

in-fact claim based on the CIDP diagnosis (although Respondent contested its accuracy)—and to that end, the parties have submitted a number of expert reports and briefs.

Now, and for the reasons stated below, I hereby deny entitlement. The overall record preponderantly supports the CIDP diagnosis. But the record and expert reports do *not* also support the finding that the flu vaccine caused Petitioner’s injuries, *or* (and most importantly) that Petitioner’s onset of neurologic symptoms occurred in a medically acceptable timeframe when measured from the date of vaccination.

I. Factual Background and Medical History

Pre-Vaccination Medical History

Before Mr. Nieves ever received the flu vaccine that is the basis for his claim, he had repeatedly visited healthcare professionals over many years, complaining of symptoms that to some extent echo some of his post-vaccination maladies. Mr. Nieves has a past medical history significant for sinusitis, fibromyalgia rheumatica with myositis, obesity, status post-lap band surgery, anxiety, cervical spinal stenosis with related radiculopathy, muscle spasms, and right carpal tunnel syndrome. Ex. 2 at 2–4; Ex. 3 at 3–121; Ex. 4 at 1–222; Ex. 7 at 1–22. He had been prescribed neuropathic pain relief medication for some of his symptoms, but it provided little relief. Ex. 2 at 29.

In 2013, Mr. Nieves saw a neurosurgeon with complaints of pain radiating to both shoulders and arms with sensations of heaviness, weakness, numbness, and tingling. Ex. 2 at 29–30. This was associated with some difficulty buttoning shirts, tying shoes, and accompanied by frequent falling. *Id.* In addition, he reported limb weakness, pain (neck and back), and other neurologic-like symptoms (arm and leg weakness, numbness, and difficulty walking) in late 2014. Ex. 4 at 12–15. Cervical decompression surgery was planned. *Id.* at 15; *see also id.* at 114 (operative report for Dec. 10, 2014 surgery).

Even in the nine-plus months prior to vaccination, Petitioner continued to display some symptoms echoing what he claims occurred post-vaccination. Thus, in February 2015, Petitioner had a follow-up visit after his cervical spine surgery, and at that time it was recorded that he had been prescribed medication for fibromyalgia, although the Petitioner at this time denied the diagnosis, maintaining instead he merely experienced muscle spasms. Ex. 3 at 97–98.

October 2015 Vaccination and Subsequent Medical History

On October 28, 2015, Mr. Nieves (then 57 years old) received a seasonal flu vaccine in his right arm. Ex. 1 at 10. That same day, he alleges, he began feeling “feverish and a little achy.” Affidavit, dated November 16, 2020, filed as Ex. 19 (ECF No. 41-2) (“Nieves Aff.”) at 2. The next

day (October 29, 2015),³ Mr. Nieves recalls “waking up with general malaise and flu like symptoms with some aches and pains all over [] [his] body.” *Id.* He presented to his primary care physician that day with complaints of a mild allergic response four hours after receiving the flu shot, representing that it had resolved but that he was experiencing “generalized arthralgias without arthritis.” Ex. 5 at 2. Petitioner otherwise reported “[f]eeling generally well,” however, and his exam revealed no specific or acute neurologic issues this time. *Id.* He was advised to take ibuprofen and a muscle spasm relaxant/sedative, with follow-up as needed. *Id.*

Mr. Nieves alleges that a bit more than three days after the vaccination (or by the late evening of October 31st), he began “feeling strange,” and noticed that his “walking was a little different.” Nieves Aff. at 2. He also recalls that he began losing some sensation in his hands and feet. *Id.* He experienced more sensory issues and tingling/numbness in his hands and feet the next day (November 1, 2015). *Id.* These symptoms are arguably initial manifestations of a neurologic problem. (An entry in Mr. Nieves’s affidavit recounting his condition as of November 4, 2015, also expresses his suspicion that the back pain he later began to feel was attributable to an altered gait, which in turn began “when I started feeling paresthesia and loss of sensation in my feet dating back to *November 1.*” Nieves Aff. at 3 (emphasis added).

On November 2, 2015, Mr. Nieves reported to the emergency room with complaints of fatigue, leg pain, and lower back pain, informing treaters that his symptoms had existed for five days (which, if correct, meant the vaccination date (October 28th) was their start). Ex. 3 at 89–90. He also noted difficulty walking due to his symptoms. *Id.* at 91. (Petitioner’s affidavit also states that as of this date he was having a hard time walking, and felt like he was losing sensation when “grabbing and holding things.” Nieves Aff. at 2). A neurologic exam at this time did reveal weakness in Petitioner’s extremities, but Petitioner was ultimately discharged. *Id.* at 92, 97.

The next day (November 3, 2015), Petitioner was seen by neurologist Dr. Anna Marieta Moise. Ex. 3 at 83–87. He reported at this time “paresthesias in his distal extremities since he got his flu shot one week ago.” *Id.* at 84 (emphasis added). One week would approximately place onset on October 27–28th (or right around the day of vaccination). He also stated that he was having difficulty walking/balance issues, plus a headache predating vaccination. *Id.* The record from this visit includes the notation “fibromyalgia?” in its brief medical history recitation but no comment on the term is provided. *Id.* Dr. Moise’s exam showed normal strength and reflexes, however, and only “subjective decreased sensation,” plus a normal gait. *Id.* at 86.

Based on the exam and Petitioner’s presentation, Dr. Moise opined that his paresthesias were most likely due to nutritional deficiencies connected to Petitioner’s lap-band surgery earlier that year—but “not consistent with AIDP [the most common GBS variant]⁴ given normal strength

³ Petitioner’s affidavit appears to shift from reporting dates in 2015 to 2016, within the same month. *See* Nieves Aff. at 1. But the 2016 references are very likely merely a typographical error.

⁴ AIDP stands for “acute inflammatory demyelinating polyradiculoneuropathy.”

and normal reflexes.” Ex. 3 at 86. She proposed only that Petitioner receive vitamin/nutritional supplements. *Id.* Another treater (Dr. Ethelyn Johnson) saw Petitioner on November 3, 2015, and Petitioner informed her as well that he had experienced “multiple complaints since receiving flu shot 1 week ago,” including walking issues, limb weakness, and paresthesias. *Id.* at 87.⁵ This treater’s impression expressed doubt as to the propriety of a GBS diagnosis, since Petitioner displayed “good strength on exam, gait minimally impaired, reflexes +1 bilat.” *Id.* at 89.

On November 4, 2015, Mr. Nieves returned to the emergency room and was again seen by Dr. Johnson. Ex. 3 at 76–78. He now reported worsening back pain, generalized weakness, and ongoing difficulty walking, comparable to his complaints from the day before. *Id.* at 76. However, the exam revealed normal results. *Id.* at 77. Petitioner was assessed with pain, given medications for treatment, and referred to a pain clinic for additional follow-up. *Id.* Less than a week later (November 9, 2015), Mr. Nieves again returned to the emergency room, reporting worsening low back pain radiating up to his neck and shoulders and down to his lower extremities. Ex. 3 at 67. However, he denied “weakness, numbness, or tingling in the lower extremity.” *Id.* at 67–68. He was assessed with chronic back pain—and it was again proposed that his condition was not likely reflective of a primary neurological problem, whether GBS or cord compression. *Id.*

On November 10, 2015, Mr. Nieves returned to his primary care physician, now complaining of weakness, fatigue, and generalized muscle pain he claimed had manifested after receipt of the flu vaccine. Ex. 5 at 1. He denied any ascending/progressive weakness at this time, however. *Id.* An exam revealed normal strength and reflexes, and hence no neurologic issues, although his gait was classified as “slow.” Petitioner was nevertheless referred to a hospital emergency department for further evaluation and neurology care. *Id.* at 7.

Later that day, Mr. Nieves was admitted to Methodist Hospital in San Antonio, Texas, where he remained an in-patient for ten days. Ex. 3 at 65; Ex. 9 at 16–18. His history (consistent with what is reviewed above) noted fever and present myalgias “[s]ince the flu shot,” with “progressive tingling, numbness,” falls, weakness, and fatigue. *Id.* at 16. At this point, “some concern for Guillain-Barre” was expressed by a treater. Ex. 9 at 16, 17. A neurologic evaluation at the hospital revealed some weakness and areflexia, plus ascending numbness. *Id.* at 16. In addition, a lumbar puncture showed mildly elevated protein levels, and based on the foregoing Mr. Nieves was diagnosed with GBS and treated with IVIG.⁶ *Id.*

In the course of these treatments, Petitioner experienced some improvement, and he was discharged 10 days later, on November 20, 2015, with a treatment plan to continue physical therapy

⁵ This record specifically observes that Petitioner’s history referenced fibromyalgia, but that Mr. Nieves disputed the diagnosis. Ex. 3 at 87.

⁶ “Intravenous Immunoglobulin” is defined as “a therapy treatment for patients with antibody deficiencies. It is prepared from a pool of immunoglobulins (antibodies) from the plasma of thousands of healthy donors.” *Intravenous Immunoglobulin*, American College of Rheumatology, <https://www.rheumatology.org/I-Am-A/Patient-Caregiver/Treatments/Intravenous-Immunoglobulin-IVIG> (last visited Apr. 17, 2023).

(“PT”) and occupational therapy (“OT”). Ex. 3 at 65. Diagnosis at discharge remained GBS. Ex. 6 at 107–117; 122–125; 150–154. Petitioner began PT that same late fall, but did not find it to result in objective improvement, although his weakness subsided a bit. Ex. 3 at 48, 50; Ex. 6 at 35, 80.

Subsequent Symptoms and CIDP Diagnosis

Two months later, on January 8, 2016, Mr. Nieves was seen by neurologist Dr. Adetoun Musa. Ex. 3 at 44–48. Mr. Nieves indicated he was experiencing worsening paresthesias, balance issues, and that his weakness had not improved with PT/OT, or since his prior IVIG treatment in November the prior year. *Id.* at 45. Dr. Musa’s exam revealed some weakness and sensory issues, but normal reflexes, and he ultimately opined that Petitioner had the AIDP GBS variant—although given the prolonged duration of symptoms, Dr. Musa also proposed CIDP as a potential counter-explanation. *Id.* at 47. He noted the need for Petitioner to undergo an EMG.⁷ *Id.* at 48.

The scheduled EMG testing occurred on February 8, 2016, and it revealed the presence of generalized sensorimotor polyneuropathy, predominately demyelinating in type and mild in degree. Ex. 3 at 43. These findings were deemed to be consistent with an acquired segmental demyelinating polyneuropathy, like that seen in CIDP and related disorders. *Id.* On February 11, 2016, Mr. Nieves returned to Dr. Musa who agreed the EMG results were confirmatory of CIDP, and proposed that Petitioner undergo another round of IVIG. *Id.* at 38–42.

Subsequent History and Questions About Diagnosis

Not long after the February consultation with Dr. Musa, Petitioner returned again to the hospital emergency room on February 16, 2016, now complaining of weakness in all extremities and back pain that “shoots down his lower extremities with pressure or movement.” Ex. 3 at 28. His exam revealed largely normal reflexes, but weakness deemed secondary to pain, and he was admitted with CIDP and received additional IVIG treatment. Mr. Nieves was thereafter discharged, with a plan to continue outpatient treatment at the infusion clinic. *Id.* at 25–38; Ex. 11 at 1–17. In March, Petitioner went back to Dr. Musa, and although he complained of generalized neuropathic pain, his exam was again mostly normal. Dr. Musa repeated his prior impression of CIDP, and proposed a treatment plan including recurring monthly IVIG infusions for six months plus a repeat EMG/NCS at that time. Ex. 3 at 15–18; Ex. 8 at 76–80.

By mid-April 2016, Petitioner had completed an IVIG treatment, but found that its benefits wore off after a few weeks, leaving him with painful muscle spasms and cramps involving his

⁷ “Electromyography” 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; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals. *Electromyography*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography> (last visited Apr. 17, 2023).

neck, shoulders, and lower back. Ex. 8 at 59–62. In May, he started a second round of PT. Although he struggled with increased pain during the sessions, his therapist proposed that his reaction might reflect an “elevated pain response” to the activity itself. *Id.* at 47. Ultimately, the chronic muscle spasms and cramps Petitioner experienced while active prevented his successful utilization of PT, and he was eventually discharged due to lack of progress. *Id.* at 18–30; Ex. 11 at 69–98.

In September of that same year, Petitioner now reported to a different set of neurologic treaters that despite six months of treatment, he did not deem the IVIG to be helpful. Ex. 11 at 67. On September 20, 2016, petitioner underwent a second EMG, as planned by Dr. Musa in March. But this study now showed no evidence of an acquired segmental demyelinating polyneuropathy, inconsistent with what would be expected for a chronic and ongoing neuropathic condition like CIDP. Ex. 11 at 63 (“NO evidence of an acquired segmental demyelinating polyneuropathy like that can be seen in chronic inflammatory demyelinating polyradiculopathy (CIDP) and related disorders”). In addition, the EMG revealed bilateral median neuropathy, or carpal tunnel syndrome. *Id.* at 63–65. A third EMG performed in December 2016 was also inconsistent with CIDP, while confirming the presence of a mild case of carpal tunnel syndrome. Ex. 11 at 63.

On December 21, 2016, petitioner saw neurologist Dr. Nidhi Kasatwar, complaining of “continued cramps in his hands and legs and also his abdomen with fluttering of muscles.” Ex. 11 at 61. Examination showed fairly normal strength, however (4/5 on his right side, likely limited due to pain, and 5/5 on the left), with normal sensation and reflexes, although Petitioner displayed an abnormal gait and required a cane for ambulation. *Id.* at 62. Dr. Kasatwar took note of the recent EMG that had been negative for CIDP (while supportive of a carpal tunnel diagnosis), and settled on an impression focusing on Petitioner’s history of cervical radiculopathy, obesity, and fibromyalgia, with continued cramps and muscle pain. It was now proposed that Petitioner cease the IVIG infusions, and instead seek primary care treatment for chronic pain. *Id.*

Limited records have been offered detailing Petitioner’s subsequent course. For example, after this case’s initiation, Petitioner visited yet another neurologist, Dr. Juan Bahamon, in August 2019, reporting to Dr. Bahamon that he had experienced CIDP (although the record from this visit notes that Dr. Bahamon lacked “independent medical records” to corroborate what he was told about Petitioner’s history). Ex. 14 at 1. Dr. Bahamon expressed uncertainty as to whether “we are dealing with an incomplete recovery of a monophasic [GBS],” but expressed doubt about CIDP given an exam revealing the existence of deep tendon reflexes.” *Id.* at 7.

II. Expert Reports

A. Petitioner’s Experts

1. *Marcel Kinsbourne, M.D.*

Dr. Kinsbourne, a pediatric neurologist, submitted three written reports addressing both the proper diagnosis for Mr. Nieves’s condition as well as the underlying causation dispute. *See*

generally Report, dated Jan. 27, 2020, filed as Ex. 17 (ECF No. 23-2) (“Kinsbourne Rep.”); Report, dated Nov. 14, 2020, filed as Ex. 20 (ECF No. 41-3) (“Second Kinsbourne Rep.”); Report, dated Dec. 4, 2021, filed as Ex. 26 (ECF No. 55-1) (“Third Kinsbourne Rep.”).

Dr. Kinsbourne received his medical degree from Oxford University in England, along with his Bachelor of Arts, and his Master of Arts. *See* Kinsbourne Rep. at 1.⁸ He then received his M.D. from the State of North Carolina. *Id.* Thereafter, Dr. Kinsbourne did several years of different post-doctoral training in neurology, pediatrics, and chest diseases, and is a member of the American Board of Pediatrics and Royal College of Physicians. *Id.* at 1–2. Dr. Kinsbourne was previously a professor of psychology, professor of pediatrics, lecturer in neurology, adjunct professor of linguistics and cognitive science, adjunct professor of occupational therapy, director of the behavioral neurology department at the Eunice Kennedy Shriver Center, and other positions related to neurologic and cognitive studies. *Id.* at 2–3. He also has held positions on several editorial boards, professional societies, and administrative assignments. *Id.* at 4–6. Dr. Kinsbourne has conducted research into pediatric disorders, developmental delays and factors, cerebral deficiencies, learning disabilities, therapies, and epilepsy. *Id.* at 6–39. Importantly, however, Dr. Kinsbourne has not treated patients, pediatric or otherwise, for almost thirty years.⁹

First Report

Dr. Kinsbourne began with an overview of Mr. Nieves’s medical history. *See generally* Kinsbourne Rep. at 2–5. Significantly, he observed in the record multiple instances in which (despite initial concerns for GBS) treaters interpreted test results, coupled with Petitioner’s persistent symptoms, to be consistent with CIDP. *Id.* at 5. Petitioner’s CIDP eventually “stalled,” but left him with motor/sensory limits as well as chronic pain. *Id.*

Dr. Kinsbourne deemed the CIDP diagnosis consistent with the record evidence. In particular, he emphasized that CIDP could exist even in the presence of active/normal reflexes, noting that a number of articles revealed that to be common, and that Petitioner’s treaters had deemed other testing consistent with some form of polyneuropathy despite an absence of areflexia. Kinsbourne Rep. at 5. He also suggested that Petitioner’s relatively-quick onset and initial symptoms (which misled treaters to deem GBS a possibility) might reflect an acute onset form of CIDP). *Id.* at 6. Indeed, Dr. Kinsbourne embraced the view that CIDP is “closely similar” to GBS, mainly distinguishable by the former’s chronic nature. *Id.* at 6–7; M. Dalakas, *Pathophysiology of Autoimmune Polyneuropathies*, 42 *Presse Med.* e181, e182 (2013), filed as Ex. 17 Ref. 3 (ECF No. 24-1) (“Dalakas”). Otherwise, CIDP and GBS share the same immunologic mechanisms and

⁸ Although Petitioner purported to file a separate CV for Dr. Kinsbourne (*see* Ex. 16 (ECF No. 23-1)), the document so labeled was simply an extra copy of his expert report—and it does not appear Petitioner ever subsequently filed a CV for Dr. Kinsbourne. But the first report begins with a paragraph labeled “qualifications,” and I therefore refer to it for this information about his education and professional experience

⁹ *See L.M. v. Sec’y of Health & Hum. Servs.*, No. 14-714V, 2019 WL 4072130 (Fed. Cl. Spec. Mstr. July 23, 2019) (discussing Dr. Kinsbourne’s more recent practice experience).

many clinical features—and the fact that less is known about triggering agents for CIDP could simply be the product of the fact that “onset of CIDP is usually far in the past and easily forgotten,” whereas GBS is acute and monophasic in course. Kinsbourne Rep. at 7.

Next, Dr. Kinsbourne attempted to grapple with a record that was (as Respondent’s experts argue) in some ways inconsistent with CIDP, proposing that Petitioner had experienced a particular CIDP variant, characterized by an acute onset but manifesting mostly “toward sensory deficit.” Kinsbourne Rep. at 7. Mr. Nieves did not display weakness *per se*, but instead a hesitancy “to exert and to maintain full muscular effort” given the pain he was experiencing—as evidence by his inability to complete physical therapy. *Id.* Thus, Dr. Kinsbourne explained instances (such as at an August 2019 neurologic visit) when Petitioner displayed “giveaway weakness”¹⁰ as reflecting Petitioner’s involuntary unwillingness to engage in painful exertions. *Id.*; Ex. 14 at 1–7.

Dr. Kinsbourne cited several items of literature that he maintained recognized a “pure sensory” CIDP variant. *See, e.g.,* S. Oh et al., *Chronic Sensory Demyelinating Neuropathy: Chronic Inflammatory Demyelinating Polyneuropathy Presenting as a Pure Sensory Neuropathy*, 55 J. Neurol., Neurosurg. and Psych. 677 (1992), filed as Ex. 17 Ref. 14 (ECF 26-4) (discussing ten relevant patient cases). He also noted that pain was a common, “long-term residual symptom,” in addition to CIDP’s other symptom features (although he mainly supported this contention with reference to case series reports). *See, e.g.,* S. Boukhris et al., *Pain as the Presenting Symptom of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Study of 11 Cases*, 10 J. Periph. Nerv. Syst. 329 (2005), filed as Ex. 17 Ref. 1 (ECF No. 23-3); K. Kuitwaard et al., *Recurrences, Vaccinations and Long-Term Symptoms in GBS and CIDP*, 14 J. Periph. Nwerv. Syst. 4, 310 (2009), filed as Ex. 17 Ref. 7 (ECF No. 25-2) (“Kuitwaard”) at 311. Mr. Nieves’s sequelae were all consistent with CIDP’s features. Kinsbourne Rep. at 8.

Dr. Kinsbourne did not accept the possibility that Petitioner’s CIDP had eventually remitted, or that his longer-term symptoms course undermined the diagnosis. He particularly questioned the negative electrodiagnostic results from testing performed on Petitioner in the fall of 2016, maintaining that such testing had a variable sensitivity, and that “CIDP may continue to be active” even when this kind of testing did not reveal its presence. Kinsbourne Rep. at 8; Y. Rajabally et al., *Validity of Diagnostic Criteria for Chronic Inflammatory Demyelinating Polyneuropathy: A Multicenter European Study*, 80 J. Neurol., Neurosurg. and Psych. 1 (2009), filed as Ex. 17 Ref. 16 (ECF No. 27-1) (“Rajabally”) at 1 (comparing different criteria, including electromyographic testing, for levels of sensitivity in establishing CIPD diagnoses).

