

did so in a medically-acceptable timeframe. Indeed, it is more likely, given the facts of this case, that his CIDP onset predated vaccination.

I. Factual Background

Pre-vaccination History

Mr. Mason was born on February 9, 1948. Ex. 1 at 1 (“Mason Affidavit”); Ex. 3 at 78. His pre-vaccination medical history included anemia, arthritis, bronchial asthma, carpal tunnel syndrome, degenerative disc disease, diverticulosis, gastroesophageal reflux disease, hiatal hernia, hypothyroidism, hyperlipidemia, insomnia, neck pain and stiffness, plantar fasciitis, pancreatitis, sciatica, skin cancer, and spinal stenosis. Ex. 3 at 58–59, 64, 81, 70, 143, 157–58. Mr. Mason’s records also generally document treatment of ongoing back pain throughout 2013.³

Starting in January 2014, Mr. Mason began seeing podiatrist, Dr. Steven Bailey, D.P.M., for right foot pain that had begun three months prior (presumably in October 2013), and Petitioner continued seeing Dr. Bailey for this pain even after the vaccination at issue. Ex. 4 at 1–7. He also reported to Dr. Bailey some foot paresthesias and numbness, among other things, in the months before the vaccination. *See e.g.*, Ex. 4 at 3 (March 10, 2014: Mr. Mason reported pain and intermittent swelling in his “great toe”), 5–6 (August 18 or 19, 2014: Mr. Mason complained of right and left foot pain and two weeks earlier he felt a tingling in his right foot and the swelling in his left foot, but his neurological sensations were intact to pinpoint and vibratory sensation bilaterally), and 7 (September 30, 2014: Mr. Mason reported ongoing pain in his left medial foot and numbness in his right lateral forefoot). During this time, Mr. Mason presented to primary care physician Adrian Carreon, M.D., for his hypothyroidism, sciatica, and degenerative disc disease. *See, e.g.*, Ex. 3 at 58, 64, 74, 76, 78, 81, 143.

Mr. Mason saw orthopedic surgeon Dr. Gabriel Garcia-Diaz twice in May 2014, for back pain that Mr. Mason said began in 2011 and worsened in 2014. Ex. 9 at 1. Mr. Mason reported acute onset of right-sided back pain. *Id.* He also described lower extremity pain in the buttock and on the right side that was made worse by standing, walking, or changing positions. *Id.* He reported limb numbness but no limb weakness. *Id.* After Mr. Mason underwent bilateral sacroiliac joint blocks on June 2, 2014, he reported improvement on June 9, 2014. *Id.* at 17, 68–69.

³ On April 24, 2013, Petitioner reported that he had physical therapy for six weeks with no recurrence of neck and back pain. Ex. 3 at 96. However, on July 12, 2013, he reported back spasms, and on July 24, 2013, he again complained of left upper back pain. *Id.* at 88, 92. Because his job as a truck driver “contribute[d] to his delayed recovery,” Dr. Carreon recommended that Mr. Mason take “at least 10-14” days off. *Id.* at 90. The following month Mr. Mason followed up with Dr. Carreon on August 15, 2013, reporting that he had been attending physical therapy with some improvements but was unable to perform his usual activities such as swimming, walking, biking, or running. *Id.* at 84. He stated that he had worsening back spasms that got worse in the afternoon and later in the day. *Id.* Dr. Carreon approved Mr. Mason’s disability forms, with a modified disability date of September 3, 2013. *Id.* at 86. Mr. Mason ended up retiring in 2013. Ex. 10 at 74.

Of note, during the months leading up to vaccination Mr. Mason had lab work done, and on September 23, 2014, his lab work revealed his sedimentation rate (“ESR”) was 60 and his c-reactive protein (“CRP”) was 5.4. Ex. 3 at 141. Both are biomarkers of inflammation.

Vaccination and Onset of Symptoms

On October 9, 2014, Mr. Mason (now 62 years old) received the flu vaccine. Ex. 2 at 1. During this appointment with primary care physician Miguel Ollada, M.D., Mr. Mason discussed the side effects he was experiencing to anti-inflammatory medication that he was receiving, and his recent elevated ESR result. Ex. 20 at 113. Mr. Mason was assessed with having osteoarthritis, hyperlipidemia, and asthma. *Id.* He was instructed to discontinue the medication that was causing him problems but to continue using his inhalers. *Id.* There is no record evidence close-in-time to this visit of any vaccine reaction.

Later that month (now 19 days after vaccination), on October 28, 2014, Mr. Mason was examined for bilateral foot pain by rheumatologist Meghal Parikh, M.D. Ex. 5 at 37–42. He had previously reported that this pain had persisted for the “last several years”—although no specific time period was mentioned, nor did he mention how long the pain complained of at this time had been occurring. *Id.* at 39. He also reported right foot numbness in his toes and right foot cramping. *Id.* The records note that previously Petitioner had undergone Morton’s neuroma surgery.⁴ *Id.* Dr. Parikh documented bilateral foot pain with paresthesias and prescribed a trial of Gabapentin. *Id.* at 41.

Mr. Mason’s bilateral foot pain and right foot numbness continued thereafter, and he also began to experience swelling as well, causing him to walk more slowly. Ex. 4 at 9; Ex. 6 at 3–4. Accordingly, on November 17, 2014, Mr. Mason returned to his podiatrist, Dr. Bailey, complaining of left foot pain, along with pain on both lateral sides of his feet and toe numbness. Ex. 4 at 9. Dr. Bailey noted that Mr. Mason had “generalized swelling of his foot and there [was] swelling up into his leg.” *Id.* Dr. Bailey was concerned that Mr. Mason’s symptoms could be attributed to a vascular etiology due to the amount of swelling (noting that the swelling was a likely explanation for the increased pain), and Petitioner was instructed to see a vascular surgeon. *Id.*

On November 25, 2014, Mr. Mason was seen by Mehrdad Amirhamzeh, M.D., a vascular doctor. Ex. 6 at 3–4. Mr. Mason reported that he had numbness, pain, swelling, and tingling in his legs and feet bilaterally (although similar to the record from his first to Dr. Parikh, no specific date was listed in this record as to when these symptoms began). *Id.* at 3. Dr. Amirhamzeh ordered Mr. Mason to have a duplex ultrasound of his bilateral lower extremities. *Id.* at 4. The ultrasound

⁴ Although the record states Morton’s “neuroma” surgery occurred, it most likely meant to refer to Morton neuralgia. This is defined as “a form of foot pain, metatarsalgia caused by compression of a branch of the plantar nerve by the metatarsal heads; chronic compression may lead to formation of a neuroma.” See *Dorland’s Illustrated Medical Dictionary* (33d ed. 2020) at 1164 [hereinafter *Dorland’s*].

revealed that all results from this study were within normal limits, and there was no evidence of any significant lower extremity peripheral arterial disease requiring treatment or intervention. *Id.* at 7.

On December 1, 2014, Petitioner saw neurologist Diana J. Hylton, M.D., for assessment of his aching feet and tight neck. Ex. 7 at 2. Mr. Mason told her that these symptoms “started last summer [and] kept getting worse.” *Id.* An electromyography (“EMG”) and nerve conduction study (“NCV”) of Mr. Mason’s lower extremities showed that the “[m]otor nerve amplitudes [were] decreased, consistent with an axonal neuropathy” and “absent L sural but the R sural and R superficial peroneal is normal” *Id.* at 18.⁵ A few days later, Petitioner went back to Dr. Parikh to discuss his recent lab work and his EMG/NCV study. Ex. 5 at 25. Mr. Mason denied improvement despite taking neuropathic-treating medications. *Id.* Dr. Parikh proposed that Mr. Mason’s foot pain was probably due to some form of polyneuritis, with his left foot being more symptomatic than his right, and also was occurring contemporaneously with elevated inflammatory markers. *Id.* at 23. Dr. Parikh increased Mr. Mason’s Gabapentin dosage and ordered lab work to check for vasculitis. *Id.*

On December 10, 2014, Mr. Mason had a follow up appointment with Dr. Carreon regarding his cholesterol medication. Ex. 3 at 54. Mr. Mason now reported having foot pain when walking, numbness and tingling in both feet, and soreness in his neck. *Id.* Dr. Carreon assessed Mr. Mason with having idiopathic peripheral neuropathy without a clear etiology. *Id.* at 56. Dr. Carreon recommended increasing Mr. Mason’s dosage of Gabapentin, although he also noted that it appeared that such pharmaceutical treatment was only working partially. *Id.* at 56–57.

Treatment in 2015 and Search for Explanation

On January 5, 2015, Mr. Mason followed up with Dr. Parikh, again complaining of neck pain and “persistent bilateral foot and left leg pain almost daily.” Ex. 5 at 14. He was assessed with bilateral foot pain “probably due to polyneuritis.” *Id.* at 16. Dr. Parikh recommended a sural nerve biopsy to assess Mr. Mason for vasculitis. *Id.* He was also told to change his medications and to have a consultation for a left sural nerve biopsy. *Id.*

Four days later, on January 9, 2015, Mr. Mason saw Dr. Carreon to follow up on his recent lab results. Ex. 3 at 50. He now complained of aching in both distal legs and feet, along with associated numbness and tingling. *Id.* Dr. Carreon noted that Mr. Mason’s inflammatory markers were still high, but that all extensive serologic tests had produced unremarkable results. *Id.* at 50, 53. Dr. Carreon referred Mr. Mason to Stanford University for a biopsy, opining that Mr. Mason’s “hypothyroidism [might be] secondary to an autoimmune thyroiditis, likely Hashimoto’s,” and ordered more lab work. *Id.* at 53.

⁵ Also documented in Ex. 5 at 30.

On January 27, 2015, neurosurgeon Gordon Li, M.D. conducted a pre-operative examination (prior to Mr. Mason's left sural nerve biopsy). Ex. 8 at 1. Dr. Li noted that Mr. Mason had "a one-year history of progressive bilateral neuropathic foot pain." *Id.* (This would place onset well before the October 2014 vaccination). The next day, on January 28, 2015, Mr. Mason had a follow up appointment with Dr. Carreon regarding his lab results, and he reported his symptoms continued and were resistant to medicinal treatments. Ex. 3 at 46. Dr. Carreon diagnosed Mr. Mason with Hashimoto's thyroiditis, which was confirmed by positive antibody testing. *Id.*

A few days later, on January 30, 2015, Mr. Mason underwent a left sural nerve biopsy procedure at Stanford. Ex. 8 at 3–6. After reviewing the results of the biopsy, a radiologist proposed (in a February 17, 2015 report) that they reflected CIDP, "in which a mixed axonal and demyelinating pathology is common." Ex. 7 at 21. The report particularly noted that "[s]egmental demyelination can be seen in some indolent axonal neuropathies (supported by clinical history of neuropathic pain) or as a feature of primary demyelination neuropathies. The presence of perineurium inflammation may be supportive of [CIDP] in which a mixed axonal and demyelinating pathology is common. . ." *Id.*

Post-CIDP Diagnosis and Treatment

A few months later, Mr. Mason returned to Dr. Parikh on March 5, 2015, noting that his symptoms continued unabated. Ex. 5 at 5–6. Dr. Parikh concluded that Mr. Mason's foot pain was "probably due to polyneuritis, left [foot] more symptomatic than right and associated with significant elevated inflammatory markers." *Id.* at 5. These records also stated that Mr. Mason's sural nerve biopsy confirmed his CIDP diagnosis. *Id.* Later that same month, Petitioner saw Dr. Hylton again, and (based on biopsy, testing, and clinical evidence) he concurred with the CIDP diagnosis. Ex. 7 at 28.

