In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-420V

(to be published)

JOY HOUSTON, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Chief Special Master Corcoran Dated: August 19, 2021

Kristi Schubert, Lamothe Law Firm L.L.C., New Orleans, LA, for Petitioner.

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

ENTITLEMENT DECISION[1]

On March 22, 2018, Joy Houston filed a petition for compensation under the National Vaccine and Injury Compensation Program (the "Vaccine Program").[2] (ECF No. 1) ("Petition"). The Petition alleged that Ms. Houston experienced chronic inflammatory demyelinating polyneuropathy ("CIDP") after receipt of a tetanus-diphtheria-acellular pertussis ("Tdap") vaccine administered on October 6, 2016, and/or a Measles, Mumps, & Rubella II ("MMR") vaccine on October 17, 2016.

The parties have requested an entitlement determination based solely on the filed medical records, plus expert reports and associated medical articles and literature, and have waived the opportunity to file briefs in support of their respective positions. Having reviewed the record, I find that Petitioner has not carried her evidentiary burden. Although Petitioner's CIDP diagnosis

[1] This Decision shall be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012)). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public. *Id*.

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

is not disputed, she has not preponderantly established that the Tdap vaccine (the vaccine Petitioner's expert focuses on) can cause CIDP, or did so here.

I. Factual Background

Pre-vaccination History

Ms. Houston was 33 years-old at the time of her vaccinations, and pregnant for the fifth time. Her prior medical history included uncontrolled insulin-dependent type 2 diabetes with neuropathy. Ex. 2 at 428. As early as 2011 (five years before the vaccinations at issue) she was experiencing tingling in her hands, and it had been proposed by endocrinologists at that time that she had a diabetic neuropathy. Ex. 20 at 271-75. She experienced more tingling incidents in 2013, and was assessed as having a peripheral neuropathy. *Id.* at 12-15, 40-49. Petitioner's preexisting diabetes (which is not alleged to have been exacerbated by vaccination) and its sequelae loom large over her alleged post-vaccination injury.

Ms. Houston was pregnant when she was vaccinated in October 2016, and the kinds of diabetic issues endemic to her general pre-vaccination medical history also impacted her pregnancy. Ex. 2 at 268-69, 274-76, 963-67, 980. Throughout this time period (spring and summer 2016), her medical treatment included both standard prenatal care and repeat visits to the Ochsner Medical Center ("OMC") emergency department, where she reported complaints of lower extremity edema and cellulitis. Ex. 3 at 9-11, 68-70, 92-94. By September 2016 she returned to OMC, now complaining of epigastric pain associated with hot and cold feeling, generalized malaise, and body aches for a day, and also sought treatment for this in connection with her OB-GYN visits. *Id.* at 119-22; *see also* Ex. 2 at 847, 850-51. Ms. Houston was eventually admitted to Tulane Medical Center, and physicians there proposed she might be experiencing preeclampsia due to increased urine protein above her baseline, treating her in part with insulin (which she had in the past irregularly taken). *Id.* at 715-16.

In mid-September, at another prenatal care visit, Petitioner's blood pressure was deemed elevated, and she was admitted as an inpatient again after back pain complaints. Ex. 2 at 477-78, 493, 682-84. Petitioner now complained of right hip/lumbosacral pain radiating to the right foot, with associated pins and needles sensation (paresthesia), and right upper abdominal pain that was associated with movement. The exam noted patellar reflexes 1+ and trace non-pitting bilateral pedal edema. *Id.* at 480-81. Ms. Houston received Gabapentin (used for nerve pain) and a narcotic, and was given instructions on relieving back strain in pregnancy, then discharged. *Id.* at 492-93, 502, 506, 509, 569-70. She returned to Tulane Medical Center again at the end of September, complaining of abdominal cramping plus epigastric pain that had been constant throughout her pregnancy, but there was no concern for severe features of preeclampsia, and she was discharged. *Id.* at 434, 436-38.

Vaccinations and Initial Post-Partum Health

On October 6, 2016, Ms. Houston received the Tdap vaccine in her left deltoid at her 33-week OB visit. Ex. 2 at 428-30. A week later, she was admitted to Tulane Medical Center for right upper abdominal, back, and right lower extremity pain. She was no longer taking nerve pain medication. On October 13, 2016, she delivered her baby (a healthy girl) by caesarian section at 34 weeks gestational age—somewhat prematurely due to concerns for pre-term labor, preeclampsia, and diabetes. The day after delivery, Petitioner complained of right hip and groin pain, with radiation to the anterior thigh of moderate severity that was associated with heaviness in the right lower extremity, but no weakness or calf pain—noting to treaters that the pain had been present for the prior three months, and thus had been deemed sciatica. She also displayed 4/5 strength bilaterally "appropriate for post-operative state," intact sensation, and normal knee reflexes. She was instructed in measures for increased back support, and several days later, her back pain was improved. *Id*. at 133, 142, 152, 302-05; Ex. 5 at 207, 217, 228.

Ms. Houston was thereafter discharged with her baby on October 17, 2016. Her postpartum course was deemed complicated by elevated blood pressure along with her persistently poorly-controlled blood sugar, but was considered stable enough for release. Ex. 2 at 131-32, 305. She received the MMR vaccine at discharge. *Id.* at 349.

Five days later, on October 22, 2016, Petitioner took herself back to the Tulane Medical Center emergency department with complaints of lower abdominal/pelvic and back pain involving the thoracic and lumbar regions, but no trouble walking, numbness, or focal weakness. Her exam showed diffuse abdominal tenderness, no back tenderness, and negative straight leg raise, and her pain was deemed consistent with her postpartum condition. Her blood sugars were elevated, however, and she admittedly was not compliant at this time with prescribed insulin usage. Detailed instructions for care of preeclampsia, high blood pressure, and diabetes were given, and she was discharged. Ex. 2 at 80, 95-107. She had two additional emergency room visits that same month (October 22nd and 27th) complaining primarily of abdominal pain each time, but was discharged after treaters deemed her symptoms attributable to constipation or her poorly-controlled diabetes. Ex. 4 at 1243-47, 1301; Ex. 5 at 19-29; Ex. 2 at 34, 37, 45-63.

Evidence of New Neuropathic Symptoms

On October 28, 2016, Ms. Houston went again to a hospital emergency department, now complaining of abdominal pain plus acute onset of constant and severe bilateral lower extremity numbness for three days, with some associated weakness that did not interfere with her ability to walk. Her exam noted slight weakness with lifting the legs off of the bed, but no plantar or dorsiflexion weakness, and she denied saddle paresthesias. She displayed high glucose and blood pressure readings, plus elevated glucose and protein in urine. An MRI of the lumbar spine (with and without contrast) showed no evidence of nerve root enhancement or cord lesions, but retained

fecal material was found throughout the colon by x-ray. Ex. 3 at 150-159.

Two days later, on October 30, 2016, Petitioner was admitted to the hospital. The exam showed mild upper abdominal tenderness but bilateral lower extremity weakness. A CT scan of the abdomen showed a fluid collection (approximately 5x2 cm.) in the right rectus sheath near the C-section site that was concerning for abscess, but the absence of tenderness in this area or fever and normal white blood cell counts were not consistent with infection. A thoracic spine CT scan was also unremarkable. Ex. 3 at 849-53.

