to the point where he can currently drive his truck and car with hand controls, boat, fish, walk unassisted, and cross-country ski Tr. at 33. He has not had any relapses, but sometimes finds himself tired after days where he "overdid" exercising. *Id.* at 34. Petitioner recalls his treating provider, Dr. Shouman, even informed him that in January of 2025, that his EMG test was his best improvement to date. *Id.* However, Petitioner complains that he still suffers residual effects, like losing sensitivity from touch, having to play adaptive handicapped sports instead of regular sports, walking with a cane, and not being able to drive his car without hand controls. *Id.* at 35–36.

2. Dr. Norman Latov – Dr. Latov is a board-certified neurologist who offered three written expert reports on behalf of the Petitioner and testified at the hearing. Report, dated May 30, 2023, filed as Ex. 21 (ECF No. 23-1) ("First Latov Rep."); Report, dated Jan. 11, 2024, filed as Ex. 55 (ECF No. 34-1) ("Second Latov Rep."); Report, dated Jan. 14, 2025, filed as Ex. 85 (ECF No. 62-1) ("Third Latov Rep."). Dr. Latov opines that Petitioner developed axonal GBS as a consequence of receipt of the Hep. B vaccine.

Dr. Latov earned his M.D. from the University of Pennsylvania, School of Medicine in 1975. Curriculum Vitae, dated May 31, 2023, filed as Ex. 52 (ECF No. 29-1) ("Latov CV") at 1. After earning his M.D., Dr. Latov completed an internship at Boston City Hospital and his Residency in Neurology at Columbia University. *Id.* at 2. He followed this up with a fellowship in immunology where he looked T cell regulation of B cells before joining the faculty at Columbia University's Department of Neurology. *Id;* Tr. at 43. Dr. Latov also directed a laboratory that conducted research into the mechanism of autoimmune peripheral neuropathies that is credited with discovery of certain antibodies. First Latov Rep. at 1. Dr. Latov has and continues to treat patients to this day, and he currently serves as a Professor of Neurology at Columbia University. Latov CV at 1; Tr. at 47. Over the course of his forty-year career, Dr. Latov has authored over one-

hundred fifty publications. Latov CV at 1–14.

Dr. Latov began his testimony describing the diagnostic criteria and symptoms treating providers look for to diagnose a peripheral neuropathy. Known causes of neuropathy include diabetes, infections, toxins, and more. Tr. at 49. When presented with a patient with symptoms of weakness, sensory loss, or pain, providers order tests like blood tests, spinal taps, and nerve conduction studies to evaluate the type of neuropathy (axonal or demyelinating) and its severity. *Id.* at 48–50. A constellation of tests and symptoms are needed to make a diagnosis of GBS or CIDP. *Id.* at 51.

Dr. Latov also discussed the competing diagnoses at issue in this case. GBS is different from CIDP, he maintained, noting that the former can be an axonal or demyelinating disease that runs a monophasic course—so patients decline, stabilize, and improve. *Id.* CIDP, by contrast, is always demyelinating, and has a prolonged course and includes remissions and exacerbations of symptoms. *Id.* Diagnostic criteria for GBS are divided into two levels of certainty, but the criteria Dr. Latov focused on were bilateral and flaccid weakens of the limbs, decreased or absent deep tendon reflexes, and monophasic pattern with a nadir between twelve hours and twenty-eight days. *Id.* at 51–54 (discussing J. Sejvar et al., *Guillain-Barre Syndrome and Fisher Syndrome: Case Definitions and Guidelines for Collection, Analysis, and Presentation of Immunization Safety Data*, 29 Vaccine 599, 604 (2011), filed as Ex. A-2 (ECF No. 30-3).

Dr. Latov also testified that ninety percent of GBS patients will reach nadir by four weeks, but there are outlier cases where nadir is reached months after onset. Tr. at 56 (citing A. Asbury & D. Cornblath, *Assessment of Current Diagnostic Criteria for Guillain-Barre Syndrome*, 27 Ann. Neurol. Supp. S21, S21–S22 (1990), filed as Ex. 23 (ECF No. 23-3) ("Asbury & Cornblath")). Diagnostic criteria for CIDP is different, typically presenting with symmetric proximal or distal progressive neuropathy involving at least two limbs developing over at least eight weeks and absent or reduced tendon reflexes. Tr. at 59–60 (citing P. Van den Bergh et al., *European Academy of Neurology/Peripheral Nerve Society Guideline on Diagnosis and Treatment of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force-Second Revision,* 28 Eur. J. Neurol. 3556, 3560 (2021), filed as Ex. 87 (ECF No. 63-2) ("Van den Bergh")).

Based on Petitioner's medical records, Dr. Latov opined that the most appropriate diagnosis was axonal GBS. Tr. at 64. Petitioner started developing sensory symptoms indicative of neuropathy within five weeks of his second Hep. B vaccine dose, followed by sensory loss and weakness. *Id*. at 65–66. Petitioner's spinal tap showed increased protein concentration and normal white blood cell counts, and Petitioner's EMG results showed abnormalities indicative of

sensorimotor neuropathy,[6] which is demyelination secondary to axonal disease. *Id.* at 66–70. Further, Petitioner's treating provider, Dr. Shouman, noted that Petitioner likely had "acute to subacute inflammatory demyelinating neuropathy," advised Petitioner not to take a third vaccine dose, and prescribed IVIg treatment to the Petitioner (a standard treatment for GBS). *Id.* at 72–74 (quoting Ex. 1 at 297). Dr. Latov also noted that Petitioner's records showed he was improving with IVIg treatment. *Id.* at 74 (citing Ex. 3 at 22).

By contrast, Dr. Latov deemed the record to be inconsistent with CIDP. His EMG findings, for example, did not reflect primary demyelinating abnormalities, as would be the case with CIDP. Tr. at 69–70. Indeed, such abnormalities should be evident even in the midst of ongoing treatment. *Id.* at 71–72 (discussing R. Chin et al., *Follow-up Nerve Conduction Studies in CIDP after Treatment with IGIV-C: Comparison of Patients with and without Subsequent Relapse*, 52 Muscle Nerve 498, 500 tbl. 2 (2015), filed as Ex. 86 (ECF No. 63-1) ("Chin")). Petitioner's EMGs actually showed continued improvement. *Id.* at 78–80 (citing Ex. 1 at 62,65; Ex. 4 at 129). In addition, Dr. Latov recalled that Petitioner's treating provider, Dr. Shouman, assessed the Petitioner with acute to subacute inflammatory demyelinating neuropathy, and eventually diagnosed him with GBS. *Id.* at 72–73. Further, Dr. Shouman advised Petitioner to not take a third dose of the Hep. B vaccine (underscoring the potential causative relationship). *Id.* at 74.