For the remainder of 2015, Mr. Mason continued to follow up with Drs. Parikh and Hylton. Ex. 5 at 1–3; Ex. 7 at 30, 33, 36, 39, 45. With treatment, his foot pain had decreased, and his joints appeared normal overall. Ex. 5 at 1–3; Ex. 7 at 30, 33. As of April 30, 2015, he was "feeling better all the time," as Dr. Hylton noted, but he remained on Prednisone due to continued leg weakness. Ex. 7 at 30. After decreasing his Prednisone dosage his tingling returned in June 2015. *Id.* at 33.

In the fall of 2015, Petitioner received a five-day course of intravenous immunoglobulin ("IVIG") treatments. Ex. 10 at 510. He also again saw Dr. Carreon on November 13, 2015. Ex. 3 at 34. His treatments were noted and some modifications suggested. *Id.* On December 1, 2015, when obtaining an evaluation for transient anemia (later deemed to be likely a side effect of some of his other treatments), he reported that his neuropathy symptoms had begun in March 2015 (or five-plus months post-vaccination). Ex. 11 at 1, 3.

Throughout 2016, Mr. Mason periodically reported foot pain and numbness, and he continued to receive IVIG treatments and take Prednisone. *See, e.g.*, Ex. 7 at 49, 52, 54–55, 57–59, 61–62, 66–68, 71,73; Ex. 10 at 374–77, 380–82, 409–12, 415–17, 444–45, 449, 456. A note

from Dr. Hylton, dated August 30, 2016, summarized Petitioner's clinical course and condition as follows:

[Mr. Mason] has a demyelinating neuropathy which was diagnosed by nerve conduction study in 2014, EMG exam, and nerve biopsy at Stanford which diagnosed a condition called CIDP which requires therapy with Gamunex. [He] has had recurrent flare-ups and is going to require Gamunex 25 g daily for five days intermittently until symptoms are resolved. He continues on prednisone at this time.

Ex. 7 at 63. By 2017, Petitioner developed an ulcer likely related to taking Prednisone, leading him to stop taking it while continuing IVIG, which he received into 2019. Ex. 7 at 78, 83; Ex. 15 at 2-10; Ex. 16 at 11-12, 14-16; Ex. 48 at 6-7.

Mr. Mason's symptoms are ongoing, and he reports that he continues to receive IVIG infusions. Pet. at 8. Mr. Mason's strength, power, and endurance have all deteriorated. *Id.* He used to be a triathlete running miles under seven minutes but can no longer run uphill and has instability when he walks upstairs. *Id.* He also reports constant fatigue, stiffness, and pain. *Id.*

II. Expert Reports

A. *Petitioner's Expert – Lawrence Steinman, M.D.*

Dr. Steinman, an adult and pediatric neurologist, prepared two written reports for the Petitioner. Report, dated October 14, 2019, filed as Ex. 22 (ECF No. 26-1) ("Steinman First Rep."); Report, dated August 23, 2020, filed as Ex. 49 (ECF No. 36-1) ("Steinman First Rep."). Dr. Steinman opines that Mr. Mason suffers from CIDP caused by his October 9, 2014 vaccination.

Dr. Steinman received his undergraduate degree from Dartmouth College, and his medical degree from Harvard Medical School. CV, filed as Ex. 47 (ECF No. 29-5) ("Steinman CV") at 1. He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past 39 years. *Id.*; Steinman First Rep. at 1. He is board certified in neurology from the American Board of Psychiatry and Neurology. Steinman CV at 2. Dr. Steinman has also published hundreds of peer-reviewed publications on neurology and autoimmune disease. *Id.* at 5-47. He holds several patents related to the diagnosis and treatment of autoimmune and demyelinating diseases. *Id.* at 2-3. He presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics at Stanford University. *Id.* at 1.

First Expert Report

Dr. Steinman's initial report begins with a several-page summary of Mr. Mason's medical history and treatment course, including the months leading up to his October 2014 vaccination. Steinman First Rep. at 3-10. Dr. Steinman also detailed Mr. Mason's cross-country racing, and

highlighted how his training regime deteriorated after the vaccination to the point where Petitioner could hardly walk, let alone compete in a triathlon. *Id.* at 9–10.

Diving into his medical theory for how the flu vaccine could trigger CIDP, Dr. Steinman described the mechanisms behind molecular mimicry. Molecular mimicry occurs as a result of amino acid sequences shared between a vaccine’s viral or bacterial particles and structures in human tissue. Steinman First Rep. at 10. The immune system’s reaction to the foreign vaccine antigens produces immune cells that in turn can cross-react against the similar human tissue antigens, mistakenly identifying self structures as foreign. *Id.*; L. Steinman, *Autoimmune Disease*, *Scientific Am.* 106–14, 109 (1993), filed as Ex. 29 on Oct. 18, 2019 (ECF No. 27-7). The theory of molecular mimicry is accepted in the medical community, and can be driven by B cell-produced antibodies or autoreactive T-cells. *See* R. Fujinami et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 *Clinical Microbiology Rev.* 80–94, 8081 (2006), filed as Ex. 31 on Oct. 18, 2019 (ECF No. 27-9). Dr. Steinman admitted that “cross-reactive immune responses between viruses and host are relatively common,” since demonstrating structural/sequential homology between foreign antigens and self structures is easy to do. Steinman First Rep. at 11. Thus, for an autoimmune disease to occur through the mechanism of molecular mimicry, the autoimmune attack must occur “between the virus and host at a ‘disease-related’ epitope.” *Id.*

Science has shown the existence of autoimmune processes occurring via molecular mimicry and resulting in peripheral neuropathies. Steinman First Rep. at 11. One of the strongest examples is provided by the anti-ganglioside antibody response triggered by the bacterium *Campylobacter jejuni*, the cytomegalovirus, and/or the H1N1 influenza virus. The resulting autoimmune process has been demonstrated to be causally associated with Guillain-Barré syndrome (“GBS”)—an acute peripheral neuropathy somewhat akin to CIDP. *See generally* I. Namchamkin et al., *Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome*, *J. Infectious Diseases* 226–33, 231–32 (2008), filed as Ex. 32 on Oct. 18, 2019 (ECF No. 27-10); C.W. Ang et al., *Structure of Campylobacter jejuni Lipopolysaccharides Determines Antiganglioside Specificity and Clinical Features of Guillain-Barré and Miller Fisher Patients*, *Infection & Immunity* 1202–208, 1206–207 (2002), filed as Ex. 33 on Oct. 18, 2019 (ECF No. 28-1); L. Wang et al., *Association of Anti-Ganglioside Antibodies and Anti-CMV Antibodies in Guillain-Barré Syndrome*, *Brain & Behav.* 1–6, 4–5 (2017), filed as Ex. 34 on Oct. 18, 2019 (ECF No. 28-2).

In the instance of any of these identified viruses or bacteria, prior infectious exposure typically precedes the development of GBS. *See* H. Hartung, *Infections and the Guillain-Barré Syndrome*, *J. Neurology, Neurosurgery & Psychiatry* 277, 277 (1999), filed as Ex. 35 on Oct. 18, 2019 (ECF No. 28-3) (“Hartung”). As of Hartung’s publication 20 years ago, the frequency of association with GBS and *Campylobacter jejuni* was 33% “in most parts of the western world,” but in China and Japan the figure was higher, at 45%-60% with GBS, 8%–10% with Epstein Barr virus and GBS, and 10-15% with GBS and cytomegalovirus. Hartung at 277. In this case, however,

the Petitioner had no established pre-onset infection with any of the above (eliminating them as potential causes). Steinman First Rep. at 11–12.

Dr. Steinman then discussed what degree of homology between viral amino acids and myelin basic protein (“MBP”)—the anticipated situs of attack for a demyelinating condition—would be necessary to support his theory. Steinman First Rep. at 12–13. He noted that there were specific types of homologies recognized as sufficient to trigger paralysis in experimental encephalomyelitis (“EAE”), an animal model used to simulate this kind of autoimmune disease. *See e.g.*, A.M. Gautam et al., *A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis*, *J. Experimental Med.* 605-09, 607 (1992), filed as Ex. 36 on Oct. 18, 2019 (ECF No. 28-4) (“Gautam I”) (showing that a peptide with 4 of 11 amino acids induced neuroinflammation as frequently as a native 11 amino acid myelin peptide); A.M. Gautam et al., *Minimum Structural Requirements for Peptide Presentation by Major Histocompatibility Complex Class II Molecules: Implications in Induction of Autoimmunity*, 91 *Proc. Nat’l Acad. of Sci.’s USA* 767–71, 770 (1994), filed as Ex. 37 on Oct. 18, 2019 (ECF No. 28-5) (“Gautam II”) (finding that a six amino acid peptide (with identity at five amino acids) was sufficient to trigger neuroinflammation); A.M. Gautam et al., *A Viral Peptide With Limited Homology to a Self-Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis*, *J. Immunology* 60–64, 63 (1998), filed as Ex. 38 on Oct. 18, 2019 (ECF No. 28-6) (“Gautam III”) (identifying that five of eleven amino acids were sufficient to trigger EAE with involvement of the spinal cord even though only three of the amino acids were consecutive) (collectively, the “Gautam Literature”).

Relying on the above, Dr. Steinman conducted database research (using the Basic Local Alignment Search Tool⁶ (“BLAST”)) into the components of the 2014-15 flu vaccine, looking for shared sequence homologies with MBP as well as the axon proteins at the Node of Ranvier, known as contactin and neurofascin. Steinman First Rep. at 14. He chose these target antigens because of his assumption that they are a likely situs for autoimmune attacks resulting in inflammatory neuropathies like GBS and CIDP. *See, e.g.*, R.A.C. Hughes et al., *Immune Responses to Myelin Antigens in Guillain-Barré Syndrome*, *J. Neuroimmunology* 1–10, 7–9 (1984), filed as Ex. 39 on Oct. 18, 2019 (ECF No. 28-7) (“Hughes”); P.E. Marchiori et al., *Cerebrospinal Fluid and Antiphospholipid Antibodies in Multiple Sclerosis, Guillain-Barré Syndrome and Systemic Lupus Arthematosus*, *Arq Neuro-Psiquiatr* 465–68, 465–66 (1990), filed as Ex. 40 on Oct. 18, 2019

⁶ BLAST is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, <https://blast.ncbi.nlm.nih.gov/Blast.cgi> (last visited Dec. 29, 2021); *see I.J. v. Sec’y of Health & Hum. Servs.*, No. 16-864V, 2021 WL 1232733, at *10, n.17 (Fed. Cl. Jan. 4, 2021), *review granted sub nom. J. v. Sec’y of Health & Hum. Servs.*, 155 Fed. Cl. 20 (2021). Research undertaken to identify such homology has been previously described (by none other than Dr. Steinman) as an “in silica” study—meaning that the research can be conducted via a desktop or personal computer. *See Blackburn v. Sec. of Health & Hum. Servs.*, No. 10-410V, 2015 WL 425935, at *10 (Fed. Cl. Spec. Mstr. Jan. 9, 2015).