The flu vaccine Petitioner received was in Dr. Kinsbourne’s view likely causal of Petitioner’s CIDP. He maintained that the immunologic mechanism relevant to GBS—molecular mimicry between vaccine components and myelin surface gangliosides, resulting in an antibody-

¹⁰ “Giveaway” weakness has been defined as “a lack of effort, or an impression by the examiner that the effort was poor.” *Sanchez v. Sec’y of Health & Hum. Servs.*, No. 18-1012V, 2022 WL 1013264, at *7 n.15 (Fed. Cl. Spec. Mstr. Mar. 11, 2022).

driven cross-reaction against those gangliosides—applied also to CIDP. Kinsbourne Rep. at 8–10. At most, any pathogenic difference between GBS and CIDP was attributable to a lymphocyte receptor that was effective in “switching off an ongoing immune response by causing T cell apoptosis.” *Id.* at 9; C. Comi, *Fas-Mediated T-cell Apoptosis in Chronic Inflammatory Demyelinating Polyneuropathy*, 16 (Supp.) *J. Periph. Nervs. Syst.* 45 (2011), filed as Ex. 7 Ref. 2 (ECF No. 23-4). While some individuals would realize the “benefit” of this receptor (and thus experience only GBS), others do not, with the attendant result of a chronic form of polyneuropathy, CIDP. Thus, a “host variable, independent of the causation” of the illness explained why a person would experience the more chronic form of polyneuropathy. Kinsbourne Rep. at 9.

Otherwise, Dr. Kinsbourne argued that while good epidemiologic evidence did not exist to establish an association between a “rare disorder” like CIDP and the flu vaccine, case reports established such a link. Kinsbourne Rep. at 9; J. Pritchard et al., *Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy Following Immunisation*, 73 *J. Neurol., Neurosurg. and Psych.* 348 (2002), filed as Ex. 17 Ref. 15 (ECF No. 26-5) (“Pritchard”) (questionnaire distributed by GBS patient support group in U.K. identified 65 of 179 CIDP patients who received vaccinations after their diagnosis, and two experienced relapse after receipt of the flu vaccine). Another study that took into account VAERS¹¹ data also observed instances of post-vaccination neuropathies. D. Vellozzi et al., *Safety of Trivalent Inactivated Influenza Vaccines in Adults: Background for Pandemic Influenza Vaccine Safety Monitoring*, 27 *Vaccine* 2114 (2009), filed as Ex. 17 Ref. 23 (ECF No. 27-7) (“Vellozzi”). Vellozzi did not specifically look for reported instances of CIDP, and it overall deemed the version of the flu vaccine it analyzed to be safe, but it did observe enough reported instances of GBS post-vaccination to warrant closer review. Vellozzi at 2118–19.

Dr. Kinsbourne also proposed that the onset timeframe was medically-acceptable. Kinsbourne Rep. at 10. He allowed for the fact that Petitioner had reported an “apparently transitory respiratory allergic reaction” within hours of vaccination, as well as leg and lower back pain five days later. *Id.* at 2. In his reading of the record, however, Mr. Nieves had presented with CIDP/GBS “prodromal manifestations” five days post-vaccination, with more specific neurologic symptoms (toe numbness) the next day (possibly meaning the day after Mr. Nieves’s ER visit on November 2, 2015). *Id.* at 6. Such timing was consistent with an acute onset-form of CIDP, and was also consistent with the “temporal interval for neuroimmune polyneuropathies.” *Id.* at 6, 10. Dr. Kinsbourne discounted Petitioner’s documented identification (on November 3, 2015) of symptoms (a “one-week history of paresthesias in his distal extremities”), since that would place

¹¹ The Vaccine Adverse Event Reporting System (“VAERS”) is a national passive-reporting warning system designed to detect safety problems in U.S.-licensed vaccines. *See About VAERS*, VAERS, <https://vaers.hhs.gov/about.html> (last visited Mar. 22, 2023). It is managed by both the CDC and the FDA. VAERS monitors and analyzes reports of vaccine related injuries and side effects from both healthcare professionals and individuals. *See generally Carda v. Sec’y of Health & Hum. Servs.*, No. 14-191V, 2017 WL 6887368, at *6 (Fed. Cl. Spec. Mstr. Nov. 16, 2017).

onset *before* vaccination—contrary to Petitioner’s contention (made, it should be emphasized, in *non-contemporaneous* witness statements) that he was symptoms-free at that time.

Second Report

Dr. Kinsbourne’s second report was filed in reaction to Respondent’s arguments that onset of Petitioner’s symptoms did not fit the timeframe for a Table flu-GBS claim.¹² He noted that the medical community accepted the likelihood of post-vaccination malaise (soreness at the injection situs; fever; and aches, among other things), and that such symptoms could manifest in reaction to the speedy cytokine upregulation encouraged by vaccinations generally. Second Kinsbourne Rep. at 1–2. The effects of this increase in cytokines, associated with the immune system’s innate response to an external trigger (including vaccines), could be seen within hours to two days post-vaccination. *Id.* at 2–3. Here, Mr. Nieves had reported some initial “allergic reaction” within hours of receiving the flu vaccine, along with joint pain, and these non-neurologic symptoms were all consistent with the expected vaccine reaction. *Id.* at 3. They thus were distinguishable from the kind of *neurologic* symptoms reflective of CIDP.

Later records did reveal CIDP manifestations—just not as close-in-time to vaccination. However, Dr. Kinsbourne was somewhat vague in specifying an onset date for these neurologic symptoms. Thus, in distinguishing Petitioner’s immediate, vaccine-induced malaise, Dr. Kinsbourne acknowledged that Petitioner’s CIDP onset could not have been the day of, or after, vaccination, but instead “must have begun later.” Second Kinsbourne Rep. at 2. He also noted that even at the time of Petitioner’s November 2, 2015 ER visit, CIDP “could not yet be reliably *diagnosed*.” *Id.* (emphasis added). Indeed, he disputed Petitioner’s own reporting of symptoms having begun close-in-time to vaccination (as reflected in the contemporary records). *Id.* at 4–5.

Instead, Dr. Kinsbourne emphasized Petitioner’s complaints about “progressive tingling, numbness and frequent falls” as reflective of CIDP—but these were only reported November 9, 2015. Second Kinsbourne Rep. at 5. Ultimately, he embraced this timeframe (which might place manifestation of neurologic symptoms as late as 10–12 days post-vaccination) as consistent with “the 3-42 day risk period for the onset of GBS/acute onset CIDP”—even though this opinion was not only inconsistent with Dr. Kinsbourne’s first report (*See* First Kinsbourne Rep. at 10 (“[t]he onset of Mr. Nieves’s polyneuropathy followed the influenza vaccination *after five days*”)), but also with the literal medical record (even allowing for the possibility that Petitioner mistook his immediate malaise as associated with his later neurologic issues).

Third Report

The final written report submitted by Dr. Kinsbourne addressed the first expert report offered by one of Respondent’s experts, Dr. Brian Callaghan. He began by revisiting his opinion

¹² The special master to whom the case was originally assigned had observed the record evidence (also reviewed above) suggesting an onset too close-in-time to vaccination to be consistent with the 3–42 day time interval required for a Table flu-GBS claim. *See generally* Order, dated May 1, 2020 (ECF No. 30).

about Petitioner’s diagnosis, dismissing Dr. Callaghan’s argument out of hand that Mr. Nieves’s documented, pre-existing comorbidities had anything to do with his CIDP. Third Kinsbourne Rep. at 1. He emphasized the difficulty of diagnosing CIDP, casting the issue in a different light: in his view, it was less *misdiagnosed* than *underdiagnosed*. *Id.*; U. Chaudhary and Y. Rajabally, *Underdiagnosis and Diagnostic Delay in Chronic Inflammatory Demyelinating Polyneuropathy*, 268 J. Neurol. 1366 (2021), filed as Ex. 26 Ref. 3 (ECF No. 59-3). Thus, the mere fact that not all criteria for the condition were precisely met in Petitioner’s case did not mean there was “a more fitting diagnosis.” Third Kinsbourne Rep. at 2. And the sudden appearance of Petitioner’s post-vaccination neurologic symptoms only confirmed that he had experienced an “acute onset” form of CIDP (although such individuals could go on to experience the kind of chronic symptoms more characteristic of CIDP). *Id.* at 2, 5–7.¹³ Dr. Kinsbourne also disagreed with Dr. Callaghan’s effort to distinguish GBS and CIDP, arguing that despite their many distinctions (which he deemed “mainly why they have been given different names”) it remained reasonable to view CIDP as “chronic” GBS. *Id.*

Dr. Kinsbourne also attempted to bulwark his prior assertions that the flu vaccine could cause CIDP. He made the general argument that epidemiologic evidence would be unable to detect rare events like autoimmune-driven polyneuropathies, that such evidence was required to prevail in Vaccine Program cases in any event, and that even the evidence cited for this point by Dr. Callaghan at least provided “weakly positive” evidence in favor of causation. Third Kinsbourne Rep. at 3–5; P. Doneddu et al., *Risk Factors for Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP): Antecedent Events, Lifestyle and Dietary Habits. Data from the Italian CIDP Database*, 27 Eur. J. Neurol. 1, 3 (2019), filed as Ex. 26 Ref. 6 (ECF No. 59-6) (“Doneddu I”) (seven of 411 CIDP patients reported pre-onset receipt of flu vaccine). With respect to what antibodies likely drive CIDP, Dr. Kinsbourne deemed any distinction having “no obvious relevance to the issue of vaccine causation,” since Respondent had not shown that the kind of antibody likely to drive CIDP was different. Third Kinsbourne Rep. at 8. He also cited literature that he said revealed “a small minority of patients” with CIDP displayed anti-ganglioside antibodies in any event. L. Querol and C. Lleixa, *Novel Immunological and Therapeutic Insights in Guillain-Barré Syndrome and CIDP*, *Neurotherapeutics* (2021) <https://doi.org/10.1007/s13311-021-01117-3>, filed as Ex. 26 Ref. 7 (ECF No. 59-7) (“Querol”), at 4 (noting that “anti-ganglioside antibodies have been reported in some CIDP cohorts,” but deeming it a “weak association,” and acknowledging that “other meaningful clinical-immunological correlations with anti-ganglioside antibodies have not been established yet” for CIDP).

¹³ In the context of this discussion, Dr. Kinsbourne repeated his prior assertion that the main explanation for CIDP’s chronicity was a host genetic deficiency—the inability to “inhibit the continuation of the inflammatory polyneuropathy at nadir.” Third Kinsbourne Rep. at 7. But Dr. Kinsbourne not only referenced Dr. Akbari’s opinion here, but personally lacks the immunologic qualifications to comment reliably on this topic in comparison to Dr. Akbari, and therefore I do not deem this portion of his opinion worthy of substantial weight.

2. *Joseph Jeret, M.D.*

Dr. Jeret, a neurologist with expertise in EMG/NCS studies and clinical familiarity with peripheral neuropathies like CIDP, offered an expert report in reaction to that provided by Respondent's first expert, Dr. Callaghan, plus a supplemental report. Report, dated December 12, 2021, filed as Ex. 23 (ECF No. 53-2) ("First Jeret Rep."); Report, dated Feb. 9, 2022, filed as Ex. 27 (ECF No. 72-1) ("Second Jeret Rep."). Dr. Jeret offered his own interpretation of Petitioner's medical records, and agreed with the CIDP diagnosis.

Dr. Jeret received his undergraduate degree from CUNY Brooklyn College in Brooklyn, New York in 1984, and his medical degree from SUNY Health Science Center at Brooklyn in 1988. Curriculum Vitae, filed as Ex. 22 (ECF No. 53-1) ("Jeret CV") at 1. He completed a one-year general internal medicine preliminary year at Maimonides Medical Center, followed by a three-year residency in Neurology and one-year fellowship in Clinical Neurophysiology at SUNY Downstate. Jeret CV at 1; First Rep. at 1. He is board certified in Neurology by the American Board of Psychiatry and Neurology and is currently employed by the Icahn School of Medicine at Mount Sinai Medical Center in New York as an active physician in the Department of Neurology. *Id.* He is also on staff at two community hospitals—South Nassau Community Hospital and Mercy Medical Center. *Id.* Dr. Jeret has published numerous articles in areas related to neurology, reflecting his broad general neurology practice. *Id.*

First Report

The first part of Dr. Jeret's report consisted of an explanation of CIDP, plus the role EMG and NCS testing play in diagnosing it. First Jeret Rep. at 2–6. He repeated Dr. Kinsbourne's general contentions that CIDP could be understood as a "chronic counterpart" to GBS, despite the differences between the two. *Id.* at 3. He also noted that, as a general matter, less than two thirds of CIDP patients would otherwise meet electromyographic clinical criteria for CIDP (and hence other data, like the results of a lumbar puncture, were needed to confirm the diagnosis in many cases). *Id.*; K. Gorson and A. Ropper, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP): A Review of Clinical Syndromes and Treatment Approaches in Clinical Practice*, 4 J. Clin. Neuromusc. Dis. 4:174 (2003), filed as Ex. 23 Ref. 11 (ECF No. 57-1) ("Gorson & Ropper"), at 178.¹⁴ And he discussed the specific EMG/NCS testing findings necessary to confirm CIDP. *Id.* at 4. But because the technical CIDP criteria were in many cases unmet, despite other indicia that CIDP explained a patient's condition, several "atypical CIDP" variants had been proposed. First Jeret Rep. at 4; P.E. Doneddu et al., *Atypical CIDP: Diagnostic*

¹⁴ Gorson & Ropper also identified vaccines as "an associated systemic medical disorder," including them among a list of medications (presumably which have been associated with CIDP). See Gorson & Ropper at 177 Table 2. However, the article does not provide any context at all for this assertion, and Table 2 more generally discusses CIDP as a "secondary symptomatic" inflammatory demyelinating neuropath[y]," adding that "the precise relationship of the underlying illness to the neuropathy varies from case to case." *Id.* at 176

Criteria, Progression and Treatment Response. Date from the Italian CIDP Database, 90 J. Neurol. Neurosurg. Psychiatry 125 (2019), filed as Ex. 25 Ref. 5 (ECF No. 60-5) (“Doneddu II”).

Next, Dr. Jeret engaged in a granular evaluation of Mr. Nieves’s medical history, including diagnoses and treatments he had received well before vaccination. First Jeret Rep. at 7–18. He acknowledged, for example, concerns for fibromyalgia expressed for Petitioner as early as 2012, but maintained that this diagnosis had never been formally confirmed. *Id.* at 7–8. He further observed EMG testing consistent with carpal tunnel syndrome from 2013, but emphasized that evidence of polyneuropathy had not been identified at that time. *Id.* at 8–9. Petitioner had also been treated for ongoing neck pain, but Dr. Jeret noted that relevant records seemed to focus on some form of spondylosis¹⁵ or cervical stenosis as the proposed cause, rather than a neurologic explanation for Petitioner’s symptoms. *Id.* at 8–9. Treatment sought by Petitioner in late 2013 in reaction to episodes of falling had revealed no loss of reflexes. *Id.* at 9.

At a neurologic evaluation toward the end of 2013, Dr. Jeret acknowledged, Petitioner had reported a long history of leg numbness, progressively worsening in the immediate four to five months and featuring loss of leg sensation. First Jeret Rep. at 9. He also at this time revealed absent reflexes, but no other major neurologic symptoms. *Id.* The months thereafter, treatment seemed focused on Petitioner’s cervical stenosis/neck pain. However, Petitioner obtained yet another neurologic consultation in November 2014, at which time he again reported numbness and weakness in his limbs. *Id.* at 10. Moreover, exam revealed diminished reflexes and sensation (although treatment again seemed oriented toward his cervical spine issues). *Id.* But by the spring of 2015, Petitioner reported improvement in his neck pain symptoms and had an otherwise normal exam. *Id.* at 11. Dr. Jeret deemed this to be Petitioner’s “pre-vaccine baseline,” characterized by an absence of neurologic complaints (despite a several-year history to that point complaining of a large variety of such issues). *Id.*

By contrast, Dr. Jeret emphasized a number of post-vaccination neurologic symptoms findings that were consistent with CIDP. First Jeret Rep. at 18. By November 3, 2015, Petitioner was reporting limb weakness and worsening paresthesias (consistent with his informing neurologists at this time that he had experienced “a one-week history of paresthesias” since his vaccination). *Id.* at 11. At this time, he also displayed some reduced sensation in distal upper and lower extremities, plus reduced reflexes in the arms and ankles. *Id.*; Ex. 3 at 83–87. The next day, when he took himself to the ER, Petitioner now displayed a normal neurologic exam overall, but Dr. Jeret questioned its thoroughness. *Id.* at 11–12.

¹⁵ “Cervical Spondylosis” is defined as “degenerative joint disease affecting the cervical vertebrae, intervertebral disks, and surrounding ligaments and connective tissue, sometimes with pain or paresthesia radiating along the upper limbs as a result of pressure on the nerve roots.” *Cervical Spondylosis*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=107846&searchterm=cervical%20spondylosis> (last visited Apr. 17, 2023).

When Petitioner returned to the ER on November 9, 2015, he again demonstrated no weakness, numbness, or tingling—but after his admission to the hospital the next day, he reported a history (since the vaccine) of progressive tingling, numbness and weakness—and at this time (after an exam revealed diminished strength in the distal lower extremities) GBS was raised as a concern. First Jeret Rep. at 12. A neurologic consultation on November 11th (which included an exam revealing areflexia, limb weakness, and lack of sensory levels) confirmed the need for a lumbar puncture. That testing confirmed the presence of a neuropathy—whether GBS or acute CIDP. *Id.* at 13.

Dr. Jeret provided some specific discussion of the various EMG/NCS testing performed on Petitioner. The February 2016 electromyographic testing results, for example,¹⁶ found several indicia of CIDP, including “acquired segmental demyelination,” “conduction block,” and “absent F-wave.” First Jeret Rep. at 13–14; Ex. 3 at 43–44. The second post-vaccination EMG/NCS testing, performed in September 2016, by contrast, revealed normal F-waves and no conduction block, but Dr. Jeret deemed this consistent with “successfully-treated CIDP.” First Jeret Rep. at 17. Dr. Jeret did not comment on the results of the third such study, other than to suggest it was merely a prerequisite for establishing care with a new neurologist. *Id.* at 18.

Given such a record, Dr. Jeret deemed Petitioner’s CIDP diagnosis to have ample credibility and accuracy. Numerous specific symptoms Petitioner reported reflected neurologic issues, and were confirmed by the February 2016 EMG/NCS testing. First Jeret Rep. at 18. Resolution of symptoms in the months to come (as reflected in the follow-up EMG/NCS testing) was attributable to successful treatment. *Id.* And this diagnosis had been embraced by several of Petitioner’s treating neurologists. *Id.* at 18–19, 23. By contrast, only Dr. Callaghan rejected the CIDP diagnosis—but in Dr. Jeret’s totality review of the record, there was ample evidence to support the CIDP diagnosis even if not all of the strictest criteria could be shown to be met. *Id.* at 22.

Dr. Jeret did not accept other explanations for Petitioner’s symptoms. For example, he took issue with the idea that Petitioner’s symptoms reflected fibromyalgia, as found in a record from March 2016, arguing that this diagnosis was not derived from a doctor’s speculation but simply existed in records due to “something said by the patient and then repeatedly cloned” in subsequent records. First Jeret Rep. at 15, 23–24. In fact, Dr. Jeret felt that records from this timeframe actually cast doubt on fibromyalgia as an explanation, and (unlike CIDP) no contemporaneous treaters ever proposed it as an alternative diagnosis. *Id.* at 23–24; Ex. 3 at 13. He also maintained that fibromyalgia would feature diminished reflexes on the testing findings from Petitioner’s first 2016

¹⁶ Dr. Jeret’s report confusingly identifies this as the “second” study; the record establishes it was the first EMG/NCS testing performed on Petitioner *post-vaccination*. First Jeret Rep. at 13. Petitioner had received an EMG study in November 2014, pre-vaccination—but it only demonstrated cervical radiculopathy, consistent with his neck pain complaints, rather than polyneuropathy consistent with CIDP. *Id.* at 10. The record thus does not contain EMG/NCS evidence supporting a CIDP *diagnosis* any time before the vaccination—but it is the post-vaccination EMGs that are most relevant to Petitioner’s claim.

EMG or cerebrospinal fluid tests, while radiculopathy would not feature the EMG testing “improvements” observed in the second 2016 EMG. *Id.* at 19. And he denied IVIG had been unsuccessful, noting improvement over time in Petitioner’s strength, reflexes, and sensation throughout the course of his receipt of such treatments. *Id.* at 16.