During her admission, Petitioner was seen by gastroenterology specialists, to whom she reported her symptoms and history since giving birth earlier that month. New testing revealed the possibility of chronic active gastritis (likely thought to be the product of medications she took) and suspicion for an H. pylori infection. Ex. 3 at 883-87, 928. Ms. Houston was also now seen by a neurologist, Dr. Ruben Juarbe, and she informed him of her overall symptoms. The exam revealed some strength deficits (3/5 proximal strength and 4/5 strength distal strength), but no sensory deficits to light touch or vibration, and slightly positive bilateral upper extremity reflexes but absent lower extremity reflexes. Dr. Juarbe's impression was that Petitioner displayed possible lumbosacral plexopathy due to a demyelinating disease such as CIDP "*if this has been occurring for a longer period of time than patient thinks*." *Id*. at 891 (emphasis added).

As a result of this neurologic exam, a lumbar puncture was recommended, but Petitioner declined, and it was recommended that she see a different neurologist, Dr. Cornel Rogers of Ochsner Health System, as an outpatient. Ex. 3 at 887-91. Some in-patient physical therapy ("PT") also revealed weakness consistent with the neurology exam findings, including modified independent gait and mobility, and impaired balance, leading treaters to propose outpatient PT as well. *Id*. at 876-79. Ms. Houston was discharged on October 31, 2016, and advised to follow up with her primary care physician, Christy L. Valentine, MD, in addition to OB and neurology. *Id.* at 868-69.

On November 4, 2016, Petitioner was seen by Dr. Rogers. She now reported weakness that began in both lower extremities shortly after her C-section, with numbness and progressive weakness since. The exam was notable for 4/5 strength in the lower extremities, decreased light touch, pinprick, and vibration perception distal to the knees, normal gait, negative Romberg, normal coordination, normal upper extremity reflexes, absent lower extremity reflexes, and equivocal plantar responses. In addition, electrodiagnostic evidence suggested the presence of a length-dependent peripheral neuropathy, "likely a demyelinating neuropathy." Ex. 10 at 23-34. IVIG treatment every four weeks was now planned. Ex. 3 at 960.

That same November (and prior to beginning IVIG), Petitioner had two additional ER visits for the same kinds of complaints as before (lower back pain, lower extremity numbness/weakness, generalized body pain). Ex. 3 at 190-96; 214-15. Around the time of the third such visit (November 14, 2016), Petitioner began receiving IVIG. *Id*. at 1086-87, 1116, 1119.

Toward the end of the month, Petitioner had additional emergency department visits, both of which resulted in prompt discharge. Ex. 3 at 268-80, 305-07.

The second of the aforementioned visits (on November 25, 2016) resulted in additional hospitalization. For later that day after being discharged, Ms. Houston returned to the emergency department after falling down at home, complaining of progressively-worsening bilateral extremity weakness that day that she alleged was not improved with IVIG. Exam revealed diffuse paraspinal tenderness, and 3/5 lower extremity strength with 1+ patellar reflexes (in comparison to bilateral upper extremity strength of 4+/5 with 2+ biceps reflexes). An MRI showed enhancement of the cauda equina, but no cord abnormality. After a lumbar puncture, Petitioner was given a steroidal treatment and admitted, where she received a large variety of serologic tests, none of which seemed to identify an infectious cause, although other testing revealed positive inflammation biomarkers. Ex. 3 at 1129-34, 1261-1266, 1269. The nonspecific diffuse abnormal enhancement of the cauda equina nerve roots was deemed highly suspicious for failed treatment of CIDP. *Id.* at 1141. Petitioner noted significant improvement after five days of steroids, and by discharge on November 30, 2016, was ambulating with a rolling walker and required minimal assistance with self-care. Her back pain was also under better control based on a course of several medications. Ex. 3 at 1141-46, 1197-98.

As with prior months, December 2016 featured Petitioner making numerous visits to emergency departments for treatment of the same overall constellation of symptoms that had plagued her since giving birth (if not also before). *See, e.g.*, Ex. 3 at 354-56 (December 3rd visit); 380-90 (December 4th visit); 411-14 (December 5th visit); 447-50, 453 (December 9th visit); Ex. 4 at 637-41, 645, 709 (December 11th visit); 574-76 (December 26th visit); 597-600 (December 29th visit). In each instance, she was treated and promptly released. In the intervening period, however, Ms. Houston again saw Dr. Rogers, and his exam revealed distal bilateral lower extremity strength of 3/5, normal sensation, uncoordinated heel to shin maneuver, and areflexia throughout. Dr. Rogers assessed Petitioner with CIDP, prescribed appropriate medications, and proposed follow-up in three months. Ex. 10 at 78-85. Dr. Rogers examined Petitioner again after a subsequent emergency department visit on December 19, 2016, but largely reached the same conclusions (although he did observe a high glucose level at this time). Ex. 3 at 477-80.

Treatment and Diagnostic Assessment in 2017-18

Although Ms. Houston received an additional IVIG infusion in the first half of January 2017, her pain and discomfort continued unabated, prompting her to seek emergency treatment twice that same month. Ex. 3 at 640-43, 653-54, 680-84, 1626. The following month, after Petitioner presented with new thoracic pain on her right side radiating to her right lower extremities plus leg pain similar to her chronic CIDP pain, she was admitted to OMC from February 2-14, 2017. *Id.* at 1645-53, 1656-60.

In addition to the above, Petitioner initially complained of numbness and tingling from her elbows to fingers bilaterally, coupled with an inability to stand and walk. Ex. 3 at 1672-74. The neurology team noted that in addition to CIDP, Ms. Houston had diabetes with diabetic peripheral neuropathy. *Id*. at 1725. She was treated with a variety of medications then transferred to inpatient rehabilitation on February 15, 2017. *Id*. at 1751-52, 1761, 2040-43, 2055-57. There, she received pain management consultation, and it was noted again that she had repeatedly demonstrated poor glycemic control. *Id.* at 1475. Ms. Houston was discharged from rehab on March 2, 2017.

On March 9, 2017, Petitioner again saw Dr. Rogers. She now complained of continued weakness, and worsening pain and muscle spasm, but her exam was consistent with those she had received before, and showed many of the same reflex and strength deficits. The plan was to continue IVIG, and consider other immune-modulating therapies. Ex. 10 at 154-60. Between the second half of April and mid-May, Petitioner visited the OMC emergency department three more times, largely complaining of the same issues she had raised in the past. *See, e.g.*, Ex. 3 at 717-20, 728-29, 731 (April 24th visit); 749-51, 769-71 (May 1st visit); 796-98 (May 3rd visit). At another follow-up visit with Dr. Rogers in late July 2017, Petitioner was prescribed medication for nerve pain. Ex. 10 at 244.

Later on in the fall of 2017 (and after yet another emergency department visit (Ex. 2 at 12-16)), Ms. Houston went to see a family practice physician on September 27, 2017. At this time, she provided a history of diabetes with neuropathy but no serious medical issues, although she did not her CIDP diagnosis. She could not walk unassisted now. She was prescribed some antidepressants, and then a month later (after calling about neuropathic back and leg pain), some pain relievers as well. Ex. 13 at 15-17. She saw the same treater again in February, and reported improvement across the board. *Id.* at 14-15.

On May 17, 2018, Petitioner returned to her primary care treater, noting that she had received no physical therapy for months and now needed pain management. She also had not been compliant with diabetes care, and requested supplies and a glucometer. Lab results, among other things, revealed mild anemia, urine positive for protein and glucose, and fairly high glucose levels. Ex. 13 at 11-13, 17-18. She saw him again in mid-July 2018, reporting pain in her back and foot that was not resolved with medication, although her walking was improving with physical therapy. She also complained of psoriasis and vitiligo, the latter of which involved her elbows, face, and scalp. "Trace" hyperthyroidism was noted, and she was referred to dermatology and pain management. Ex. 13 at 7-11.