Further disputing Respondent's argument for a CIDP diagnosis, Dr. Latov asserted that the Petitioner's medical records did not demonstrate that Petitioner's health was backsliding after improvement. Tr. at 75–84. Physical therapy notes where Petitioner reported his struggling with his recovery at certain times were consistent with GBS, Dr. Latov opined. *Id.* at 76, 83. It is normal in recovery to have good and bad days based on energy levels and emotional state. *Id.* at 76–77, 83. Rather, Dr. Latov testified that muscle exams and EMGs were more objective evidence as to whether the condition is getting worse or not. *Id.* at 77. In this case, Petitioner's EMGs showed improvement and that Petitioner's neuropathy was not worsening. *Id.* at 78–79, 84.

The Hep. B vaccine likely caused Petitioner's axonal GBS, Dr. Latov opined. Tr. at 85. One of the mechanisms by which the vaccine could have done so was molecular mimicry, which occurs when there is a structural or sequential homology between a component in the vaccine and the host, resulting in the immune system attacking the body itself rather than the disease. *Id.* at 87–88. The best example of molecular mimicry causing GBS if from the *Campylobacter jejuni* bacterial infection, since the antigens of the bacterium contain structures comparable to gangliosides found in peripheral nerves. *Id.* at 90–92 (citing B. Soliven, *Animal Models of Autoimmune Neuropathy*, 54 Ilar J. 282, 282 (2014), filed as Ex. 43 (ECF No. 54-6)). Evidence of amino acid sequential homology was also present with respect to myelin oligodendrocyte

[6] Dr. Latov later clarified that acute axonal sensorimotor neuropathy ("AMSAN") is the motor form of GBS, which Petitioner's treating providers missed. Tr. at 161–162. Dr. Latov continued to explain that Petitioner did not have acute motor axonal neuropathy ("AMAN"). *Id.* at 161.

glycoprotein ("MOG") found in the central nervous system and proximally in the peripheral nervous system. *Id.* at 93–94 (citing D. Bogdanos et al., *A Study of Molecular Mimicry and Immunological Cross-Reactivity Between Hepatitis B Surface Antigen and Myelin Mimics*, 12 Clinical & Developmental Immunology 217, 222 (2005), filed as Ex. 82 (ECF No. 61-3) ("Bogdanos")). Dr. Latov conceded that it is not proven that this is the "cross-reactive antigen and Guillain-Barre in [petitioner's] case, but it is a potential[,]" even for the axonal variant of GBS. *Id.* at 95–96.

Dr. Latov offered other scientific literature in support of his theory that the vaccine caused Petitioner's GBS. Tr. at 98–99. He pointed to multiple case studies where patients who had received the Hep. B vaccine went on to develop GBS. *Id.* (discussing N. Souayah et al., *Guillain-Barre Syndrome After Vaccination in United Sates: Data from the Centers for Disease Control and Prevention/Food and Drug Administration Vaccine Adverse Event Reporting System (1990-2005)*, 11 J. Clin. Neuromuscul. Dis. 1, 2 (2009), filed as Ex. 45 (ECF No. 30-39) (identifying ninety-four instances of GBS after receiving a the Hep. B vaccine, with the majority of those cases experiencing symptom onset within six weeks of administration); C. Vital et al., *Postvaccinal Inflammatory Neuropathy: Peripheral Nerve Biopsy in 3 Cases,* 7 J. Peripheral Nervous System 163, 163 (2002) filed as Ex. 71 (ECF No. 55-8) ("Vital") (case studies where two of the three patients with GBS had previously received a Hep. B vaccine); M. Khamaisi et al., *Guillain-Barré Syndrome Following Hepatitis B Vaccination*, 22 Clinical Experimental Rheumatology 767, 767–68 (2004), filed as Ex. 72 (ECF No. 55-9) ("Khamaisi") (case study where the patient developed GBS 8 weeks after receiving a Hep. B vaccine, but tables other case reports that show onset of GBS symptoms occur in many patients as soon as one day after receiving the Hep. B vaccine); H. Muller, *Inflammatory Infiltrates in the Spinal Cord of Patients with GBS*, 106 Acta Neuropathol. 509, 509 (2003), filed as Ex. 84 (ECF No. 61-5) ("Muller") (investigation of a case where patient developed GBS nine days after receiving the Hep. B vaccine and was found to have inflammatory infiltrates and histopathological changes in the spinal cord)).

Respondent had offered a large-scale epidemiologic study that facially seemed to contradict a vaccine association. R. Baxter et al., *Lack of Association of Guillain Barre Syndrome with Vaccinations*, 57 Clin. Infect. Dis. 197, 200–01 (2013), filed as Ex. A-36 (ECF No. 30-37) ("2013 Baxter") (retrospective study concluding no causal association between vaccines and development of GBS from results of only twenty-five instances of vaccine administration within six-weeks of GBS symptom onset out of four-hundred fifteen confirmed cases of GBS). But Dr. Latov deemed Baxter to be full of biases that allowed the study to come to that conclusion. Tr. at 100–01.

Turning to specific causation and the appropriate medical timing for vaccine-caused GBS, Dr. Latov opined that a five-week onset from time of vaccination to onset of neuropathy symptoms was appropriate and was supported by the medical literature submitted for this case. Tr. at 101–02

(discussing L. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977*, 110 Am. J. Epidemiol. 105, 105 (1979), filed as Ex. 12 (ECF No. 8-2) ("Schonberger")). Further, Dr. Latov stated that he did not believe that Crohn's disease, ulcerative colitis, or Humira causes GBS. *Id.* at 103. While Crohn's disease and ulcerative colitis are autoimmune, and people with autoimmune diseases tend to develop other afflictions, that does not mean that one causes the other. *Id.* at 103–04. Dr. Latov disagreed that Humira causes GBS because there is no evidence that it causes GBS more frequently than any other comparable TNF[7] inhibitor. *Id.* at 104–05. Respondent's offered literature supporting a connection between the two is fundamentally flawed, Dr. Latov opined, because a majority of the patients in the case studies recorded had a preceding infection, which is another possible explanation for the development of GBS. *Id.*

On cross, Dr. Latov conceded that his testimony was the first time he had opined that Petitioner had axonal-oriented GBS. Tr. at 110–12. When pressed on what is the classical presentation of GBS, Dr. Latov acknowledged that the majority of GBS patients reach nadir four weeks after onset and also reach post-nadir recovery within two to four weeks. *Id.* at 108–09 (discussing Asbury & Cornblath). Petitioner, however, was improving as late as early 2021—longer than would be expected for the most common form of GBS. *Id.* at 124. Dr. Latov also agreed that Dr. Shouman at one point opined that the Petitioner was suffering from a chronic form of demyelinating inflammatory polyneuropathy, or CIDP, and that Petitioner's EMGs weakly supported the presence of ongoing demyelination. *Id.* at 129, 135–36.