Next, Dr. Jeret proposed that the flu vaccine had likely caused Petitioner’s CIDP. He observed as a general matter that aberrant immune responses could produce autoimmune diseases, mediated by antibodies (produced in reaction to a foreign pathogen—or vaccine antigen) that (in this case) would mistakenly attack peripheral nerve myelin. First Jeret Rep. at 19; J. Brostoff et al., *Post-Influenza Vaccine Chronic Inflammatory Demyelinating Polyneuropathy*, 37 *Age and Ageing* 229 (2007), filed as Ex. 23 Ref. 5 (ECF No. 56-5) (case report involving single patient). This process could specifically be set into motion if a vaccine’s peptides mimicked “the body’s own peptides.” First Jeret Rep. at 19. Here, items of literature filed in the case demonstrated that CIDP was associated with a number of reported antecedents, including the flu vaccine. P. McCombe et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical and Electrophysiological Study of 92 Cases*, 110 *Brain* 1617 (1987), filed as Ex. 23 Ref. 18 (ECF No. 57-8) (one-third of patients in study reported antecedent infection; vaccines not discussed); Kuitwaard at 312 (questionnaire directed at GBS/CIDP patients revealed that (out of 76 CIDP patients who had responded to the survey) eight reported a pre-onset vaccination (most often the flu vaccine), and five of 24 CIDP patients who received the flu vaccine post-vaccination reported a symptoms worsening).

In addition, case reports not only elucidated instances of CIDP after receipt of the flu vaccine, but suggested relapses or symptoms worsening was possible in the wake of vaccination. P. Kelkar, *Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) with Rapid Progression after Influenza Vaccination: A Report of Three Cases*, 8 *J. Clin. Neuromusc. Dis.* 20 (2006), filed as Ex. 23 Ref. 23 (ECF No. 57-6); Pritchard at 348 (two of 46 CIDP patients responding to questionnaire reported relapse after receipt of flu vaccine). And indirect evidence of a causal link was supplied by the fact that the “GBS/CIDP Organization” recommended that individuals who experienced one of those two related neuropathies after vaccination not receive a vaccine a second time, since this “assumes that one may receive CIDP from a vaccine.” First Jeret Rep. at 20.¹⁷ Otherwise, since a CIDP diagnosis was so much more difficult, more direct proof of causation was extremely hard to generate or identify. First Jeret Rep. at 20.

¹⁷ The link provided to the webpage purportedly containing this view does not work. However, another link of the “GBS/CIDP Foundation International” page states that “[f]or the rare person who developed GBS within four to six weeks of receiving an immunization, the Foundation advises to avoid the same immunization in the future.” See <https://www.gbs-cidp.org/support/resources/flu-shots-and-vaccinations/> (last accessed Apr. 17, 2023). Thus, this admonition is specific only to those individuals who *first* develop GBS after vaccination, and only indirectly applies to this case (since Petitioner does not allege he previously experienced a flu vaccine-caused neuropathy).

With respect to onset, Dr. Jeret proposed that the record established Petitioner’s neurologic symptoms began no sooner than approximately five days post-vaccination—consistent with an accepted “several day latency” (although he seemed to rely on Dr. Kinsbourne for this timeframe). First Jeret Rep. at 20. In so maintaining, Dr. Jeret differentiated Mr. Nieves’s immediate symptoms from what came later. Mr. Nieves initially reported a “mild allergic reaction” and arthralgias the day after vaccination, but no numbness, weakness, or areflexia. First Jeret Rep. at 11, 20–21. But the history contained in Petitioner’s November 11, 2015 neurologic evaluation “suggests that the symptoms began ‘several days’ after the flu shot.” *Id.* at 13. And Dr. Jeret emphasized Dr. Musa’s “precise history” taken in March 2016 (five months post-vaccination), which described non-neurologic/subjective symptoms one week post-vaccination, with weakness “a week after that” plus areflexia (suggesting an onset of *more* than one week after vaccination). *Id.* at 15.

But Dr. Jeret struggled to differentiate contrary records suggesting an earlier onset. Thus, he noted that the Petitioner’s own chronology (prepared after the claim’s filing) identifies a loss of sensation in his hands and feet (something neurologic) the evening of October 31, 2015—a little over *three days* post-vaccination. First Jeret Rep. at 21; *see generally* Nieves Aff. But the medical record also indicates that Petitioner not only reported a number of neurologic-like symptoms on November 3rd (six days post-vaccination), but claimed he had been experiencing them for a week, or at least “since” the October 28th vaccination. *Id.* at 11. Dr. Jeret also admitted that although at the time of Petitioner’s mid-November 2015 hospitalization, he reported the kind of malaise-like post-vaccination symptoms that could be distinguished from neurologic complaints, he *also* reported progression of the latter kinds of issues *from the time of vaccination*, and hence (even at this point) “the onset of the neurological symptoms is not clearly delineated.” First Jeret Rep. at 12.¹⁸

Second Report

Dr. Jeret prepared a succinct additional report *less than two months* after the filing of his first. But while it purports to “address several issues that have been addressed by the other physicians,” it appears to have been filed mostly in reaction to my ordering the parties to brief the case for a ruling on the record—and thus recapitulates points Dr. Jeret already covered in his initial report.

For example, Dr. Jeret repeated his contention that Petitioner likely experienced “acute” CIDP, noting that his medical history reflected CIDP in all regards except for its abrupt onset

¹⁸ Dr. Jeret more generally offered the comment that medical records (especially those that are created via electronic entry into a computer file) often “clone,” or repeat verbatim, prior entries about patient histories without comment or consideration, that doctors will truncate a series of symptoms into a streamlined temporal reference for ease, and otherwise that “sloppy medical documentation” is common. First Jeret Rep. at 21. Of course, from the perspective of fact finding *in this case*, this simply means that the inconsistent and vague record presented on this topic needs to be closely scrutinized, differentiating between evidence deserving of weight from proof less trustworthy—and these comments do not undermine the reasonability of giving immediately-contemporaneous proof *more weight* than summaries or after-the-fact statements made after the claim’s filing.

(which made it resemble GBS). Second Jeret Rep. at 1. He then questioned the application of one item of literature touted by Dr. Callaghan to this case, maintaining that it lacked statistical significance and epidemiologic reliability (in the absence of a comparison control group), and that it relied on inexact data about the studied patients' symptoms onset. *Id.* at 1–2; Doneddu I at 2 (deeming it unlikely that antecedent events, including vaccination, could be risk factors for CIDP, based on questionnaire). He maintained that case report-derived “anecdotal reports” of post-vaccination CIDP were “the highest level of evidence that currently exists,” and therefore “[s]trict *Daubert* principles cannot be applied” in evaluating causation (a questionable opinion for a *non-legal medical expert* in a Vaccine Program case to advance). *Id.* at 2.

Dr. Jeret also maintained that no diagnosis other than CIDP was possible given Petitioner's medical history. The combination of symptoms experienced by Petitioner and that reflected CIDP—“areflexia, elevated CSF protein, and the multiple EMG findings” from the February 2016 testing could not be explained by fibromyalgia or radiculopathy. Second Jeret Rep. at 2. And numerous qualified treaters embraced the CIDP diagnosis (not to mention Drs. Jeret and Kinsbourne). *Id.* at 3.

Third Report

Dr. Jeret's concluding report was also succinct, and endeavored to rebut points made both by Respondent's neurologic expert, Dr. Callaghan, and immunologic expert, Dr. Mark Tompkins. Dr. Jeret's comments on the latter's opinion were mainly limited to the observation that Dr. Tompkins's lack of clinical expertise cast doubt on his assertion that the “GBS/CDIP Organization” warning about the danger of vaccination after vaccine-induced illness, especially since the article Dr. Tompkins relied upon involved a much smaller group of opining neurologists. Third Jeret Report at 1.

The remainder of this report addressed comments by Dr. Callaghan. First, Dr. Jeret disclaimed Dr. Callaghan's interpretation of the course of EMG testing as inconsistent with CIDP, noting that (a) Respondent's own literature established that IVIG treatment was not always successful, but (b) in fact the record showed Petitioner had improved, as reflected by “the objective EMG” results, and (c) the IVIG treatment would have been abandoned had treaters “truly doubted” the CIDP diagnosis. Third Jeret Rep. at 1. Otherwise, the first post-vaccination EMG confirmed the presence of sufficient criteria for CIDP to support the diagnosis, although Dr. Jeret admitted that the results had never been made available in this case¹⁹—forcing all experts to rely on descriptions of such testing in the medical records. *Id.* at 1–2. Dr. Jeret reemphasized the different clinical factors from the record that were consistent with a CIDP diagnosis (and in particular the proof of absent reflexes and the CSF testing results). *Id.*

¹⁹ Petitioner attempted to obtain some of the EMG results and file them into the record of this case, but could not do so. See Ex. 21 (ECF No. 48-1).

Second, Dr. Jeret rejected Dr. Callaghan’s emphasis on Petitioner’s allegedly-incompatible “multitude of symptoms,” deeming them “unrelated” to his CIDP. Third Jeret Rep. at 2. That diagnosis had not been rejected, he maintained, and it was corroborated by the fact that IVIG treatment was never discontinued (since this suggested it was deemed to be beneficial). *Id.* In a similar vein, Dr. Jeret did not accept fibromyalgia as an alternative diagnosis, proposing that the criteria for it had not been met, that it appeared to have been cloned in computerized medical records based on incorrect reporting by Mr. Nieves, and that it did not exclude a CIDP diagnosis regardless. *Id.* And no contemporaneous treater had considered “anything other than nerve demyelination” as explanatory for Petitioner’s condition. *Id.*

Dr. Jeret also sought to rebut Dr. Callaghan’s attacks on vaccine causation. He noted that even literature offered by Dr. Callaghan (and discussed below) allowed for a post-vaccination risk of CIDP. Third Jeret Rep. at 2–3. What limited epidemiologic evidence existed on the subject did not categorically reject the possibility of causation. In addition, Dr. Jeret again argued that the CIDP/GBS similarities outweighed their differences, and since molecular mimicry was “an accepted theory cited repeatedly in multiple peer-reviewed publications,” there was no reason not to embrace it in this context as well (as supported by literature filed by Dr. Jeret). *Id.* at 3; *see also* First Jeret Rep. at 19 (citations omitted).

3. *Omid Akbari, PhD.*

Petitioner filed two reports from Dr. Omid Akbari, Ph.D. *See generally* Expert Report, dated December 17, 2021, filed as Ex. 25 (ECF No. 54-2) (“First Akbari Rep.”); Report, dated August 1, 2022, filed as Ex. 29 (ECF No. 88-2) (“Second Akbari Rep.”). Dr. Akbari opined that Mr. Nieves’s CIDP was likely caused by the flu vaccine.

Dr. Akbari is a professor of allergy and immunology at Keck School of Medicine at the University of Southern California. Akbari First Rep. at 1–3; Omid Akbari CV, filed as Ex. 24 (ECF No. 54-1) (“Akbari CV”). He received his bachelor and master’s degrees from University College London. Akbari CV at 1. He then received a Ph.D. in cellular and molecular immunology from the National Institute for Medical Research in London before completing a postdoctoral fellowship at Stanford University. *Id.* He has and continues to serve on the editorial board of several journals, and he has numerous publications in the area of immunology and allergy research. *Id.* at 4–5, 9–13. He has particular experience on the subjects of “immune tolerance and how immune cells induce autoimmune and allergic diseases.” First Akbari Rep. at 2. Dr. Akbari is not a medical doctor, however, and therefore he does not diagnose or treat patients with neurological diseases in a clinical setting.

First Report

The initial part of Dr. Akbari’s first report contained an overview of Petitioner’s medical history, and it was consistent with the observations of Petitioner’s two neurologic experts. First Akbari Rep. at 3–6. He then discussed CIDP’s features, emphasizing (consistent with Drs.

Kinsbourne and Jeret) how difficult it was to identify consistent elements of it, as well as the fact that CIDP often presented acutely. *Id.* at 6. He otherwise classified CIDP (like some subgroup/variants of it) as a “chronic inflammatory neuropathy,” with a “distinct pathophysiology” involving the “breakdown of myelin” attributable to T cells and macrophages. *Id.* at 6–7. And he agreed with Dr. Kinsbourne that host susceptibility was likely a major (and most significant) factor in whether a person was likely to incur an autoimmune disease like CIDP—and in turn (since these diseases were themselves rare) why vaccine-induced instances of disease rarely occurred (since most individuals were not susceptible). *Id.* at 21–22.

However, Dr. Akbari disputed Dr. Callaghan’s contention that CIDP was distinguishable from GBS in a larger sense, emphasizing that “many features of immune cells and GBS and CIDP are indeed comparable.” First Akbari Rep. at 8–9. He also denied that the fact that fewer “diagnostic biomarkers” (meaning antibodies), if any, were associated with CIDP in comparison to GBS was a meaningful basis for distinguishing the two, proposing that *all* autoimmune peripheral neuropathies were mostly T cell and macrophage-mediated (and that in any event GBS-associated antibodies were not even present in approximately half of GBS patients). *Id.* at 10. In a similar vein, Dr. Akbari maintained that because “the target antigens for CIDP patients remain elusive,” it was very difficult to even identify the antibodies that were likely central to it, as recent studies acknowledged. *Id.*; Querol at 4. Overall, however, GBS and CIDP had more in common than not—although Dr. Akbari allowed (somewhat consistent with arguments made by Dr. Kinsbourne) that “alteration of the suppressive function” of certain regulatory immune cells likely contributed to a host propensity for autoimmune disease generally, and more specifically why CIDP would become chronic in nature, rather than be monophasic as in the case of GBS. First Akbari Rep. at 12–13, 15–16.

Next, Dr. Akbari proposed a mechanism for how the flu vaccine could lead to CIDP. He began by noting that environmental stimuli (for example, infections) could trigger an autoimmune process, allowing for the possibility that vaccines could as well. First Akbari Rep. at 22. He also observed how frequently neuropathies followed vaccination. *Id.* at 13; D. Karussis and P. Petrou, *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, 13 *Autoimmun Rev.* 3:215 (2014), filed as Ex. 25 Ref. 36 (ECF No. 64-6) (“Karussis”) (primarily if not exclusively discussing central nervous system neuropathic illnesses, like transverse myelitis). Other studies had suggested that flu vaccine peptide components derived from the wild virus could stimulate “autoreactive T cells” to attack nerve gangliosides, and Dr. Akbari referenced evidence for amino acid sequence homology between such components (in particular, hemagglutinin) and the target (although he offered studies involving multiple sclerosis (“MS”)—a central nervous system even *more* distinguishable from CIDP than CIDP is from GBS—to support this contention). First Akbari Rep. at 13–14; S. Markovic-Plese et al., *High Level of Cross-reactivity in Influenza Virus Hemagglutinin-specific CD4+ T-cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis*, 169 *J Neuroimmunol.* (1-2):31 (2005) filed as Ex.

25 Ref. 37 (ECF No. 64-7) (“Markovic-Plese”) (T helper cells²⁰ generated in lab in reaction to influenza peptides taken from a patient suffering from multiple sclerosis (“MS”) revealed high level of potential cross-reactivity, based on homology of amino acid sequences, with viral and human peptide sequences, including some myelin-related peptides). Any cross-reactive autoimmune process would be attributable to molecular mimicry, and Dr. Akbari noted that “structural homology” was likely as important as sequential to causing a cross-reaction between a foreign antigen and mimicked self-structure. First Akbari Rep. at 14–15. In fact, auto-reactive T cells already possessed by a person could be involved as well. *Id.* at 15.

Dr. Akbari’s causal theory also touched briefly on the concept that vaccination could stimulate some existing regulatory T cells that normally protect against autoimmunity, thereby opening the door to disease. First Akbari Rep. at 16–17. However, Dr. Akbari offered little to support this contention. Rather, he cited Kuitwaard, which only showed some symptoms increase in vaccinated individuals already diagnosed with CIDP (and moreover based solely on their *individually-reported circumstances*, rather than verified and independent evidence of vaccine association). Kuitwaard at 312. And he referenced literature noting that in elderly populations, a breakdown in the effectiveness of some T-regulator immune cells lead to a decreased responsiveness to the flu vaccination—an observation that said nothing about whether the vaccine was likely to *cause* an autoimmune disease by interference with these protective cells. First Akbari Rep. at 17; I. Herrero-Fernandez et al., *Effect of Homeostatic T-cell Proliferation in the Vaccine Responsiveness Against Influenza in Elderly People*, 16 *Immunity & Ageing* 14:1, 8 (2019), filed as Ex. 25 Ref. 46 (ECF No. 65-6) (“Herrero-Fernandez”).

Thereafter, Dr. Akbari described the more typical conception of a causal mechanism involving vaccines and injuries like CIDP: that they could stimulate the production of antibodies resulting in an autoimmune cross-reactive damage to myelin. He noted the extent to which reliable science had identified antibodies against myelin gangliosides in patients with peripheral neuropathies, and that the ganglioside targets were the locus for the start of the myelin destruction. First Akbari Rep. at 17; G. Zhang et al., *Erythropoietin Enhances Nerve Repair in Anti-Ganglioside Antibody-Mediated Models of Immune Neuropathy*. 6 *PLoS One*10:e27067 (2011), filed as Ex. 25 Ref. 48 (ECF No. 65-8) at 9. Vaccines had been observed to be so associated in triggering such an autoimmune process as well (albeit in studies not specific to CIDP). First Akbari Rep. at 18; N. Agmon-Levin et al., *Transverse Myelitis and Vaccines: A Multi-Analysis*, 18 *Lupus* 1198 (2009), filed as Ex. 25 Ref. 53 (ECF No. 66) (“Agmon-Levin”) (37 cases of post-vaccination transverse myelitis (“TM”), obtained from 40-year literature search).²¹

²⁰ “Th Cells: T helper cells” are defined as “T cells [that] trigger reactions in other immune system cells . . . [t]hey are activated in the lymph nodes by dendritic cells, which causes them to proliferate. After proliferation, they mature into effector T cells.” See *Snyder v. Sec’y of Health & Hum. Servs.*, No. 01-162V, 2009 WL 332044 (Fed. Cl. Spec. Mstr. Feb. 12, 2009).

²¹ Agmon-Levin is often cited by Vaccine Program claimants as evidence of CNS-oriented autoimmune demyelinating diseases after vaccination, but has been criticized. Its limited findings had to be dredged from a lengthy reporting

Dr. Callaghan had proposed (as discussed below) that antecedent infection was far less implicated in CIDP than in GBS, but Dr. Akbari argued that items of literature previously offered by Dr. Kinsbourne, such as Rajabally, did identify a meaningful number (10 percent) of infectious associations for the studied 268 CIDP patients. First Akbari Rep. at 18; Rajabally at 657–58. This, plus the fact that other vaccines had been shown associated with demyelination, bulwarked the conclusion that the flu vaccine could be as well. Karussis at 215, 221. More generally, however, Dr. Akbari questioned whether studies of any kind could accurately determine whether “an adverse event would be directly linked to a vaccine,” given the impracticability of identifying trustworthy biomarkers specifically, or the more general difficulty in *both* identifying rare adverse events (which he suggested passive surveillance like VAERS could track) *and* linking those events to specific biological mechanisms capable of explaining them. First Akbari Rep. at 23–24. It was simply too difficult, scientifically-speaking, to conduct reliable studies that could analyze such rare instances and draw conclusions from the testing with any degree of reliability. *Id.* at 24–26.²² Respondent was, in Dr. Akbari’s view, demanding certainty. *Id.* at 26–27.

Regarding onset and timing, Dr. Akbari proposed that Mr. Nieves’s symptoms began within five to six days of vaccination, relying to some extent on the reports his co-experts had provided and their review of the medical history. First Akbari Rep. at 27–29. He deemed this consistent with “what is described in the literature,” although he primarily relied on studies involving GBS. *Id.* at 27; L. Polakowski et al., *Chart-Confirmed Guillain-Barré Syndrome After 2009 H1N1 Influenza Vaccination Among the Medicare Population, 2009-2010*. 178 Am. J. Epidemiol. 6:962 (2013), filed as Ex. 25 Ref. 75 (ECF No. 68-5), at 968–69 (post-vaccination risk highest 8–21 days post-vaccination, although risk up to six weeks/42 days was still “slightly increased” from a standpoint of statistical significance).

Dr. Akbari particularly pointed to one study in which the majority of peripheral neuropathy cases identified from a South Korean patient sample occurred within a week/seven days of vaccination (comparable to Petitioner’s experience). Y. Park et al., *Clinical Features of Post-Vaccination Guillain-Barré Syndrome (GBS) in Korea*, 32 J. Korean Med. Sci. 7:1154 (2017), filed as Exhibit 25 Ref. 78 (ECF No. 68) (“Park”). Park considered post-vaccination GBS cases submitted for compensation to the Korean Advisory Committee on Vaccination Injury Compensation between 2002 and 2014 as part of the National Immunization Program in South Korea. Park at 1154–55. In fact, of the 48 flu-GBS cases approved for compensation in South Korea during that period, more than half (25) involved onset of neurological symptoms within *two*

period, suggesting not just that the injury is rare but that it is preponderantly unlikely. *See, e.g., Pearson v. Sec’y of Health & Hum. Servs.*, No. 16-9V, 2019 WL 3852633, at *14 (Fed. Cl Spec. Mstr. July 31, 2019) (giving limited weight to Agmon-Levin in a case alleging that flu vaccine caused TM, since Agmon-Levin referenced only two post-flu vaccine TM cases, despite the number of years of data considered).