On July 31, 2018, Petitioner saw Dr. John Englund of LSU Neurology for a second opinion regarding her CIDP diagnosis. She gave a history of receiving a brief trial of IVIg for two months, as well as a trial of high-dose Solu-Medrol for five days, plus her physical therapy and other medications. She felt that her symptoms had improved since the initial diagnosis, but that they were poorly managed. Her history of uncontrolled diabetes was also noted. And she reported difficulties walking plus sharp, throbbing pains in her feet and lower legs that did not radiate

proximally past the knees. Exam revealed 4/5 strength of the intrinsic hand muscles, proximal lower extremity strength of 4/5 and distal lower extremity strength of 5/5, except for ankle dorsiflexion which was zero. Triceps reflexes were trace and all others were absent. The impression was a combination of diabetic distal symmetric polyneuropathy and superimposed inflammatory polyneuropathy, with improved glucose control urged as treatment. Ex. 12 at 14, 20-22.

On September 14, 2018, Petitioner received EMG/NCS testing which showed chronic sensory motor polyneuropathy with both demyelinating and axonal features, deemed likely from a combination of diabetic polyneuropathy with superimposed CIDP. Ex. 12 at 29-30. She was seen at Ochsner Foundation Hospital for nausea with vomiting on October 4, 2018. Ex. 21 at 111. Petitioner also presented to West Jefferson Medical Center for muscle wasting and atrophy on October 16, 18, and 23, 2018. *Id.* at 111-112. Throughout January of 2019, Petitioner was seen at various facilities for her CIDP as well as muscle wasting an atrophy. *Id.* at 112.

II. Expert Reports

A. *Petitioner's Expert – Cornel Rogers, M.D.*

In addition to having actually treated Ms. Houston, Dr. Rogers prepared two "to whom it may concern" letters on her behalf, as well as a written causation report. Letter, dated June 5, 2018, filed as Ex. 10 at 14-15 (ECF No. 20-2); Letter, dated July 10, 2018, filed as Ex. 9 (ECF No. 11-2) ("July 10th Letter"; Report, dated July 1, 2020, filed as Ex. 27 (ECF No. 37-2) ("Rogers Rep.").[3]

Dr. Rogers obtained his medical degree from Louisiana State University ("LSU") Health Sciences Center in 2010, and has an undergraduate degree in biology from Xavier University. CV, filed as Ex. 28 (ECF No. 37-3) ("Rogers CV") at 1. He did his residency in neurology, plus a fellowship in neurology at LSU from 2014-15, and has since worked as a staff neurologist at Ochsner Health System. Rogers CV at 1. He does not, however, appear to be formally board certified in neurology (and indeed he characterizes himself in his report as only "board eligible"). Rogers Rep. at 2. Dr. Rogers also has not written about CIDP, nor does he appear to have conducted research into the illness, or other comparable peripheral neuropathies. Dr. Rogers

[3] Prior to submission of a formal expert report, Petitioner filed a "narrative report" from Dr. Rogers, consisting of a three-page opinion to which were appended several items of literature. Narrative Report, dated March 2, 2020, filed as Ex. 26 (ECF No. 31-5). The special master to whom this case was initially assigned, however, deemed this submission inadequate for purposes of establishing causation, and instructed Petitioner to file a report consistent with what is typically required for non-Table causation-in-fact claims in the Vaccine Program, providing a several-page explanation of what was required. Order, dated March 4, 2020 (ECF No. 33). Petitioner thereafter filed the revised and more lengthy report addressed herein. I am treating the second-filed report (rather than its earlier iteration) as Petitioner's primary expert report, and concur with the special master previously assigned to this case that the Narrative Report was substantively inadequate (although I have reviewed it and considered its arguments, which largely overlap the second version).

reports that at present he is treating approximately 150 patients suffering from neuropathies, with ten experiencing CIDP or something comparable. *Id.*

Letters Prepared for Petitioner

On June 5, 2018 (after the case's March 2018 initiation), Dr. Rogers wrote a letter on Ms. Houston's behalf, noting that she had been diagnosed with CIDP after developing an onset of weakness in the hospital following a C-section and concurrent receipt of the vaccines at issue, and stating as follows:

> From the time of her hospitalization until she was diagnosed[,] she experienced progressive weakness and showed clinical and electrodiagnostic evidence that her symptoms were due to a chronic demyelination which is a result of immune mediated destruction of the nerves. Based on the patient's history and lack of any evidence of any previous autoimmune diseases it is *plausible* that the symptoms stem from her vaccination.

Ex. 10 at 14-15 (emphasis added).

Dr. Rogers prepared a second opinion letter on July 10, 2018. Ex. 10 at 15-16 (also filed as Ex. 9 at 2). This letter stated that Ms. Houston had been diagnosed with CIDP on November 7, 2016, and that her condition "more likely than not" stemmed from the receipt of the Tdap and MMR vaccines. July 10th Letter at 2. Dr. Rogers did not specify which vaccination caused petitioner's onset of symptoms, however, and did not describe any theory of how either vaccine could cause CIDP. Ex. 10 at 15-16.

Expert Report

Dr. Rogers's report begins with a several-page summary of Ms. Houston's medical history and treatment course, including the years prior to the October 2016 vaccinations. In so doing, he highlighted a number of different facts. First, he noted that Petitioner had been determined to suffer from Type II diabetes rather than Type I, since she did not display diabetic ketoacidosis after ceasing medications. Rogers Rep. at 2; Ex. 20 at 274. Second, he observed that in many instances in which Petitioner revealed what looked like neurologic injuries, the record was less settled than appeared. Thus, for example, the tingling Petitioner experienced in 2011 was not associated with other abnormalities after a neurologic exam (Ex. 20 at 682-86), and this was also true other times in her pre-vaccination history. Rogers Rep. at 2-3. He went so far as to opine that in October of 2013 Petitioner had been "misdiagnosed" with a diabetic neuropathy, given the overall mildness of symptoms and lack of corroboration. *Id.* at 3.

Next, Dr. Rogers focused on the time around the vaccinations in question. In September 2016, Petitioner's neuropathic complaints were accompanied by normal reflexes, and she even

began to feel better after seeking treatment in mid-September. Rogers Rep. at 3. She also displayed normal reflexes when she was admitted in mid-October (after receiving the Tdap vaccine), at which time she gave birth, and subsequent hip and groin pain was not accompanied by abnormal strength, sensation, or reflexes. *Id.* at 4. The same was true of her ER evaluations on October 25th and 27th. *Id.*

Petitioner's October 28, 2016 ER visit, however, was deemed by Dr. Rogers to reflect a change in her health distinguishable from prior incidents. Now, she reported bilateral leg numbness for three days (placing onset on the 25th—19 days post-vaccination—although Dr. Rogers contradictorily did not find significant Petitioner's two ER visits before the 28th). Rogers Rep. at 4; Ex. 6 at 232. That weakness was now objectively confirmed after neurologic exam. Rogers Rep. at 4. Then, after her admission to the hospital, the neurologic picture was more mixed—and indeed, as Dr. Rogers acknowledged, Dr. Juarbe speculated at this time that a CIDP diagnosis would be more likely assuming Petitioner's symptoms had been "occurring for a longer period of time" than she believed. *Id.* at 5; Ex. 6 at 893.

