In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 17-1112V
Filed: May 9, 2025

EILEEN SCHMIGEL,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Paul R. Brazil, Muller Brazil, LLP, Dresher, PA, for petitioner.
Dorian Hurley, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On August 18, 2017, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that she suffered chronic inflammatory demyelinating polyneuropathy ("CIDP") following receipt of an influenza ("flu") vaccination on October 21, 2015. (ECF No. 1.) For the reasons discussed below, I find that petitioner is *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute;

[1] Because this document contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the document will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10, *et seq.*

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received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury.

In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B). In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In that context, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

CIDP is not an injury listed on the Vaccine Injury Table relative to any vaccination. Guillain-Barre Syndrome ("GBS"), which petitioner's expert invokes as partial support for his theory of causation, is a Table injury if onset occurs 3-42 days following receipt of a flu vaccine. 42 C.F.R. § 100.3(a)(XIV)(D). However, a diagnosis of CIDP is listed among the exclusionary criteria for a Table Injury of GBS. 42 C.F.R. § 100.3(c)(15)(vi). To succeed on a claim that petitioner's flu vaccine caused CIDP, petitioner must satisfy the burden of proof for "causation-in-fact."

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" with the logical sequence being supported by "reputable medical or scientific explanation." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (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. *Id*.

A petitioner may not receive a Vaccine Program award based solely on his or her assertions, but may support the petition with either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa-13(b)(1). A petitioner may also rely upon circumstantial evidence. *Althen*, 418 F.3d at 1280. In that regard, the *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3. Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). 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." § 300aa-13(b)(1). The special master is required to consider the entirety of the record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

This case was reassigned to the undersigned in August of 2019. (ECF No. 46.) By that time, respondent had already filed his Rule 4 Report, recommending against compensation (ECF No. 28), and the parties had filed expert reports, with neurologist Nizar Souayah, M.D., opining on petitioner's behalf and neurologist Peter Donofrio, M.D., opining on respondent's behalf. (ECF Nos. 40, 42; Exs. 30, A.) After the case was reassigned, I held a fact hearing on September 25, 2020 (*see* Transcript of

Proceedings ("Tr.") at ECF No. 67), and issued a finding of fact regarding onset of petitioner's symptoms on November 19, 2021. (ECF No 74; *see also Schmigel v. Sec'y of Health & Human Servs.*, No. 17-1112V, 2021 WL 5905687 (Fed. Cl. Spec. Mstr. Nov. 19, 2021).)

The parties subsequently filed several additional rounds of expert reports to account for the facts as I had found them. Petitioner filed three reports by neurologist and virologist Maria Fangchun Chen, M.D., Ph.D. (ECF Nos. 79, 86, 95; Exs. 47, 67, 131) and respondent filed three further reports by Dr. Donofrio (ECF Nos. 84, 88, 96; Exs. N, W, Y). Respondent also filed a report by immunologist William Hawse, Ph.D. (ECF No. 96; Ex. DD.) Petitioner then filed a motion for a ruling on the written record on February 27, 2024. (ECF No. 97.) That motion is fully briefed. (ECF Nos. 98, 100.)

I have concluded that the parties have had a full and fair opportunity to develop the record and that it is appropriate to resolve this case on the existing record. *See Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (citing *Simanski v. Sec'y of Health & Human Servs*., 671 F.3d 1368, 1385 (Fed. Cir. 2012)); *see also* Vaccine Rule 8(d); Vaccine Rule 3(b)(2).