²² It was for this reason that the IOM had (as previously referenced by Dr. Kinsbourne) been unable to confirm or deny a vaccination link with CIDP. First Akbari Rep. at 26.

days of vaccination. *Id.* at 1155–56 and Fig. 1. Park does not, however, discuss whether that timeframe was medically acceptable (or any other for that matter), nor does it set forth what criteria applied in awarding injury compensation in these Korean cases. Park at 1155. Dr. Akbari deemed reliance on GBS-specific studies reasonable, given the overlap between GBS and CIDP, and the attendant difficulty in separating one from the other. First Akbari Rep. at 27.

Dr. Akbari’s first report also included several pages devoted to a discussion of CD4+ T cells, or “T helper cells”—a kind of T cell understood not to directly attack foreign pathogens, but instead to either assist with the production of antibodies by B cells or to induce inflammation generally in the context of an infection. First Akbari Rep. at 11–13;9–21. He noted that one version of a T helper cell (the Th17 cell) was observed in increased levels for patients with peripheral neuropathies like CIDP, and thus likely was a factor in encouraging autoimmune disease. *Id.* at 11, 19–20, 21; S. Li et al., *IL-17 and IL-22 in Cerebrospinal Fluid and Plasma are Elevated in Guillain-Barré Syndrome*, 2012 *Mediators Inflamm.* Article ID 260473 (2012), filed as Ex. 25 Ref. 57 (ECF No. 66-7) at 4 (proposing that certain cytokines associated with GBS and CIDP likely had an association with the Th17 helper cells, especially since “GBS is classically regarded” as being mediated in part by T helper cells).

Dr. Akbari further posited that both a wild flu infection and vaccination could promote/upregulate this class of T cell. First Akbari Rep. at 12; Y. Lin et al., *Th17 Cytokines and Vaccine-Induced Immunity*, 32 *Semin. Immunopathol.* 79 (2010), filed as Ex. 25 Ref. 29 (ECF No. 62-9) (“Lin”) at 86 (review article observing importance of Th17 T helper cells in immune response to pathogens, and examining ways vaccines might aid their function, such as through development of “cytokine adjuvants”). Lin, however, does not find that vaccines cause pathogenic *increases* in this class of T helper cells; instead (and at most), it discusses vaccines in which upregulation of a specific cytokine (IL-17) is a *goal* of vaccination—in particular for vaccines seeking protection against various bacteria. Lin at 80–82. And Lin considered only a DNA version of the flu vaccine (distinguishable from the inactivated version at issue in this case) as upregulating cytokines. *Id.* at 82 (Table 1) and 84.

Dr. Akbari linked this discussion to studies looking at the effects of including adjuvants in a vaccine, like alum, and the role certain immune response pathways (the “inflammasome”)²³ play in increasing an inflammatory milieu when stimulated by adjuvants. Akbari First Rep. at 19–20. However, the flu vaccine (at least as administered in the U.S., and the version at issue herein) *does not contain an adjuvant*—and Dr. Akbari’s discussion of this topic did not clarify why he deemed

²³ I have previously discussed at length the role the inflammasome (a protein complex thought to provoke inflammation as part of the innate/initial immune response) is theorized to play in the human immune system response. See generally *Olson v. Sec’y of Health & Hum. Servs.*, No. 13-439V, 2017 WL 3624085 (Fed. Cl. July 14, 2017) (dismissal of case alleging HPV vaccine caused rheumatoid arthritis), *mot. for review den’d*, 135 Fed. Cl. 670 (2017), *aff’d*, 758 F. App’x 919 (Fed. Cir. 2018).

science positing a role for adjuvants to interfere with the immune process had anything to do with this case.

Second Report

Dr. Akbari's final report (at thirty pages plus references) exceeded the length of his first report (raising the reasonable question of what he left out the first time).²⁴

Dr. Akbari added considerable detail to defending his contention that CIDP could be triggered after the flu vaccine via the mechanism of molecular mimicry. After offering a caveat that his theory could not be proven with certainty given the incomplete nature of the relevant science on the topic, he reiterated his arguments that peptide sequences in the wild flu virus could be recognized, due to mimicry, by T cells, disputing Dr. Tompkins's claim that the T cells had not been shown to be specific to the flu peptides, and stressing the reliability of these findings and their utility in providing a means of establishing cross-reactivity potential in the context of differing viral infections. Second Akbari Rep. at 10–11. He also defended literature involving the animal model for central nervous system demyelinating diseases, “experimental autoimmune encephalomyelitis,” or “EAE”²⁵ as a reasonable means for testing immunologic function in the presence of demyelinating disease, noting that Dr. Tompkins had himself utilized it. *Id.* at 11–13.

In addition, Dr. Akbari offered some more recent literature addressing molecular mimicry as a pathologic mechanism relevant to GBS. Second Akbari Rep. at 17–18; J. Laman et al., *Guillain-Barré Syndrome: Expanding the Concept of Molecular Mimicry*, 43 Trends in Immunol. 4:296 (2022), filed as Ex. 29 Ref. 21 (ECF No. 90-1) (“Laman”). Laman is a review article/opinion piece embracing GBS as “the best-supported example of true molecular mimicry at the B cell level,” and that a better understanding of the immunologic/pathogenic processes that drive it could result in innovations in diagnostic techniques and treatment therapies “for other antibody-driven neurological diseases.” Laman at 296. Dr. Akbari thus invoked Laman as evidence that “molecular mimicry and structure mimicry do exist and are capable of causing . . . peripheral demyelination.” Second Akbari Rep. at 18. However, the reliability of that contention is less in dispute herein than whether molecular mimicry explains *CIDP* after vaccination—a subject Laman does not address. In fact, Laman acknowledges that “the targets of neuropathogenic antibodies (or T cells) in patients

²⁴ The parties (primarily due to the conduct of Petitioner and his counsel) also wasted time in the spring of 2022 with needless motions practice about the propriety of additional expert filings, and the scope of what they would be permitted to do. Recognizing that Petitioner should have the opportunity to rebut Dr. Tompkins's report, I allowed the filing of a final responsive report from Dr. Akbari. Order, dated May 23, 2022 (ECF No. 81) at 2. But I indicated (in the hopes that the responsive report would be succinct, and not lard the record with even more secondary literature) that the only additional articles filed should be recently-published items from 2022, that could not have been offered before. *Id.* Incredibly (and part and parcel with the unnecessarily lengthy second report), Dr. Akbari identified, and filed, nearly 20 such publications—though few (if any) reflect new discoveries about CIDP or its purported association with the flu vaccine.

²⁵ EAE is an animal model study allowing researchers to explore immune function in the context of an MS-like central nervous system autoimmune disease. See *Harrington v. Sec'y of Health & Hum. Servs.*, No. 14-43V, 2018 WL 4401976 (Fed. Cl. Spec. Mstr. Aug. 14, 2018).

not harboring antiganglioside antibodies” remain unknown, even for patients presenting with demyelination (such as most CIDP patients). Laman at 305.

Another recent article revealed that “broadly reactive influenza antibodies” (some of which could be produced in response to vaccination) “increases autoreactive antibodies . . . and induces demyelinating diseases.” Second Akbari Rep. at 19–20; L. Labombarde et al., *Induction of Broadly Reactive Influenza Antibodies Increases Susceptibility to Autoimmunity*, 38 Cell Rep. 10:1 (2022), filed as Ex. 29 Ref. 24 (ECF No. 90-4) (“Labombarde”). Labombarde looked at this question both via autoimmune disease experimental models like EAE (injecting the mice subjects directly with lab-created autoreactive influenza antibodies) and by a comparison of levels of similar antibodies in humans who had been infected by different wild virus flu strains from different seasons. Labombarde at 4–12. Its authors did find that “the induction of autoreactive antibodies in conjunction with broadly reactive antibodies, exacerbated autoimmunity.” *Id.* at 12. However, Labombarde also forthrightly noted that this exacerbation only occurred “in the presence of inflammation or underlying defects in tolerance,” so that “*an increase in broadly reactive influenza antibodies alone is not sufficient to induce autoimmune disease.*” *Id.* at 13 (emphasis added).

In other words (and contrary to Dr. Akbari’s assertion), *in the absence* of an existing inflammatory setting or demonstrated immune tolerance inhibition, external triggers that might activate autoreactive immune cells were not *also* likely to encourage disease (or at least have not been shown to have that capacity). Nor did Labombarde say anything about the role *current* versions of the flu vaccine might play in encouraging the development of these antibodies; on the contrary, its authors spoke on vaccination only in the context of the goal of generating a “universal influenza vaccine” that does not yet exist. Labombarde at 13. While its authors proposed that such a new vaccine formulation could help “generate durable, broadly reactive influenza antibodies,” there was a danger that upregulation of these antibodies could in certain circumstances make autoimmune disease more likely (albeit under the conditions mentioned above). Labombarde does not conclude or propose that an inactivated form of the flu vaccine promotes disease in this manner.

Dr. Akbari also defended the amount of amino acid homology necessary for mimicry to spark a cross-reaction, against Dr. Tompkins’s arguments. Second Akbari Rep. at 18–19. Structural homology, he maintained, was as critical to causing a cross-reaction as sequential similarity, and therefore rendered the latter less significant to whether an autoimmune process due to mimicry was likely to occur. *Id.* at 19. But he stressed that his theory was not limited to the concept that vaccine-induced autoimmune disease occurred as a result of molecular mimicry, but that he also relied on “regulation of immune homeostasis”—and in particular for this case, the failure of such regulatory processes (thereby permitting the *chronic* demyelination characteristic of CIDP). Second Akbari Rep. at 13, 21. Properly-functioning immune regulation in the majority of cases prevents cross-reactivity from causing autoimmune disease. *Id.* at 13–14, 20–21. But literature offered by other experts, like Pritchard, established that patients with both GBS and CIDP displayed decreased levels of these regulatory immune cells, and that their diminishment likely was “a main factor resulting in a person’s predisposition to develop autoimmune disease

during activation of the immune system such as by receipt of a vaccine.” *Id.* at 15. Dr. Akbari also offered a number of more recently-published articles about the significance of regulatory immune cells in an environment of neuro-inflammatory diseases (although none advance the contention that any vaccination would *cause* the dysregulation of such cells sufficient to trigger a peripheral neuropathy like CIDP). *Id.* at 23–27 (citations omitted).²⁶

But Dr. Akbari admitted he could not provide “the exact mechanisms responsible for the reduction” in regulatory cells. Second Akbari Rep. at 15. Indeed, he largely reiterated his prior assertions that patients with demyelinating diseases often displayed high levels of T helper cells and certain pro-inflammatory cytokines in their blood, leading to the conclusion that both played a role in disease pathogenesis. *Id.* at 21–22. But he did not show that the flu vaccine pathogenically increased these amounts, seeming instead to assume that those susceptible to disease *due* to immune dysregulation specific to their host genetic variability would in turn suffer merely from the increases in such immune cells attributable *generally* to vaccination. And he denied that the flu vaccine could not cause such increases, taking issue with literature offered by Dr. Tompkins for those points. *Id.* at 22–23. But in doing so, Dr. Akbari could only reference recent studies specific to the COVID-19 vaccine (which mechanistically induces immune protection in a manner wholly distinguishable from how the flu vaccine functions), arguing that they revealed an association with demyelinating disease, and that the inflammatory response to vaccination was (consistent with his theory herein) associated with the functioning of immune pathways as well as immune system dysregulation. *Id.* at 27–29.

Dr. Akbari also offered a number of additional points in an effort to rebut narrower aspects of the arguments of Respondent’s experts, but which are not central to this case’s resolution. For example, the first five pages of his final report were devoted to casting doubt on the weight to be given to two “low impact”²⁷ articles cited by Respondent’s immunologic expert, Dr. Tompkins, as undermining a flu-CIDP association. Second Akbari Rep. at 1–5; *See, e.g.,* S. Greene et al., *Near Real-Time Surveillance for Influenza Vaccine Safety: Proof-of-Concept in the Vaccine Safety Datalink Project*, 171 *Am. J. Epid.* 2:177 (2009), filed as Ex. D Tab 9 (ECF No. 84-9) (“Greene”) (case-control study relying on data from patients enrolled in eight large medical care organizations, comparing risk of adverse events (including peripheral neuropathies) for millions of flu vaccine doses administered in three seasons between 2005 and 2008 revealed no statistically significant

²⁶ These articles merely bulwark Dr. Akbari’s general points about the role immune regulation plays in autoimmune disease, and therefore I do not include extensive discussion of them herein. To illustrate their less-than-ringing support for his theory, however, I note that one such article discussed the capacity of a certain formulation of the flu vaccine *not at issue in this case* to enhance vaccine immunogenicity via the T helper cell response. Second Akbari Rep. at 27; L. Moise et al., *Novel h7N9 Influenza Immunogen Design Enhances Mobilization of Seasonal Influenza T Cell Memory in H3N2 Pre-Immune Mice*, *Human Vacc. & Immunother.*, <https://doi.org/10.1080/21645515.2022.2082191> (2022), filed as Ex. 29, Ref. 37 (ECF No. 90-17) (“Moise”). Dr. Akbari noted only that Moise suggested that “suitable conditions” could trigger *dysregulated* immune responses—and he proposed flu vaccine administration might constitute such conditions—but Moise’s focus was on improving the efficacy of a totally different kind of flu vaccine.

²⁷ Dr. Akbari defined this to mean the overall credibility or quality of a particular publication. Second Akbari Rep. at 2 n.2.

relationship). Dr. Akbari maintained that articles like Greene were biased given their pharmaceutical industry funding, reflected design bias in the studies' methodologies (which resulted in larger vaccine coverage rates for the subject pools than would be seen in an unbiased, general population study), and thus produced unreliable results that under-detected likely adverse vaccine events. Second Akbari Rep. at 2–5. At bottom, Dr. Akbari maintained that it was “unethical and possibly illegal” for Respondent’s experts to rely on any studies attributable to the manufacturers of the vaccine “that caused injury in Mr. Nieves.” *Id.* at 6.

In addition, Dr. Akbari defended his discussion of immune pathway/inflammasome stimulation by the flu vaccine as contributing to injury, despite Dr. Tompkins’s accurate observation that the vaccine itself is not adjuvanted (and thus discussions of the impact of adjuvants in stimulating that pathway were irrelevant in the context of this case). Second Akbari Rep. at 6–9. In reaction, Dr. Akbari maintained that viral infections could *themselves* stimulate these pathways, and that he never intended to refer only to adjuvants as having this potentiality. *Id.* at 6–8. This stimulation would lead to the production of pro-inflammatory cytokines that could in turn contribute to demyelinating disease, but more likely induce the class of T helper cells he had previously identified as pathogenic. *Id.* at 8–9. But it is far from clear that Dr. Akbari’s argument on this point does anything more than *describe* either how the immune system generally reacts to a vaccine, or how the T helper cells he deems important to his theory arguably function in CIDP—this aspect of his theory did not establish that the vaccine *would likely* trigger a pathologic process, even if immune pathway responses bear generally on how the body responds to vaccines and infections.

Dr. Akbari pointed as well to several recent publications specific to an entirely different issue: whether the Epstein-Barr virus (“EBV”) is associated with MS. Second Akbari Rep. at 15–16 (citations omitted). He maintained these new articles demonstrated that T cells (albeit not the helper cells discussed in his theory) that recognized the EBV could also recognize influenza A virus, and likely “may respond to several flu antigens” as well. Second Akbari Rep. at 16. As a result, evidence about EBV’s association with MS (a form of demyelinating disease) via molecular mimicry was relevant to this case, and pointed toward a means of understanding “the pathophysiological roots for the induction of GBS/CIDP after exposure” to the flu vaccine. *Id.* Dr. Akbari did not, however, reference a *comparable* study specific to the injury at issue in this case; indeed, the mere *existence* of the EBV studies somewhat undermines the contention that epidemiology of this level of reliability could not be performed for other demyelinating injuries.

Finally, Dr. Akbari again emphasized the extent to which a wild flu infection *or* flu vaccine had been shown in reliable studies to correlate with an increase in the T helper cells and associated cytokines that were connected to GBS. Second Akbari Rep. at 22–24 (references omitted). He specifically noted a more recent study demonstrating an increase of the Il-17 cytokine after vaccination. *Id.* at 23; R. Bernard-Valnet et al., *Influenza Vaccination Induces Autoimmunity Against Orexinergic Neurons in a Mouse Model for Narcolepsy*, 145 *Brain* 2018 (2022), filed as Ex. 29 Ref. 13 (ECF No. 90-13) (“Bernard-Valnet”). But not only does Bernard-Valnet involve a

completely distinguishable, *nondemyelinating* injury (narcolepsy) that occurs in the brain (not the peripheral nervous system), but its observations were specific to the *Pandemrix* flu vaccine—a version that is *adjuvanted*, that is not administered in the United States, and that was not received by Petitioner. Bernard-Valnet at 2021.²⁸

B. Respondent’s Experts

1. *Brian Callaghan, M.D., M.S.*

Dr. Callaghan, a neurology professor with specific expertise in peripheral neuropathies like GBS and CIDP, offered two written expert reports on behalf of Respondent. Report, dated July 24, 2021, filed as Ex. A (ECF No. 50-1) (“First Callaghan Rep.”); Report, dated April 17, 2022, filed as Ex. C (ECF No. 74-1) (“Second Callaghan Rep.”). He disputed the accuracy of Petitioner’s CIDP diagnosis, and secondarily denied that the flu vaccine could cause it.

Dr. Callaghan received his undergraduate degree from the University of Michigan, his medical degree from the University of Pennsylvania in 2004, and his Master’s in Science from the University of Michigan in 2011. Curriculum Vitae, filed as Exhibit B (ECF No. 50-2) (“Callaghan CV”) at 1. He is board certified in psychiatry/neurology as well as electrodiagnostic medicine. Callaghan CV at 1. Dr. Callaghan was appointed to be a clinical lecturer at the University of Michigan Health System’s Department of Neurology in 2009 and has been an Associate Professor of Neurology there since 2018. *Id.* He has published more than 100 articles and medical book chapters, most of which focus on neuropathies, and his research interest lies in diagnostic evaluation and testing of peripheral neuropathies. First Callaghan Rep. at 1; Callaghan CV at 2, 11–20. Dr. Callaghan reports to treat approximately 30 patients with CIDP per year. First Callaghan Rep. at 1.

First Report

Like Dr. Kinsbourne before him, Dr. Callaghan engaged in a lengthy review of the medical record. *See generally* First Callaghan Rep. at 1–3. But he noted numerous aspects of that history that he deemed inconsistent with a CIDP diagnosis. First, he pointed to evidence that Mr. Nieves had experienced pre-vaccination symptoms congruent with his post-vaccination condition. *Id.* at 3–4 (noting pre-vaccination history of radiculopathy, chronic pain, and numbness/tingling; seen for “numb legs and falls” in December 2013, reporting symptoms for many years, and displaying absent reflexes; seen in November 2014 for radiating neck pain plus numbness and weakness).

²⁸ I have in prior cases discussed at great length the distinctions between Pandemrix and the attenuated/inactivated version of the flu vaccine administered in the U.S.—including the fact that there are wild viral antigens common to both does *not* mean that studies specific to Pandemrix bear on other vaccine versions. *See, e.g., D’Tirole v. Sec’y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *20–21 (Fed. Cl. Spec. Mstr. Nov. 28, 2016), *mot. for review den’d*, 132 Fed. Cl. 421 (2017), *aff’d*, 726 F. App’x 809 (Fed. Cir. 2018).

Second, Dr. Callaghan emphasized the changing findings over time in Petitioner’s EMG/NCS testing results. The first post-vaccination EMG (from February 2016—and thus performed approximately three months after onset) “revealed a generalized polyneuropathy with predominantly demyelinating features” that Dr. Callaghan agreed was consistent with the CIDP diagnosis. First Callaghan Rep. at 2. The next such testing did not occur until seven months later, in September 2016—but Petitioner now showed “no evidence of demyelination or CIDP.” *Id.* at 3, *citing* Ex. 11 at 64. A third EMG/NCS testing round produced comparable negative results. Second Callaghan Rep. at 3.

Finally, Dr. Callaghan took note of Petitioner’s overall symptoms progression. When Petitioner first sought emergency care in November 2015, reported pain levels were high, he was ataxic, displayed absent reflexes, and complained of ascending weakness and tingling in his limbs. First Callaghan Rep. at 2. IVIG was started, but proved over time ineffective—and substitute treatments for neuropathic symptoms, like oral steroids, did no better. *Id.* By February 2016, Petitioner was not displaying much weakness as opposed to pain, and other symptoms (specific to the cervical or lumbar spine) continued to present that could not be deemed associated with CIDP. *Id.* In the spring of 2016, IVIG’s only half-ameliorative nature was again observed, and while Petitioner did display some symptoms comparable to what he had in the past, he also displayed giveaway weakness. *Id.* at 3.