Thereafter, the medical record revealed additional evidence in support of the CIDP diagnosis (in particular EMG testing). Rogers Rep. at 5. IVIG treatment was initiated but was only partially successful, and Petitioner's progress was uneven. *Id.* Steroids, however (which are understood to be effective in the treatment of CIDP), helped Ms. Houston to start to feel better by December 2016. Unfortunately, her personal circumstances and insurance limitations interfered with consistent treatment, and thus she has not been able to see the kind of meaningful recovery that might otherwise be possible. *Id.* at 6. Indeed, Dr. Rogers felt that the demyelination damage Ms. Houston experienced was likely now irreversible, with arresting further regression the best she could hope for. *Id.*

Dr. Rogers then outlined his understanding regarding how to weigh the different kinds of evidence that might bear on vaccine causation. Rogers Rep. at 6-7. Although he deemed epidemiologic studies the best kind of support, he also observed that they could not typically "detect the risk of a rare event" like a vaccine injury, reducing their value in this context. *Id.* at 9. Dr. Rogers also noted, however, that he could not identify any epidemiologic studies relevant to the different possible explanations he proposed for causation (discussed below). *Id.* Rather, he proposed "biological plausibility" was a sufficient basis for finding causation, adding that case reports of post-vaccination injury would constitute reliable proof under the circumstances. *Id.* at 9-10. Dr. Rogers had earlier, however, deemed case report evidence to be in the third tier of reliability in establishing vaccine causation. *Id.* at 6.

Applying the above as framework, Dr. Rogers arrived at the Tdap vaccine as causal by a process of elimination. He posited that three occurrences might account for Ms. Houston's CIDP: (a) neurosarcoidosis—a rare, chronic inflammatory disease of the central nervous system that

causes demyelination,[4] (b) her preexisting diabetes, in which case her CIDP was a form of diabetic neuropathy, or (c) the Tdap vaccine itself. Rogers Rep. at 10-11. Neurosarcoidosis, Dr. Rogers maintains, was considered "early on" by treaters as potentially explaining Petitioner' symptoms—although the medical record overview in his report makes no mention of the term, and he offers no citation to the record substantiating this contention. Nevertheless, Dr. Rogers opined that it could be excluded from Petitioner's differential, because she lacked the lung involvement most common to neurosarcoidosis, and also because her presentation was not consistent with it. *Id.* at 10.

Dr. Rogers also disputed that Petitioner's well-established diabetes had any connection to her CIDP. As a threshold matter, he denied that she was even properly diagnosed with type I diabetes, but also contended that the "symmetrical distal sensory loss" (in which sensory deficits arise bilaterally from the feet upward, with hand parasthesias beginning later) characteristic of a diabetic neuropathy was not present in Ms. Houston's case. Rogers Rep. at 10; R. Pop-Busui et al., *Diabetic Neuropathy: A Position Statement by the American Diabetes Association*, 40(1) Diabetes Care 136-154 (2017), filed as Ex. 34 on July 1, 2020 (ECF No. 37-9). In so arguing, Dr. Rogers reviewed in detail multiple incidents from Ms. Houston's medical history, going as far back as 2013, and consistently denying that these findings were consistent with diabetic neuropathy. Rogers. Rep. at 11-12. By contrast, Dr. Rogers felt the findings from Petitioner's October 28, 2016 ER exam were consistent with CIDP ("weakness across the whole leg") over diabetic neuropathy. *Id.* at 12. At best, Dr. Rogers allowed that "some of the symptoms [Petitioner] experiences today do appear to be related to her poorly controlled diabetes," but he limited this concession to things like cardiac arrythmias, sensory symptoms, or gastroparesis. *Id.*

In opining that the Tdap vaccine could cause CIDP, Dr. Rogers relied on several different arguments and items of evidence. He noted initially that "an infectious cause has long been postulated to explain the development of autoimmunity," and that the mechanism that could propagate such a disease process was molecular mimicry (in which "antigens that are immunologically similar to the host antigens" induce a cross-reactive response). Rogers Rep. at 13; F. Epstein et al., *Molecular Mimicry and Autoimmunity*, New England J. of Med. 2068-74 (1999), filed as Ex. 32 on July 1, 2020 (ECF No. 37-7) ("Epstein"); R. Yu et al., *Ganglioside Molecular Mimicry and Its Pathological Roles in Guillain-Barré Syndrome and Related Diseases*, 74(12) Infection and Immunity 6517-27 (2006), filed as Ex. 31 on July 1, 2020 (ECF No. 37-6) ("Yu").[5] This cross-reaction would cause the demyelination that is central to a neuropathy like CIDP, by attacking the myelin basic protein that constitutes a building block of nerve myelin (the

[4] Demyelination is the destruction, removal, or loss of the myelin sheath of a nerve or nerves. *Dorland's Medical Dictionary* 480 (33rd ed. 2020); E. Hoitsma et al., *Neurosarcoidosis: A Clinical Dilemma*, 3 Lancet Neurology 397-407 (2004), filed as Ex. 29 on July 1, 2020 (ECF No. 37-4).

[5] Guillain-Barré syndrome ("GBS") is recognized as several typically-acute and monophasic disorders characterized by an immune-mediated attack on peripheral nerves, particularly in the myelin sheath or Schwann cells of sensory and motor nerves. Yu at 6517.

sheath covering nerves). Rogers Rep. at 13, 15. Molecular mimicry is recognized as an "attractive conceptual link" that reliably explains, at least as a model, how autoimmune-driven disease processes might unfold. *Id;* Epstein at 2068.[6]

CIDP, Dr. Rogers proposed, could be triggered by infection or vaccination. In support, he offered a study that he purported showed 11 percent of CIDP patients had been vaccinated before their disease onset. Rogers Rep. at 14; K. Gable et al., *Distal Acquired Demyelinating Symmetric Neuropathy After Vaccination*, 14(3) J. of clinical Neuromuscular Disease 117-22 (2013), filed as Ex. 30 on July 1, 2020 (ECF No. 37-5) ("Gable"). Gable, however, only *cited* to a different study (not filed in this case) for this contention, so its reliability cannot be ascertained. Otherwise, Gable described only two subjects relevant to this case. The first, a 35-year-old healthy man, developed progressive distal symmetric numbness and paraesthesias two weeks after receipt of a Tdap vaccine. Gable at 117. The other, a 31-year-old healthy man, developed slowly progressive distal symmetric paresthias and numbness two months after influenza vaccination. *Id.* at 119. Gable's authors concluded that vaccine-associated CIDP was a rare but poorly characterized event that should not discourage at-risk individuals from being vaccinated. *Id.* at 121. Nonetheless, they acknowledged that neuropathy can occur after vaccine and suggested consideration of this diagnosis for patients with new sensory symptoms after vaccination. *Id.*

More specifically, Dr. Rogers deemed it likely that the Tdap vaccine could specifically trigger CIDP (and he favored it as causal over the MMR). Rogers Rep. at 14. The fact that the Tdap vaccine contained tetanus toxoid was significant in his view. *Id.* In support, he noted case reports associating the Tdap vaccine to CIDP where it was proposed that the toxoid was responsible. *Id;* G. Fenichel, *Assessment: Neurologic Risk of Immunization – Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology*, 52:1546 Nurology 1-8 (1999), filed as Ex. 35 on July 1, 2020 (ECF No. 37-10) ("Fenichel"). Dr. Rogers highlighted a passage in Fenichel discussing one "unique patient [who] developed three episodes of Guillain-Barré syndrome (GBS) after three doses of tetanus toxoid." *Id.* at 3. The patient subsequently experienced additional relapses without prior immunization and was diagnosed as having CIDP. *Id.* However, Fenichel acknowledges that "[i]t is not possible to know whether the tetanus toxoid caused or triggered CIDP in a susceptible individual." *Id.*