Dr. Latov further conceded on cross that multiple pieces of scientific literature offered in this matter did not address the relationship between the Hep. B vaccine and any form of GBS. *See* Tr. at 141–46. And he admitted that some of the evidence submitted that *did* pertain to the Hep. B vaccine did not involve GBS or CIDP, or could not otherwise be connected to the relevant form of vaccine. *Id.* at 146–51. *See, e.g., Adverse Effects of Vaccines: Evidence and Causality*, Institute of Medicine (K. Stratton et al., eds. 2012), filed as Ex. 57 (ECF No. 42-2) ("2012 IOM Rep.") (showing homology between myelin basic protein and Hep. B virus polymerase, not Hep. B *surface* antigen which is used in Hep. B vaccines); McCoy et al., *Multiple Sclerosis and Virus Induced Immune Responses - Autoimmunity Can be Primed by Molecular Mimicry and Augmented by Bystander Activation*, 39 Autoimmunity 9, 9 (2006), filed as Ex. 37 (ECF No. 24-8) (discussing multiple sclerosis).

More fundamentally, Dr. Latov agreed that articles *he had co-authored* supported the idea of a "primary immune-mediated neuropathy as an extra-intestinal disorder associated with IBD and not merely a co-occurrence with CIDP." Tr. at 153 (quoting F. Gondim et al., *Peripheral*

[7] Tumor necrosis factor ("TNF") is defined as "either of two lymphokines that are capable of causing in vivo hemorrhagic necrosis of certain tumor cells but not affecting normal cells." *Tumor necrosis factor*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74613 (last visited Nov. 5, 2025).

Neuropathy in Patients with Inflammatory Bowel Disease, 128 Brain 867, 867 (2005), filed as Ex. A-18 (ECF No. 30-19) ("2005 Gondim")). In other words, Dr. Latov allowed that the kind of neuropathy Petitioner had experienced *could itself occur in association with Crohn's disease*. Dr. Latov maintained, however, that 2005 Gondim had underscored that even if men with IBD were more susceptible to CIDP, causation between the two had not been demonstrated. *Id.* (discussing 2005 Gondim).

Dr. Latov criticized Respondent's expert testimony on rebuttal, reemphasizing his opinion that the Hep. B vaccine had likely caused Petitioner's GBS. The EMG results did not return any numbers or data that would support demyelination (as would be consistent with CIDP), especially since there was no evidence of an abnormal temporal dispersion greater than 30%. Tr. at 309–12. Those same testing results, however, revealed continued improvement after the acute event. *Id.* at 313. The fact that the improvement was not more rapid did not undermine a GBS diagnosis because nerves often do not regenerate completely, and thus abnormalities can be evident over time even while recovery is underway. *Id.* at 313–14. EMG results consistent with CIDP, Dr. Latov argued, would show new abnormalities arising persistently (although he referenced in support of this contention an article not filed in this case). *Id*. at 315.

Dr. Latov also reiterated the view that there are many theories explaining how the Hep. B vaccine could cause GBS, like molecular mimicry and MOG cross-reactivity. Tr. at 314–318. Dr. Latov once again likened the Hep. B vaccine to the flu vaccine—which is accepted in the program as causing GBS—opining that if one can cause GBS, the other can as well. *Id.* at 317–18. Also, Dr. Latov pointed to Bogdanos as support for potential homology between MOG and Hep. B surface antigen to further a molecular mimicry theory. *Id.* at 318. And, lastly, Dr. Latov commented on the relevance of MOG cross-reactivity in the absence of a diagnosis of MOG-associated disease as supporting the conclusion that the Hep. B vaccine is capable of causing GBS. *Id.* 314–16. Multiple studies, he maintained (although not submitted in the present case) found damage to the proximal nerve root, which contains MOG, in several patients who possessed anti-MOG antibodies and who developed peripheral neuropathies. *Id.* This supports a theory that inflammatory reaction in the proximal nerve root caused axonal damage and an abnormal EMG reading. *Id.* at 315–16.

B. *Respondent's Witnesses*

1. Dr. Dara Jamieson – Dr. Jamieson is a board-certified neurologist who submitted one expert report in this case, and testified at trial on behalf of the Respondent. Report, dated Sep. 25, 2023, filed as Ex. A (ECF No. 30-1) ("Jamieson Rep."). Dr. Jamieson opines that Petitioner had CIDP that was not caused by his receipt of a Hep. B vaccine.

Dr. Jamieson received her M.D. from the University of Pennsylvania School of Medicine.

Curriculum Vitae, filed as Ex. A-42 (ECF No. 30-43) ("Jamieson CV"). She followed her M.D. with a neurology residency and a cerebrovascular fellowship at the Hospital of the University of Pennsylvania. *Id.* at 1. She later taught and participated in clinical practice in Philadelphia at multiple facilities, including Temple University hospital; Thomas Jefferson University, University of Pennsylvania hospital; and Pennsylvania Hospital where she gained close to twenty years of experience. *Id.* at 1–2. Dr. Jamieson is currently a Clinical Associate Professor of Neurology at Weill Cornell Medicine where she teaches medical students and residents. *Id.* at 1. She has authored twenty-two peer reviewed medical articles and contributed to approximately fifty other pieces of medical literature regarding neurology. *Id.* at 10–14.

Dr. Jamieson began her testimony asserting her conclusion that the Petitioner's proper diagnosis is CIDP—not GBS. Tr. at 172. Axonal GBS resembles AIDP—the most common form of GBS—at first because of the sudden onset of symptoms and clinical course. *Id*. at 175–76. However, early EMGs of axonal GBS will show *axonal* features, and the patient's condition will be longer and worse than someone with AIDP. *Id.* at 176. CIDP is also prolonged, and characterized as chronic, relapsing-remitting, and fluctuating. *Id.* at 177.