By the early fall of 2016, however (now nearly one year since the vaccination at issue), it seemed more evident to treaters that IVIG was not helping ameliorate Petitioner’s symptoms. First Callaghan Rep. at 3. In addition, he now displayed normal strength, reflexes, and sensation. *Id.* By the end of the year, IVIG was discontinued, and Petitioner’s symptoms were more oriented to lower back pain radiating into his legs plus cramps and spasms, with neurology treaters recommending treatment for “symptoms related to fibromyalgia.” *Id.* As of a neurologic visit in August 2019, it appeared treaters had begun to lean *against* CIDP as a diagnosis. *Id.*, *citing* Ex. 14 at 1.

From the foregoing, Dr. Callaghan opined that Petitioner’s CIDP diagnosis was more likely than not inapt. First Callaghan Rep. at 4–5. Mr. Nieves had experienced a host of pre-vaccination symptoms that seemed consistent with what he displayed after vaccination. *Id.* at 4. The kinds of treatments commonly employed for CIDP (or GBS for that matter), like IVIG and steroids, did not prove effective. *Id.* at 4–5. EMG tests were only confirmatory for a neuropathy at the outset of his illness, with follow-up testing not revealing the existence of a chronic demyelinating condition. In addition, Petitioner’s exams revealed other “atypical features” like giveaway weakness, “spasms that appeared more voluntary or functional,” unreasonable sensitivity to touch, and sensory issues at the thoracic spinal cord level inconsistent with CIDP, not to mention many other distinguishable symptoms. *Id.* at 4–5.

In Dr. Callaghan’s view, these factors (plus the record history of neurologists coming to doubt the diagnosis) all undermined the CIDP diagnosis. First Callaghan Rep. at 5. In so opining,

he noted that even if Dr. Kinsbourne had offered literature descriptions of “atypical presentations of CIDP,” he had not adequately defended the possibility that so many “atypical presentations occurring together” in the instance of Petitioner’s medical history could still meet the diagnostic criteria. *Id.* In fact, CIDP was frequently *misdiagnosed*. J. Allen and R. Lewis, *CIDP Diagnostic Pitfalls and Perception of Treatment Benefit*, 85 *Neurol.* 498 (2015), filed as Ex. A Tab 3 (ECF No. 51-3) (diagnostic criteria often not met, patients frequently reported improvement from immunotherapy regardless of accuracy of diagnosis, and electrophysiologic evidence often reviewed with liberal eye favoring diagnosis).

In addition to the foregoing, Dr. Callaghan offered an opinion on causation (although, and as with Dr. Kinsbourne, it somewhat exceeded his otherwise-demonstrated neurologic expertise). He noted that Dr. Kinsbourne relied heavily for this aspect of his opinion on “case reports and series”—a kind of evidence Dr. Callaghan deemed to provide little value on the question of causation, especially because case reports mostly only demonstrated a temporal relationship with the vaccination and alleged injury. First Callaghan Rep. at 5. The survey studies Dr. Kinsbourne offered only showed low percentages of post-vaccination CIDP (and in small sample groups as well), and were of limited significance otherwise, as their authors admitted. *Id.* at 5–6.

By contrast, Dr. Callaghan pointed to what he deemed “the best study investigating antecedent events,” Doneddu I, which he noted did not support a causal association. First Callaghan Rep. at 6; Doneddu I at 3, 6 (only seven out of 411 CIDP patients (1.5 percent) in survey reported receipt of vaccine (all flu vaccine) before onset, suggesting a causal relationship was unlikely). Another review article discussing CIDP generally noted that although its pathogenesis was thought to feature a synergistic interaction between the initial/innate “humoral” immune response and the subsequent “cell-mediated” response, little was known about triggers, with “no infectious agent” linked consistently to the disease’s start. E. Mathey et al., *Chronic Inflammatory Demyelinating Polyradiculopathy*, 0 *J. Neurol. Neurosurg. Psych.* 1 (2015), filed as Ex. A Tab 1 (ECF No. 51-1) (“Mathey”), at 3.

The purported mechanistic and pathologic congruence between CIDP and GBS was also, in Dr. Callaghan’s view, far less certain than Dr. Kinsbourne argued. He noted, for example, that literature suggested CIDP actually occurred *less* often after antecedent infection or trauma (in comparison to GBS), thus diminishing the likelihood that it occurred via molecular mimicry (as was more generally understood to occur with GBS). First Callaghan Rep. at 6; E. Ubogu, *Inflammatory Neuropathies: Pathology, Molecular Markers and Targets for Specific Therapeutic Intervention*, 130 *Acta Neuropathol.* 4:445, filed as Ex. A Tab 5 (ECF No. 51-5) (“Ubogu”) at 15. Ubogu also noted that “[a]ntibodies against peripheral nerve myelin proteins or node of Ranvier components are too infrequently detected in the sera of CIDP patients to be considered pathogenic or molecular markers of disease,” and the same was true for “[a]ntibodies against complex gangliosides.” *Id.* at 15. The two conditions also likely had distinguishable pathogenic courses, since “compromised immune tolerance” or immune regulatory dysfunction was relevant to CIDP’s chronicity. *Id.* Indeed, Dr. Kinsbourne’s observations about the host variances in T cell apoptosis

that likely contributed to CIDP underscored their distinction. And literature identifying antibodies against myelin surface gangliosides as driving GBS did not similarly propose that CIDP was mediated in this fashion. First Callaghan Rep. at 6.²⁹

Dr. Callaghan also commented on questions raised within the medical records about Petitioner's onset. Although Petitioner's history seemed to differentiate the initial symptoms Petitioner reported close-in-time to vaccination from his later neurologic symptoms, Dr. Callaghan did take note that at the time of Petitioner's November 3rd ER visit, he specifically reported a week of paresthesias—and obtained at that time a neurology consultation as well—suggesting that neurologic symptoms had occurred far closer in time to vaccination than Dr. Kinsbourne allowed. First Callaghan Rep. at 1. Yet Dr. Callaghan also stressed the degree to which Petitioner's pre-existing comorbidities explained his post-vaccination symptoms. *Id.* at 4–5. As a result (and in keeping with his view overall that Petitioner was improperly diagnosed with CIDP), Dr. Callaghan did not directly opine on when Petitioner's arguably-neurologic symptoms likely first manifested.

Second Report

Dr. Callaghan's second report reacted to the supplemental reports filed by Petitioner's two neurologic experts (Drs. Jeret and Kinsbourne). Beginning with the former, he noted that he had been unable to review the precise EMG/NCS findings from Petitioner's February 2016 testing, making it difficult for him to comment specifically on whether contemporaneous interpretations of them were accurate, or what they otherwise revealed. But even to the extent the first post-vaccination electromyographic testing confirmed the CIDP diagnosis, the other studies did not—and Petitioner's experts had failed to persuasively explain why this was the case, even though Petitioner's recovery remained incomplete at these later times. Second Callaghan Rep. at 1.

Dr. Callaghan also commented on assertions about the Petitioner's "atypical" form of CIDP—noting that even if some patients did not meet all clinical criteria, "the vast majority do," and thus those criteria had value in weighing if Petitioner's presentation was actually consistent with CIDP. Second Callaghan Rep. at 1. Here, the combination of Petitioner's consistently atypical presentation, plus his many "incompatible" symptoms (which in general predated vaccination or were not specific to CIDP), all added up, in Dr. Callaghan's estimation, to an individual who had not likely been properly diagnosed with CIDP. *Id.* at 1–2. As further support for this opinion, Dr. Callaghan reiterated his prior observations that Mr. Nieves had not responded favorably to polyneuropathy-specific treatments like IVIG, that the EMG/NCS testing performed after February 2016 was inconsistent with the diagnosis, and that the record revealed treater doubt about the accuracy of CIDP later in the course of Petitioner's illness. *Id.* at 2. He rejected Dr. Jeret's

²⁹ I also note that both sides discussed whether a section of the Institute of Medicine's Report on vaccine causation of CIDP after the flu vaccine supported, or contradicted, causation. *See* First Kinsbourne Rep. at 9; First Callaghan Rep. at 6; K. Stratton et al., *Adverse Effects of Vaccine: Evidence and Causality*, Institute of Medicine, National Academies (2012), filed as Ex. 23 Ref. 28 (ECF No. 58-8) (the "IOM Report"). In this case, however, I find the IOM Report to be too equivocal in its conclusions specific to this case to meaningfully "move the needle" on the causation question—in either direction.

proposal that the subsequent negative EMG testing results reflected the success of IVIG treatment, observing that the tests occurred “because [Petitioner] was not doing well with persistent symptoms.” *Id.* at 3 (emphasis added).

In addition, Dr. Callaghan stressed his prior assertion that CIDP was not the same as GBS, even if the two could appear similar at the outset and thus be difficult initially to distinguish, since both ultimately had “different pathophysiologies.” Second Callaghan Rep. at 2. He again noted that literature observed that CIDP was far less associated with antecedent infection, and that the anti-ganglioside target believed integral to GBS’s autoimmune course had no analogue in the context of CIDP. *Id.* at 2–3. And he took issue with Dr. Kinsbourne’s conception of CIDP and GBS as being triggered in the same way (and thus “indistinguishable at onset”), but then diverging mainly because of host genetic variability, emphasizing his own view that they were ultimately not “the same disease.” *Id.* at 3.

Dr. Callaghan identified his own contemporaneous literature that he maintained did not support a CIDP-vaccine association. Second Callaghan Rep. at 2; H. Köller et al., *Chronic Inflammatory Demyelinating Polyradiculopathy*, 352 N. Engl. J. Med. 1343 (2005), filed as Ex. C Tab 1 (ECF No. 74-2) (“Köller”); J-M. Vallat et al., *Chronic Inflammatory Demyelinating Polyradiculopathy: Diagnostic and Therapeutic Challenges for a Treatable Condition*, 9 Lancet Neurol. 402 (2010), filed as Ex. C Tab 2 (ECF No. 74-3) (“Vallat”). Neither article makes such an affirmative representation, however, although both cast doubt on aspects of Petitioner’s theory concurrent with Dr. Callaghan’s arguments. *See, e.g.*, Köller at 1348 (noting that little is known about “antigen specificity” driving CIDP). Vallat does not discuss vaccine causality either, but mentions that “by contrast with GBS, a single triggering antigen has not yet been found” for CIDP. Vallat at 402.³⁰

In addition, Dr. Callaghan criticized the extent to which Petitioner’s experts mostly looked to case reports—“valuable tools in flagging areas for future study,” he agreed, but inadequate otherwise to establish causation. Second Callaghan Rep. at 3. Dr. Callaghan also defended his invocation of Doneddu I as unsupportive of causation, deeming it “The best study on the subject” despite limitations, and reiterating that it revealed a very low-positive association with vaccination and CIDP in comparison to other antecedent occurrences. Doneddu I at 3. Petitioner’s experts ultimately admitted that they could do no better than point to case reports—underscoring, in Dr. Callaghan’s opinion, the lack of vaccine association (since no more reliable scientific studies had yet been produced).

³⁰ Köller, however, does acknowledge the possibility that in rare circumstances molecular mimicry between gangliosides and antigenic mimics could explain CIDP’s pathogenesis, although its authors limited that to the context where the CIDP patient was shown to have been infected with the *Campylobacter jejuni* bacteria known already to trigger some forms of GBS via this process. Köller at 1351. It is undisputed that the flu vaccine does not contain any aspects of *C. jejuni*.

2. *S. Mark Tompkins, PhD.*

Dr. Tompkins acted as Respondent’s primary expert on immunologic issues raised in this case, and prepared a single written report. Report, dated June 1, 2022, filed as Ex. D (ECF No. 83-1) (“Tompkins Rep.”). He maintained the flu vaccine had not been reliably shown to be causal of CIDP.

Dr. Tompkins received his B.S. in Microbiology at the University of Illinois in 1990, and his Ph.D. in Immunology and Molecular Pathogenesis at Emory University in 1997. Curriculum Vitae, filed as Ex. E (ECF No. 83-2) (“Tompkins CV”) at 1. Dr. Tompkins then completed post-doctoral training in Immunology at Northwestern University in Chicago and later in Virology/Immunology at CBER/FDA in Bethesda, Maryland. *Id.*; Tompkins First Rep. at 1. He has held several academic positions since 2005. Tompkins CV at 2. Currently, Dr. Tompkins is a Professor of Infectious Diseases in the Center for Vaccines and Immunology at the College of Veterinary Medicine, University of Georgia, where he teaches graduate students immunology and virology, as well as train pre- and post-doctoral fellows in his laboratory. *Id.*; Tompkins First Rep. at 1. In addition, Dr. Tompkins has co-authored approximately more than 100 peer-reviewed papers and book chapters in the fields of immunology and virology. *Id.*

Like the other experts in this case, Dr. Tompkins included in his report an evaluation of Petitioner’s medical history before turning to his actual opinion. Tompkins Rep. at 1–2. Given his admitted lack of clinical or neurologic-specific expertise, Dr. Tompkins did not offer an opinion on diagnosis, but instead focused almost wholly on causation. *Id.* at 2. Overall, he disputed that a theory relying on molecular mimicry could explain how the flu vaccine would cause CIDP, as embraced by Drs. Kinsbourne and Jeret, or that a combination of mechanisms amounting to a “dysregulated immune response” (increases in pro-inflammatory T-helper cells plus imbalances in the protections usually provided by regulating immune cells), as proposed by Dr. Akbari, would produce the same pathogenic result. *Id.* at 3.

First, Dr. Tompkins denied that Petitioner’s experts had offered any general reliable evidence connecting the inactivated/unadjuvanted version of the flu vaccine (the one at issue in this case)³¹ and CIDP. Tompkins Rep. at 3–5. One article relied upon for such an association was subject to reporting bias (since it employed patient reports of post-vaccination CIDP for proof rather than independent evidence (*see generally* Kuitwaard), while another involved merely two patients out of 46 as experiencing even arguably-associated flu vaccine-induced relapses. *See, e.g.*, Pritchard at 348.

³¹ Dr. Tompkins also included in his report a section discussing Dr. Akbari’s argument about vaccine immune pathway stimulation attributable to vaccine adjuvants, noting (as I have already observed) that the flu vaccine contains no adjuvant. Tompkins Rep. at 8.

Case reports were also in Dr. Tompkins’s estimation weak proof of causation, since they only established temporal associations. Tompkins Rep. at 3–4. An article like Agmon-Levin (which did not even focus on CIDP) required a *40-year* review of literature—and even then could identify only 37 instances of temporal associations between autoimmune injuries and vaccination, underscoring the unlikely nature of a causal connection. *Id.* at 4; Agmon-Levin at 1199, 1202. And Dr. Tompkins deemed the recommendation of the “GBS/CIDP Organization” not to obtain a vaccine after incurring (purportedly) vaccine-caused disease to be not only lacking in scientific substantiation, but contradicted by other recent publications revealing a consensus among neurologists that vaccination was not likely to be harmful. *See, e.g.,* B. Roy et al., *Influenza Vaccination in Autoimmune Neuromuscular Diseases: A Survey of Current Practices and Perceptions*, 63 *Muscle & Nerve* 918 (2021), filed as Ex. D Tab 2 (ECF No. 84-2) “(Roy)”. Roy (relying on questionnaires, as with Kuitwaard) found that 60 to 66.7 percent of surveyed neurology practitioner specialists treating CIDP and other peripheral neuropathies recommended the flu vaccine to any patient without regard for the possibility of an adverse impact. Roy at 920. By contrast, broader evaluations of more than four million flu vaccine doses for adverse events, *including* CIDP, found no increased risk. *See generally* Greene.

Second, Dr. Tompkins questioned the reliability of Dr. Akbari’s view that the flu vaccine could somehow cause autoreactive T cells to spark an autoimmune attack resulting in CIDP. Tompkins Rep. at 4–6. He noted that to support this contention, Dr. Akbari invoked *in vitro* studies involving MS to show homology between wild flu peptides and T helper cells, and that the process of demonstrating the homologies was the result of an experimentally-driven “artificial selection process,” rather than reflecting what was likely to happen *in vivo*, and/or in response to the vaccine itself. *Id.* at 4; *see generally* Markovic-Plese. It could not be assumed any cross-reactivity was disease-causing due to myelin destruction, even if homology could be demonstrated. Tompkins Rep. at 6. Moreover, the specificity of these T cells to the flu peptide was uncertain, since the studies showed they responded to many other different viruses. Tompkins Rep. at 4; Markovic-Plese at 35 Table 2. The “diversity in influenza hemagglutinin sequences” of amino acids meant that consistent homology with vaccine components could often not be demonstrated, despite the experimental results of studies like Markovic-Plese. Tompkins Rep. at 5,6.³²

³² Dr. Tompkins also leveled a common criticism about literature offered by petitioners involving EAE. As he observed, EAE utilizes a particularly strong kind of adjuvant to elicit disease far in excess of what a normal vaccine would contain (and of course the flu vaccine has no adjuvant as already observed). Tompkins Rep. at 6. But this means that not only does the EAE model directly *create* “extreme inflammatory responses,” but also permits the animal immune system to encounter far greater numbers of antigenic peptide sequences, greatly increasing the likelihood of a mimic and/or cross reaction. *Id.*

I do not deem EAE studies *per se* underserving of weight simply because they involve a model that exaggerates circumstances for purposes of research. EAE is, after all, designed specifically to permit scientists to *model* an inflammatory autoimmune context in order to test various hypotheses, and the findings it can derive have significance even if obtained in an intentionally-experimental context. However, Dr. Tompkins’s criticisms illuminate why their findings are not wholly comparable to what would be experienced after vaccination.

Dr. Tompkins also observed that as a general matter, sequential homology was common in nature, but alone could not establish a likelihood for cross-reactivity leading to disease. Tompkins Rep. at 6. In fact, because it was not all that challenging for researchers to *demonstrate* the potentiality for molecular mimicry as a foundational mechanism for cross-reactivity, the real question was why autoimmunity did not regularly occur—and produce disease. Tompkins Rep. at 6; C. Benoist and D. Mathis, *Autoimmunity Provoked by Infection: How Good is the Case for T Cell Epitope Mimicry?* 2 Nat. Immunol. 9:797 (2001), filed as Ex. D Tab 4 (ECF No. 84-4) (“Benoist”). Benoist, a review article discussing two commonly-cited examples³³ of mimicry as the driver of autoimmune diseases, aimed to highlight the inherent contradiction between the putative *occurrence* of disease proceeding via this mechanism and the fact that the autoreactive T cells that would drive the disease process “remain innocuous unless somehow activated.” Benoist at 797. After discussing the case for mimicry as pathogenically-determinative in both instances, and giving due regard to the evidence favoring the contention, Benoist nevertheless concluded that “the case is not yet convincing enough to espouse,” and that the ease with which mimics could be identified only complicated the process of weighing if in fact mimicry was to blame. *Id.* at 800.

Third, Dr. Tompkins disputed the strength of Dr. Akbari’s argument that the flu vaccine could cause the upregulation of purportedly-pathogenic T helper cells, and/or create an inflammatory setting (due to cytokine increases) conducive for autoimmune disease. Tompkins Rep. at 7. Authority offered to support the latter point came from a study involving a wild flu virus infection—a context distinguishable from vaccination, since the infectious process would be far more widespread in the human body, and thereby implicate far more of an innate response (during which time cytokines would be greatly increased). J. Bermejo-Martin et al., *Th1 and Th17 Hypercytokinemia as Early Host Response Signature in Severe Pandemic Influenza*, 13 Critical Care 6:1 (2009), filed as Ex. 25 Ref. 28 (ECF No. 62-8) (“Bermejo-Martin”). Bermejo-Martin looked at levels of cytokines in the blood sera of 35 patients suffering from mild as well as severe H1N1 wild-virus-infected patients, finding that the severely-ill patients displayed cytokines associated with TH17 helper cells “commonly linked to the pathogenesis of autoimmune/inflammatory diseases,” although its authors could not identify the “exact role” these T helper cells played—including whether they were “detrimental or beneficial.” Bermejo-Martin at 2. Thus, Bermejo-Martin arguably undercuts the conclusion that the T helper cells highlighted by Dr. Akbari are necessarily pathogenic (while not supportive of the contention that vaccination stimulates them in the first place).