[6] In a few places in his report, Dr. Rogers also proposed that "bystander activation"—in which "T cells unrelated to the [foreign] antigen presented can be activated" in connection with "signals derived from the ongoing response directed against the vaccine-antigen"—could mechanistically explain the pathogenesis of vaccine-caused CIDP. *See, e.g.*, Rogers Rep. at 15, 16. But he did not flesh out this contention, or provide any evidence that would substantiate its relevance herein. I also have in other cases noted the fact that to some extent bystander activation presupposes an initial cross-reaction driven by the vaccine antigen having already occurred. *Bender v. Sec'y of Health & Hum. Servs.*, 141 Fed. Cl. 262, 267 (2019). So absent a showing that this has happened, bystander activation is not a compelling mechanism for vaccine causation, even if it has medical/scientific reliability as a mechanistic concept. Dr. Rogers otherwise did not flesh out this aspect of his opinion.

The medical record also, in Dr. Rogers's view, bulwarked his causation contention, since it confirmed the Tdap vaccine had likely "induced" Petitioner's symptoms. Rogers Rep. at 15. He found significant the fact that Ms. Houston, a woman, was as a general rule less likely to experience CIDP than a man, and also that she had no prior history of autoimmunity. *Id.* She should not have likely experienced CIDP in the first place—and therefore probably did so because of the vaccine. In addition, vaccination is more closely associated with the "classic" presentation of CIDP, consistent with Petitioner's experience (which he reiterated had been properly so diagnosed). *Id.* at 15-16; Gable at 117-119. And Petitioner's response to IVIG underscored the vaccine's role in her disease. In Dr. Rogers's view, an aberrant immune response triggered by a vaccination (as purportedly here) would result in overproduction of "autoreactive cells" that would render IVIG treatment less effective if CIDP was not vaccine-caused. Rogers Rep. at 16. He did not, however, offer any independent medical or scientific substantiation for this contention.

Finally, Dr. Rogers discussed the timeframe in which Petitioner experienced post-vaccination CIDP, deeming it medically acceptable. Again, due to an absence of epidemiologic studies on the subject, he referenced case reports to support his opinion. He thus cited Fenichel, noting how the subject discussed therein experienced three instances of demyelinating neuropathy after three doses of tetanus toxoid-containing vaccine, in timeframes of nine days to three weeks (with each subsequent period shorter). Rogers Rep. at 17; Fenichel at 3. Because Petitioner received the Tdap vaccine on October 6, 2016, and likely had in his view an onset of October 25th, 19 days separated the two events—a timeframe consistent with this case report. Dr. Rogers also expressed doubt that Petitioner's CIDP could have predated vaccination. He reiterated his view that Petitioner's pre-vaccination history did not reveal true CIDP, in the form of symmetrical/bilateral sensorimotor symptoms. Rogers Rep. at 13. Rather, there was no such evidence until after vaccination, in late October 2016. *Id.*

B. *Respondent's Expert – Brian Callaghan, M.D., M.S.*

Dr. Callaghan, an associate professor of neurology and specialist in treatment of neuropathies like CIDP, prepared a single written report for Respondent. Report, dated November 25, 2020, filed as Ex. A (ECF No. 41-1) ("Callaghan Rep."). Dr. Callaghan accepted Ms. Houston's CIDP diagnosis, but disputed that the flu vaccine could have caused it. Callaghan Rep. at 5.

Dr. Callaghan received his undergraduate degree from the University of Michigan, his medical degree from the University of Pennsylvania in 2004, and Masters in Science from the University of Michigan in 2011. CV, filed as Exhibit B on November 30, 2020 (ECF No. 41-2) ("Callaghan CV") at 1. He is boarded in psychiatry/neurology as well as electrodiagnostic medicine. Callaghan CV at 1. He 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. Callaghan Rep. at 1; Callaghan CV at 2, 11-20. Dr. Callaghan has averred that he treats approximately 30 patients with CIDP per year. Callaghan Rep. at 1.

Dr. Callaghan's report does not contain a description of CIDP, and he does not seem to dispute Dr. Rogers's discussion of it. However, Dr. Callaghan did pinpoint differences between CIDP and other peripheral neuropathies involving demyelination that he considered important to this case. In particular, Dr. Callaghan maintained that pathogenic "mechanisms that have been described for GBS" could *not* simply be applied to CIDP, even though both conditions were peripheral neuropathies driven by demyelination. Callaghan Rep. at 5.

Rather, Dr. Callaghan noted that "multiple reviews of the literature" established that CIDP was not understood to be triggered by antecedent infection (unlike GBS), making it far less likely that the molecular mimicry mechanism applied. Callaghan Rep. at 5*;* E. Ubogu, *Inflammatory Neuropathies: Pathology, Molecular Markers and Targets for Specific Therapeutic Intervention,* 4 Acta Neuropathol. 130, 445-68 (Oct. 2015), filed as Ex. E (ECF No. 41-5) ("Ubogu"), at 459; E. Mathey et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Pathology to Phenotype*, J. Neurol. Neurosurg. Psychiatry 1-13 (2015), filed as Ex. F on Nov. 30, 2020 (ECF No. 41-6) ("Mathey"). Discussing the immunopathogenesis of CIDP, Mathey found that although some patients have reported antecedent infections prior to onset of neurological symptoms, neither the targets nor the trigger for the autoimmune response has been identified and no infectious agent has been consistently linked with initiation of CIDP. Mathey at 3. Indeed, Dr. Callaghan proposed that "there is no evidence supporting molecular mimicry" as the autoimmune driver of CIDP. Callaghan Rep. at 5. And Dr. Rogers's assertions about the tetanus toxoid component of the vaccine as potential mimic for amino acid sequences/structures in myelin basic protein were not corroborated with any science or medical evidence showing its role in triggering CIDP. *Id.*

Other differences between CIDP and GBS further diminished application of what was known about the latter to the former. For example, autoantibodies thought to be associated with CIDP were *not* also associated with GBS. Callaghan Rep. at 5; Mathey at 6-7. In addition, the GBS-associated autoantibodies attacked gangliosides (antigenic receptor molecules found on the cell surface of nerve myelin),[7] whereas the CIDP-specific autoantibodies have different suspected targets. R. Lewis, *Chronic Inflammatory Demyelinating Polyneuropathy: Etiology, Clinical*

[7] Gangliosides are glycosphingolipids that contain sialic acid and are present in many cell types, but most abundantly within neural tissues along their linings (myelin). Mayo Clinic Laboratories, *Ganglioside Antibody Panel, Serum*, https://neurology.testcatalog.org/show/GM1B (last visited Aug. 6, 2021). Among the immune-mediated peripheral neuropathies, autoantibodies to gangliosides represent an important class of noncancer-associated drivers of certain autoimmune peripheral neuropathies. *Id.* Depending on the specific ganglioside autoantibody found and the antibody titer, in the appropriate clinical context, these findings may be supportive of a specific clinical diagnosis and may also be prognostic for treatment. *Id.*

Features, and Diagnosis (UpToDate, Inc., current through April, 2020), filed as Ex. 33 on July 1, 2020 (ECF No. 37-8), at 4 ("Lewis"). These pathogenic differences, moreover, are reflected in the distinctions between the conditions. CIDP evolves slowly over time, whereas GBS is acute and usually monophasic, and the two neuropathies also differ in treatment efficacy, with steroid proving very effective in treatment of CIDP but not GBS. Callaghan Rep. at 5. CIDP's causes remained largely unknown, along with its risk factors. *Id.* at 4; Lewis at 2.