Dr. Jamieson nevertheless opined that Petitioner has CIDP, noting that his symptoms appeared at five weeks (which is beyond when 90% of GBS patients would experience onset), his weakness began in his hands, and Petitioner was not showing signs of wheelchair dependance until two months after symptoms onset—all inconsistent with a GBS diagnosis. Tr. at 179. In addition, he was experiencing distal sensory symptoms, it took over eight weeks from onset for Petitioner to reach his nadir after onset, and IVIg treatment provided before nadir did *not* improve his symptoms or prevent him from getting worse *Id.* at 180–82. GBS patients usually improve over weeks to months with treatment, whereas the Petitioner was receiving treatment for a longer time. *Id.* at 183.

Other medical records similarly supported a diagnosis of CIDP. Petitioner's neurologic exam was unremarkable initially except for distal sensory loss, and recorded an "absence of weakness." Tr. at 184–85 (citing Ex. 2 at 7, 9). These kind of features are far more consistent with CIDP. *Id.* at 186. In addition, Dr. Jamieson opined that Dr. Latov had misinterpreted Petitioner's EMG results, which when properly read supported a diagnosis of CIDP. *Id.* at 188. Dr. Latov had focused on motor nerve results, when he should have been looking at on *sensory nerve results*—and the record showed that Petitioner was experiencing sensory symptoms before motor symptoms. *Id.* at 188–89. The EMGs reflect more sensory nerve abnormalities than motor nerve abnormalities, which point to demyelinating peripheral neuropathy, like CIDP. *Id.* at 189–90, 206–07. And overall Petitioner's condition was not monophasic. *Id.* at 199. Rather, Petitioner reported his hands and feet worsening over the course of weeks, reflecting sensory abnormalities and not a monophasic progression. *Id.*

Testifying to specific causation, Dr. Jamieson opined that Petitioner's injury was more likely attributable to his Humira medication and/or Crohn's disease. Tr. at 213–14. Crohn's disease, she testified, is an irritable bowel disorder/autoimmune disease that can be associated with secondary neurologic disorders—like peripheral neuropathies or demyelinating axonal conditions. *Id.* at 213–16, 219 (*citing* 2005 Gondim at 867; F. Gondim et al., *A Case-Control Study of the Prevalence of Neurological Diseases in Inflammatory Bowel Disease (IBD)*, 73 Arq. Neuropsiquiatr. 119, 120 (2015), filed as Ex. A-19 (ECF No. 30-20). Humira has also been associated with demyelinating peripheral neuropathies and demyelinating axonal diagnoses. *Id.* at 214–15, 221–23 (discussing C. Gill et al., *Neurological Complications of Therapeutic Monoclonal Antibodies: Trends from Oncology to Rheumatology*, 17, Curr. Neurol. Neurosci. Rep. 75, 75 (2017), filed as Ex. A-22 (ECF No. 30-23) ("Gill") (a review of neurological complications associated with monoclonal antibody-based therapies, including Humira); N. Natividade et al., *Guillain-Barre Syndrome in a Patient on Adalimumab for the Treatment of Psoriasis*, 92 An Bras Dennatol. 85, 85 (2017), filed as Ex. A-24 (ECF No. 30-25) ("Natividade") (case report detailing the instance of GBS developing in a 45 year-old male patient who was taking Humira)). So regardless of the correct diagnosis, it was the impact of Petitioner's Crohn's disease (and/or the medication used to treat it) that led to his peripheral neuropathy, with the former making Petitioner susceptible and the latter actually triggering the neuropathy. *Id.* at 215; *see* 2005 Gondim at 877 ("[I]t is likely that there is a primary immune-mediated neuropathy as an extra-intestinal disorder associated with IBD and not merely a co-occurrence with CIDP.").

On cross, Dr. Jamieson was asked whether Petitioner's ambulatory difficulties started before or after his IVIg treatments on November 20, 2020. *See* Tr. at 224–31. Dr. Jamieson conceded that Dr. Loftus did note that Petitioner displayed an "unsteady gait" under his physical examination notes during Petitioner's November 16, 2020 visit, and that Petitioner confirmed he had been using assistive devices to walk at the time of his November 18, 2020 fall risk screening. *Id.* at 226–27 (citing Ex. 1 at 289, 352). However, Dr. Jamieson stated that she did not see anything in the Mayo Clinic records, as documented by Petitioner's treating neurologist, that Petitioner had either complaints or ambulatory difficulties on examination consistent with the degree of gait abnormality later documented on December 17th. *Id.* at 230–31.

When questioned about the EMG readings, Dr. Jamieson maintained that Petitioner met the criteria for CIDP and not GBS. She emphasized the abnormal temporal dispersions on Petitioner's November 16, 2020 EMG reading, with subsequent testing continuing to show abnormalities over time. Tr. at 232–34, 239 (citing Ex. 1 at 420; Van den Bergh at 6). Abnormal temporal dispersions can be indicative of demyelination if they are shown to be above 30% duration increase between proximal and distal negative peak CMAP amplitude in two or more nerves. *Id.* at 236; Van den Bergh at 6. In this case, however, the literal EMG findings had not been produced, requiring reliance on the conclusions of the treater who performed the EMG. Tr. at 236.

Dr. Jamieson admitted that subsequent EMG results showed the Petitioner's motor nerve action potentials were improving, but she maintained that they also continued to show abnormalities consistent with sensory nerve issues years after symptom onset. Tr. at 239–41. The fact that the EMGs did not normalize was otherwise expected with CIDP, Dr. Jamieson opined. *Id.* at 241. Dr. Jamieson did concede that the EMG results on April 5, 2024, showed improvement and a halt in active demyelination, but this had occurred over *three years* after onset, and thus could not in her opinion reflect the results for a patient with severe axonal GBS. *Id.* at 243.

2. Dr. William Hawse – Dr. Hawse is an academic immunologist who submitted one expert report in this case and testified at trial on behalf of the Respondent. Report, dated Sep. 25, 2023, filed as Ex. B (ECF No. 31-1) ("Hawse Rep."). Dr. Hawse opined that the Hep. B vaccine could not have caused Petitioner's injury.