(ECF No. 28-8) (“Marchiori”); J. J. Devaux et al., *Nodal Proteins are Targets in Guillain-Barré Syndrome*, *J. Peripheral Nervous Sys.* 62–71, 67–70 (2012), filed as Ex. 41 on Oct. 18, 2019 (ECF No. 28-9) (“Devaux”); J. Kira et al., *Anti-Neurofascin Autoantibody and Demyelination*, *Neurochemistry Int’l* 1–7, 1 (2019), filed as Ex. 42 on Oct. 18, 2019 (ECF No. 28-10) (“Kira”).

Significantly, however, several of these items of literature focused on GBS, trying to draw parallels with CIDP despite its different progression. *See, e.g.,* Hughes, Marchiori. An animal study, Devaux, was also mostly GBS-focused, looking at the role autoantibodies might play in attacking certain “adhesion molecules playing a central role in the formation of nodes of Ranvier,” and thus contributing to GBS’s pathogenesis. Devaux at 62-63, 68. However, Devaux tested for the presence of these autoantibodies in both 100 GBS patients plus 50 CIDP patients, finding their existence in a third of the tested CIDP sample. *Id.* at 62. Devaux therefore has more direct relevant to the issues in this case. But Devaux’s authors specifically allowed that “[t]he causes generating these autoantibodies in GBS remain . . . unknown,” and that the autoantibodies were not associated with any known antecedent illnesses, and thus whether molecular mimicry might explain their development could not yet be ascertained. *Id.* at 69.

Kira, by contrast, did directly consider autoantibodies proposed to mediate CIDP’s pathologic course. *See, e.g.,* Kira at 4-6 (discussing IgG4 autoantibodies against paranodal nerve proteins discovered in “a fraction” of CIDP patients suffering from a specific phenotypic form of the disease). However, the cases of “antibody-positive CIDP” were most common in a demographic group distinguishable from Mr. Mason (younger onset and overall more severe evidence of gait disturbance and tremor). *Id.* at 5. More significantly, Kira’s authors admitted that “[t]he mechanism by which [the relevant] autoantibodies . . . can cause each IgG4 antibody-specific [disease] feature remains to be elucidated,” as well as “the mechanism by which IgG4 antibodies to nodal and paranodal proteins emerge.” *Id.* at 6. Thus, Kira does not implicate vaccines in the production of these autoantibodies. Moreover, its authors speculated that a chronic inflammatory setting might be a *prerequisite* to antibody-driven pathogenesis—suggesting in turn that the studied autoantibodies would play a secondary role in the overall disease process. *Id.*

Dr. Steinman’s BLAST searches revealed several common sequences, such as GSASGVSECRF (shared between MBP and the target antigen of the 2014-2015 flu vaccine), which had five of eleven identical amino acids, and he proposed as a result that this homology with vaccine components was sufficient for a damaging cross-attack by immune cells. Steinman First Rep. at 14–15; *see generally* Gautam Literature. Other BLAST searches found homologies listed above three out of six identical amino acids YTDTYH, which in his view also reached the threshold outlined in cited literature. Steinman First Rep. at 14; *see generally* Gautam Literature. Other searches were comparably fruitful in his estimation. Steinman First Rep. at 15–17. Thus, Dr.

Steinman opined that sufficient homology existed between the components of the flu vaccine and his chosen target antigens for a cross-reaction to occur.⁷ *Id.* at 22.

As noted above, however, Dr. Steinman acknowledged that most people do not develop autoimmune demyelinating diseases simply from the existence of homology. Steinman First Rep. at 22; K. Ota et al., *T-cell Recognition of an Immunodominant Myelin Basic Protein Epitope in Multiple Sclerosis*, *Nature* 183–87, 183–84 (1990), filed as Ex. 44 on Oct. 18, 2019 (ECF No. 29-2); M. Pette et al., *Myelin Basic Protein-Specific T Lymphocyte Lines from MS Patients and Healthy Individuals*, *Neurology* 1770–776, 1775 (1990), filed as Ex. 45 on Oct. 18, 2019 (ECF No. 29-3). Rather, other genetic and environmental factors were necessary before these self-reactive immune response to myelin could trigger inflammatory neuropathies. Steinman First Rep. at 22. It was his contention that such additional factors, such as vaccination, must be present for Petitioner (although he did not identify what they were beyond the very fact that Petitioner received a vaccine and developed CIDP subsequently after). *Id.*

Dr. Steinman next considered record evidence pertaining to Mr. Mason specifically, in an attempt either to show how it was consistent with his theory—or in the alternative how it did not rebut that same theory. Only after Petitioner received the flu vaccine, for example, did he start complaining of tingling in his legs and difficulty walking. Steinman First Rep. at 23; Ex. 4 at 9; Ex. 6 at 3–4. Petitioner also complained of these symptoms occurring bilaterally combined with severe pain. He could no longer run or exercise like he had before the vaccine. Steinman First Rep. at 23; Ex. 19 at 15–16. Thus, Dr. Steinman believed that the onset of Petitioner’s neurologic-like symptoms likely post-dated vaccination.

Petitioner’s pre-vaccination symptoms, by contrast, could be attributed to other ailments. For example, Dr. Steinman associated Petitioner’s previous fractured toe, foot strain, and collapsed arch to his athletic training regime. Steinman First Rep. at 23; Ex. 4 at 5–6. Petitioner’s back pain and pain down his right side was also more likely the result of Petitioner’s SI Joint Syndrome. Steinman First Rep. at 23; Ex. 9 at 8, 13–14. Additionally, Petitioner’s lumbar pain was prevalent since 2011, providing another explanation of his continued pain. Steinman First Rep. at 23; Ex. 9 at 1. And the raised inflammatory markers could have easily been attributed to his osteoarthritis. Steinman First Rep. at 23; Ex. 3 at 58–61, 141. Otherwise, some of these symptoms were inconsistent with a peripheral neuropathy like CIDP, since they manifested asymmetrically, with Petitioner complaining of numbness on his right side. *Id.*; Ex. 3 at 62; Ex. 4 at 7. And before receipt of the vaccine, treaters had not even speculated that Mr. Mason could have a peripheral neuropathy, although they did so thereafter. Steinman First Rep. at 23; Ex. 3 at 56.

⁷ Dr. Steinman also tried to identify molecular mimics between components of the flu vaccine received by Petitioner and antigens that were targeted in GBS by looking at the areas of alignment between those components and either MBP or contactin that were identified in the BLAST search and used a filtration step outlined in the Gautam Literature. Steinman First Rep. at 18–19; *see generally* Gautam Literature. He concluded that this analysis supported his conclusions, as well.

Finally, Dr. Steinman deemed the timing and onset of Petitioner’s symptoms to be medically acceptable when evaluated against the date of vaccination. He placed onset of any neuropathic symptoms to be during the interval of October 17 to November 8, 2014 (or eight days to five weeks post-vaccination), when Petitioner purportedly began experiencing issues with walking.⁸ Onset specifically in his view likely began close to vaccination, gradually worsening over the next three weeks. Steinman First Rep. at 23-24. Dr. Steinman also opined that his onset views were supported by Petitioner’s October 28, 2014 visit with Dr. Parikh, who at that time noted that Petitioner had paresthesia in his feet. Ex. 5 at 41. According to cited literature, the timeframe for onset of any form of inflammatory neuropathy following the influenza vaccination was consistent with Petitioner’s experience. Schonberger et al., *Guillain Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. Epidemiology 105–23, 110–11 (1979), filed as Ex. 46 on Oct. 18, 2019 (ECF No. 29-4) (“Schonberger”). Notably, however, Schonberger *only* discusses GBS, and (as of the filing of the first report) nothing offered by Dr. Steinman spoke about anticipated CIDP onset.

Second Expert Report

Dr. Steinman’s supplemental report began with a several-page summary of Petitioner’s medical history and treatment course, aimed at rebutting Dr. Jamieson’s counter-diagnosis. Steinman Second Rep. at 2. Dr. Steinman opined that Petitioner’s objective testing in the period before vaccination—specifically, Petitioner’s September 30, 2014 sensory exam—established that his earlier symptoms were due to his intense long distance running and triathlete training regime, not initial evidence of peripheral neuropathy or CIDP. Steinman Second Rep. at 2; Ex. 4 at 7–8. Similarly, Dr. Steinman could not find support in the medical records for Dr. Jamieson’s assertion that Petitioner had preexisting small fiber sensory peripheral neuropathy with pure sensory symptoms, because prior to vaccination Petitioner did not “have” peripheral neuropathy or CIDP. *Id.* Dr. Steinman once again argued that Petitioner’s intense training regime was the explanation for his prior back pain (SI Joint Syndrome), right sided leg pain, and differing bilateral foot pain. *Id.*

Dr. Steinman also addressed Petitioner’s elevated ESR and CRP measurements (obtained the month prior to vaccination), opining that they were reflective of hypothyroidism and osteoarthritis. Steinman Second Rep. at 2. He noted that subclinical hypothyroidism and osteoarthritis are a known cause for elevation of ESR and CRP. G. Gupta et al., *Study on Subclinical Hypothyroidism and its Association with Various Inflammatory Markers*, J. Clinical & Diagnostic Res. 4-6, 5 (2015), filed as Ex. 50 on Aug. 24, 2020 (ECF No. 36-2) (“Gupta”). Specifically, for hypothyroidism, Dr. Steinman pointed to several visits that he believed supported this diagnosis. First, he mentioned the February 20, 2014 visit to Dr. Carreon, where lab work

⁸ In fact, the record does not talk about issues of walking, but rather indicates that Petitioner complained primarily of foot pain. *See* Ex. 3 at 54. This foot pain may have occurred *when* walking, but the record does not show that the walking itself was deemed a separate issue (i.e. that Petitioner’s gait had independently become problematic). *Id.*

revealed that Petitioner was euthyroid and had subclinical hypothyroidism. Ex. 3 at 76–77. Second, on July 23, 2014, Dr. Carreon had proposed that based on Petitioner’s symptoms of stiffness in his neck, back pain, chest pain, and right foot pain, Petitioner’s symptoms could reflect polymyalgia or arthralgia, or alternatively might be related to diffuse osteoarthritis. Ex. 3 at 58–61. Dr. Carreon did not detect any systemic abnormality except for hypothyroidism which was still in the subclinical state. *Id.* Thus, there were other known causes for elevated markers of inflammation besides a preexistent inflammatory neuropathy. Steinman Second Rep. at 3.