Based on an overview of Petitioner's history and testing results, Dr. Callaghan agreed that CIDP was the proper diagnosis. Callaghan Rep. at 5. He deemed it likely to have begun around the time of Petitioner's c-section, or October 13, 2016 (a week after receipt of the Tdap vaccine). *Id.* at 4. He also highlighted the fact of Petitioner's pre-vaccination diabetes and her poor control of it, but his report does not formally connect her CIDP diagnosis to these prior symptoms. *Id.* at 1-2. Dr. Callaghan did, however, note that more recent records continued to identify diabetic neuropathy as associated with Petitioner's symptoms. *Id.* at 3; Ex. 12 at 29 (September 14, 2018) ("[c]hronic sensory motor polyneuropathy with both demyelinating and axonal features. This is most likely a combination of diabetic polyneuropathy with superimposed CIDP"). Dr. Callaghan did not find significant Petitioner's partial responsiveness to IVIG, noting that "CIDP responds to IVIG regardless of the mechanism" initiating it. Callaghan Rep. at 5.

Dr. Callaghan specifically disputed that the Tdap vaccine could cause CIPD. In so doing, he noted that Dr. Rogers primarily relied on case reports, which he characterized as "anecdotal," providing "low-level evidence" of causation in comparison to other classes of proof. Callaghan Rep. at 4. At bottom, case reports mostly established a temporal relationship between the injury and vaccination, and typically their own authors acknowledged their causal limitations. *Id.* at 3, 4; Fenichel at 2.

In addition, a recent case-control study greatly undermined the contention that *any* vaccine could cause CIDP. Callaghan Rep. at 4; P. Donnedu, et al., *Risk Factors for Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)): Antecedent Events, Lifestyle and Dietary Habits. Data from Italian CIDP Database*, 0 Eur. J. of Neurol. 1-8 (2019), filed as Ex. D on Nov. 30, 2020 (ECF No. 41-4) ("Donnedu"). In Donnedu, data on antecedent events occurring 1-42 days prior to onset were collected from the medical information of 411 CIDP patients from an Italian database. Donnedu at 1. Antecedent events were reported by 15.5% of these patients, including infections in 12% and vaccinations in 1.5%. But less than two percent of the 411 CIDP patients—seven subjects in total—had been vaccinated within six weeks of onset, and each of these individuals had received the flu vaccine. *Id.* at 3. Donnedu's authors concluded from this study that "antecedent events are unlikely to play a role in the risk on CIDP." Callaghan Rep. at 4; Donnedu at 6.

III. Applicable Law

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, a petitioner must prove that: (1) they suffered an injury falling within the Vaccine Injury Table (i.e., a "Table Injury"); or (2) they suffered an injury actually caused by a vaccine (i.e., 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 & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006). In this case, Petitioner does not assert a Table claim.

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) (explaining that mere conjecture or speculation is insufficient under a preponderance standard). On one hand, proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). But on the other hand, 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 & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non–Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Human Servs.,* 418 F.3d 1274 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

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 & Human 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. However, the Federal Circuit has *repeatedly* stated that the first prong requires a preponderant evidentiary showing. *See Boatmon v. Sec'y of Health & Human*

Servs., 941 F.3d 1351, 1360 (Fed. Cir. 2019) ("[w]e have consistently rejected theories that the vaccine only "likely caused" the injury and reiterated that a "plausible" or "possible" causal theory does not satisfy the standard"); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010). This is consistent with the petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted). If a claimant must *overall* meet the preponderance standard, it is logical that they be required also to meet each individual prong with the same degree of evidentiary showing (even if the *type* of evidence offered for each is different).

Petitioners may offer a variety of individual items of evidence pertaining to the first *Althen* prong, without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or even a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). The individual items of proof offered for the "can cause" prong, however, must *each* reflect or arise from "reputable" or "sound and reliable" medical science. *Boatmon,* 941 F.3d at 1359-60.

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 & Human 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 & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views 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 & Human 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 also be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human 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 & Human Servs.*, No. 06–522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356–57 (2011), *aff'd without opinion*, 475 F. App'x. 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 & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide 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 & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. Den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11–355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11I(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 & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records 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 & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Human Servs*., 468 F. App'x 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 & Human Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms.").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). 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 v. Sec'y of Health & Human Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, there are 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 & Human 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 & Human 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 & Human Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Human 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 & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

However, in the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings—e.g., the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human 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*, 618 F.3d at 1347 (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. At 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 91997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08–601V, 2012 WL 3609993, at *17 (Fed.

Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Human Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Human Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

E. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record, as per the parties' request, and without the parties' submission of briefs. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs*., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. CIDP as Vaccine Injury

There was no dispute in this case as to the diagnostic features of CIDP, or the more fundamental fact that Ms. Houston suffered from it, although the experts did not concur as to its pathogenesis or how it relates to comparable neuropathies. Dr. Rogers defined CIDP as "an immune mediated neuropathy that affects nerve roots in addition to peripheral nerves," with a relapsing-remitting course. Rogers Rep. at 8, *citing* Lewis at 3. CIDP typically is symmetric, and features demyelination leading to motor deficiencies rather than sensory. Rogers Rep. at 8. Symptoms progress over a period of months, and will feature absent or lessened deep tendon reflexes, weakness, and difficulties walking. *Id.* at 8-9.

Importantly for purposes of deciding this case, the filed literature establishes that CIDP should not be viewed as merely a "chronic" form of GBS, even though both are peripheral neuropathies involving nerve demyelination. Ubogu at 455 (deeming the conception that CIDP is chronic GBS to be "overly simplistic" despite the symptomatic overlap between the two). Differences in their clinical presentation distinguish the two—and they also cannot be assumed to have the same pathogenic mechanisms. Thus, while it is believed that certain autoantibodies specific for amino acid sequences found in myelin basic protein may be a source of the autoimmune attack resulting in GBS, "antibodies against peripheral nerve myelin proteins . . . are too infrequently detected in the sera of CIDP patients to be considered pathogenic or molecular markers of disease." *Id.* at 457 (adding that "[a]ntibodies against complex gangliosides . . . are rarely detected in CIDP patient [blood] sera or cerebrospinal fluid"); Mathey at 6 ("[t]he pursuit of autoantibodies reactive to the major compact myelin proteins in CIDP has thus far been somewhat unproductive"). Little is also known about CIDP's most likely causes, triggers, or pathogenesis, in comparison to GBS. Ubogu at 459; Mathey at 6 ("for the majority of patients the specific target of the autoantibody response is unknown"). Indeed, it may be more correct to think of CIDP as arising "in the *setting* of a dysregulated immune system" than to be directly *driven* by an aberrant immune response. Ubogu at 459 (emphasis added).

There are ample prior decisions associating different vaccines with CIDP (in particular the flu vaccine), and petitioners have settled many such cases on favorable terms.[8] *See, e.g.*, *Jastisan v. Sec'y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). I have myself acknowledged their existence in my own prior decisions, and the fact that such determinations should be given *some* consideration as persuasive guidance. *See Strong v.*

[8] Prior decisions from different cases do not control the outcome herein. *Boatmon*, 941 F.3d at 1358–59; *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). But special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39 (2007) ("[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim") (emphasis added). They would therefore be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions.