Dr. Hawse is an Assistant Professor in the Department of Immunology at the University of Pittsburgh School of Medicine. See Curriculum Vitae, filed as Ex. B-12 (ECF No. 31-13) ("Hawse CV") at 1. He earned his Ph.D. in Biophysical Chemistry at Johns Hopkins and currently runs a laboratory studying CD4+ T-cell generation and immune tolerance to inform therapeutic strategies for autoimmune diseases. Hawse Rep. at 1. Dr. Hawse has published multiple peer-reviewed articles on the subject. Hawse CV at 2–4. He is not, however, a medical doctor or experimental clinician, and thus does not offer commentary on diagnosis. *See id.*

Dr. Hawse's testimony focused on causation. Petitioner's injury—whether CIDP or GBS—was not likely caused by the Hep. B vaccine. CIDP is a chronic disease, which lends itself to not being a vaccine-induced injury because the viral load administered in a vaccine dissipates with time, and the administered vaccine would be gone from the body within three years. Tr. at 270–71.

If there were an association between the Hep. B vaccine and CIDP or GBS, Dr. Hawse maintained, there would be epidemiologic evidence of a signal between the two, comparable to the signal connecting the 1970s flu vaccine GBS. Tr. at 272–73. But Petitioner had failed to provide any epidemiologic or convincing evidence of a causal relationship between the Hep. B vaccine and his injury. *Id.* at 274–80. Rather, Petitioner put forward retrospective studies, studies using VAERS[8] data, and case reports—all of which Dr. Hawse deemed weak causation proof. *Id. See, e.g.,* Chin; Vital. The most relevant epidemiologic evidence specific to the Hep. B vaccine in this case was 2013 Baxter—a case-centered study that allowed focus on transient effects that have a duration window after receiving a vaccine. *Id.* at 274–75. Its authors also took preventative

[8] VAERS is the Vaccine Adverse Event Reporting System, a database maintained by the Centers for Disease Control. VAERS collects information about adverse events that occur after the administration of licensed vaccines in the U.S. See About VAERS, Vaccine Adverse Event Reporting System (VAERS), https://vaers.hhs.gov/about/index (last visited October 31, 2025).

measures to remove bias from the study population. 2005 Baxter at 868. They ultimately concluded there was no causal association between the Hep. B vaccine and GBS. *Id.* at 276; 2013 Baxter at 200–01 (determining no causal association between vaccines, including the Hep. B vaccine, and GBS where only one patient received a Hep. B vaccine within six weeks of GBS symptom onset out of a study of four-hundred fifteen confirmed cases of GBS).

Dr. Hawse also maintained that Petitioner's proposed mechanisms of bystander activation or molecular mimicry could not credibly explain how the Hep. B vaccine could spark an autoimmune neuropathic condition. Tr. at 289–90. In his opinion, there was an absence of evidence demonstrating that bystander activation is a causative mechanism for developing an autoimmune pathology. *Id.* The same is true for molecular mimicry. *Id.* at 289. Neither Petitioner nor Dr. Latov had identified a component of the nerve axon that serves as a mimic with any antigenic component of the Hep. B vaccine. *Id.* at 282. At most, Dr. Latov did put forward a proposed mimic of MOG, with Bogdanos as support, but Dr. Hawse opined that this study was not indicative of a molecular mimic between humans and the Hep. B vaccine because no patients looked at in the study developed demyelinating disorders after vaccination. *Id.* at 284–86 (discussing Bogdanos).

Briefly discussing specific causation, Dr. Hawse proposed that the Hep. B vaccine was likely not the trigger for Petitioner's injuries. Tr. at 292. He could identify no record medical evidence that would explain why the Petitioner did not experience an adverse reaction to his first dose. *Id.* at 291. Dr. Hawse also acknowledged that there were alternative causes for Petitioner's injury, such as his Crohn's disease, which likely increased Petitioner's levels of pro-inflammatory cytokines and contributed to systematic inflammation more so than a vaccine would. *Id.* at 291–93.

On cross, Dr. Hawse acknowledged that molecular mimicry is a reliable scientific mechanism in certain cases, like how a *C. Jejuni* infection causes GBS. Tr. at 294–95. When asked about whether molecular mimicry was the mechanism that caused GBS from influenza vaccines, Dr. Hawse was unsure, but rejected comparing the flu vaccine to the Hep. B vaccine. *Id*. at 295–96 He conceded it was possible, but there was no evidence to suggest it was *plausible*. *Id.* at 300–01. In that same vein, Dr. Hawse noted that a genetic susceptibility to GBS is possible, as well as other factors that could lead to the development or susceptibility to GBS (although he did not deem these factors germane to the case). *Id.* at 301–02. Dr. Hawse also agreed that the 2012 IOM Report had stated that subgroup analysis or focused epidemiologic studies may miss rare, adverse events, and that no epidemiologic studies have looked at an association between the Hep. B vaccine causing GBS. Tr. at 304–306. But Dr. Hawse also noted that the 2012 IOM Report did not find any indication that the Hep. B vaccine can cause GBS or CIDP. Tr. at 307.

III. Procedural History

As noted, this matter was initiated in April 2022. After Respondent filed his Rule 4(c) Report opposing entitlement, the parties began the process of obtaining expert opinions in support of their respective positions, with the final such report filed on the eve of hearing in the early winter of 2025. I set the matter for hearing in February 2025, and the case went to trial as scheduled. The claim is now ripe for resolution.

V. 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 ex rel. 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).[9] There is no Table claim for GBS or CIDP caused by the Hep. B vaccine.

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). 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 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).

[9] 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).

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*, 418 F.3d at 1278: "(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 even 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 v. Sec'y of Health & Hum. Servs*, 121 Fed. Cl. 230, 245 (2015), *vacated and remanded*, 844 F.3d 1363 (Fed. Cir. 2017).