Dr. Tompkins detected no support from other items of literature offered by Dr. Akbari for the concept that the flu vaccine could trigger a response from pathogenic T helper cells. Lin, for example, only discussed such a potentiality in the context of an “experimental DNA vaccine” that in turn caused upregulation of cytokines specific to a kind of T helper cell (and in turn suggested

³³ One involved Lyme arthritis (in which the inflammatory process continues even after eradication/elimination of the underlying Lyme tick-borne bacterium), and the other a herpes virus-caused corneal inflammation. Benoist at 798–800.

that these T cells could be beneficial in future iterations of the flu vaccine). Lin at 80–82. A more recent study actually revealed that a different version of the flu vaccine *intended* to possess greater immunogenicity only increased some cytokine levels but not others, and that “the proportion of specific cytokine-secreting cells [the T helper cells] did not change post-vaccination.” Tompkins Rep. at 7; D. Skribinski et al., *Induction of Human T-cell and Cytokine Responses Following Vaccination with a Novel Influenza Vaccine*, 8 Sci. Rep. 18007:1 (2018), filed as Ex. D Tab 5 (ECF No. 84-5) (“Skribinski”), at 2. The current inactivated version of the vaccine, by contrast (and the one at issue in this case) was deemed by Skribinski to be “poor at eliciting [T helper cell] responses.” *Id.*

Fourth, Dr. Tompkins maintained that the flu vaccine did not likely result in “dysregulation” of certain immune cells, like T effector cells, thought to contribute to autoimmune processes when their moderating function is inhibited. Tompkins Rep. at 7–8. He noted that Dr. Akbari relied in part for this contention on evidence that CIDP symptoms had in certain articles been reported to recur after vaccination—but the literature invoked, like Kuitwaard, involved self-reporting of symptoms but with no meaningfully-increased evidence of risk. Kuitwaard at 311–13. Dr. Akbari had also offered an item of literature, Herrero-Fernandez, involving an elderly cohort’s inhibited immune response as evidencing dysfunction by the regulating immune cells, but the authors had actually observed “no statistically significant differences between vaccine responders and non-responders” post-vaccination. Tompkins Rep. at 7; Herrero-Fernandez at 2. And regardless, this latter point did not support the conclusion that the flu vaccine could *cause* immune regulatory cell dysfunction sufficient to result in CIDP—only that “Treg cells have a role in maintaining peripheral tolerance and can influence induction and recall of adaptive immune responses.” Tompkins Rep. at 7–8.

Dr. Tompkins concluded by considering the question of Petitioner’s post-vaccination onset timeframe from the standpoint of how long an immunologic process would take to unfold. Tompkins Rep. at 8. The Petitioner, he noted, had in witness statements identified an onset no later than the evening of October 31st, or a little more than three days post-vaccination. *See* Ex 19 at 2. But (and referencing the IOM Report), Dr. Tompkins maintained that antibody response to vaccination would occur no sooner than 7-10 days post-vaccination, and perhaps within three to five days if the response was hastened due to recall from a prior exposure. IOM Report at 58. Even if the shorter timeframe was applicable, Dr. Tompkins deemed three days too fast for a pathogenic T helper cell-driven reaction sufficient to “elicit clinical signs” (which here were what led Petitioner to seek medical treatment). Tompkins Rep. at 8. Even under a study creating experimental “optimal conditions used to elicit autoimmune disease” (the aforementioned EAE model) and involving the “passive transfer” of pathogenic T helper cells in an animal model, *ten days passed* before disease was elicited. *Id.*; I. Stromnes & J. Goverman, *Passive Induction of Experimental Allergic Encephalomyelitis*, 1 Nat. Protocols 4:1952, filed as Ex. D Tab 8 (ECF No. 84-8) (“Stromnes & Goverman”) at 1958. Such a timeframe did not match Petitioner’s

circumstances—and since the flu vaccine he received did not contain an adjuvant, it was likely a disease process would take even longer to manifest in symptoms. Tompkins Rep. at 8.

III. Procedural History

As noted above, this case was initiated in October 2018, and it was originally assigned to a different special master. After Respondent indicated his intent to contest entitlement, Petitioner filed Dr. Kinsbourne’s first report. The parties were subsequently ordered to brief the disputed issue of onset, since it was relevant to whether a viable flu vaccine-GBS claim had been presented. *See* Order, May 1, 2020 (ECF No. 30). While such filings were pending, this case was reassigned to me. *See* Docket Entry, July 20, 2020 (ECF No. 32).

Once that matter was briefed, I determined that no Table claim was tenable because of the CIDP diagnosis, and ordered such a claim dismissed in January 2021. ECF No. 42. However, I also observed that a causation-in-fact claim alleging that the flu vaccine had caused CIDP remained, and to that end the parties filed additional expert support and literature. They later briefed their respective positions, based on my determination that a ruling on the record was an appropriate means for deciding the case. Scheduling Order, dated December 28, 2021 (ECF No. 63). Both sides have filed their briefs, and the matter is ripe for resolution. Petitioner’s Memorandum, dated April 1, 2022 (ECF No. 73) (“Mot.”); Respondent’s Memorandum, dated June 6, 2022 (ECF No. 85) (“Opp.”); Petitioner’s Reply, dated August 31, 2022 (ECF No. 92) (“Reply”).

IV. Parties’ Arguments

A. *Petitioner*

1. Initial Memorandum — Petitioner’s brief addresses both the disputed diagnosis and the causation prongs set forth under *Althen v. Sec’y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). In support of his contention that he was properly diagnosed with CIDP, he notes that both of his neurologic experts concur with the diagnosis, and that his initial presentation and February 2016 EMG/NCS results are all consistent with the conclusion that he experienced an acute form of CIDP, presenting much like GBS at first glance. Mot. at 1–3. Its acute onset did not preclude it from being considered CIDP, especially in light of Petitioner’s subsequent course. *Id.* at 2. And Petitioner’s contemporaneous treaters all agreed on the diagnosis as well. *Id.* at 2.

Regarding the first *Althen* prong, Petitioner breaks down his causation theory into the following components: (1) the immune cells in CIDP and GBS are comparable or have similar functions in driving the respective diseases; (2) certain T helper cells secrete cytokines involved in demyelination, and those cytokines are increased by the flu vaccine; (3) immune pathway complexes a/k/a the “inflammasome” are also involved (although Petitioner again spoke of its

stimulation by adjuvants *not included in the relevant version of the flu vaccine*);³⁴ and (4) autoreactive T cells get stimulated due to sequential and structural homology between a protein component of the vaccine and myelin antigens. Mot. at 5–7. He also maintained that CIDP’s chronic features was attributable to a secondary failure of immune regulatory cells independent of the initial autoimmune process. *Id.* at 7–8.

Petitioner added that he was not in this case arguing for a cytokine-driven pathogenic process. Mot. at 20. In addition (and in an effort to address points I have made in prior cases),³⁵ he stressed that he was *not* simply maintaining that the science applicable to the flu-GBS relationship applied (although he nevertheless maintained that the fact that this case involves the flu vaccine means “there will be a stronger case for the concept that the influenza vaccination is capable in general of causing demyelinating neuropathies”). Mot. at 4.

The “can cause” prong, Petitioner maintained, was also satisfied. Treaters not only confirmed the CIDP diagnosis but ruled out other explanations (although Petitioner identified no record instances in which vaccine causation was *proposed* or considered). Mot. at 24–25. He also noted that experts like Dr. Jeret had rebutted the possibility of a fibromyalgia diagnosis, observing that it seemed to be mentioned/repeated in the record mainly as an artifact of computer records-keeping. *Id.* at 25. Otherwise, Petitioner reiterated support for the diagnosis discussed previously in the brief.

The timeframe of Petitioner’s onset was, he maintained, medically acceptable in relationship to his vaccination date. Mot. at 3–4, 25–27. He noted that Dr. Kinsbourne proposed a five-day onset (or November 2, 2015), relying on his outline of Petitioner’s initial course, and differentiating Mr. Nieves’s malaise-like symptoms (which were not deemed related) from what came later. *Id.* at 25–26. At the same time, however (and reflecting the confusion on the issue that the two neurologic/diagnostic reports submitted by Petitioner reveal), Petitioner contended that the first “objective manifestation of polyneuropathy” did not occur until November 10, 2015—twelve days post-vaccination, but still “a reasonable time interval” (and adding that five or twelve days was medically acceptable under his theory). *Id.* at 26. He also maintained that because his own “oral history” (which places onset *either* before or very close in time to vaccination) was inconsistent with the records, the latter deserve more weight in this case. *Id.* at 26–27.

Petitioner also endeavored to address some of the cases likely relevant to this one, plus a number of items of literature filed by both sides. *See generally* Mot. at 2–5, 8–24. I do not

³⁴ *See, e.g.*, Mot. at 6–7 (“there is a domino effect *from aluminum adjuvants* to inflammasome and cytokines in the IL family which enhance the amplification of Th17 cells . . . and upset the ratio of Treg / T effector cells leading to CIDP”) (emphasis added). Indeed, Petitioner’s initial memorandum goes on to note all of the literature supporting the effect of the non-included aluminum adjuvant. *Id.* at 17–20.

³⁵ *Houston v. Sec’y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *16–17 (Fed. Cl. Spec. Mstr. 2021).

summarize the arguments based on these references, however, since they often amount simply to reiterating what an item of literature *said*.

2. *Reply* — Petitioner offered a “reply” that was inexplicably longer than his initial brief (and far longer than a reply should ever be). *See generally* Reply. He stressed again that the CIDP diagnosis had preponderant support, noting the credentials of his experts to interpret EMG findings, that the primary diagnostic criteria for CIDP were met, and that ample medical record support established “hard findings” in support of the diagnosis. Reply at 1–6.

Petitioner also addressed (again) the *Althen* prongs and his success in meeting them. *See generally* Reply at 16–33. In defending against the assertion that Petitioner had not shown that the flu vaccine could cause T helper cells to release certain cytokines implicated in CIDP, he complained that Dr. Akbari had (by my order) been limited to 2022 literature (begging the question of why Dr. Akbari’s *first* report—which was under no such limitation—did not address this critical causation issue in that 30-plus page report—which was then under no such filing constraint).³⁶ *Id.* at 18. But he maintained that items like Lin, or articles on a different version of the vaccine (involving a strain common to the trivalent vaccine, but which is *adjuvanted*), confirmed the vaccine’s stimulatory capacity. *Id.* at 19–20.

Petitioner further clarified that his theory is not that the flu vaccine “dysregulates” the immune regulatory system, but instead that it impacts the “balance” of these cells (a distinction without a difference to no small extent). *Id.* at 21. He again referenced Bernard-Valnet, although (as his extensive discussion reveals) the article involved not only a different condition (narcolepsy), but an H1N1-specific version of the vaccine different from that at issue in this case. *Id.* at 22–23. He again reviewed the purported impact of the flu vaccine on the immune pathways, arguing that “still holds true” despite the fact that the vaccine lacks an adjuvant, repeating his complaint about the unfairness of literature limits (and ignoring the fact that perhaps Petitioner *should have known* the relevant vaccine lacked an adjuvant before Dr. Akbari advanced arguments depending on the adjuvant effect). *Id.* at 23–26. And he maintained that the vaccine could via molecular mimicry stimulate autoreactive T cells, even going so far as to repeat a chart contained in Dr. Akbari’s supplemental report to show the process. *Id.* at 28. He further cited the recent studies on the EBV association with MS as indirect proof of how autoreactive T cells could be stimulated. *Id.* at 29–30.

³⁶ Petitioner grossly misconstrues the context in which “limitations” on his use of literature were placed. As the docket indicates, I became concerned in 2022 that the filings in this case were starting to exceed reasonable amounts; indeed, Dr. Akbari alone offered 78 *individual items of literature* in support of his first report. I therefore warned Petitioner that any “supplemental” report from Dr. Akbari needed to avoid adding even more articles “to the pile” for the sake of a large record, but be concise and specific to anything Dr. Tompkins prepared. *See* Order, dated May 23, 2022 (ECF No. 81). Instead, Dr. Akbari coupled his excessively long responsive report with 20 “recent” items of literature—few, if any, of which are all that specific to the vaccine’s propensity to injure.

Regarding timeframe, Petitioner urges that more weight be given to medical record evidence (and in particular to the views of treaters as to when “objective” proof of neurologic symptoms were apparent to them) than to Petitioner’s affidavit and contemporaneous statements about his symptoms (although he does not explain why in this case the latter are untrustworthy). Reply at 6–7. Here, “true evidence of peripheral neuropathy” is not found before November 10, 2015 (based on the conclusions at that time of examining neurologists), and this accordingly represents the correct onset. *Id.* at 7.

Other points previously asserted in Petitioner’s initial brief were given a more expansive discussion or amplified. For example, Petitioner argued that epidemiologic evidence simply was insufficiently powered to detect the rare event of a vaccine injury—and evidence Respondent offered was either unreliable (Doneddu I) or outweighed by Petitioner’s evidence (Rajbally, Kuitwaard, and the case report evidence cited). Reply at 8–9. He re-emphasized his claim that even if GBS and CIDP were in some respects distinguishable, they could have the same etiologic trigger. *Id.* at 10–13. In so arguing, he strained to highlight the similarities between the two, and (like many other claimants before) seemed to view CIDP’s chronicity as not a meaningful distinction. *Id.* at 14 (“[o]utside of the time course, GBS and CIDP are similar”), 15–16. He deemed Respondent responsible for proving GBS and CIDP *cannot* have the same trigger (as opposed to his burden to prove the vaccine *was* the trigger). *Id.* at 16.

B. *Respondent*

Respondent contests both the propriety of the CIDP diagnosis as well as Petitioner’s success in satisfying the *Althen* prongs. Regarding the former issue, Respondent underscored Dr. Callaghan’s opinion that Petitioner likely “never had CIDP,” given that (1) IVIG did not appear overall to have been effective, (2) the diagnosis found less support over time (and particularly by late 2016); (3) EMG testing after the first (admittedly confirmatory) results in February 2016 did not support the diagnosis; and (4) Petitioner’s overall complex presentation and history of comorbidities went beyond the concept of “atypical” CIDP. *Opp.* at 16–17

Petitioner’s causation theory was also inadequately substantiated, Respondent argued. Despite Petitioner’s protestations to the contrary, his theory *was* heavily based on molecular mimicry as the pathogenic mechanism—but it had not been linked with enough specificity to the circumstances of this case. *Opp.* at 17–18. Thus, it was speculative to assume the flu vaccine’s propensity to increase cytokines (at least during the innate immune response) was pathogenic, and not otherwise shown to make a cross-attack due to mimicry more likely. *Id.* at 18–19. Indeed, the presence of specific cytokines or T helper cells in the context of CIDP did not mean they were *initially causal* of the disease due to vaccination. *Id.* at 19. And arguments about stimulation of the immune pathways were dependent on the presence of adjuvants that are not found in the flu vaccine. *Id.* at 19.

Respondent, however, had offered reasonable and reliable epidemiologic studies undercutting any association between the flu vaccine and CIDP. Opp. at 19–20, *discussing* Doneddu, Ubogu, and Mathey. And case report evidence filed by Petitioner was unworthy of significant weight, as recognized in prior Program decisions. *Id.* at 20.

The second *Althen* prong was also unmet in Respondent’s estimation. Because CIDP has no known “prodrome ‘triggers’” it was speculative to deem the vaccination preceding it in this case causal. Opp. at 21. The record contained no instances of treaters associating the vaccine with Petitioner’s CIDP. *Id.* at 22. And the onset timeframe was not medically acceptable. Even if Petitioner’s immediate malaise-like post-vaccination reaction was unrelated, Petitioner began displaying arguably-neurologic symptoms close-in-time nevertheless, as he contemporaneously reported to treaters. *Id.* at 22–23. And his own affidavit identified onset as late on October 31st—three days post-vaccination. *Id.* at 23. Dr. Tompkins had deemed such a timeframe far too soon for an immune response reliant on the adaptive, secondary generation of antibodies and immune cells to occur. *Id.*

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 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).³⁷ In this case, Petitioner was unable to advance a Table claim because of his CIDP diagnosis (as I observed in dismissing that version of the claim). See ECF No. 42.

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; see also *Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Hum. Servs.*, 931 F.2d

³⁷ 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).

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

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 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 a generally accepted medical theory. *Andreu v. Sec’y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245 (“[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one” (emphasis in original)).

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

Moberly, 592 F.3d at 1322)); *see also Howard v. Sec'y of Health & Hum. Servs.*, No. 16-1592V, slip op. (Fed. Cl. Feb. 27, 2023) (“[t]he standard has been preponderance for nearly four decades”). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (“medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

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

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical

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

B. *Legal Standards Governing Factual Determinations*

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

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and “complete” (i.e., presenting all relevant information on a patient’s health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec’y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) (“[g]iven the inconsistencies between petitioner’s testimony and his contemporaneous medical records, the special master’s decision to rely on petitioner’s medical records was rational and consistent with applicable law”), *aff’d sub nom. Rickett v. Sec’y of Health & Hum. Servs.*, 468 F. Appx. 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec’y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec’y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff’d*, 993 F.2d at 1525 (Fed. Cir. 1993) (“[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter’s symptoms”).

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

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

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

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec’y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the

factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec’y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec’y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec’y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. See *e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner’s case. Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen v. Sec’y of Health & Hum. Servs.*, 618 F.3d 1339,1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); see also *Isaac v. Sec’y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff’d*, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert’s credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); see also *Porter v. Sec’y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

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

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner’s case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec’y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec’y of Health & Hum. Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

E. *Determining Matter on Record Rather Than at Hearing*

I have opted to decide this case based on written submissions and evidentiary filings, including the numerous expert reports that have been submitted, despite Petitioner’s preference for a hearing. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions (or components of a claim) on the papers rather than via evidentiary hearing, where (in the exercise of their discretion) they conclude that the former means of adjudication will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The Federal Circuit has recently affirmed this practice. *Kreizenbeck v. Sec’y of Health & Hum. Servs.*, 945 F.3d 1362, 1365–66 (Fed. Cir. 2020). It simply is not the case that every Vaccine Act claim need be resolved by hearing—even where the petitioner explicitly so requests.

ANALYSIS

I. An Overview of Relevant Medical Terms and Prior Decisions

A. *GBS vs. CIDP*

As noted above, CIDP has been defined as a progressive, immune-mediated peripheral neuropathy that occurs due to an autoimmune attack. Dalakas at e182; Mathey at 1. It results in weakness, numbness, paresthesia, and sensory ataxia that presents as relapsing-remitting, stepwise progressive, or gradually progressive, and more often than not involves motor and sensory nerve dysfunction. Ubogu at 11; Mathey at 1. These symptoms tend to be symmetrical and involve lower and upper limbs, although variants can involve different phenotypic presentations. Dalakas at e182; Mathey at 1 (clinical presentations of variants depend on differing “immunogenetic variations”).

There is no doubt that GBS and CIDP overlap, and that the latter has often been viewed as the “chronic counterpart” of the former. Dalakas at e182. However, reliable science supports the conclusion that “those conceptions may be oversimplistic.” Ubogu at 11. In particular, GBS has an acute onset, is monophasic, and is not steroid-responsive in comparison to CIDP. Vallat at 402. CIDP can *present* acutely, but has a relapsing and meandering course—and it can be difficult to diagnose in part due to its somewhat insidious and smoldering character. *Id.* In addition, far less is known about the autoantibodies that might drive CIDP, what their human nerve tissue targets would be, and even whether those targets are shared with GBS. Mathey at 6; Ubogu at 13. Indeed, some CIDP-focused research seems to have identified nerve “nodal regions” as likely targets for attack—and (importantly) that damage to the node of Ranvier or paranode might explain some of CIDP’s presentation—whereas GBS is more generally thought to involve attacks on nerve surface myelin. Mathey at 6–7, 9 (“disruption of nodal function is likely to interfere with normal nerve excitability and membrane potentials, contributing to conduction failure”); Dalakas at e182 (“GBS represents several syndromes based on the degree of involvement of the motor or sensory nerve fibers and the myelin sheath or axon”).

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

Petitioner suggests that a focus on the differences between GBS and CIDP is mistaken, but his extensive effort to draw a line between disease “etiology” and “pathogenesis” obscured more than clarified the issue. To this end, Petitioner noted a number of reliable explanations for CIDP’s persistent/chronic course (for example, immune dysregulation (*see* Mathey at 5, Ubogu at 15)).

But such contentions are not only tentative and somewhat speculative (even if that speculation has an informed quality), but they do not reflect a clear medical consensus that GBS and CIDP are alike in all regards (including triggers) *except* for regulatory interference as explaining temporal course. At bottom, not enough is known about the complex interplay of immune processes leading to GBS to conclude that *all* peripheral neuropathies start the same way, but then diverge only due to other unrelated factors, like immune dysregulation. Indeed—even GBS *itself* is characterized by phenotypically-distinguishable variants that have totally different likely pathogenic triggers or initiators, with one not interchangeably explanatory for another.³⁸

Petitioner’s arguments about “immune cell profiling”—that “many features of immune cells participating in both GBS and acute CIDP are the same” (Mot. at 7)—were also unpersuasive. The mere fact that autoimmune diseases may feature common immune cells (macrophages, B cells, T cells, etc.) is *to be expected* in common diseases, but does not mean that the diseases share the same pathogenesis *for Program purposes*. It must be noted: in the Program, special masters seek to evaluate if it has been preponderantly demonstrated that a specific vaccine “can cause” a particular disease—and just as vaccines have different antigenic components intended to impact the immune system in different ways, so too do disease processes unfold differently, even if they have some commonalities inherent to *any* immune reaction. It simply proves too much to argue, as Petitioner does, that such commonalities erase important distinctions between different diseases that themselves often have different external triggers.