Sec'y of Health & Hum. Servs., No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018).

However, I have identified no recent[9] *reasoned* decisions in which a special master explained how or why the Tdap vaccine was likely causal of the claimant's CIDP. And there are several decisions in the past ten years that suggest the strength of a vaccine association with CIDP is far weaker than what may have previously been presumed. In a 2014 case, for example, a petitioner was unsuccessful in claiming her ongoing neurological condition was aggravated by two influenza vaccinations. *Jacunksi v. Sec'y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422 (Fed. Cl. Spec. Mstr. Sept. 23, 2014). The special master highlighted a prestigious Institute of Medicine report (among other things) which specifically found insufficient available evidence to support an association between influenza vaccine and CIDP. *Jacunski,* 2014 WL 5168422, at *14.

II. Petitioner Has Not Carried Her Burden of Proof

The parties' experts agree that Ms. Houston experienced CIDP post-vaccination, but disagree whether the Tdap vaccine (which Dr. Rogers unquestionably focused his opinion upon) could have caused it, or did so here. I find that none of the three *Althen* prongs have been met.

Althen Prong One

Petitioner has not established on this record that the Tdap vaccine could cause CIDP. As a threshold issue, I note that Dr. Callaghan was demonstrably more qualified to opine on the nature and treatment of CIDP, and he succinctly set forth in his expert report why he felt it unlikely that the Tdap vaccine could cause it. In particular, Dr. Callaghan persuasively distinguished the disease mechanisms of GBS and CIDP. Callaghan Rep. at 5. Multiple reviews of the literature state that "unlike AIDP [GBS], antecedent infections or trauma rarely precipitates CIDP, reducing the likelihood that molecular mimicry serves as a trigger…" *Id.* (citing Mathey). And CIDP and GBS have different underlying causes, clinical presentations, and treatments. *Id.* CIDP can be caused by autoantibodies to contactin and neurofascin, while GBS has been associated with different autoantibodies such as those targeting gangliosides. *See Tomsky v. Sec'y of Health & Hum Servs.*, No. 17-1132V, 2020 WL 5587365 at *8 (Fed. Cl. Spec. Mstr. Aug. 24, 2020); *Isaac v. Sec'y of*

[9] In a case that is now 16 years old, the Court granted a motion for review reversing a special master's determination that a tetanus toxoid-containing vaccine had not been shown to cause CIDP. *Kelley v. Sec'y of Health & Hum. Servs.*, 68 Fed. Cl. 84 (Fed. Cl. 2005). *Kelley,* however, is distinguishable legally as well as factually. The injured party in that case had presented to his pediatrician's office with complaints of lower back stiffness and numbness and tingling of both hands and feet, as well as dizzy spells at night, which had begun during the second week post-vaccination. *Id.* at 97. More significantly, *Kelley* relied on older literature that seemed to assume that CIDP and GBS were two sides of the same coin—whereas the more-recently-generated literature filed in this case stands strongly for the contrary conclusion. *See, e.g.*, Mathey. Thus, although I am not bound by the Court's findings in *Kelley,* I deem it to be based on now-superseded science and medical views, and do not afford it persuasive value in deciding this case.

Health & Hum. Servs., 108 Fed. Cl. 743, 751 (Fed. Cl. 2013). And while there is evidence supporting molecular mimicry as a disease mechanism driving GBS, Dr. Callaghan explained that there is no comparable evidence associating molecular mimicry with CIDP. *Id.*

Dr. Rogers is a treater by contrast, and he did not possess the same degree of expertise necessary to opine on the immunologic issues raised by a vaccine causation theory (although I acknowledge that neither expert had a robust, demonstrated background in immunology). Certainly his opinion was deserving of serious consideration, especially given his professional focus on neurology and direct knowledge of Petitioner's treatment history, and he raised some compelling points about Petitioner's overall course that support her claim. This is not a case where the report submitted on a claimant's behalf was frivolous—to the contrary, the opinion offered by Dr. Rogers was sober and detailed. But in the end, Dr. Rogers lacks Dr. Callaghan's demonstrated focus on understanding peripheral neuropathies not only from a treatment standpoint, but from the vantage of a researcher as well. I therefore inherently have given Dr. Callaghan's opinion greater weight.

Yet even ignoring such facial distinctions between expert qualifications, the core opinion advanced by Dr. Rogers was not on its own medically/scientifically reliable, or sufficiently based in reputable scientific views about CIDP, even if elements of it passed muster. As discussed above, CIDP and GBS likely have different root causes and proceed via different pathogenic mechanisms. Callaghan Rep. at 5. Far less is known about CIDP than other neuropathies such as GBS, and it is not the case that what is true for GBS is also wholly true for other injuries such as CIDP. Moreover, the Tdap vaccine and its wild virus counterparts are not even closely associated with GBS—let alone Petitioner's actual injury. But even if it were otherwise, Petitioner has made no showing comparable to what would be required to prove that this association translates to an association between Tdap and CIDP. At most, Dr. Rogers has relied heavily on case reports—and not only does he offer only a handful of them, but as a class of evidence they are not typically given substantial weight in the Program. *See K.O. v. Sec'y of Health & Hum. Servs.*, No. 13-472V, 2016 WL 7634491, at *11-12 (Fed. Cl. Spec. Mstr. July 7, 2016) (discussing appellate precedent on case reports). Dr. Rogers's own report admits as much. Rogers Rep. at 6 (classifying case reports as occupying a third tier of persuasive evidence on causation).

It is true that Respondent's expert has not come close to conclusively establishing that the Tdap vaccine *cannot* cause CIDP. Indeed, some aspects of Dr. Callaghan's opinion make the classic Program mistake of deeming the rarity of vaccine injury (and thus comparative safety of the Tdap vaccine) as rebutting Petitioner's case—when in fact rarity of injury is the "coin of the realm" in Vaccine Act cases, and does not by itself *ever* preclude a showing that a vaccine could cause harm, simply because it is safe for the majority of recipients. *Cordova v. Sec'y of Health & Hum. Servs.*, No. 17-1282V, 2021 WL 3285367, at *18 (Fed. Cl. Spec. Mstr. June 23, 2021). As a result, I do not give substantial weight herein to articles like Donnedu, cited for the proposition

that "antecedent events" like vaccination were not likely causal—since all Donnedu's authors observed was that a *small percentage* of CIDP patients in the study had been vaccinated before onset. (More troubling for Petitioner, however, if the fact that none of these subjects received the Tdap vaccine).

Nevertheless—the preponderant burden to satisfy the "can cause" prong falls first on claimants, and the test can be failed even where doubt remains about causation (just as a successful showing does not render causation a certainty). Here, Respondent and his expert have overall rebutted Petitioner's somewhat-conclusory showing, even if not all of Respondent's defenses warranted substantial weight.

Althen Prong Two

The record does not permit the conclusion that the Tdap vaccine likely "did cause" Ms. Houston to experience CIDP, even if I had found it *could* theoretically. No treaters other than Dr. Rogers ever proposed any association between the October 2016 vaccinations and Petitioner's subsequent diagnosis. And while I credit Dr. Rogers's treater opinion to some extent, the aforementioned discussion of its deficiencies undermines its evidentiary weight substantially. Otherwise, the record only reveals a temporal association between receipt of the Tdap vaccine on October 6, 2016, and Petitioner's subsequent onset and diagnosis (first proposed by Dr. Juarbe toward the end of that month). The intervening record may provide increasing information that (as Dr. Rogers explained) better supported a CIDP diagnosis than before, but it does not show how the vaccine was playing a role in causing this to occur (and Petitioner has not established how that record is consistent with the limited case report evidence filed in this matter).