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 Cerrone v. Sec'y of Health & Hum. Servs.*, 146 F.4th 1113, 1121 (Fed. Cir. 2025) (arguing that *Althen* prong one requires only a showing of plausibility "understates the burden [a petitioner] bears under the first factor in the *Althen* formulation"); *Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *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"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). 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 Dept. 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 den'd*, 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. den'd 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. den'd* (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) (determining that 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).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *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*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to 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 v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often 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 v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (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.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) 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. *La Londe 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 Pharm., 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). Under *Daubert*, the 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).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases 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 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. 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 review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 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").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor 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.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 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. App'x 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. Overview of GBS, CIDP, and Their Treatment in Prior Program Cases

GBS

GBS has been defined as an acute, monophasic peripheral neuropathy involving rapidly-progressive and ascending weakness and paralysis, and which is thought to have an autoimmune mechanism. Monohan & Brannagan III at 122. It typically presents with weakness in proximal and distal muscles, sensory loss in extremities, loss of deep tendon reflexes. *Id.*; Asbury & Cornblath at S21. Cranial nerve involvement, autonomic dysfunction, respiratory weakness, and pain may be associated as well. Monohan & Brannagan III at 122. Autonomic involvement can involve evidence of cardiac arrhythmia, urinary retention, and unstable blood pressure. A. Walling & G. Dickson, *Guillain-Barre Syndrome*, 87 Am. Family Physician 191, 193 (2013), filed as Ex. 50 (ECF No. 55-1) ("Walling & Dickson"). But symptoms like fever, malaise, respiratory, or gastrointestinal symptoms tend to subside before neuropathic symptoms set in. Asbury & Cornblath at S23.

Axonal GBS is a rare form of GBS that is characterized by axonal loss rather than nerve surface demyelination. Asbury & Cornblath at S23; Walling & Dickson at 193. Axonal loss affects motor function and presents as muscle weakness. Asbury & Cornblath at S23; Walling & Dickson at 193. On EMG tests, evidence of axonal GBS can be seen by reduced amplitude but relatively preserved conduction velocities. Asbury & Cornblath at S23. AIDP, on the other hand, is

characterized by demyelination which results in sensory symptoms, such as paresthesia in the feet and hands. Walling & Dickson at 193. Compared to axonal GBS, EMG results for demyelinating forms of GBS show severe reduced conduction velocity or full conduction blockage. Asbury & Cornblath at S23–S24.

GBS can be vaccine-caused, specifically by the flu vaccine (although the risk from wild flu *infection* is much greater). *See* Walling & Dickson at 191–92. Consistent with this, a large body of reasoned Program decisions[10] recognize an association between the flu vaccine and GBS (as well as other related peripheral neuropathies). Indeed, there is a Table claim for GBS due to receipt of a flu vaccine. 42 C.F.R. § 100.3.14. This means the Government accepts that sufficiently-probative and reliable science on the topic exists to justify conceding causation, at least for Program purposes. *Haskins v. Secretary of Health & Hum. Servs.*, No. 18-1776V, WL 2020 1870279 (Fed. Cl. Spec. Mstr. Mar. 13, 2019). Even in cases where a Table element for such a claim cannot be met (for example, when onset is too short or too long to fit within the timeframe of 3–42 days set for the claim), any subsequent causation-in-fact analysis performed by the special masters rarely requires the claimant to offer proof in support of the first *Althen* prong, "can cause" element; instead, it is reasonably assumed to be satisfied already. *See Welch v. Sec'y of Health & Hum. Servs*. No. 18-494V, 2019 WL 349360 (Fed. Cl. Spec. Mstr. July 2, 2019).

Other vaccines have also been found causal of GBS, although there is disagreement among the special masters as to the preponderant strength of these proposed associations. *See generally Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075, 2022 WL 9669651, at *36–37 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (finding the pneumococcal vaccine caused GBS); *but see Dennington v. Sec'y of Health & Hum. Servs.,* No. 18-1303V, 2023 WL 2965239 (Fed. Cl. Spec. Mstr. Apr. 17, 2023) (Tdap vaccine not shown to be causal of GBS), *mot. for review den'd*, 167 Fed. Cl. 640 (2023), *appeal dismissed*, No. 2024-1214, 2024 WL 1255318 (Fed. Cir. Mar. 25, 2024); *Gatto v. Sec'y of Health & Hum. Servs.,* No. 21-924V, 2025 WL 1235088, at *1 (Fed. Cl. Mar. 31, 2025) (meningococcal vaccine not causal of GBS); *Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at *30 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review den'd*, 167 Fed. Cl. 127 (2023) (pneumococcal vaccine was not shown to cause GBS); *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *3 (Fed. Cl. Spec. Mstr. Dec. 9, 2022) (same). It thus cannot be said that the Program has developed a consistent view as to what the science preponderantly "says" about GBS vaccine causation when the flu vaccine is not involved. Instead, it appears that the outcome in such cases is mostly a function of the evidence before the special master (along with the special master's individual view about the applicability of causation theories to different vaccines), with no clear trend one way or the other.

[10] Although prior decisions from different cases do not control the outcome herein, special masters may reasonably take into account, for guidance, the logic of such reasoned determinations. In fact, it is wise to do so, given how often similar causation theories or fact patterns arise in Vaccine Program cases.

At the same time, I acknowledge there exists Program support for the conclusion that the Hep. B vaccine can cause demyelinating conditions. *See, e.g.*, *Osso v. Sec'y of Health & Hum. Servs*., No. 18-575V, 2023 WL 5016473, (Fed. Cl. Spec. Mstr. July 13, 2013); *Stevens v. Sec'y of Health & Hum. Servs.,* No. 99-594V, 2006 WL 659525, at *1–3 (Fed. Cl. Spec. Mstr. Feb. 24, 2006). Indeed, on October 13-14, 2004, former Special Master (and later Chief Judge of the Court of Federal Claims) Margaret Sweeney held a hearing—which became known as the "Hepatitis B— Neurological Demyelinating Omnibus Proceeding"—to determine whether a causal association exists between the Hep. B vaccine and several demyelinating illnesses, including GBS. *Stevens,* 2006 WL 659525; *Werderitsh v. Sec'y of Dept. of Health & Hum. Servs.,* No. 99-638V, 2006 WL 1672884 (Fed. Cl. Spec. Mstr. May 26, 2006); *Peugh v. Sec'y of Dept. of Health & Hum. Servs.,* No. 99-319V, 2007 WL 1531666 (Fed. Cl. Spec. Mstr. May 8, 2007); *Gilbert v. Sec'y of Dept. of Health & Hum. Servs.,* No. 04-455V, 2006 WL 1006612 (Fed. Cl. Spec. Mstr. Mar. 30, 2006). These cases were then reassigned to former Special Master Laura Millman, who found that in all four cases, causation was established. *Peugh*, 2007 WL 1006612, at *1, 17–18. While these matters are not binding on the outcome herein, I do not totally disregard them either.