III. Factual Summary

The pertinent facts regarding onset of petitioner's symptoms are set forth in greater detail in the prior Finding of Fact. (ECF No. 74, pp. 2-10; 2021 WL 5905687, at *2-8.) Petitioner, who had a relevant prior history of insulin-dependent diabetes, received a flu vaccination on October 21, 2015. For the reasons discussed throughout the Finding of Fact, I resolved conflicting evidence to conclude that she subsequently experienced numbness and tingling in her extremities around late November of 2015. *Schmigel*, 2021 WL 5905687, at *12. However, although there was evidence to suggest petitioner was also experiencing generalized pain and fatigue, the medical records documented normal strength up to a June 30, 2016 medical encounter at which "adequate" strength and muscle tone were noted. *Id.* (discussing Ex. 9, pp. 1-3; Ex. 5, p. 2; Ex. 6, p. 2). Thereafter, weakness "of any degree" was first detected on physical exam on July 20, 2016, about nine months post-vaccination.[3] *Id.* at *12 (discussing Ex. 10, p. 4). At that encounter, petitioner had full strength in all four extremities but had 4+ toe extension and 5- toe flexion. *Id.* at *4 (discussing Ex. 10, p. 4). Petitioner's treating physicians had difficulty determining whether her condition was better explained by CIDP or diabetic neuropathy. *Id*. at *4-5 (discussing Ex. 10, p. 4; Ex. 9, p. 6; Ex. 19, p. 65). However, she was ultimately diagnosed with CIDP, *id*. at *5 (discussing Ex. 17, pp. 32-38), though respondent disputes the accuracy of that diagnosis. (ECF No. 98, pp. 24-31.)

[3] In one location within the analysis portion of the Finding of Fact, petitioner's encounter with Dr. Papsdorf is incorrectly stated as being July 27, rather than July 20. 2021 WL 5905687, at *12. However, the encounter is correctly dated in the explanation of petitioner's medical history, *id.* at *4, and the typographic error does not affect the analysis.

Petitioner sought care from a number of different physicians during the first half of 2016. She established care with a primary care provider, Dr. Schudy, on June 30, 2016. (Ex. 9, p. 1.) By that time, petitioner had undergone an EMG/NCS of her upper extremities[4] in May of 2016 and a lower extremity EMG/NCS in June of 2016.[5] (Ex. 7, p. 1; Ex. 19, p. 54.) Dr. Schudy was the first physician to become concerned that petitioner may have CIDP. (Ex. 9, pp. 2-3.) In particular, he felt that petitioner's June NCS was "highly suggestive" of CIDP. (*Id*. at 3.)

Petitioner first presented to a neurologist, Dr. Papsdorf, on July 20, 2016, for an evaluation of possible CIDP as suspected by Dr. Schudy. (Ex. 10, p. 1.) Petitioner reported that her neuropathy was "very fast" in onset, and she was concerned it was related to her flu vaccination. (*Id.* at 3.) However, she also reported a 1.5 year history of diabetes, which was by then "[v]ery controlled." (*Id.*) She explained that her A1C started at 8.8% when she was diagnosed, but that she had lost weight and brought it down to 5.6%. (*Id.*) Thus, Dr. Papsdorf was concerned that petitioner had both diabetic neuropathy, which she noted to be the most common peripheral neuropathy in the United States, as well as "insulin neuritis," which is a sudden onset of neuropathy that appears during the treatment of diabetes. (*Id.* at 4-5.) She noted that petitioner also had gait issues, but explained that the gait issues could be secondary to the neuropathy. (*Id*.) She found that petitioner had a length-dependent, symmetrical peripheral neuropathy with evidence of both small and large fiber involvement. (*Id*. at 4.) At this encounter, petitioner's strength was normal throughout her proximal and distal muscles in all four extremities, and she had normal reflexes. (*Id.*) Dr. Papsdorf ordered an NCS/EMG to rule out CIDP, though CIDP was not suspected "by exam or by history." (*Id*. at 5.) In particular, Dr. Papsdorf felt petitioner's prior NCS was more consistent with axonal loss than demyelination. (*Id.*)

Petitioner underwent a follow up electrodiagnostic study on July 27, 2016. (Ex. 10, p. 6.) However, the study was incomplete due to petitioner not being able to tolerate the needle portion of the study. (*Id*.; *see also* Ex. 19, p. 65.) This study showed a worsening of petitioner's condition as compared to her prior June study. (Ex. 19, p. 65.) The study was "suggestive of a possible demyelinating diffuse peripheral neuropathy such as [CIDP]." (Ex. 10, p. 6.) However, the study did not rule out diabetic amyotrophy without a complete needle study. (Ex. 19, p. 65.) The parties' experts disagree as to the diagnostic significance of petitioner's NCS/EMG studies, as discussed in greater detail below. Petitioner also underwent a lumbar puncture, which showed protein of 126 and glucose of 61. (Ex. 4, p. 2.)