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

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

Admittedly, there are many prior cases in which Petitioners alleging the flu vaccine caused CIDP have obtained favorable results.³⁹ *See, e.g., Jastisan v. Sec’y of Health & Hum. Servs.*, No.

³⁸ As observed in Mathey, for example, “[r]ecent work of the detection of antibodies to gangliosides in the sera of patients with GBS has demonstrated that while patients with the axonal AMAN [acute motor axonal neuropathy] disease variant have reactivity against single glycolid molecules, patients with GBS with demyelinating disease do not.” Mathey at 9; *see also id.* at 6 (antibodies that drive AMAN variant not consistently found in AIDP GBS variant). In other words, the autoimmune process that causes AMAN might be comparable to the more-common AIDP form of GBS—but the precise antigens that spark the process are different for each.

³⁹ Even though prior decisions from different cases do not control the outcome herein, special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec’y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39 (2007) (“[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special

13-937V, 2016 WL 4761950, at *1–3 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). I have myself acknowledged their existence in my own prior decisions, and the fact that such determinations should be given consideration as persuasive guidance (although *settled* cases certainly lack precedential value, in comparison to reasoned decisions). See *Strong v. Sec’y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (finding that the flu vaccine can cause CIDP); *Daily v. Sec’y of Health & Hum. Servs.*, No. 07-173V, 2011 WL 2174535, at *8 (Fed. Cl. Spec. Mstr. May 11, 2011).

However, there are few persuasive *reasoned* decisions in which a special master *explained* why a causal theory associating the flu vaccine with CIDP was persuasive. Rather, special masters have tended to lump CIDP and GBS together as virtually-interchangeable peripheral neuropathies—leading them to assume that the extensive science supporting causation for GBS after vaccination applies to CIDP, but without close consideration of the actual persuasiveness of a claimant’s prong one showing, based on expert opinions or relevant literature *specific* to the injury. See, e.g., *Tomsky v. Sec’y of Health & Hum. Servs.*, No. 17-1132V, 2020 WL 5587365, at *15 (Fed. Cl. Spec. Mstr. Aug. 24, 2020) (“for purposes of this decision I merely assume but do not decide that petitioner has established a medical theory causally linking the flu vaccine to CIDP”); *Strong*, 2018 WL 1125666, at *22. As I have recently noted, this presumption is worthy of more careful analysis, if not full reconsideration. *Mason v. Sec’y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (finding a flu vaccine-CIDP causal relationship was established—but noting that “the fact that reliable science establishes an association between GBS and the flu vaccine does not inerrantly lead to the conclusion that CIDP can also be deemed to be similarly-associated”).⁴⁰

II. Petitioner Has Preponderantly Established the CIDP Diagnosis

It is often appropriate for a special master to first determine whether the alleged injury has evidentiary support before applying the *Althen* test—particularly when the injury is disputed, so that “the special master [can] subsequently determine causation relative to the injury.”

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.

⁴⁰ Some decisions from the past ten years have at least started that reconsideration process. In a 2014 case, for example, a petitioner was unsuccessful in claiming her ongoing neurological condition was aggravated by two influenza vaccinations. *Jacunksi v. Sec’y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422, at *14 (Fed. Cl. Spec. Mstr. Sept. 23, 2014) (flu vaccine did not significantly aggravate CIDP; noting distinction between strength of evidence supporting flu vaccine-GBS association and flu vaccine-CIDP connection).

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

Broekelschen, 618 F.3d at 1346. In some cases, determining the injury obviates entirely the need for an *Althen* analysis, since the petitioner’s claim, and causation theory, is dependent on a finding of a specific injury. *Id.*

Petitioner’s claim relies on the determination that he was properly diagnosed with CIDP.⁴¹ Respondent makes several persuasive points against that conclusion. I agree that subsequent electromyographic testing was not just inconsistent with the diagnosis, but eventually began to suggest Petitioner’s neuropathy had resolved. Petitioner’s lack of improvement from common CIDP treatments also undercuts the diagnosis, even if some other symptoms arguably *improved* due to the benefits of treatment. And many of Petitioner’s pre-existing comorbidities seem relevant to his presentation, complicating the conclusion that he was suffering from an independent polyneuropathy. Indeed, it appears that Petitioner’s treaters began to favor *other* explanations for his injury, such as fibromyalgia, or deemed his symptoms the secondary result of his chronic back pain and related cervical spine issues, while evidence that his CIDP was still unresolved diminished. Dr. Callaghan certainly possessed ample expertise on peripheral neuropathies, entitling his reading of Petitioner’s medical history to weight.

At the same time, the record provides substantial support for the CIDP diagnosis—at least from November 2015 to the summer or fall of 2016 (a timeframe more than sufficient to meet the Act’s six-month “severity” requirement). Of particular importance is the fact that contemporaneous treaters with neurologic expertise deemed his presentation to feature enough neuropathic symptoms to eventually lead them to propose a CIDP diagnosis—confirmed by initial EMG/NCS testing results that even Dr. Callaghan did not dispute. While subsequent testing, or Petitioner’s symptoms course, did not continue to corroborate this diagnosis fully, I accept Dr. Jeret’s explanation for why these results do not discredit the initial diagnosis. That diagnosis was never formally rejected (although it became less certain). Clearly initial treaters had trouble finding sufficient clinical signs to *embrace* the diagnosis at first, but eventually they did.

Otherwise, Dr. Callaghan’s reading of the medical record, while reasoned and based on his experience, is not derived from direct examination of the Petitioner, and occurs after the fact. I am loathe to substitute the view of an outside expert on diagnosis for that of contemporaneous treaters who saw Petitioner “in real time,” and whose thinking on his presentation should be afforded weight.

Weighing all of the above together, the record preponderates in favor of Petitioner’s contention that he likely suffered from some kind of “atypical” CIDP—a form that presented acutely enough to look (initially) a bit like GBS except for its subsequent chronic (if fairly brief

⁴¹ I note that (at the time of my dismissal of the flu vaccine-GBS Table claim), I did not previously *find* that CIDP was established as the proper diagnosis based on the record. Rather, I indicated that the evidence that this was the injury (as affirmatively proposed by Dr. Kinsbourne) meant that a flu-GBS Table claim was not tenable. *See* Decision Dismissing Table Claim, dated Jan. 11, 2021 (ECF No. 42), at 5. But in dismissing the Table claim, I observed that Respondent did not agree with CIDP as the proper diagnosis in any event, and had raised fact disputes relevant thereto. *Id.* at 6.

for CIDP) course, and one that featured pain and some sensory issues over other features. As special master, I am not called upon (nor do I possess the necessary qualifications) to diagnose claimants myself; rather, I evaluate the evidence to see whether it preponderantly supports a proposed diagnosis. Here, despite Respondent’s fair questions, I find that evidence favors the CIDP diagnosis.

This finding underscores how preponderance works in “real world” contexts. In this case, ample reliable evidence is inconsistent with the CIDP diagnosis, but the *overall* balance of evidence supports Petitioner on this issue. This is the essence of the weighing special masters perform; a finding for one side over the other on a specific disputed issue occurs when the evidence “tips” the scale in one direction, despite reasonable evidence going the other way. The same method of weighing—in which items of reliable evidence exist on both sides, but weighing of all items *in toto* favors one outcome over the other—applies to the *Althen* prongs, but in this case does *not* support causation, as discussed below.

III. Petitioner Has Not Carried His Causation Burden of Proof⁴²

A. Althen Prong Three

Entitlement in the Program cannot be established unless all three *Althen* prongs are preponderantly satisfied. *Althen*, 418 F.3d at 1278. In this case, the strongest basis for dismissal is the fact that the timing of Petitioner’s post-vaccination neurologic symptoms onset has not been shown to be medically acceptable (assuming for a moment that the flu vaccine can cause CIDP).

There is no defined period for what would constitute a medically acceptable onset for CIDP after receipt of the flu vaccine. Persuasive authority suggests, however, that an onset beyond a week has sufficient medical and scientific support to meet the third *Althen* prong. *See, e.g., Kelley v. Sec’y of Health & Hum. Servs.*, 68 Fed. Cl. 84, 102 (Fed. Cl. 2005) (CIDP onset approximately two weeks after vaccination); *Daily*, 2011 WL 2174535, at *9 (finding that onset of CIDP within a few weeks of vaccination was a medically acceptable timeframe). Timeframes that are substantially longer, by contrast, would not be medically acceptable. *Patel v. Sec’y of Health & Hum. Servs.*, No. 16-848V, 2020 WL 2954950, at *18-21 (Fed. Cl. Spec. Mstr. May 1, 2020) (seven months for vaccine-caused CIDP too long); *Strong*, 2018 WL 1125666, at *21 (four months between flu vaccine and onset of CIDP was too long).

It is also the case, however, that *short* onsets are grounds for dismissal, where the timeframe from vaccination to onset is too fast for an aberrant immune response to have likely occurred. *Rowan v. Sec’y of Health & Hum. Servs.*, No. 17-760V, 2020 WL 2954954 (Fed. Cl. Spec. Mstr. Apr. 28, 2020) (36-hour GBS onset after receipt of flu vaccine not medically acceptable); *Orton v. Sec’y of Health & Human Servs.*, No. 13-631V, 2015 WL 1275459, at *3–4 (Fed. Cl. Spec. Mstr. Feb. 23, 2015) (one-day onset of GBS after flu vaccine administration not substantiated with

⁴² I address the *Althen* prongs in order of their significance to my determination.

expert opinion). While there are some contrary determinations, they are specific to the facts presented, and thus do not stand as persuasive evidence that medical science would support a very abrupt post-vaccination onset. *Compare Davis v. Sec'y of Health & Hum. Servs.*, No. 14-978V, 2022 WL 1654743, at *55 (Fed. Cl. Spec. Mstr. Apr. 27, 2022) (one-day onset of CIDP after flu vaccine found medically acceptable; petitioner suffered from uncontrolled Type II diabetes that would have made it more likely the vaccine synergistically interacted with claimant's existing diabetic neuropathy, and record established that this had occurred).

An initial deficiency with Petitioner's prong three showing is his over-reliance on "GBS science" to establish a medically acceptable period for post-vaccination onset. Petitioner did offer some reliable literature and testimony regarding the expected post-vaccination onset of GBS. *See generally* First Akbari Report at 27–28; Park at 11, 55–56. But as discussed, I do not accept as preponderantly established the contentions of Petitioners' experts that GBS and CIDP differ only as to chronicity. Indeed, the acute nature of GBS somewhat suggests that onset will be *faster* there than in CIDP (and the "acute CIDP" characterization of Petitioner's injury does not wholly obviate that problem). It is also significant that GBS and CIDP have not been shown to share the same antecedent triggers; indeed, it is not even evident what CIDP's most likely triggers are.

As a result, "what is known about GBS's onset timeframe cannot simply be borrowed as a template for how long vaccine-caused CIDP would take." *Mason*, 2022 WL 600415, at *25. Nor is the Table timeframe for flu vaccine-GBS onset applicable in the context of a non-Table case. *Grant*, 956 F.2d at 1148 ("similarity of a petitioner's injury to those listed on the Table does not show causation in fact"); H.R. Rep. No. 908, 99th Cong., 2d Sess., pt. 1, at 15 (1986), *reprinted in* 1988 U.S.C.C.A.N. 6344, 6356 ("the petition must affirmatively demonstrate that the injury or aggravation was caused by the vaccine. *Simple similarity to conditions or time periods listed in the Table is not sufficient evidence of causation*") (emphasis added).⁴³ And this is true even if, as I have found, Petitioner experienced a form of CIDP that presented acutely—"acutely-presenting CIDP" is still not GBS.

Dr. Akbari provided the most substantive opinion on the nature of the immune processes that might begin with vaccination and later result in CIDP. That causal theory posits a combination of factors working in concert: that the flu vaccine stimulates T helper cells and/or certain cytokines associated with CIDP; that existing autoreactive T cells responsible for direct demyelination are also stimulated as a result of mimicry, vaccination, and/or immune pathway/inflammasome stimulation; and that the vaccine would encourage a breakdown in function of immune regulatory cells (or this would simply occur once the vaccine had started the process). While his theory does not explain with full clarity the timeframe in which this process would occur, it is reasonable to expect such a cascade of events resulting in the clinical manifestation of neurologic symptoms

⁴³ Thus, it does not avail Petitioner in this case that a *Table* flu-GBS claim has a 3-42 day onset timeframe. Not only is there no Table claim for CIDP, but (as I ruled in dismissing the flu-GBS Table claim earlier in the case's history), a CIDP diagnosis is an exclusionary factor. ECF No. 42.

reflective of an ongoing demyelination process would take at minimum several days to a few weeks to unfold. *See also Blackburn v. Sec'y of Health & Hum. Servs.*, No. 10-410V, 2015 WL 425935, at *22 (Fed. Cl. Spec. Mstr. Jan. 9, 2015) (discussing CIDP onset); Tompkins Rep. at 8.

Petitioner thus maintains that onset occurring within five to ten days post-vaccination, or even a bit longer, would constitute a medically acceptable timeframe. Mot. at 25–27. And I agree that preponderant evidence establishes that such a timeframe is reasonable. *But the record in this case does not allow for the conclusion that Petitioner's onset occurred within such a timeframe.* Rather, it supports onset beginning the late evening of October 31, 2015—as specifically alleged by Petitioner. *See Nieves Aff.* at 1–2. This is slightly more than 72 hours, or *three days*, post-vaccination. It was at this time Petitioner first recalls experiencing the kinds of symptoms that might seem neurologic in character.

The record clearly supports Petitioner's contention that his *immediate* malaise-like symptoms were distinguishable from what came after, and thus did not constitute his CIDP onset. But Petitioner consistently reported to treaters that he had been experiencing symptoms “since” vaccination—not that there was a quiescent period before his neurologic symptoms emerged.⁴⁴ Thus, even if there was some handoff-overlap between common post-vaccination issues, like fever, and the neurologic onset symptoms reflecting CIDP, Petitioner likely experienced onset too soon for the proposed complex immune process to have begun due to vaccination *and* result in the manifestation of symptoms that could have been observed. *See Tompkins Rep.* at 8, *citing Stromnes & Goverman*. Importantly, Stromnes & Goverman involved the EAE model, in which artificial conditions for an autoimmune reaction are lab-created for experimental purposes—and *even there* (where a notably-strong adjuvant⁴⁵ is employed in order to create the circumstances for an observable disease process), the aberrant reaction takes *more than five days* to occur (and likely longer). Stromnes & Goverman at 1958.

Petitioner's neurologic experts also proposed an onset date, but they did not persuasively establish a later onset was more likely—and even disagreed to some extent as to what the likely onset was in the first place. Dr. Kinsbourne, for example, initially vouched for a *five-day* onset. First Kinsbourne Rep. at 10. Yet in his next report (when confronted with the fact that Petitioner had reported neurologic-like symptoms in a timeframe that would place them as *beginning* at the

⁴⁴ Petitioner attempts to deny the accuracy of his multiple statements to treaters of neurologic symptoms “since” the vaccine. *See, e.g.*, Ex. 3 at 67–69, 76–77, 96; Ex. 5 at 1. However, while I agree that he has established that his *immediate* post-vaccination malaise was unrelated, I do not find persuasive his efforts to vary contemporaneous evidence of what he told treaters. There is ultimately a symptoms “continuum” in this case, beginning the day of vaccination itself—and the record preponderantly supports the conclusion that his neurologic symptoms began October 31st, which is consistent with his reports of symptoms “since” vaccination.

⁴⁵ EAE utilizes “complete Freund's Adjuvant,” an “oil-in-water emulsion containing Mycobacterial cell wall components, [and] is regarded scientifically as an effective means of potentiating humoral antibody response to injected immunogens.” *Complete Freund's Adjuvant*, Office of Research and Economic Development, <https://www.csulb.edu/office-of-research-and-economic-development/policy-use-of-complete-freunds-adjuvant-cfa> (last visited Apr. 17, 2023).

time of vaccination, and certainly sooner than five days) he equivocated, arguing for an onset closer to November 9th, or nearly *12 days* post-vaccination—but in doing so seemed to rely more on when symptoms could be *understood* by treaters as CIDP, rather than the first manifestation of a neurologic issue. Second Kinsbourne Rep. at 5. Dr. Jeret similarly focused on when Petitioner began to receive “the most reliable neurological examination[s]” (First Jeret Rep. at 13), since, in his estimation, competent specialists were better able to make sense of Petitioner’s course. Petitioner’s briefing also seems to put far more faith in the “objective findings” by treaters than Petitioner’s sworn statements. *See, e.g.*, Mot. at 26.

But all of these arguments make the mistake of conflating *onset* with *diagnosis*. It is well understood in the Program that the two are not the same. *Wetz v. Sec’y of Health & Hum. Servs.*, No. 07-633V, 2012 WL 3967106, at *3 (Fed. Cl. Spec. Mstr. May 31, 2012) (citing *Brice v. Sec’y of Health & Hum. Servs.*, 36 Fed. Cl. 474, 477 (Fed. Cl. 1996)). Any initial symptom or manifestation is sufficient to constitute onset, no matter whether the claimant or treating medical professionals *identify it* as significant or as the basis for a specific diagnosis. *See Tenneson v. Sec’y of Health & Hum. Servs.*, 142 Fed. Cl. 329, 338 (2019). Thus, the date a treater affirmatively *recognizes* that a collection of ambiguous symptoms supports a particular diagnosis is distinguishable from when any individual symptom *first* manifested—with only the latter being relevant to onset, for Program purposes.

Here, Petitioner’s treaters may not have grasped that Mr. Nieves’s initial symptoms were neurologic, and may have given some evidence or exam results more weight than others in trying to explain his condition. They clearly were not comfortable with embracing the CIDP diagnosis at first (consistent with Dr. Callaghan’s observation that CIDP is prone to misdiagnosis). But this does not mean onset of his allegedly vaccine-caused symptoms had not likely already *occurred*. On the contrary—Petitioner (accurately, in hindsight) *understood* his neurologic symptoms to be different from mere vaccination malaise, so much so that he repeatedly sought care for them in the span of two weeks post-vaccination. The record thus strongly establishes that Mr. Nieves experienced neurologic symptoms *far too soon to implicate the vaccine*. His diagnosis firmed up over time, but that does not shift onset to a later date more favorable to Petitioner.⁴⁶

⁴⁶ There is also the substantial issue of Petitioner’s numerous pre-vaccination symptoms, many of which echo his “atypical” CIDP presentation. CIDP is well-understood to often have an insidious onset. *Blackburn*, 2015 WL 425935, at *22. Indeed, experts like Dr. Kinsbourne stated that one reason antecedent causes like an infection are often not identified for CIDP is precisely *because* its symptoms may take a substantial period of time to manifest after an earlier, but ignored or mistakenly-disregarded, trigger. Kinsbourne First Rep. at 4–5.

Given his medical history, it is conceivable Petitioner’s CIDP actually *predated* vaccination by some period of time, but was not recognized as CIDP until after vaccination. I emphasize that *I do not so preponderantly determine this* from the record, since there is no convincing/preponderant evidence of pre-vaccination neurologic symptoms consistent with what was later relied upon for his CIDP diagnosis, and Respondent’s experts have not otherwise shown persuasively that his complicated health history provides an alternative cause explanation. But Petitioner’s numerous and documented comorbidities complicate his arguments about onset.

By contrast, Dr. Tompkins persuasively (and quite succinctly) explained what was deficient about the onset timeframe in this case. *See generally* Tompkins Rep. at 8; Ex 19 at 2. A three day post-vaccination response was simply too fast for *any* of the processes Petitioner’s experts outlined (some combination of vaccine stimulation of T helper cells and cytokines leading to autoreactive T cell attacks, coupled with stimulation of immune pathways and interference with immune regulation) to begin, complete, *and* manifest with outward neurologic symptoms. *See Rowan*, 2020 WL 2954954, at *17 (discussing timeframe “lag and log” components in which immune processes resulting in autoimmune attack will unfold—and that the process would not occur within a day or two). The time it would actually take for an autoimmune process leading to disease was better illustrated by the kind of experimental animal models invoked in many of Petitioner’s filed items of literature. Stromnes & Goverman at 1958. Even in that context (where provoking an autoimmune disease response was the *intended* result of experiments, aided by use of a powerful adjuvant), the timeframe involved was considerably longer. Tompkins Rep. at 8.

Petitioner offers two arguments against an early onset finding, but both are unavailing. First, he proposes that I should effectively disregard his affidavit (or contemporaneous statements to treaters regarding his onset for that matter), and focus instead on the records in which treating neurologists *confirmed* objective evidence on exam that supported the CIDP diagnosis. Reply at 6–7. Putting aside the fact that this argument is highly unusual for a claimant to embrace in a Program case,⁴⁷ and that it partially relies on the conflation of treater confirmation/diagnosis with onset discussed above, it misapprehends the weighing of evidence that special masters perform when evaluating fact issues like onset. That process is based on a number of interdependent core concepts: that records are *not* presumptively correct, even if they deserve weight; that contemporaneous record statements about disputed issues (especially reflecting what a claimant told a treater about the symptoms he was experiencing) should be in most cases read as honest statements of a claimant’s health status at the time, even if they report subjective issues (like symptoms nature or timing) not formally confirmed by tests or treaters; and that witness statements, while not sufficient *alone* to prevail on entitlement, can in tandem with record evidence be highly probative of a fact issue.