B. *Respondent’s Expert – Dara G. Jamieson, M.D.*

Dr. Jamieson, a neurologist with extensive experience in the diagnosis and treatment of patients with neuropathies, prepared two written report for Respondent. Report, dated January 4, 2020, filed as Ex. A (ECF No. 31-1) (“Jamieson First Rep.”); Report, dated September 30, 2020, filed as Ex. C (ECF No. 38-1) (“Jamieson Second Rep.”). Dr. Jamieson did not accept Mr. Mason’s CIDP diagnosis, and opined that Mr. Mason suffered from a small fiber peripheral neuropathy. Jamieson First Rep. at 15. She also disputed that the flu vaccine could have caused it. *Id.*

Dr. Jamieson received her undergraduate degree from George Washington University and her medical degree from the University of Pennsylvania School of Medicine. CV, filed as Exhibit B on January 13, 2020 (ECF No. 31-2) (“Jamieson CV”) at 1. She then proceeded to a neurology residency and a cerebrovascular fellowship at the Hospital of the University of Pennsylvania. Jamieson CV at 1. She was a practicing neurologist for 32 years in academic medical centers before transitioning to a teaching appointment as the Clinical Professor of Neurology at Weill Cornell Medicine. *Id.* at 2. She also currently holds editorship positions for Neurology Alert as the headache editor and Current Treatment Opinions in Neurology as the cerebrovascular editor. *Id.* She received specialty board certificates in neurology and vascular neurology by the American Board of Psychiatry and Neurology, neurosonology by the American Society of Neuroimaging, and headache medicine by the United Council for Neurological Subspecialties. *Id.* at 2. Dr. Jamieson has published papers in peer reviewed journals, authored two books and other book chapters, and reviewed articles of multiple neurological topics. *Id.* at 11–16; Jamieson First Rep. at 1.

First Expert Report

Dr. Jamieson extensively reviewed Petitioner’s medical summary, documenting each of Petitioner’s doctor’s visits in a manner consistent with this Decision’s review of the factual record. First Jamieson Rep. at 2–10. She then discussed peripheral neuropathies, or diseases of the peripheral nervous system (“PNS”). *Id.* at 11. These diseases involve damage or loss of the axon (the actual nerve) and/or its myelin covering, and present clinically with “weakness and/or sensory symptoms and generally show muscle atrophy, distal sensory loss, distal greater than proximal weakness, and/or reduced to absent deep tendon reflexes on neurological examination.” *Id.*; B.

Morrison, *Neuromuscular Diseases*, 36 *Seminars in Neurology* 409–18, 408 (2016), filed as Ex. A, Tab 1 on Feb. 27, 2020 (ECF No. 33-1) (“Morrison”).

EMG and NCV studies are used to diagnose and characterize PNS diseases. Jamieson First Rep. at 11; Morrison at 411. An EMG/NCV study can specifically help to differentiate between the two major groups of peripheral neuropathies (axonal vs. demyelinating). Jamieson First Rep. at 11; Morrison at 411. The NCV study works by “electrically stimulating a nerve . . . and recording the response of that nerve . . . [through] examining the amplitude of the response and conduction velocity of the action potential generated, the investigator can determine the number of axons present in the nerve and their state of myelination.” Morrison at 411. As Dr. Jamieson noted, some types of neuropathies, like small fiber sensory neuropathies, may not be associated with any abnormalities on EMG/NCV studies. Jamieson First Rep. at 11.

Next, Dr. Jamieson described CIDP, the injury alleged by Petitioner. She defined CIDP as “a progressive immune-mediated peripheral neuropathy that generally presents with gradually worsening symmetrical numbness and paresthesia (i.e. tingling) followed by prominent . . . distal (e.g. hands and feet) and proximal (e.g. shoulder and thigh), extremity weakness.” Jamieson First Rep. at 12; *See, e.g., P. Dyck & J. Tracy, History, Diagnosis, and Management of Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, *Mayo Clinic Proceedings* 777–93, 778 (2018), filed as Ex. A, Tab 2 on Feb. 27, 2020 (ECF No. 33-2) (“Dyck & Tracy”); A. Peltier & P. Donofrio, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Bench to Bedside*, *Seminars in Neurology* 187–95, 187–88 (2012), filed as Ex. A, Tab 3 on Feb. 27, 2020 (ECF No. 33-3) (“Peltier & Donofrio”). Dr. Jamieson also discussed its clinical presentation, noting that motor weakness is the most prominent neurological symptom showing up on neurological exams, but that it can feature extremity pain as well. Jamieson First Rep. at 12; Dyck & Tracy at 779. Exams can also show some subtle sensory abnormalities, with patients typically presenting with decreased vibration and proprioception (large fiber sensory functions), although sensory loss is rarely prominent. Jamieson First Rep. at 12; Dyck & Tracy at 780. Additionally, deep tendon reflexes in the extremities are decreased or absent. *Id.*

Diagnostic evidence of CIDP can be provided by elevated inflammatory markers such as ESR and CRP (although both are seen in a multitude of other neurological and non-neurological disorders involving inflammation), and there are no known specific serum biomarkers for CIDP at present. Jamieson First Rep. at 13. NCV studies can reveal CIDP-associated demyelination,⁹ which can manifest as “(i) slowing of motor conduction velocities; (ii) lengthening of distal motor latencies; (iii) prolonged minimal F wave latencies, and (iv) partial conduction block. *See J. Vallat et al., Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Diagnostic and Therapeutic Challenges for a Treatable Condition*, 9 *Lancet Neurology* 402–12, 405 (2010), filed as Ex. A, Tab 4 on Feb. 27, 2020 (ECF No. 33-4) (“Vallat”). Segmental demyelination and remyelination to a variable degree on teased fiber preparations, subperineural or endoneural edema, onion-bulb

⁹ Defined as “destruction, removal, or loss of the myelin sheath of a nerve or nerves.” *Dorland's* at 480.

formation, and endoneurial inflammatory mononuclear cell infiltrates are seen on biopsies of affected nerves as additional supportive criteria for CIDP. *Id.* at 405. Spinal fluid protein levels are also usually elevated in CIDP patients (although this, like the presence of inflammatory biomarkers, is common to many other neurological disorders). *See* Dyck & Tracy at 778.

Because many neuropathies can resemble CIDP, to ensure the correct course of treatment it is important to determine the nature of suspected peripheral nerve damage (and whether it is reflective of CIDP or a different PNS condition). Jamieson First Rep. at 12. Dr. Jamieson stated that testing by routine blood work, spinal fluid analysis, EMG/NCV studies, and nerve biopsies could be appropriate in place of more involved or specific testing.¹⁰ *Id.* Initial treatment of CIDP can include intravenous immunoglobulin with steroids, plasma exchange, and steroid-sparing immune-modulating drugs (used in treatment-resistant cases). *See* P.E. Doneddu & E. Nobile-Orazio, *Management of Chronic Inflammatory Demyelinating Polyradiculopathy*, 31 *Current Op. Neurology* 511–16, 511 (2018), filed as Ex. A, Tab 5 on Feb. 27, 2020 (ECF No. 33-5) (“Doneddu I”). Since there are no widely-recognized laboratory biomarkers for CIDP, Dr. Jamieson believed that treatment must be fashioned in light of objective evidence of disease course, like improvement of motor weakness or EMG/NCV abnormalities. Jamieson First Rep. at 13; Doneddu I at 2.

Dr. Jamieson also differentiated between CIDP and acute inflammatory demyelinating polyradiculoneuropathy (“AIDP”), a common form of GBS. Jamieson First Rep. at 13. Though both GBS/AIDP and CIDP are inflammatory neuropathies presenting with predominant motor symptoms, CIDP has an indolent and chronic course of more than eight weeks to maximum weakness, while in AIDP weakness progressively worsens to its maximum in half the time—less than four weeks. Vallat at 402. Additionally (and importantly), patients with CIDP respond to steroid treatment, unlike AIDP patients. *Id.* at 407–08. This is further evidence that these admittedly-related conditions are nevertheless distinct.

Dr. Jamieson then described sensory neuropathies, the diagnostic classification she favored for Petitioner’s injury. Jamieson First Rep. at 13. Sensory neuropathies are caused by injury to the small, thinly myelinated, and unmyelinated sensory nerve fibers. *Id.* They can present clinically as pain distally in the arms and legs, but most patients have painful sensations limited to the feet. *Id.*; A.J. Terkelsen et al., *The Diagnostic Challenge of Small Fibre Neuropathy: Clinical Presentations, Evaluations, and Causes*, 16 *Lancet Neurology* 934–44, 934–35 (2017), filed as Ex. A, Tab 6 on Feb. 27, 2020 (ECF No. 33-6) (“Terkelsen”).

Exams for small fiber sensory peripheral neuropathy include temperature and pin prick to assess loss of sensation to primary small fiber sensory modalities. Jamieson First Rep. at 13; Terkelsen at 935. This may show a decrease in reflexes, and less likely a subtle distal extremity

¹⁰ Dr. Jamieson listed specific examples as metastatic bone surveys for myeloma, serum electrophoresis with immunofixation for monoclonal gammopathies, heavy metal screening for toxic neuropathies, and human immunodeficiency virus for HIV associated sensory and motor neuropathies when CIDP or another peripheral neuropathy was suspected. Jamieson First Rep. at 12.

weakness. Jamieson First Rep. at 13; Terkelsen at 935. Other evaluations, like blood work, electrodiagnostic studies, cerebrospinal fluid analysis, and histopathologic analysis of nerve and skin biopsy specimens can rule out other causes of PNS symptoms. Jamieson First Rep. at 13; Terkelsen at 936–38. However, there are no specific blood markers, abnormalities on electrodiagnostic studies, or elevated levels on spinal fluid analyses that can be relied upon to diagnose small fiber sensory neuropathies. Jamieson First Rep. at 13.

Diagnostic criteria for small fiber sensory neuropathies can be variable but include the following: the presence of length-dependent symptoms, clinical signs of small fiber damage, a normal nerve conduction study, abnormal autonomic testing (such as abnormal QST thresholds at the foot), and reduced intraepidermal nerve fiber density on skin biopsy. *Id.*; Terkelsen at 941. Dr. Jamieson noted that while some small fiber sensory polyneuropathies are associated with underlying toxic, metabolic, infectious, inflammatory, or autoimmune causes, their etiology remains unknown. Jamieson First Rep. at 13; K.G. Gwathmey & K.T. Pearson, *Diagnosis and Management of Sensory Polyneuropathy*. The BMJ 1–23, 3 (2019), filed as Ex. A, Tab 7 on Feb. 27, 2020 (ECF No. 33-7) (“Gwathmey”). She also stated that “the treatment of sensory polyneuropathies can be directed toward the presumptive cause, or can be purely symptomatic, without any specific curative therapy.” Jamieson First Rep. at 13; Gwathmey at 13.