Petitioner self-referred to another neurologist, Dr. Bittle, in September of 2016. (Ex. 4, p. 1.) Dr. Bittle concluded that petitioner was suffering CIDP, though he noted

[4] The EMG was normal, but the NCS showed bilateral median sensory/motor neuropathy consistent with carpal tunnel syndrome, as well as bilateral ulnar motor neuropathy. (Ex. 7, p. 1.)

[5] The June study showed peroneal and tibial motor neuropathy, as well as sural and superficial peroneal sensory neuropathy with evidence of sensorimotor axonopathic and demyelinating peripheral neuropathy. (Ex. 19, p. 54.)

that her strength was "fairly good" relative to her EMG/NCS findings and also noted that her neuropathic pain was less common for CIDP (reported in less than 20% of patients). (*Id*. at 6.) Dr. Bittle's assessment also noted petitioner's CSF protein of 126. (*Id.*) Thereafter, petitioner continued to seek treatment for CIDP, including IVIG. As discussed below, the parties' experts disagree as to whether petitioner's condition responded to IVIG.

Petitioner consulted a third neurologist, Dr. Aggarwal, in August of 2017, who maintained the impression of CIDP. (Ex. 17, pp. 32-38.) She consulted a fourth neurologist, Dr. Hunter, beginning in November of 2017. Dr. Hunter also recorded an impression of CIDP. (Ex. 19, pp. 2-3.) These encounters appear to have been primarily focused on management of petitioner's CIDP, rather than revisiting diagnosis. Although petitioner's prior neurology evaluations were reviewed, no further electrodiagnostic studies were performed.

Although petitioner repeatedly reported to her physicians that her condition arose post-vaccination, none of the physicians specifically addressed whether her condition was casually related to her vaccination.

IV. Expert Opinions

a. Nizar Souayah, M.D., for petitioner[6]

Dr. Souayah prepared a single report for this case, which was filed prior to issuance of my finding of fact regarding onset. (Ex. 30.) Dr. Souayah's opinion as to specific causation assumed that, within five weeks of her vaccination, petitioner experienced onset of a constellation of symptoms consistent with CIDP, including diffuse muscle pain, numbness and tingling, and weakness. (*Id.* at 5, 7, 16-17.) Accordingly, because this assumption is inconsistent with my findings of fact, Dr. Souayah's opinion is not informative with regard to specific causation. Dr. Souayah's opinion remains relevant with respect to general causation; however, for the reasons discussed below, I do not find it necessary to reach that question. Important to the discussion that follows, Dr. Souayah opines that CIDP can be causally linked to flu vaccinations occurring up to eight weeks prior to onset of the condition. (*Id.* at 10 (citing Karissa L. Gable et al., *Distal Acquired Demyelinating Symmetric Neuropathy After Vaccination*, 14 J. CLINICAL NEUROMUSCULAR DISEASE 117 (2013) (Ex. 111)).)

[6] Dr. Souayah is board certified in neurology with a subspecialty in neuromuscular medicine, and he maintains an active medical license in New Jersey. (Ex. 30, p. 1.) He currently works as a professor of neurology at Rutgers-New Jersey Medical School. In his medical practice, he regularly diagnoses and treats patients with neurological conditions, including CIDP. In his research capacity, he investigates the causal relationship between vaccines and adverse events with a particular interest in the incidence of neurological adverse reactions related to vaccination. (*Id.*)

b. Maria Fangchun Chen, M.D., Ph.D., for petitioner[7]