Here, I have engaged in such a weighing of disparate items of evidence. What I conclude from this analysis is that *in addition* to Petitioner’s reporting of post-vaccination malaise (which I agree is not evidence of CIDP onset), Petitioner made several contemporaneous statements post-vaccination that his onset had occurred close to the date of vaccination or not long after. *See generally* Ex. 3 at 67–69, 76–77, 96; Ex. 5 at 1. The symptoms he described therein were

⁴⁷ In my experience as a special master, Petitioners usually hope to *vary* (or at least add nuanced details to) what a record says (especially when it directly rebuts their preferred onset date), by offering either their own sworn statement or affidavits/declarations from other witnesses. Special masters are routinely urged to give more, or at least equal, weight to such witness statements. And as my discussion of the legal considerations involved in fact-finding in Program cases reveals, there is a well-developed line of decisions going all the way to the Federal Circuit evaluating the circumstances in which witness statements deserve weight when compared to contemporaneous record evidence.

qualitatively different from mere vaccination malaise. In addition, he *chose* in his October 2020 affidavit (created two years after the case’s filing) to provide a detailed history that placed onset *three days* post-vaccination.⁴⁸ This is more than enough to determine, based on the preponderance standard, that his onset was likely earlier than what is now argued, even if treaters did not *diagnose* his CIDP until they obtained more concrete proof of neurologic issues (in the form of their own exams of Petitioner).

Second, Petitioner observes that in the course of his obtaining neurologic-specific treatment, there were instances in which he did not display “objective” neurologic symptoms or signs on exam. Reply at 7. In particular, he was negative for numbness, tingling, or weakness on November 9, 2015, but objectively demonstrated it the next day. *Id.* at 7; Ex. 3 at 67–70; Ex. 9 at 19–20. But this inconsistency does not preclude an earlier onset date. Any illness might feature days in which symptoms surge or are more difficult to endure than other days—especially CIDP, which is known to feature relapse and/or waxing and waning of symptoms. And while it is true on this record that Petitioner’s presentation was confusing overall, making it difficult for treaters to identify an explanation for his illness, it remains the case that treater *confirmation* of illness does not determine onset. I also reiterate that Petitioner’s history shows he was consistently concerned about his health post-vaccination, enough to seek medical intervention repeatedly. The relapsing, intermittent nature of CIDP is consistent with a waxing and waning of symptoms over time- and with symptoms appearing, subsiding, and then appearing again.

In short, the proposed *general* timeframe for CIDP’s onset under Petitioner’s theory has some reliable scientific support—but this record does not permit me to conclude that *Petitioner’s* CIDP more likely than not occurred *within* that five-to seven-plus day timeframe—or that it could have begun so close in time to the vaccination date. The vaccine-triggered immune processes posited to have caused his CIDP would not have had time to begin, and *also* cause the symptoms manifestations reported as of October 31, 2015, barely three days after vaccination.

B. Althen Prong Two

The medical record in this case does not allow for the conclusion that the flu vaccine likely “did cause” Mr. Nieves’s CIDP. First, no treaters ever proposed any association between the October 2014 vaccinations and Petitioner’s subsequent diagnosis. Just as I have credited treater opinions that Petitioner’s overall presentation was consistent with CIDP, despite Dr. Callaghan’s reasoned objections, the *lack* of treater support for a vaccine association cannot be ignored at the same time. Instances in the record in which Petitioner himself *reported* an association during a medical visit are of course not probative, since his personal views are not informed by medical or

⁴⁸ Because that affidavit was prepared at a time when the focus in this case was on whether *Table* onset for GBS (3-42 days) could be met, Petitioner may have had an incentive to stress that timeframe. But Petitioner cannot have his cake and eat it too, abandoning his prior sworn statements because they no longer assist him after the claim has been defined more narrowly.

scientific training—and the same goes for memorialization of such instances by treaters in history sections of subsequent records.

Second, other than the fact that Petitioner received a CIDP diagnosis within a month or so of his vaccination, nothing in the record corroborates the theory that an aberrant immune response was occurring *due* to vaccination at this time. No exam determination, test result, or other diagnostic findings⁴⁹ provide reason to accept the argument that the vaccine was more likely than not the *explanation* for Petitioner’s neurologic complaints. Petitioner did experience some malaise-like reaction to the flu vaccine, but that has not been shown to be associated with Petitioner’s later-diagnosed neurologic injury. On the contrary: experts like Dr. Kinsbourne went out of their way to *differentiate* this initial malaise from Petitioner’s other symptoms (in the aim of rebutting the conclusion that he had experienced onset too close in time to vaccination for the vaccine to be causal). There is simply no evidence to corroborate the theory working “in real time.”

In addition, Petitioner’s medical history is replete with reference to a number of comorbid conditions that greatly complicate Petitioner’s success in satisfying the second *Althen* prong. Even before the vaccination in question, Mr. Nieves had experienced cervical issues, ultimately requiring surgery, that explained persistent neck pain, and he was thought to possibly have suffered from fibromyalgia as well (although Petitioner’s experts correctly observe that this diagnosis is not fully corroborated). Ex. 2 at 2–3; Frist Jeret Rep. at 7–8. He also experienced some neurologic-like symptoms in the year before vaccination. As Dr. Callaghan opined, the number of “atypical” features of Petitioner’s CIDP were noticeably large—and by the time Petitioner had undergone several follow-up EMG/NCS tests, treaters were revisiting the possibility of alternative explanations beyond a polyneuropathy. Second Callaghan Rep. at 2–3.

I have found it more likely than not that Petitioner experienced some kind of CIDP, and Petitioner’s myriad comorbidities cannot be preponderantly established as an alternative cause for his neurologic condition. But the nature of Petitioner’s health history greatly confounds a narrative of *vaccine-caused* CIDP, since woven into that history before and after vaccination are many strands of non-neurologic symptoms and conditions that cannot be neatly separated herein, and which may have had some relationship to his post-vaccination symptoms—as missed by treaters as Petitioner’s initial neurologic symptoms were not understood to reflect CIDP. Petitioner displayed a wide array of pre-vaccination symptoms that reasonably could be associated with a meandering, progressive neuropathic course that did not fully manifest until after after vaccination (with receipt of the vaccine merely occurred in the *midst* of this unfolding disease process).⁵⁰ This

⁴⁹ For example, proof of the presence of allegedly-causal antibodies is lacking (although the absence of such evidence is ameliorated somewhat by a lack of scientific awareness of what the specific autoantibodies associated with CIDP would be in the first place).

⁵⁰ In fact, Petitioner’s smoldering history of consistent issues pre-vaccination, some of which echoed as neurologic in character, is somewhat consistent with the waxing and waning associated with CIDP, and a pathologic course that could slowly, intermittently progress over a period of weeks to months before treaters could identify it fully based on clinical presentation and testing. *Blackburn*, 2015 WL 425935, at *27 (denying compensation due to petitioner’s CIDP

aspect of the record weighs somewhat against Petitioner (although I cannot find that this constitutes an “alternative cause,”⁵¹ and I do not give this evidence as much weight as the aforementioned considerations).

I am also unpersuaded by the contention that the lack of an identified alternative cause (like proof of an infectious process) leaves the vaccine as the only likely cause. Claimants do not prevail merely because no other potential cause has been identified, let alone proven. *Bender v. Sec’y of Health & Hum. Servs.*, No. 11-693V, 2018 WL 3679637, at *34–35 (Fed. Cl. Spec. Mstr. July 2, 2018) (noting that just because other alternatives are ruled out did not mean the vaccine caused the theory), *mot. for review denied*, 141 Fed. Cl. 262, 267 (2019). In the end, Petitioner mostly invokes the *post hoc* fact of his illness as proof the vaccine was causal—a circular argument that goes against the Program’s recognition that *not all post-vaccination illnesses are vaccine-caused*—even when there is a sound reason to associate the vaccine with the injury. *See Capizzano*, 440 F.3d at 1327.

C. Althen Prong One

Evaluation of the first *Althen* prong presents a far thornier question, and not simply because of the unnecessarily-vast amount of evidence offered by Petitioner.⁵² This is particularly due to the fact that (as I discuss above) many prior Vaccine Act decisions favor a flu vaccine-CIDP association. I am strongly reluctant to disregard such prior determinations, even though it is my conclusion that their reasoning is not fully persuasive. It is mistaken to view GBS and CIDP as

beginning before vaccination, even if it could not then be diagnosed). It is not uncommon for treaters to have difficulty identifying CIDP, formally embracing the diagnosis only once it is clear that the patient’s symptoms are not proceeding in a monophasic fashion, but instead have become chronic in nature. *Daily*, 2011 WL 2174535, at *1 (petitioner began to experience neurological symptoms that were initially diagnosed as GBS, but after several relapses and years of partially effective or ineffective treatment, his diagnosis was changed to CIDP). I do not on this record, however, purport to find that Petitioner’s CIDP predated vaccination, as there is insufficient preponderant evidence to allow that conclusion.

⁵¹ It is well-established in the Program that *if* a petitioner carries his initial *Althen* burden, the burden shifts to Respondent to prove an alternative cause. *Stone v. Sec’y of Health & Hum. Servs.*, 676 F.3d 1373 (Fed. Cir. 2012). Petitioners are not required to eliminate evidence of an alternative cause, or fully rebut it in their case in chief. But this does *not* mean that evidence pertaining generally to a claimant’s health bearing on causation can only be considered after a prima facie case for entitlement has resulted in such burden shifting. *Snyder v. Sec’y of Health & Hum. Servs.*, 553 F. App’x 994, 1000 (Fed. Cir. 2014) (“ ‘no evidence should be embargoed from the special master’s consideration simply because it is also relevant to another inquiry under the statute’ ”) (*quoting Stone*, 676 F.3d at 1380); *see also de Bazan*, 539 F.3d at 1353 (“[t]he government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner’s evidence on a requisite element of the petitioner’s case-in-chief”). Rather, it has some relevance to a petitioner’s success in establishing a vaccine-caused injury, and I may include it in my weighing generally.

⁵² The Petitioner filed a total of 168 articles in this case, along with two reports from his primary prong one expert, Dr. Akbari, exceeding 60 pages in total. This is facially excessive—especially given my admonition to Petitioner to limit new filings in association with Dr. Akbari’s second report.

opposite sides of the same coin, as has too often been assumed—but this view is not without *some* logic.

Dr. Akbari was the only one of Petitioner’s experts with sufficient expertise to opine credibly on the immunologic issues relevant to causation.⁵³ But his overall theory amounts to a confusing, inter-related patchwork of contentions, backed up with proof that obscured more than illuminated. He attempted to spin medical evidence about *features* of CIDP (often relying on limited studies regarding the blood sera findings for CIDP patients) into an explanation for how an aberrant immune process caused by a vaccine could *lead* to CIDP. But evidence that vaccines might stimulate production of some immune cells, or even interact with them, does not mean that the vaccine causes the injury—even given *other* reliable proof that these same immune cells are found in the blood sera of CIPD or GBS patients. Petitioner’s theory simply assumes that one is connected to the other (and invokes the rarity of vaccine disease⁵⁴ to avoid close scrutiny of any evidentiary deficiencies). Elsewhere, the causation theory merely assumes that a showing about how the immune system is *expected* to function explains the potentiality for aberrant responses—but without proof for the former.

There were also several erroneous foundational contentions in Dr. Akbari’s opinion. In particular, his first report (as well as Petitioner’s initial brief in this case) mistakenly assumed that the flu vaccine at issue is adjuvanted, and relied on literature discussions of how the *adjuvant* impacts the inflammasome for this aspect of the theory. *See, e.g.*, First Akbari Rep. at 20–21; Mot. at 6, 17, 19–20. Petitioner subsequently recognized his error and attempted to address it, but it is clear that the arguments about inflammasome stimulation did depend on studies involving adjuvant impact.

⁵³ I do not give the prong one causation opinions of Drs. Kinsbourne or Jeret nearly the same weight—and they do not bear detailed discussion as a result. While both experts had sufficient *neurologic* expertise to opine on the question of diagnosis (and to offer their own readings of the medical record to that end), neither have demonstrated backgrounds in molecular biology or immunology. In fact, Dr. Kinsbourne’s opinion arguably deserves *even less* weight than Dr. Jeret’s. Respondent correctly observed that the aspect of Dr. Kinsbourne’s reports devoted to the “can cause” question largely reflected a literature search he personally performed for this case, and did not flow from his demonstrated expertise studying immunology and/or the role vaccines might play in the pathogenesis of a peripheral neuropathy. Resp. at 17 n.5. Dr. Kinsbourne’s value as a Program expert has in fact been reasonably questioned for many years. He has been criticized in prior cases for lacking up-to-date experience seeing patients, and certainly is not even a demonstrated expert in the topic of peripheral neuropathies, despite his medical background in neurology. *Stone v. Sec’y of Health & Hum. Servs.*, 676 F.3d 1373, 1376 (Fed. Cir. 2012).

⁵⁴ The fact that vaccine injuries are generally rare cannot be converted into a “rare cases” defense, shielding Petitioner from his evidentiary obligations. The oft-quoted phrase “in a field bereft of complete and direct proof of how vaccines affect the human body” simply does not free claimants from proving preponderantly that the vaccine can cause the injury in question. *Althen*, 418 F.3d 1280. The phrase simply means that a variety of items of circumstantial evidence may be offered (since direct proof will be hard to come by), and that collectively they can add up to a preponderance. At bottom, a petitioner’s evidentiary burden does not function as a “sliding scale,” adjusting downward whenever the state of scientific knowledge on a question of causation is incomplete or limited. *Caves v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 143 (2011), *aff’d*, 463 F. App’x 932 (Fed. Cir. 2012).

Petitioner’s invocation of molecular mimicry was especially confounding. Dr. Akbari did not offer the more-common expert contention often heard in Program matters: that the vaccine’s antigens contain protein sequence mimics of self-antigenic targets, with the result that antibodies manufactured by the immune system in reaction to the vaccine would mistakenly cross-attack the self-antigens due to the similarity. Indeed, he protested that it was too difficult to do so. Instead, he attempted to leapfrog the antibody causation “step” entirely, focusing on existing “autoreactive” T cells that drive disease (which he proposed were more critical in the nerve damage wrought by neuropathies in any event). *See, e.g.*, First Akbari Rep. at 13–14, 17, 21. But he still maintained that molecular mimicry had some role to play in the disease process—without specifying how. *What* in the vaccine was a mimic (whether sequentially or structurally) in the vaccine, and to *what* antigenic target? Why would this mimic stimulate existing autoreactive T cells? It remains wholly unclear from the Petitioner’s theory, which seems to hope that a combination of discussion of cytokines, T cells, and the impact of vaccination, adds up to causation.

The invocation of immune cells responsible for immune reaction regulation (T effector cells and Treg cells) as impacted by vaccination in some way was also confusingly presented, but ultimately irrelevant for causation purposes. Dr. Akbari offered reliable proof supporting the contention that CIDP’s chronicity *may* be attributable to inhibition of this regulatory function. *See, e.g.*, Dalakas at 318. And it is credible to contend, as Petitioner did, that individual genetic variance probably plays *some* role in why some individuals experience the chronicity and recurrence that characterizes CIDP. But do vaccines encourage this dysregulation, or have anything to do with it at all? It is unclear from his lengthy reports whether this can be deemed likely. At most, this discussion was merely a way to deflect the obvious difference between CIDP and GBS, by attributing CIDP’s chronicity to an independent factor (although one that at the same time could be impacted by vaccination). And it seems Petitioner would otherwise contend that because he experienced “acute” CIDP, its vaccine causation could occur even if the disease later became chronic for other reasons.

Petitioner’s experts over-relied on case report evidence—a kind of proof that may not be appropriately disregarded entirely, but which clearly does not deserve *significant* weight otherwise. *See, e.g.*, *Campbell v. Sec’y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) (“[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value ... [but] the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight.”). While I am fully cognizant that epidemiologic studies may not in every case exist (true here, despite Respondent’s invocation of Doneddu I—a study I have criticized in other cases, and which I do not give great weight to herein)⁵⁵—and certainly Petitioners are never *required* to offer epidemiologic evidence to

⁵⁵ *Houston*, 2021 WL 4259012, at *18 (giving Doneddu I limited weight since it merely found that a *small percentage* of CIDP patients in the study had been vaccinated before onset, without being able to reach reliable causation conclusions about the observed lack of relationship).

succeed—case reports are not the equivalent of a reliable large study. And if they are not bulwarked with *other* reliable proof, whatever the form or nature, case reports cannot by themselves constitute the basis for a causation finding, since they do little more than delineate an instance in which an injury *temporally followed* vaccination. It is no defense to this point to argue that case reports are the “best” evidence available (as Dr. Jeret maintained). *See* Second Jeret Rep. at 2.

In the end, I was not strongly *persuaded* by Petitioner’s causation showing, which was needlessly complex and annotated by an unreasonable number of items of literature. However, I am ultimately reluctant to disregard the fact that numerous prior Program decisions have found CIDP to be caused by the flu vaccine. In addition, Respondent’s expert on the immunologic issues in contention, Dr. Tompkins, did a better job of picking off smaller points made by Dr. Akbari than in rebutting overall the conclusion that CIDP could be flu vaccine-associated. Too often, he seemed to take refuge in the contention that “you did not prove it,” rather than making a more comprehensive case against causation, despite the GBS-CIDP links. On this matter, Petitioner’s overall showing had enough scientific reliability to be taken seriously, and raise more than a plausible possibility that *some* of the GBS findings have significance in the CIDP context, despite the difference between the two.⁵⁶

I therefore deem the showing on the first *Althen* prong in this case to be one in which an *extremely* close call is presented—and I am compelled in such circumstances to find this prong satisfied. This hardly “closes the door” on the question, however—as my foregoing discussion of my numerous reasoned reservations with the effectiveness and persuasiveness of Petitioner’s showing should make clear. Whether CIDP is likely caused by the flu vaccine is still unresolved scientifically—and a more robust showing by Respondent on that matter might tip the balance the other way, despite the Program’s history of treating flu vaccine-CIDP cases. That kind of showing was simply not made *here*.

IV. This Case Was Appropriately Decided on the Papers

In ruling on the record, I am choosing not to hold a hearing. Determining how best to resolve a case is a matter that lies generally within my discretion, but I shall explain why a hearing was not required.

Prior decisions have recognized that a special master’s discretion in deciding whether to conduct an evidentiary hearing “is tempered by Vaccine Rule 3(b),” or the duty to “afford[] each party a full and fair opportunity to present its case.” *Hovey v. Sec’y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 400–01 (1997) (citing Rule 3(b)). But that rule also includes the obligation of creation of a record “sufficient to allow review of the special master’s decision.” *Id.* Thus, the fact that a

⁵⁶ The attempt to leverage what is known about the flu vaccine’s association with GBS is far less persuasive in the context of different vaccines—and in such cases I would be disinclined to give the same weight to existing Program decisions assuming, in effect, that all vaccines equally cause demyelination.

claim is legitimately disputed, such that the special master must exercise his intellectual faculties in order to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes without a hearing—although they should only so act if a party has been given the proper “full and fair” chance to prove their claim.

The present claim could be (and was) resolved fairly without the need for live testimony from the experts. I was able to read the expert reports and filed literature, and to comprehend the theory presented, giving specific attention to some of the more recently-published items of literature. Much is known already in the Program about the flu vaccine’s relationship to GBS, and I am very familiar with how that kind of claim plays out in a non-Table context—and specifically in cases where the actual injury is CIDP. Thus, I was able to navigate the parties’ dispute, and the expert input on such matters, based on the filed record.

I am fully aware the parties disputed a number of factual issues (for example, what the EMG studies showed), but the case’s resolution did not turn on such matters. My determination on the most important fact points, like onset, could be made from careful review of the record. Petitioner may have preferred a trial, perhaps in the belief that a hearing with live witnesses would have resolved certain issues in his favor. But this is an erroneous perception. As the decider of the matter, I can forthrightly represent that hearing live from the experts would not have altered the conclusions I draw simply from the record itself and written reports, none of which presented confusing points that live testimony could clarify. Moreover, a claimant’s desire for a trial misconstrues the utility of that means of dispute resolution in the Vaccine Program. Trial does not offer advantages to claimants that decisions on the papers prejudicially deny. Trial should be reserved for circumstances when the special master ascertains that his understanding of the case will benefit (i.e., where fact witness testimony is critical, or the scientific issues are so complex that they cannot be grasped unless the expert(s) orally explain them). The degree to which parties contest points in a Vaccine Act claim does not make a hearing more necessary.

CONCLUSION

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

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.⁵⁷

⁵⁷ 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.

IT IS SO ORDERED

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