CIDP

CIDP is an immune-mediated demyelinating neuropathy that affects both large and small fiber peripheral nerves, resulting in symptoms of numbness, tingling, weakness, imbalance, loss of coordination and pain. *See* Van den Bergh at 3559–60. CIDP shares similar characteristics to GBS—but it is not simply a chronic form of GBS, for there are key differences in their clinical presentation that distinguish the two, and they cannot be assumed to have the same pathogenic mechanisms. In particular, CIDP progresses over a longer period of time than GBS, which features acute weakness within one to two weeks after onset. *See* Van den Bergh at 3559. In addition, little is known about CIDP's most likely causes, triggers, or pathogenesis, in comparison to GBS (where a variety of specific infectious triggers have been identified, as well as the situs of cross-reactive autoimmune attack). L. Querol & C. Lleixa, *Novel Immunological and Therapeutic Insights in Guillain-Barre Syndrome and CIDP*, 18 Neurotherapeutics 2222, 2223 (2021), filed as Ex. 16 (ECF No. 52-2). Thus, although many Program decisions *seem to have assumed* that what is known about GBS applies fully to CIDP given their similarities, this assumption is not well founded.

Prior decisions have associated different vaccines with CIDP (more often than not the flu vaccine), and petitioners have settled many such cases on favorable terms.[11] *See Jastisan v. Sec'y*

[11] Of course, prior decisions from different cases do not control the outcome herein (and this goes double for cases that are settled, and hence resolved without a reasoned determination). *Boatmon*, 941 F.3d at 1358–59; *Hanlon*, 40 Fed. Cl. at 630. But special masters are empowered to 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.

of Health & Hum. Servs., No. 13-937V, 2016 WL 4761950 (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 *some* consideration as persuasive guidance. *See, e.g., Mason v. Sec'y of Health & Hum. Servs.,* No. 17-1383V, 2022 WL 600415, at *21 (Fed. Cl. Spec. Mstr. Feb. 4, 2022); *Houston v. Sec'y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *16 (Fed. Cl. Aug. 19, 2021); *Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018). I have in many cases been able to find that when the *flu vaccine* is at issue, the reasoning applicable to the flu-GBS association (which is better substantiated than any other covered vaccine and GBS) can be reasonably extended to CIDP. *See Nieves v. Sec'y of Health & Hum. Servs*., No. 18-1602V, 2023 WL 3580148, at *45 (Fed. Cl. May 22, 2023), *mot. for review den'd*, 167 Fed. Cl. 422 (2023).

However, I have identified no recent *reasoned* decisions in which a special master explained how or why a vaccine other than the flu vaccine was likely causal of the claimant's CIDP. *See Houston*, 2021 WL 4259012, at *17–18 ("determining that the first *Althen* prong was not met as "[p]etitioner has made no showing comparable to what would be required to prove that [an] association [between Tdap and GBS] translates to an association between Tdap and CIDP"). And there are several decisions in the past ten years that suggest the strength of a vaccine association with CIDP is far weaker than what may have previously been presumed. 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 *7 (Fed. Cl. Spec. Mstr. Sept. 23, 2014). The special master highlighted an IOM report (among other things) which specifically found insufficient available evidence to support an association between influenza vaccine and CIDP. *Id.* at *14. I also have denied entitlement in cases alleging the Tdap vaccine caused CIDP. *See Dennington*, 2023 WL 2965239.

II. Petitioner Most Likely Experienced CIDP

A Special Master's function is not to diagnose vaccine related injuries, but to determine whether it has been shown that a vaccine caused the petitioner's injury by a preponderance of the evidence "based on the record evidence as a whole and the totality of the case. . . . " *Andreu*, 569 F.3d at 1382 (quoting *Knudsen*, 35 F.3d at 549). Nevertheless, in many cases determination of what diagnosis is best supported by the evidence bears on a case's resolution. *Broekelschen*, 618 F.3d at 1349.

Here, Petitioner understandably would rather be found to have experienced *some* form of GBS, given both the Program's generally-favorable treatment of that form of neuropathy as a vaccine injury, as well as the existence of prior (if now dated) decisions suggesting the Hep. B vaccine can cause GBS. Of course, the evidence in this case clearly *does not* support the conclusion

that Petitioner experienced AIDP—the most common form of GBS. But Dr. Latov has proposed Petitioner's GBS was slightly different: an axonal-oriented form, and featuring a longer course.

This attempt to cast Petitioner's injury as a "kind" of GBS is ultimately unsuccessful. Axonal GBS is also characterized by a monophasic course, with nadir occurring most often within four weeks of onset (consistent with AIDP—the "classic" form of GBS), and EMGs showing evidence of axonal damage predominating over demyelination. *See* Tr. at 61–64, 68; Monohan & Brannagan III at 122 ("The typical course of [GBS and GBS variants] is monophasic, with the clinical nadir between 2 and 4 weeks"). CIDP on the other hand, has an indolent course that can be marked with relapses, and has been closely linked to EMGs that demonstrate peripheral nerve demyelination. *See* Van den Bergh at 6.

Dr. Latov relied on the evidence of symptoms of sensory loss and weakness, progression of ambulatory difficulty, the opinion of treating providers, and aspects of Petitioner's EMG results for his diagnostic determination. But he only stated his embrace of axonal GBS as the proper diagnosis at hearing, somewhat reducing the persuasive value of this aspect of his opinion (since it is not clear why, if the evidence supports this diagnosis, he could not have attained that realization two years ago, at the time he prepared his initial expert report). Moreover, Petitioner's longer, intermittent temporal course here is less supportive of axonal GBS (which cannot be re-defined as another indolent form of GBS that is otherwise not CIDP). And Respondent made good arguments in favor of a CIDP diagnosis. Not merely treating providers, but also Drs. Jamieson and Latov, agreed that there was evidence of *demyelination* on the EMG, consistent with CIDP. Tr. at 63–64, 188–190; Dr. Latov distinguishes the demyelination as secondary, but Petitioner's treating providers at the Mayo Clinic and Dr. Jamieson agreed that the EMGs are indicative of a *demyelinating* peripheral neuropathy.

Thus, the mix of evidence and expert opinions is most supportive of CIDP as the likely injury Petitioner experienced—although even if it is assumed he *did* have axonal GBS, the *Althen* test was not successfully satisfied.