Dr. Chen describes CIDP as an under-diagnosed "cousin" of Guillain-Barre Syndrome ("GBS"), stressing in particular that it is difficult to diagnose due to a heterogeneous presentation. (Ex. 47, p. 11 (citing Yhojan Rodríguez et al., *Chronic Inflammatory Demyelinating Polyneuropathy as an Autoimmune Disease*, 102 J. AUTOIMMUNITY 8 (2019) (Ex. 49); Kelly Gwathmey, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy and Its Variants*, 26 CONTINUUM (MINNEAPOLIS MINN.) 1205 (2020) (Ex. 50); Y. A. Rajabally et al., *Clinical Heterogeneity in Mild Chronic Inflammatory Demyelinating Polyneuropathy*, 13 EUR. J. NEUROLOGY 958 (2006) (Ex. 51); Francisco T. Rotta et al., *The Spectrum of Chronic Inflammatory Demyelinating Polyneuropathy*, 173 J. NEUROLOGICAL SCIS. 129 (2000) (Ex. 52); Umair J. Chaudhary & Yusuf A. Rajabally, *Underdiagnosis and Diagnostic Delay in Chronic Inflammatory Demyelinating Polyneuropathy*, 268 J. NEUROLOGY 1366 (2021) (Ex. 54); Filip Eftimov et al., *Diagnostic Challenges in Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, 143 BRAIN 3214 (2020) (Ex. 55)).) It often takes years to reach a CIDP diagnosis. (*Id*. (citing Rotta et al., *supra*, at Ex. 52).) Dr. Chen acknowledges that weakness and numbness are "core" features that are required for a diagnosis of typical forms of CIDP, but suggests that other symptoms can be diagnostic of "atypical" forms of the condition. (*Id.*) She cites several sources for the proposition that fatigue can be an initial presenting symptom of CIDP. (*Id*. (citing Rajabally et al., *supra*, at Ex. 51; S. R. M. Bus et al., *Clinical Outcome of CIDP One Year After Start of Treatment: A Prospective Cohort Study*, 269 J. NEUROLOGY 945 (2022) (Ex. 56); Antonino Uncini et al., *Minimal and Asymptomatic Chronic Inflammatory Demyelinating Polyneuropathy*, 110 CLINICAL NEUROPHYSIOLOGY 694 (1999) (Ex. 57); S. Boukhris et al., *Pain as the Presenting Symptom of Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP)*, 254 J. NEUROLOGICAL SCIS. 33 (2007) (Ex. 58); Sami Boukhris et al., *Fatigue as the Main Presenting Symptom of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Study of 11 Cases*, 10 J. PERIPHERAL NERVOUS SYSTEM 329 (2005) (Ex. 59); Andrew Lawley et al., *Clinical Correlates of Fatigue in Chronic Inflammatory Demyelinating Polyneuropathy*, 62 MUSCLE & NERVE 226 (2020) (Ex. 60); Karissa L. Gable et al., *Fatigue in Chronic Inflammatory Demyelinating Polyneuropathy*, 62 MUSCLE & NERVE 673 (2020) (Ex. 61)).) She further notes a particular case report of an electrodiagnostically confirmed case of CIDP where the primary symptom was

[7] Dr. Chen received her Ph.D. in molecular virology and medical degree from the University of Pennsylvania School of Medicine in 2005 and 2007, respectively, before moving to the Hospital of the University of Pennsylvania to complete an internship in 2008 and a neurology residency in 2011. (Ex. 48, p. 1; Ex. 47, p. 1.) From there, Dr. Chen briefly worked as an attending neurologist at the Albert Einstein Healthcare Network; however, in 2013, she transitioned to a faculty position as an assistant professor of clinical neurology at Penn Medicine. (Ex. 48, p. 1; Ex. 47, p. 1.) She currently works as Associate Director of Clinical Development, Neurology and Psychiatry at Teva Pharmaceuticals Industries. (Ex. 48, p. 1.) Dr. Chen is board certified in neurology, and she maintains an active medical license in Pennsylvania. (*Id.* at 2; Ex. 47, p. 1.) In her clinical practice, Dr. Chen saw patients and supervised neurology resident physicians and medical students. (Ex. 47, p. 1.) Although she no longer sees patients, she oversees clinical research at a pharmaceutical company. (*Id.*) She has also authored four publications and researched mechanisms for how viruses, and specifically HIV, injure the nervous system. (*Id.*; Ex. 48, pp. 2-3.)

disabling fatigue without frank weakness or sensory symptoms. (*Id.* (citing Véronique Bissay et al., *Fatigue as the Presenting Symptoms of Chronic Inflammatory Demyelinating Polyneuropathy*, 38 MUSCLE & NERVE 1653 (2008) (Ex. 62)).) She opines that neuropathic pain, including painful paresthesia and muscle pain, are not uncommon features of CIDP. (*Id*. at 12 (citing Athena Michaelides et al., *Pain in Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Systematic Review and Meta-Analysis*, 8 PAIN & THERAPY 177 (2019) (Ex. 64); Andreas Goebel et al., *Pain Intensity and Distribution in Chronic Inflammatory Demyelinating Polyneuropathy*, 46 MUSCLE & NERVE 294 (2012) (Ex. 65); Peter Y. K. Van den Bergh et al., *European Academy of Neurology/Peripheral Nerve Society Guideline on Diagnosis and Treatment of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force–Second Revision*, 28 EUR. J. NEUROLOGY 3556 (2021) (Ex. 66)).) However, she is less clear as to whether these symptoms would be expected to herald the condition.