Dr. Jamieson then touched on CIDP and its links to the flu vaccine. Unlike other peripheral neuropathies like GBS, CIDP has rarely been associated in the medical literature with a single triggering antigen. Jamieson First Rep. at 14; Vallat at 402. And only a few case reports even recorded instances of CIDP or CIDP-like illnesses occurring after a flu vaccine’s receipt. *See, e.g.,* G. Remiche et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP) Associated to Hereditary Neuropathy with Liability to Pressure Palsies (HNPP) and Revealed After Influenza AH1N1 Vaccination*, Acta Neurologica Belgica 519–22, 521–22 (2013), filed as Ex. A, Tab 8 on Feb. 27, 2020 (ECF No. 33-8); S.Y. Tay & W.P. Chan, *A 9-Year-Old Female with Bilateral Leg Weakness After Influenza Vaccination*, 43 Pediatric Annals 440–41, 441 (2014), filed as Ex. A, Tab 9 on Feb. 27, 2020 (ECF No. 33-9). However, these articles were case reports examining the circumstances of individual patients, and did not reach conclusions as to causality.

At the same time, some larger studies seemed to conclude no vaccine-CIDP relationship exists. In one such article—a case-control study of data on antecedent events occurring 1-42 days before onset based on the medical information of 411 CIDP patients derived from an Italian database—32 (8%) patients had a flu-like syndrome, 9 (2%) patients had an upper respiratory infection, and 9 (2%) patients had a gastrointestinal infection, but only 7 (1.5%) patients had received the flu vaccine (with the remaining six patients having recently undergone surgery or experienced trauma). P. Donedu et al., *Risk Factors for Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP): Antecedent Events, Lifestyle and Dietary Habits. Data from Italian CIDP Database*, 0 Eur. J. Neurology 1–8, 1, 3 (2019), filed as Ex. A, Tab 12 on Feb. 27, 2020 (ECF No. 33-12) (“Donedu II”). Thus, vaccination was among the lesser-associated antecedent events to a CIDP case (at least based on this single study)—although Donedu II is no

more evidence of a lack of causation than would be similarly-reported data of a temporal relationship between vaccination and post-administration onset.

Regarding an association between sensory polyneuropathies and the flu vaccine, Dr. Jamieson commented that she could not find a single case in the medical literature connecting the two. Jamieson First Rep. at 14. However, she maintained that small fiber sensory neuropathies have rarely been reported after vaccination of any kind. *Id.* Dr. Jamieson pointed to one article where 5 patients developed paresthesias within one day to two months following vaccination for rabies, varicella zoster, or Lyme disease. N. Souayah et al., *Small Fiber Neuropathy Following Vaccination for Rabies, Varicella or Lyme Disease*, 27 *Vaccine* 7322–25, 7322–23 (2009), filed as Ex. A, Tab 13 on Feb. 27, 2020 (ECF No. 33-13) (“Souayah”). In Souayah, there was mild distal sensory loss with preserved motor strength, which was typical in small fiber neuropathies. Souayah at 7322. Additionally, electrodiagnostic studies were unremarkable and empiric immunomodulatory therapy was ineffective, but a skin biopsy showed decreased epidermal nerve fiber densities. *Id.* at 7322–23.

Based on an in-depth overview of Petitioner’s medical history and the discussion of different types of PNS diseases, Dr. Jamieson opined that Petitioner had bilateral foot pain and numbness, consistent with a pure sensory neuropathy, and that his symptoms began prior to receiving the flu vaccine on October 9, 2014.¹¹ Jamieson First Rep. at 14. His pre- and post-vaccination symptoms all evidenced a form of small fiber sensory peripheral neuropathy. *Id.* at 15; Ex. 3 at 62–65, 58–61; Ex. 4 at 1, 7–8. She particularly highlighted the fact that he suffers to this day from “decreased strength and ongoing weakness in my legs,” but saw no neurologic exam confirmation of lower extremity weakness. Jamieson First Rep. at 10; Mason Affidavit at 4.

In comparison, Dr. Jamieson noted the extent to which Mr. Mason’s symptoms were, in her opinion, inconsistent with CIDP. For example, Mr. Mason had no lower extremity weakness related to a peripheral neuropathy, which had never been documented on neurological examination. Jamieson First Rep. at 10; Mason Affidavit at 4; Ex. 9 at 25; Ex. 15 at 93. During this time, Mr. Mason was also bicycling in 2015 and 2016 and he competed in a triathlon in 2015. Mason Affidavit at 4; Ex. 9 at 25; Ex. 15 at 93. Dr. Jamieson used this to illustrate the degree to which Mr. Mason’s leg pain preceded the vaccination. Jamieson First Rep. at 10. Dr. Jamieson also stated that Mr. Mason’s symptoms and abnormal laboratory values that lead to consultations with the neurologist and the rheumatologist were present prior to his vaccination, so the symptoms were likely unrelated to the vaccine. *Id.* And Mr. Mason’s retirement was not due to symptoms related to vaccination as his disability occurred in 2013, a year before his vaccination. *Id.*; Mason Affidavit at 4.

¹¹ Dr. Jamieson also noted that Mr. Mason had long-standing low back and radiating leg pain due to lumbar spine arthritis as noted on spine imaging. Jamieson First Rep. at 15.

Dr. Jamieson thus disagreed with the CIDP diagnosis, arguing that the clinical symptomatology, the electrophysiological testing, and the nerve biopsy did not corroborate it. Jamieson First Rep. at 10. Mr. Mason did not have weakness, large fiber sensory loss with decreased vibration and proprioception, or decreased-to-absent deep tendon reflexes—all of which would be clinical criteria necessary to support the CIDP diagnosis. *Id.* at 14–15. Dr. Jamieson also specifically took issue with the extent to which Petitioner’s EMG/NCV test results were consistent with CIDP, since they did not show the presence of “slowed motor conduction velocities, lengthening of distal motor latencies, prolonged minimal F wave latencies and conduction block seen in CIDP on nerve conduction studies.” *Id.* at 15. And the neuropathological hallmarks of segmental demyelination and remyelination to a variable degree on teased fiber preparations, onion-bulb formation, subperineural or endoneural edema and inflammatory mononuclear cell infiltrates were not seen on Mr. Mason’s sural nerve biopsy. *Id.* Additionally, Mr. Mason did not respond to immunological therapy as would be expected if he had CIDP. *Id.* at 10, 15. Finally, Mr. Mason repeatedly reported a lack of symptomatic improvement, even though proper CIDP treatment would more commonly produce a decline in symptoms. *Id.* at 15.

Dr. Jamieson briefly touched on Dr. Steinman’s theory of causation, arguing that it failed to explain Mr. Mason’s pre-vaccination symptoms. Jamieson First Rep. at 11. Indeed, Dr. Steinman gave some partial credence to the fact that Petitioner *did* have prior symptoms or evidence of some kind of underlying problem, like the inflammatory biomarker findings, even if he attempted to distinguish their cause. *Id.*; Steinman First Rep. at 23.

Dr. Jamieson also took issue with Dr. Steinman’s assessment of Petitioner’s condition prior to vaccination. Although Dr. Steinman opined that Mr. Mason did not have any pre-existing peripheral neuropathy or CIDP, Dr. Jamieson argued that this was inconsistent with Mr. Mason’s medical record. Jamieson First Rep. at 10. Both Dr. Jamieson and Dr. Steinman agree that Mr. Mason’s low back pain, which radiated down his leg was due to degenerative lumbar spine disease with nerve root impingement. *Id.*; Steinman First Rep. at 24. However, Dr. Steinman believed that Petitioner’s complaints of tingling and difficulty walking began *after* vaccination, whereas Dr. Jamieson reasoned that these problems more likely started long before. Dr. Jamieson noted that in Dr. Steinman’s report alone, there was ample medical documentation of Mr. Mason’s congruent symptoms prior to vaccination. Jamieson First Rep. at 11; *See, e.g.*, Ex. 4 at 1 (January 13, 2014 visit complaining of right foot pain for the past three months); Ex. 3 at 62–65 (April 28, 2014 visit complaining of right foot and heel numbness); Ex. 3 at 58–61 (July 23, 2014 visit complaining of right foot pain); Ex. 4 at 7–8 (September 30, 2014 visit complaining of bilateral foot numbness).

Finally, Dr. Jamieson contested Dr. Steinman’s determination of onset. Dr. Steinman opined that onset occurred between October 17, 2014 and November 8, 2014, because this was the period in which a marked change in Mr. Mason’s ability to walk (approximately eight days to five weeks post-vaccination) was observed. Jamieson First Rep. at 11; Steinman First Rep. at 24. Dr. Steinman corroborated this timeframe with Mr. Mason’s October 28, 2014 visit to Dr. Parikh, which noted that Mr. Mason had paresthesia in his feet. Jamieson First Rep. at 11; Steinman First

Rep. at 24. However, Dr. Jamieson noted that this visit actually documented that Mr. Mason “has bilateral foot pain *since last several years*,” with symptoms that increased with walking or running, numbness over right sided toes, and foot cramping. Jamieson First Rep. at 11; Ex. 5 at 39 (emphasis added). Thus, the very record relied upon by Dr. Steinman revealed a pre-vaccine onset. Jamieson First Rep. at 11.

Second Expert Report

Dr. Jamieson’s supplemental report emphasized that her opinions and diagnosis remained unchanged even after reading Dr. Steinman’s Report. Jamieson Second Rep. at 1. She reasserted her view that Mr. Mason suffered from a small fiber peripheral neuropathy with pure sensory symptoms, unrelated to the flu vaccine, and that his neuropathic symptoms predated his vaccination. *Id.* at 1, 3.

Dr. Jamieson raised two key issues in reaction to Dr. Steinman’s statement that “[o]bjective testing in the period before immunization does not support Dr. Jamieson’s position. Instead, the testing is supportive of Petitioner’s case regarding his long distance running and triathlete training regime. The elevated ESR and CRP were likely indicative of hypothyroidism and osteoarthritis.” Jamieson Second Rep. at 1; Steinman Second Rep. at 2.