III. Petitioner Has Not Carried His *Althen* Burden of Proof[12]

A. *Petitioner Has Not Shown the Hep. B Vaccine "Did Cause" his Injury*

The record in this case does not support the conclusion that receipt of a dose of Hep. B vaccine on September 4, 2020, likely caused his neuropathic injury, however characterized. First,

[12] In keeping with the fact that entitlement requires that all three *Althen* prongs be met, I discuss herein only those prongs most relevant to my determination. *Dobrydnev v. Sec'y of Health & Hum. Servs*., 566 Fed. Appx. 976, 980 (Fed. Cir. 2014).

there is the fact that Petitioner experienced no reaction to vaccination—and not just the dose in question but the earlier one administered to him over one month prior. Indeed, even in the wake of the second dose, four to five weeks passed with no evidence of any kind of brewing issue—no sign of unusual inflammation or some other indicator of an adverse process underway. While there is no requirement that something adverse *must* occur in the immediate wake of a vaccination, a quiescent medical record post-vaccination does not aid a claimant in establishing the existence of an aberrant process at work that could help establish the vaccine "did cause" a subsequent injury.

The second, and even more convincing, evidentiary factor preventing a finding of specific causation is Petitioner's preexisting Crohn's disease and Humira treatments. The evidence offered in this case supports the idea that *either* Crohn's disease or Humira could result in this kind of neuropathy. Dr. Latov's own co-authored article acknowledged a likely connection between IBD-like conditions and acute and chronic we well as axonal and demyelinating peripheral neuropathies. *See* Tr. at 153, 214–15, 221–23; 2005 Gondim at 867, 868 ("[i]t is likely that there is a primary immune-mediated neuropathy as an extra-intestinal disorder associated with IBD and not merely a co-occurrence with CIDP"), 869. Similarly, there is evidence and testimony that peripheral neuropathy could be caused by Humira. *See, e.g.*¸ Natividade at 86; Gill at 75–76.

Moreover, the temporal association between Petitioner's Crohn's disease treatments and onset of neurologic symptoms was far more obvious than compared to a vaccine received five weeks before. Petitioner underwent Humira treatment for his Crohn's disease multiple times: once before his second Hep. B vaccine dose, and *thrice* after. Ex. 1 at 28, 428; 3 at 39; Ex. 9 at 2. Petitioner's last three Humira treatments occurred 30, 17, and 2 days, respectfully, before the onset of his peripheral neuropathy symptoms. *See id.* These treatments occurred much closer in time to his onset than his vaccination. *See id.*; *see also,* Ex. 6 at 2.

What of the possibility that the Hep. B vaccine *and* Petitioner's Crohn's disease, plus Humira treatments, interacted synergistically? The record does not substantiate that possibility. There is certainly no evidence in September 2020, and in the wake of receipt of the second Hep. B dose, that any aberrant immune response (for example, an increase in inflammation mediated by the combination of causal factors) was occurring. And although the exact mechanism for a neuropathy due to IBD or Humira is unknown, the connection between them is better supported than a connection between peripheral neuropathy and the Hep. B vaccine (even with the existence of some favorable Program decisions). *See generally* 2005 Gondim; 2013 Baxter. Regardless of the mechanism, insufficient evidence was offered in this case that either IBD or Humira would require additional immune stimulation (whether from infection or vaccination) to cause a peripheral neuropathy.[13]

[13] This case does not present circumstances appropriate for a "*Shyface*" analysis either. *Shyface,* 39 Fed. Cl. at 433. Under such an analytic framework (and given that a claimant need only ever prove a vaccine was a substantial factor in causing an injury), causation can be found even if the vaccine was not the sole cause – and thus even if other causal explanations are clearly present that also may have been causal. Here, however, Respondent does not concede prong

Third, there is a lack of treater support for vaccine causation. Treating provider opinions continuously reference Petitioner's Crohn's disease or Humira in conjunction with his injuries, not the Hep. B vaccine. Dr. Kass, for example, noted that Petitioner's numbness was "a possible side effect of Humira, but other possible factors need[ed] to be considered," and held off on continuing Petitioner's Humira treatment since neurologic symptoms started close in time after a treatment. Ex. 3 at 26, 39. Dr. Loftus also referenced that Petitioner had developed paresthesia "in the setting of Crohn's," adding that it was unclear whether his neurological symptoms were related to Humira. Ex. 1 at 350–51. Both Drs. Zandieh and Kissoon observed the possibility of a connection between peripheral neuropathy and autoimmune disease, and suggested that Petitioner's Crohn's disease and Humira use may have put him at an increased risk for developing a demyelinating neuropathy, consistent with Dr. Jamieson's testimony. Ex. 3 at 398–99; Tr. at 256–57.

In contrast, only Dr. Shouman speculated that the Hep. B vaccine could have been the cause of Petitioner's neuropathy, advising him not to receive the third dose. Ex. 1 at 140–142; Ex. 9 at 2. But this view was in the minority. Although not sacrosanct, statements from treating physicians can be probative in weighing causation evidence. 42 U.S.C. § 300aa-13(b)(1) (statements of treating physicians are not binding on special masters); *Snyder*, 88 Fed. Cl. at 746 n.67 (2009). While it is not the determining factor for my decision, the totality of treater views are unsupportive of vaccine causation—and thus they add further support for a finding that Petitioner has not satisfied the second *Althen* prong.

B. *Petitioner Has Not Shown His Injury Began in a Medically-Acceptable Timeframe*

Petitioner's onset timing has also not been shown to be medically acceptable, measured from the date of vaccination. No direct evidence was offered for how long it would take for an autoimmune process instigated by receipt of a Hep. B vaccine to lead to neuropathic injury, let alone whether five weeks/34 to 35 days is reasonable. Dr. Latov relied on articles like Schonberger, which is not only somewhat outdated at this point but involved the flu vaccine, and thus cannot simply be slotted into the context of a different vaccine. And the case reports filed in this matter specific to the Hep. B vaccine involved shorter timeframes as well. *See, e.g.,* Vital at 163–64 (discussing two cases where sensory disturbances appeared fifteen and twenty-one days respectively after receiving a Hep. B vaccination); Khamaisi at 768 tbl. I (noting nine out of twenty patients with GBS after a Hep. B vaccine experienced symptom onset within a month of vaccination); Muller at 509 (onset of GBS occurred nine days after receiving the Hep. B vaccine). A 37-day post-vaccination onset (from the September 4, 2020 vaccination to the October 11, 2020 onset) is weakly (at best) supported by these case reports.

---

one causation – and hence it cannot be assumed the Hep. B vaccine "can cause" axonal GBS. And there are too many reasons to doubt the vaccine played a role in Petitioner's injury (even if it is assumed it *could* cause a neuropathy).

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such a 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.[14]

IT IS SO ORDERED.

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

[14] 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.