Petitioner's history of uncontrolled diabetes and related neuropathic symptoms is also a significant stumbling block to finding that the Tdap vaccine caused her CIDP.[10] It cannot be disputed on this record that Ms. Houston suffered from such symptoms, and that they foreshadowed her more directly, CIDP-related symptoms, such as tingling in the hands and feet, long prior to vaccination. Although Dr. Callaghan has not formally proposed that her earlier problems explain her CIDP as an "alternative cause," and I do not find Respondent proved this to be the case, Ms. Houston's overall history casts considerable doubt on the contention that her CIDP was solely or primarily due to vaccination.

[10] Petitioner did not allege a significant aggravation claim—that the Tdap vaccine worsened either her diabetes or CIDP that might have predated vaccination. I therefore do not include an analysis herein of her success in so doing, although I note that her inability to prove the Tdap vaccine "can cause" CIDP negatively impacted her ability to how it could worsen a preexisting case. *Loving v. Sec'y of Health & Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009). In addition, she does not maintain what is sometimes referred to as a "*Shyface*" claim—that the vaccine was a substantial factor in causing her CIDP even if other factors also played an important role, such that one cannot be deemed predominant over any other. *Shyface v. Sec'y of Health & Hum. Servs.,* 165 F.3d 1344, 1352 (Fed.Cir.1999).

Untangling the CIDP-related neurologic symptoms that might have predated vaccination from those Petitioner experienced thereafter is extremely difficult based on this record, despite Dr. Rogers's best efforts. Petitioner seems to have sought emergency treatment regularly, and the overall impression provided by the record is not only that her health was poor, but that she persistently experienced neuropathic symptoms that may have evolved over time into the CIDP she was ultimately diagnosed with—a possibility Dr. Juarbe explicitly proposed. Ex. 3 at 891. In response, Dr. Rogers did highlight many medical record instances from Ms. Houston's past that in his view did not completely corroborate that her CIDP had begun, and argued that Petitioner's pre-vaccination symptoms could better be characterized by symmetrical distal sensory loss caused by her diabetes. Rogers Rep. at 10. He also disputed the strength of prior treater conclusions about the nature of her diabetes, and some of his assertions were reliable.

It remains the case, however, that the totality of the record suggests that (a) Petitioner's overall health was consistently impacted by uncontrolled type 2 diabetes, (b) her neuropathic-like symptoms were more likely a function of that disease than the Tdap vaccine, and (c) the many documented neuropathic symptoms she displayed pre-vaccination were likely associated with what she experienced after. All of the above looks most like the waxing and waning associated with CIDP, and is more consistent with a disease process that preceded vaccination and otherwise had nothing to do with it. Ultimately, I cannot with certainty determine *when* Petitioner's CIDP began, or to what extent it was distinguishable from any neuropathies associated with diabetes (and of course as special master I am not qualified to do so). But I *can* consider, and weigh, evidence that undermines the Petitioner's claim—and here, the evidence does so quite consistently. *See D'Toile v. Sec'y of Health & Hum. Servs.*, 726 Fed. Appx. 809, 811 (Fed. Cir. 2018).

An evidentiary misapprehension about the difference between onset and date of diagnosis may explain why Dr. Rogers's interpretation of Petitioner's pre-vaccination history was unpersuasive. Dr. Rogers put great emphasis on the exam Petitioner received on October 28, 2016, as *objective confirmation* of the CIDP diagnosis—and he may well be correct that the diagnosis could not have reasonably been embraced fully before that date. But in the Program, onset of an alleged vaccine injury is *never* dependent on the date formal diagnosis occurs. *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)). Indeed, *any* initial symptom or manifestation is sufficient to constitute onset, no matter whether the claimant or medical professionals treating him identify it as such. *See Tenneson v. Sec'y of Health & Hum. Servs.*, 142 Fed. Cl. 329, 338 (Fed. Cl. 2019). It is in fact often the case that a disease *cannot* be diagnosed solely on the basis of the first clinical symptom. It may not be until sometime thereafter—when treaters have the benefit of test results and more direct experience with the patient—that a reasonable diagnosis can be ascertained.

As a result, it does not matter if Petitioner's prior symptoms—including the multiple instances in which treaters believed she might be displaying signs of diabetic neuropathy—could not *then* fully support a CIDP diagnosis. Rather, what is significant is that Petitioner's course could have been underway long before vaccination, and/or be attributable to her diabetes. This is wholly consistent with the waxing/waning character of CIDP – a disease process that may take a long time to fully manifest and/or be properly diagnosed. *See, e.g.*, *Daily v. Sec'y of Health & Hum. Servs.*, 07-173V, 2011 WL 2174535 (Fed. Cl. Spec. Mstr. May 11, 2011) (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). Program claimants cannot show a vaccine caused their injury if onset predated vaccination. *Johnson v. Sec'y of Health & Hum. Servs.*, No. 14-113V, 2017 WL 772534, at *16-18 (Fed. Cl. Spec. Mstr. Jan. 6, 2017) (petitioner's expert conceded she could not represent that autoimmune injury more likely than not began after vaccine).

Althen Prong Three

Because Petitioner cannot meet the first two *Althen* prongs, I need not evaluate her success in preponderantly establishing that onset occurred in a medically acceptable timeframe. *Cordova v. Sec'y of Health & Hum. Servs.*, No. 17-1282V, 2021 WL 3285367, at *19 n.9 (Fed. Cl. Spec. Mstr. June 23, 2021). But I will briefly address what the evidence and expert reports said about the matter.

The experts disagreed on precise onset, with Dr. Rogers favoring 19 days post-vaccination while Dr. Callaghan proposing a single week. In either event, persuasive authority exists supporting such onsets for vaccine-induced CIDP as medically acceptable. *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). Respondent did not rebut the reasonableness of vaccine-induced CIDP beginning in either timeframe, and I have only rejected vaccine-CIDP timeframes that were substantially longer. *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).

However, because I have found that the Tdap vaccine has not been shown herein to likely cause CIDP, the reasonableness of onset is unhelpful to Petitioner. And (as discussed above) this record is rife with evidence allowing for the possibility that onset actually *predated* vaccination. As a result, although the *proposed* timeframe for onset may by itself be medically reasonable (assuming the Tdap vaccine could cause CIDP), *this* record makes it difficult to conclude that preponderant evidence establishes onset occurred *within* that otherwise-reasonable timeframe.

CONCLUSION

It cannot be assumed that because some peripheral neuropathies are closely associated with one vaccine, that *any* related condition is likely similarly attributable to *other* vaccines. Rather, claimants must do the "heavy lifting" imposed upon them in causation-in-fact cases and show how the vaccine in question could cause a different condition. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357 at 1360 (Fed. Cir. 2000). What is known about the related condition and vaccine may well supply a useful "roadmap," but in the end the claimant's showing must reliably establish causation.

This has not been accomplished in this case, as the Tdap vaccine has not been shown to likely lead to CIDP. For this reason, I am compelled to dismiss this claim.

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.[11]

IT IS SO ORDERED.

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

[11] 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.