First, based on an overview of Petitioner’s medical history and testing results, Dr. Jamieson argued that Mr. Mason’s medical records clearly indicated that in 2013 and 2014, Mr. Mason was experiencing bilateral foot pain consistent with a small fiber peripheral neuropathy, and that he was not engaged in an “intense exercise regime” as alleged by Dr. Steinman. Jamieson Second Rep. at 1. During that period, Dr. Jamieson argued that Mr. Mason was not participating in a “long distance running and triathlete training regime” that was causing his lower extremity symptoms, because Mr. Mason was on disability for back pain in 2013 and subsequently retired from his job later that year as a result of this pain.¹² Jamieson Second Rep. at 1; Ex. 3 at 86; Ex. 10 at 74. Dr. Jamieson stated that because of this disability, it “indicat[ed] that he was not physically capable of the intensive athletic training that Dr. Steinman’s contends caused his pre-vaccination neuropathic lower extremity pain.” Jamieson Second Rep. at 2. Dr. Jamieson drew upon a long history of events to substantiate her argument.¹³

¹² The record does, however, establish that Mr. Mason was participating in long distance runs in 2013 and 2014. Ex. 17 at 1. Mr. Mason competed in 14 races constituting a few triathlon’s, one duathlon, and a few shorter courses from April 20, 2013 to July 27, 2014. *Id.*

¹³ Thus, Dr. Jamieson noted that starting on April 24, 2013, Mr. Mason underwent six weeks of physical therapy for neck and back pain. Ex. 3 at 96. On August 15, 2013 Mr. Mason was undergoing physical therapy for back spasms, and had not been able to swim, walk, bike or run which were his usual activities, so his disability was continued. Ex. 3 at 86. On January 29, 2014 Mr. Mason reported bilateral foot pain and 4-6 weeks of recuperation because of his lower extremity complaints. Ex. 3 at 78–81. Mr. Mason reported on February 20, 2014 that he had not been running and was seeing his podiatrist, Dr. Bailey, for his foot problems. Ex. 3 at 74. Problems with pain in his feet continued in 2014, with Mr. Mason reporting to Dr. Bailey on March 27, 2014 that could do exercises but not run. Ex. 4 at 70. On July 23, 2014, Mr. Mason stated to Dr. Carreon that he was not feeling well, with a stiff neck, back pain, foot pain

Additionally, Dr. Jamieson disputed Dr. Steinman's contention that the inflammatory biomarker test results were the product of hypothyroidism and osteoarthritis. Steinman Second Rep. at 2–3. She noted that blood work collected from Mr. Mason on September 23, 2014, showed an elevated ESR of 60 (reference 0-20 mm/h) and CRP at 5.4 (reference < 0.8 mg/dl). Ex. 3 at 141. Although she was unable to propose the cause for these results, she commented that they predated the October 2014 flu vaccination. Jamieson Second Rep. at 2. Thus, they could not have been caused by his vaccination, or any allegedly immunological or inflammatory processes opined to have started after his vaccination. *Id.* Dr. Jamieson also commented on the Gupta article used by Dr. Steinman to support his contention for hypothyroidism and osteoarthritis, claiming that it was not fully supportive of his proposed counter-explanation. Steinman Second Rep. at 2–3. Mr. Mason's elevated CRP was admittedly in the range that was associated with subclinical hypothyroidism (5.2 ± 1.78). Gupta at 2. However, Mr. Mason's elevated ESR was significantly out of the article's range of values that were associated with subclinical hypothyroidism (20.81 ± 3.02). *Id.*

Second, Dr. Jamieson reiterated her opinion that Mr. Mason had a small fiber sensory neuropathy that was symptomatic prior to vaccination. Jamieson Second Rep. at 2. Her proposed small fiber sensory neuropathy diagnosis was based on the description of the patient's distal sensory symptoms from the record. *Id.*; Ex. 3 at 50; Dyck & Tracy at 778; Morrison at 408; Terkelsen at 934–35. There are no specific blood markers, abnormalities on electrodiagnostic studies, or elevated levels on spinal fluid analysis that diagnose small fiber sensory neuropathies. Jamieson First Rep. at 11, 13; Jamieson Second Rep. at 2. However, Dr. Jamieson noted that a skin biopsy is occasionally performed to look for a decrease in epidermal nerve fiber density, which has been suggested as a dermatopathological correlation with small fiber sensory neuropathies. Jamieson First Rep. at 13; Jamieson Second Rep. at 2; Terkelsen at 936–38. No such skin biopsy was ever performed on Mr. Mason.

III. Procedural History

Mr. Mason filed his Petition on September 29, 2017. Pet. at 1. By October 5, 2017, Petitioner filed all relevant medical records and a Statement of Completion. ECF No. 9. A year later, Respondent filed a Rule 4(c) report on October 5, 2018, contesting Petitioner's right to compensation. ECF No. 18. Petitioner then filed his expert report from Dr. Steinman on October 15, 2019. ECF No. 26. Respondent thereafter filed an expert report from Dr. Dara Jamieson on January 13, 2020. ECF No. 31. Subsequently, Petitioner and Respondent filed supplemental expert

and weight loss. He was started on thyroid replacement medication. Ex. 3 at 58–61. On August 18, 2014, Mr. Bailey believed that Mr. Mason had a fractured toe. Ex. 4 at 5–6. Then on September 30, 2014, he complained of bilateral foot pain and numbness. Ex. 4 at 7–8. When Mr. Mason consulted with a rheumatologist on October 28, 2014, he complained of bilateral foot pain lasting several years, with symptoms increasing with walking or running that according to Dr. Jamieson clearly predated the flu vaccination. Ex. 5 at 37–42.

reports from Dr. Steinman and Dr. Jamieson. ECF Nos. 36, 38. Petitioner then suggested settlement discussions, but Respondent was not amenable to settlement. ECF Nos. 39, 41.

After, the matter was transferred to me on January 26, 2021, I held a status conference with the parties and subsequently set a schedule for a ruling on the record. Later, Petitioner filed a motion for ruling on the record on May 14, 2021. ECF No. 47 (“Mot.”). Respondent filed a response to the motion for ruling on the record on August 16, 2021. ECF No. 49 (“Opp.”). Subsequently, the Petitioner filed a reply to the response for a ruling on the record on September 28, 2021. ECF No. 51 (“Reply”). The matter is now ripe for resolution.

IV. Parties’ Arguments

Petitioner argues that he was correctly diagnosed with CIDP, and that he can establish causation based upon the three-prong test set in *Althen v. Sec’y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). Mot. at 19–22; Reply at 1–2. Scientific literature, he purports, supports his contention that flu vaccines can cause peripheral neuropathies (both GBS and CIDP) via the medically-reliable mechanistic theory of molecular mimicry. Mot. at 28–33; Reply at 2–4. He compares his arguments about the propriety of molecular mimicry herein to what was advanced in three other cases. *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) (Flu/CIDP); *Strong v. Sec’y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666, at *19 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (Flu/GBS); *Daily v. Sec’y of Health & Hum. Servs.*, No. 07-173V, 2011 WL 2174535, at *8 (Fed. Cl. Spec. Mstr. May 11, 2011) (Flu/GBS and/or CIDP). *Tomsky*, however, merely assumed for sake of argument (in turn based on prior special master decisions) that CIDP’s relationship to GBS meant it plausibly could be caused by the flu vaccine—but without a reasoned evaluation of the success of the showing in the case. *Tomsky*, 2020 WL 5587365, at *15–17 (denying entitlement under the other two *Althen* prongs). And *Daily* relied on a standard of mere plausibility. *Daily*, 2011 WL 2174535, at *8.

Mr. Mason next argues that he has demonstrated that the flu vaccine did cause his CIDP, primarily due to the absence of other explanations for the injury. Mot. at 33–36; Reply at 4. He noted that although he had preexisting conditions, such as back and foot pain, they were too far removed from onset of his actual neuropathic symptoms. Mot. at 34. Finally, the timing of his onset—approximately 8-30 days after receiving his flu vaccine—constitutes a medically-acceptable timeframe, which Petitioner argued was supported by medical records, his affidavit, and other affidavits from his wife and a colleague, along with medical literature on GBS and the defined 3-42 day period for onset for a GBS Table claim in the Program. Mot. 22–27; Reply at 5.

In opposing entitlement, Respondent questions the factual basis for the alleged injury, maintaining that Mr. Mason actually suffers from a small fiber peripheral neuropathy, as proposed by Dr. Jamieson. Opp. at 9–14. Moreover, Respondent contends that even if Petitioner could establish CIDP as the proper diagnosis, the claim still fails under the *Althen* prongs. *Id.* at 15.

Under *Althen* prong one, Petitioner has not preponderantly established a reliable medical theory casually connecting his flu vaccination to CIDP, relying instead on prior caselaw and Dr. Steinman’s connection between viral infections and GBS—a related, but ultimately distinguishable peripheral neuropathy. *Id.* at 15–16. Under *Althen* prong two, the record does not support the conclusion that the flu vaccine likely caused his injury because the symptoms he complained of predated vaccination, along with the fact that no treater ever proposed his injuries were caused by the vaccine. *Id.* at 17. Finally, Petitioner’s showing under *Althen* prong three bearing on onset timeframe also fails because the record suggests onset began prior to vaccination. Petitioner’s reliance on medical literature focused on GBS and the onset of a GBS Table claim is misplaced and insufficient. *Id.* at 18.

V. Applicable Law

A. Standards for Vaccine Claims

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).¹⁴ In this case, Petitioner cannot assert a Table claim based on CIDP.

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; see also *Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec’y 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;

¹⁴ 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).

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.

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

As noted by the Federal Circuit, “[m]edical records, in general, warrant consideration as trustworthy evidence.” *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) (“[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law”), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs.*, 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) (“[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms”).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) (“[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.”)).

However, the Federal Circuit has also noted that there is no formal “presumption” that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec’y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral 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. *La Londe v. Sec’y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff’d*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec’y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec’y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec’y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). Under *Daubert*, the factors for analyzing the reliability of testimony are:

- (1) whether a theory or technique can be (and has been) tested;
- (2) whether the theory or technique has been subjected to peer review and publication;
- (3) whether there is a known or potential rate of error and whether there are standards for

controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

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

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

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

D. Consideration of Medical Literature

Both parties filed numerous items of medical and scientific literature in this case, but not 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. Standards for Ruling on the Record

I am resolving Petitioner’s claim on the filed record, and the parties have not challenged my determination to do so. Mot. at 1; Opp. at 1. 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. An Overview of Relevant Medical Terms and Prior Decisions

As noted above, CIDP has been defined as a progressive, immune-mediated peripheral neuropathy that occurs due to an autoimmune attack. Dyck & Tracy at 777–78. It results in weakness, numbness, paresthesia, and sensory ataxia that presents as relapsing-remitting, stepwise progressive, or gradually progressive. *Id.* These symptoms tend to be symmetrical and involve lower and upper limbs. *Id.* at 780. Pain is a less common symptom. *Id.* at 779.

Despite the fact that it is a peripheral neuropathy featuring demyelination—akin to the extremely common Program injury of GBS—CIDP is distinguishable, and therefore caution is warranted in assuming that what is scientifically and medically known about one applies in full to the other. In particular, GBS has an acute onset, is monophasic, and is not steroid-responsive. Peltier & Donofrio at 188; Vallat at 402. CIDP also tends to progress over a longer period of time and can be chronic. *Id.*

Dr. Jamieson has also put small fiber neuropathies into contention, so brief consideration of their characteristics is in order. Small fiber neuropathies are characterized by injury to the small, thinly myelinated, and unmyelinated sensory nerve fibers, presenting with neuropathic pain in the feet, and in some instances sensations of burning, prickling, aching, or itching. Terkelsen at 934–35. The onset of these symptoms can emerge gradually and progressively worsen or occur more rapidly. Gwathmey at 2. They can be confirmed by a skin biopsy, while the kinds of EMG or NCS testing used to evaluate CIDP have far less utility. *Id.* at 10, 12.

There are many prior cases in which Petitioners alleging the flu vaccine caused CIDP have obtained favorable results.¹⁵ See, e.g., *Jastisan v. Sec’y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950, at *1–3 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). I have myself acknowledged their existence in my own prior decisions, and the fact that such determinations should be given *some* consideration as persuasive guidance (although *settled* cases certainly lack precedential value, in comparison to reasoned decisions). See *Strong*, 2018 WL 1125666, at *20. Petitioner also correctly highlights some prior determinations, like *Daily*, that have found that the flu vaccine can cause CIDP (although *Daily* is now more than ten years old, and seems to apply a plausibility standard to the first *Althen* prong that the Federal Circuit has since uniformly rejected).¹⁶ *Daily*, 2011 WL 2174535, at *8.

However, I have identified no more-recent *reasoned* decisions in which a special master explained how or why the flu vaccine was likely causal of the claimant’s CIDP. Rather, special masters have consistently relied on the fact that CIDP and GBS have tended to be lumped together as comparable 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 CIDP. See, e.g., *Tomsky*, 2020 WL 5587365, at *15; *Strong*, 2018 WL 1125666, at *22.

There are in fact several decisions from 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

¹⁵ 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.

¹⁶ Going a bit further back, in a case that is now 17 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 (like many cases before or after) relied on older literature that seemed to assume that CIDP and GBS were two sides of the same coin.

condition was aggravated by two influenza vaccinations. *Jacunski v. Sec’y of Health & Hum. Servs.*, No. 09-524V, 2014 WL 5168422 (Fed. Cl. Spec. Mstr. Sept. 23, 2014) (flu vaccine did not significantly aggravate CIDP). The special master highlighted an Institute of Medicine report (among other things) which specifically found insufficient available evidence to support an association between influenza vaccine and CIDP (in contrast to the flu-GBS association). *Jacunski*, 2014 WL 5168422, at *14.

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.” *Broekelschen v. Sec’y of Health & Hum. Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). 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 a determination that he did in fact likely suffer from CIDP, while Respondent argues that he actually suffered from a small fiber peripheral neuropathy. On this issue, both sides raise reasonable points. Petitioner can point to several treater diagnostic conclusions from the record—a strong kind of evidence to be sure, although it is not considered sacrosanct and does not necessarily bind me. The CIDP diagnosis is, further, supported by several biopsies, as well as clinical evidence of neuropathic symptoms and EMG/NCV results. Ex. 7 at 18. And Dr. Steinman, a respected and capable neurologist, has concurred in the diagnosis as well.¹⁷ All of the above is worthy of considerable weight.

Respondent and Dr. Jamieson, by contrast, point to record evidence of symptoms (like bilateral foot pain and numbness) that long predated the vaccine, but could be consistent with a small fiber neuropathy. And although there is a lack of treater support for Respondent’s preferred diagnosis, Dr. Jamieson emphasizes Petitioner’s repeated complaints of foot pain (consistent with a small fiber neuropathy), contrasting that to an absence of some clinical proof associated with CIDP, like a lack of large fiber sensory loss with decreased vibration and proprioception, or decreased to absent deep tendon reflexes. And she denies that the EMG/NCV testing was in fact corroborative of CIDP as well.

Overall, the facts preponderate in Petitioner’s favor on the question of diagnosis. I find many of Dr. Jamieson’s arguments about CIDP persuasive, and agree that certain “classic” criteria for the diagnosis are missing. However, Petitioner has offered enough evidence for me to determine it more likely than not that he did experience CIDP. The treaters’ views, as well as the

¹⁷ I do note, however, that one of Dr. Steinman’s primary articulated basis for accepting CIDP as the proper diagnosis was merely because Petitioner had no obvious neuropathic symptoms pre-vaccination (coupled with his assertion that all prior symptoms were unrelated)—a somewhat weak basis for supporting the diagnosis. I nevertheless give his determination some weight, in light of his overall neurologic expertise.

diagnostic results, are especially reliable evidence on the point. Dr. Jamieson for her part could offer no treater support—let alone the skin biopsy so often relied on to confirm the small fiber neuropathy diagnosis. Nevertheless, my resolution of this matter in Petitioner’s favor does not at all end the analysis.

III. Petitioner Has Not Carried His Burden of Proof¹⁸

1. *Althen Prong Two*

The record does not permit the conclusion that the flu vaccine likely “did cause” Mr. Mason to experience CIDP. First, no treaters ever proposed any association between the October 2014 vaccinations and Petitioner’s subsequent diagnosis. I have in this case credited treater opinions that Petitioner’s overall presentation was consistent with CIDP, despite Dr. Jamieson’s contentions about holes in that diagnosis—but this also means that the *lack* of any treater support for the idea that his injury was linked to vaccination is deserving of attention as well.

Second, Petitioner’s medical history strongly suggests that the symptoms pointed to by Dr. Steinman as neurologic *predated* vaccination—making it impossible for Petitioner to prove the vaccine was causal. *See Johnson v. Sec’y of Health & Hum. Servs.*, No. 14-113V, 2017 WL 772534, at *16–18 (Fed. Cl. Spec. Mstr. Jan. 6, 2017) (noting that because petitioner’s expert conceded she could not represent that autoimmune injury more likely than not began after vaccine, the “did cause” element could not be established).¹⁹ In particular, the record reveals that Mr. Mason complained of foot pain, decreasing strength, weakness, and (importantly) foot paresthesias and numbness well prior to vaccination, and overall a lengthy course of time as well. The overall impression left by the record is that Petitioner persistently experienced neuropathic symptoms that may have evolved and progressed over time into the CIDP he was ultimately diagnosed with.

Dr. Steinman attempted to untangle symptoms that might have predated vaccination from those Petitioner experienced thereafter, arguing that the former were distinguishable, but his efforts were mostly unsuccessful. Thus, he highlighted many medical record instances of pre-vaccine symptoms that could be attributed to SI Joint Syndrome, hypothyroidism, and osteoarthritis, only made worse by Mr. Mason’s intense training regime. Steinman Second Rep. at 2–3. Some of his points are reasonable; not all of Petitioner’s pre-vaccine symptoms appear to reflect neurologic issues representative of CIDP, and even some of the foot pain symptoms he sought treatment for post-vaccination are not evidence of neuropathy (although this also undercuts Petitioner’s suggestion that onset could have begun at the late-October visit to Dr. Parikh). I also accept his

¹⁸ I address the causation prongs in order of their significance to my decision, rather than in the order in which they are set forth by the Federal Circuit in the *Althen* decision.

¹⁹ Petitioner did not allege a significant aggravation claim—that the flu vaccine worsened preexisting CIDP. I therefore do not include an analysis herein of his success in so doing under the prevailing standard for such a claim. *Loving v. Sec’y of Health & Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009).

specific contention that the pre-vaccination elevated inflammatory biomarkers observed after Petitioner's blood testing are not themselves specific for CIDP.

But it remains the case that the *totality of the record* establishes that the many documented symptoms Petitioner displayed pre-vaccination were more likely than not elements of a progressive neuropathic course, the nature of which could only be understood later on. The vaccination merely occurred in the *midst* of this unfolding disease process. All the above is consistent with the waxing and waning associated with CIDP, and a pathologic course that could slowly and intermittently progress over a period of weeks to months before treaters could identify it fully based on clinical presentation and testing. *See Blackburn v. Sec'y of Health & Hum. Servs.*, No. 10-410V, 2015 WL 425935, at *27 (Fed. Cl. Spec. Mstr. Jan. 9, 2015) (denying compensation due to petitioner's CIDP beginning before vaccination, even if it could not then be diagnosed). Indeed, it is not uncommon for treaters to mistake CIDP initially *to be* GBS based on presenting symptoms, with the diagnosis being modified once it is clear that the patient's symptoms are not proceeding in a monophasic fashion, but instead become chronic in nature. *Daily*, 2011 WL 2174535, at *1 (noting the 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).

The fact that Petitioner's pre-vaccination symptoms were not understood to represent neuropathic issues is irrelevant. 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.

I also give little weight to Dr. Steinman's contention that the lack of an identified alternative cause (like proof of an infectious process) means the vaccine must have caused Petitioner's CIDP.²⁰ Although preponderant evidence of an alternative explanation can weigh against a petitioner's showing, claimants do not prevail merely because no other potential cause has been identified. To argue otherwise is to contend that a temporal association between vaccine and injury is a sufficient basis for entitlement—a proposition that has been soundly rejected. *See Capizzano*, 440 F.3d at 1327 (“[t]here may well be a circumstance where it is found that a vaccine

²⁰ This issue does not totally cut in Petitioner's favor in any event. Unlike GBS, *far less* is known about possible antecedent infectious explanations for CIDP, and most of the literature Dr. Steinman offered for infectious causes of demyelinating autoimmune diseases was specific to GBS.

can cause the injury at issue and where the injury was temporally proximate to the vaccination, but it is illogical to conclude that the injury was actually caused by the vaccine”). Indeed, petitioners do not automatically prevail even when they successfully *rule out* an alternative explanation. *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).

At bottom, the record does not corroborate Petitioner’s contention that the flu vaccine initiated a disease process, mediated by the adaptive immune system through the production of autoantibodies. (Indeed, proof of the presence of the causal antibodies is also lacking, although the absence of such testing evidence is ameliorated somewhat by a lack of scientific awareness of what the specific autoantibodies associated with CIDP would be in the first place). There is no evidence in this record that Petitioner experienced any kind of post-vaccination reaction suggestive of the beginning of a pathologic immune process. The foot pain he complained of to Dr. Parikh at the end of October 2014 was no different in character from what he had been experiencing before (and indeed he reported specifically to Dr. Parikh at that time that it had been ongoing for *years*). Ex. 5 at 37. And more neurologically-specific symptoms, like numbness, were also reported before and after vaccination. The record establishes a disease process that was likely already underway by the time the flu vaccine was administered to the Petitioner.

2. *Althen Prong Three*

The experts disagreed on a precise onset date, with Dr. Steinman favoring between eight days to five weeks post-vaccination, while Dr. Jamieson proposing Petitioner’s injuries occurred prior to vaccination. On the basis of this record, however, I cannot conclude that Petitioner’s CIDP most likely began post-vaccination *at all*—let alone within the timeframe offered by Dr. Steinman (even if that timeframe is itself preponderantly established).

Petitioner did offer reliable literature and testimony regarding the expected post-vaccination onset of *comparable* demyelinating conditions like GBS. Schonberger at 110–11; Souayah at 7322–23. He also notes correctly the timeframe (3-42 days (or up to six weeks)) for a Table GBS claim—although claimants cannot “piggyback” on the Table requirements when attempting to prove a non-Table claim. *See Greene v. Sec’y of Health & Hum. Servs.*, No. 11-631V, 2018 WL 3238611, at *9 (Fed. Cl. May 7, 2018) (noting that an expert’s opinion on the timing issue of a brachial neuritis claim relied on conclusory determinations that the “Table time periods were not that far off the time period in question (something Program law says is not permitted”). However, the fact that Table claims reflect the Government’s reasoned interpretation of persuasive medical science thinking on a causation theory means they can at least be considered in deciding non-Table claims. *See generally Marino v. Sec’y of Health & Hum. Servs.*, No. 16-0622V, 2017 WL 6206383, at *2, n.6 (Fed. Cl. Spec. Mstr. Apr. 18, 2017) (even though petitioner’s claim was filed before the injury of “Shoulder Injury Related to Vaccine

Administration” was added to the Table, the special master properly relied on the Table elements in analyzing the claimant’s causation-in-fact claim).

But *this is not a GBS case*. And there are clear distinctions between GBS and CIDP, with timeframe of symptoms progression *and* manifestation being a prime difference. Peltier & Donofrio at 188; Vallat at 402. Filed literature establishes that CIDP is an evolving neuropathy that is relapsing-remitting, stepwise or gradually progressive, and typically develops over the course of eight weeks. GBS, by contrast, is shown to have an acute onset that can occur in four to six weeks or less. Thus, what is known about GBS’s onset timeframe cannot simply be borrowed as a template to understand a likely onset timeframe for CIDP. Dr. Steinman’s opinion on onset timeframe was too reliant on GBS to provide fully-reliable evidence on what would be expected for CIDP.

At the same time, Dr. Steinman’s overall causal theory (which posits that autoantibodies produced by an adaptive immune response after vaccination could precipitate CIDP) involves the kind of immune process that is often understood in Program cases to take several weeks to manifest in clinical/outward symptoms. *See Blackburn*, 2015 WL 425935, at *27. Thus, despite the lack of specific proof offered in this case relevant to CIDP, it is not unreasonable to expect the process for manufacture of autoimmune antibodies to occur in the timeframe proposed. Accordingly, Dr. Steinman’s proposed timeframe was consistent with the science and his own theory, even if it at times invoked science specific to GBS.

However, the record contains evidence that is all over the map, so to speak, when identifying possible onset dates for symptoms later deemed to be part of Petitioner’s CIDP. On the one hand, Petitioner was reported by Dr. Parikh in late October 2014 (within three weeks of vaccination) to be experiencing parasthesias along with persistent foot pain. Ex. 5 at 37-42. This could reflect the first onset of truly-neurologic symptoms, as Dr. Steinman seems to think. By early December 2014 (now two months post-vaccination), Petitioner’s symptoms had evolved enough over time for treaters to more strongly suspect a neurologic injury (rather than, say, vascular, as was suspected at one time). Yet at the same time, Petitioner repeatedly reported to treaters that the general basket of symptoms he was experiencing began *pre-vaccination* and had simply been progressing. *See, e.g., Id.* at 39 (Petitioner telling Dr. Parikh his symptoms had persisted for several years); Ex.7 at 2 (reporting to Dr. Hylton that his symptoms began “last summer”). And as already noted, Petitioner’s history is amply documented with proof of pre-vaccination neuropathic symptoms that cannot be distinguished from what later occurred.

As a result, I can find herein that the proposed timeframe for onset of antibody-driven CIDP is medically acceptable – but this record does not permit me to conclude that *Petitioner’s* CIDP more likely than not occurred *within* that timeframe. Rather, Mr. Mason’s onset likely occurred sooner than eight days post-vaccination—and in fact more likely than not predated vaccination entirely.

3. *Althen Prong One*

I am unwilling to find that Petitioner has preponderantly established a causal relationship between the flu vaccine and CIDP merely because that theory has been accepted in the Program's past. Review of prior relevant cases suggests that more often than not, that determination has been based upon the faulty supposition that GBS and CIDP are two sides of the same coin. However, and despite my reasoned doubts, the record as developed *in this case* preponderates—if barely—in Petitioner's favor on this *Althen* prong.

There are numerous weaknesses with Petitioner's causal theory. For one, 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. The evidence specific to a flu vaccine-GBS association is vastly stronger, relying on a mix of (a) knowledge about how molecular mimicry “works” in GBS's pathogenesis, (b) trustworthy animal experiments that model demyelinating injuries in the context of the molecular mimicry mechanism, and (c) solid (if somewhat old) epidemiologic evidence, like Schonberger, establishing a higher incidence of GBS after vaccination when compared to an unvaccinated population. If one were searching for the paradigm of a well-substantiated causation theory in the Program, the flu vaccine-GBS connection would serve.

But is the same true for CIDP, especially given its differences in course and even likely pathogenesis? No. The evidence offered in this case establishes, for example, that a likely locus of autoimmune attack on the myelin (the node of Ranvier) in CIDP is distinguishable from GBS, which (in its most common form, AIDP) involves primarily an attack on ganglioside structures of the MBP. Devaux at 67–70. The fact that the pathogenesis for each can likely be distinguished is also evident in the fact of their varying presentations—with GBS manifesting acutely and having a fast monophasic course, while CIDP is slower and can become chronic. Peltier & Donofrio at 188; Vallat at 402. And I am aware of no CIDP-specific studies that identify possible triggers for it, when compared to the large amount of literature on GBS's many causes.

CIDP and GBS simply have different likely underlying etiologies, clinical presentations, and treatments, making it a dubious proposition to assume the science associating vaccines with one applies to the other. Jamieson First Rep. at 12–13; *see also 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. Additionally, the suspected autoantibodies are also different, with CIDP being associated with autoantibodies to contactin and neurofascin, while GBS has been associated with different autoantibodies. *See, e.g., Tomsky*, 2020 WL 5587365, at *8; *Isaac v. Sec'y of Health & Hum. Servs.*, 108 Fed. Cl. 743, 751 (Fed. Cl. 2013). Indeed, articles offered herein like Kira identified a CIDP-specific autoantibody distinguishable in target and nature. Kira at 4–6. And nothing filed in this case supports the conclusion that the flu vaccine is understood to be associated with the production of those CIDP-specific autoantibodies (Dr. Steinman's BLAST arguments notwithstanding).

Another issue with the Petitioner's causation theory lies in his expert showing. To be sure, Dr. Steinman's theory was reasonable and supported by reliable independent literature. Although both experts were qualified to opine on the nature and treatment of CIDP, Dr. Steinman possessed superior expertise on the immunologic issues in contention, and he did attempt to meet the evidentiary standard with specific points about how the flu vaccine might cause CIDP. Dr. Jamieson's showing on causation, by contrast, was fairly limited, as she devoted far more energy to disputing the diagnosis than to rebutting Petitioner on causation. She also in part relied on literature, like Donedu II, that I have in other cases noted is not especially reliable despite its CIDP-specific discussion. *See Houston*, 2021 WL 4259012, at *18 (giving Donedu II limited weight since it merely observed 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).

But this does not mean that Dr. Steinman's opinion was particularly persuasive. Overall, he did not differentiate meaningfully between GBS and CIDP, assuming what is known about one could be applied equally to the other. Worse, he mostly relied on the same general argument—molecular mimicry explains autoimmune diseases, and homology can be shown between component X of the relevant vaccine and a posited target antigen, ergo causation—that I have in other cases rejected as insufficiently reliable to meet the preponderant standard. *See, e.g., E.S v. Sec'y of Health & Hum. Servs.*, No. 17-480V, 2020 WL 9076620, at *49 (Fed. Cl. Spec. Mstr. Nov. 13, 2020) (“the *substance* of the opinion offered was almost identical to opinions [Dr. Steinman] has offered repeatedly in prior Program cases . . .”), *mot. for review denied*, 154 Fed. Cl. 149, 157 (2021); *Tullio v. Sec'y of Health & Hum. Servs.*, No. 15-51V, 2019 WL 7580149, at *14 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *mot. for review denied*, 149 Fed. Cl. 448 (2020) (criticizing the utility of BLAST searches to support petitioner's argument); *see also McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (“[b]ut merely chanting the magic words ‘molecular mimicry’ in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or vaccine in question”) (emphasis in original), *mot. for review denied*, 76 Fed. Cl. 452 (2007)). In most respects, Dr. Steinman's causation theory was only plausible.

However, two considerations lead me to determine that (regardless of these misgivings) the first *Althen* prong was in this case preponderantly established—if only by inches. First, I am compelled to give Dr. Steinman's opinion a bit more weight than what Dr. Jamieson offered. Dr. Steinman offered *some* reliable literature (specifically Kira and Devaux) that addressed the kinds of CIDP-specific autoantibodies that could drive the disease. Although these articles by their own terms discount their success in establishing either that the autoantibodies *do* so function, or how they come to exist in the first place, they help bridge gaps in his opinion by offering evidence specific to CIDP. Dr. Steinman coupled this with a reasonable showing that (at least in theory) the flu vaccine could produce these autoantibodies, and that they could cross-react as proposed. Even

if these points were quite thinly substantiated, they were not rebutted by Respondent (other than Dr. Jamieson's conclusory contentions that more reliable and complete evidence does not exist on the topic).

Second (and inextricably connected to my first point), I take into account the prior Program findings on the issue of the flu vaccine being causal of CIDP, despite my aforementioned sense that these decisions are themselves conclusory on the subject. It seems special masters have often assumed all that is known about GBS applies to CIDP, resulting in the two conditions to be repeatedly conflated for purposes of analyzing a causation theory. *Strong*, 2018 WL 1125666, at *20. I have identified no well-reasoned prior case where the *differences* between the two are addressed and taken into account. Ultimately, I am not bound by these determinations. But I am reluctant to ignore them completely under the circumstances of this case, especially where Respondent's showing did not sufficiently touch on the immunology at issue.²¹ This, plus the adequate quality of Dr. Steinman's opinion on causation, leads me to find the "can cause" prong has been met.

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, and although the parties have not objected to my choice of this method of adjudication, 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*, 38 Fed. Cl. at 400–01 (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 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. The parties did not agree on Mr. Mason's diagnosis, but I have found in Petitioner's favor on that matter. The onset date was also disputed, but the record facts were unresponsive of Petitioner's contentions—suggesting a pre-vaccination onset was far more likely. Resolution of this question did not require formal witness testimony, but could instead be determined through careful consideration of the medical records. The question of causation itself

²¹ It would be different had Respondent offered a persuasive opinion from a qualified immunologist addressing the matters discussed above, or attempting to rebut Dr. Steinman's points on causation.

was also something that could be resolved through reading the expert reports and associated literature, and it also raised issues (the propensity of the flu vaccine to cause demyelinating injuries) with which I have extensive familiarity.

CONCLUSION

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

In the absence of a motion for review filed pursuant to RCFC Appendix B, the clerk of the court **SHALL ENTER JUDGMENT** in accordance with the terms of this decision.²²

IT IS SO ORDERED.

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

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