With respect to petitioner's own condition, Dr. Chen notes that petitioner at a September 21, 2016 encounter with her second neurologist, Dr. Bittle, reported "extreme fatigue" following her flu vaccination. (Ex. 47, p. 12 (citing Ex. 17, p. 5).) At that same encounter, Dr. Bittle attributed petitioner's neuropathic pain to CIDP. (*Id*. (citing Ex. 17, p. 10).) Because Dr. Chen places the onset of petitioner's CIDP within weeks of her flu vaccination based on fatigue and neuropathic pain, she opines that petitioner's CIDP can be causally linked to her flu vaccination based on Dr. Souayah's theory of causation. (*Id*. at 13 (citing Ex. 30).) However, she acknowledges this would be an atypical presentation. (*Id*.)

Regarding respondent's expert's competing view, Dr. Chen cites Dr. Donofrio's strict application of the European Federation Neurologic Society/Peripheral Nerve Society ("EFNS/PNS") diagnostic criteria for CIDP, by which Dr. Donofrio seeks to require hyporeflexia or areflexia, as exemplary of how CIDP is under-diagnosed. (Ex. 67, pp. 2-3 (discussing Joint Task Force of the EFNS and the PNS, *EFNS/PNS CIDP Guidelines: European Federation of Neurological Societies/Peripheral Nerve Society Guideline on Management of Chronic Inflammatory Demyelinating Polyradiculoneuropathy. Report of a Joint Task Force of the European Federation of Neurological Societies and the Peripheral Nerve Society*, 10 J. PERIPHERAL NERVOUS SYS. 220 (2005) [hereinafter 2005 EFNS/PNS CIDP Guideline] (Ex. O; *see also* Ex. D); Joint Task Force of the EFNS and the PNS, *EFNS/PNS CIDP Guidelines, European Federation of Neurological Societies/Peripheral Nerve Society Guideline on Management of Choric Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force of the European Federation of Neurological Societies and the Peripheral Nerve Society – First Revision*, 15 J. PERIPHERAL NERVOUS SYS. 1 (2010) [hereinafter 2010 EFNS/PNS CIDP Guidelines] (Ex. P; *see also* Ex. E); Peter Y. K. Van den Bergh et al., *European Academy of Neurology/Peripheral Nerve Society Guideline on Diagnosis and Treatment of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Task Force – Second Edition*, 26 J. PERIPHERAL NERVOUS SYS. 242 (2021) (Ex. Q)).) According to Dr. Chen, these criteria have sensitivity ranging from 73-84%, meaning that the criteria fail to diagnose about 1 out of every 5 patients. (*Id*. (citing Pietro Emiliano Doneddu et al., *Comparison of the*

Diagnostic Accuracy of the 2021 EAN/PNS and 2010 EFNS/PNS Diagnostic Criteria for Chronic Inflammatory Demyelinating Polyradiculoneuropathy, 93 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 1239 (2022) (Ex. 69); Satoshi Kuwabara & Tomoki Suichi, *Validation of the 2021 EAN/PNS Diagnostic Criteria for Chronic Inflammatory Demyelinating Polyneuropathy*, 93 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 1237 (2022) (Ex. 70)).) Moreover, she stresses that the criteria Dr. Donofrio cites do allow for atypical presentations to include intact reflexes. (*Id*. at 3 (citing Van den Bergh et al., *supra*, at Ex. Q, p. 4, tbl.1).) With regard to electrodiagnostic testing, although Dr. Donofrio indicates that nerve conduction velocities must be 70% or less than normal in two or more nerves to be diagnostic, Dr. Chen asserts this is not *required* as it is only one of seven electrodiagnostic abnormalities that will support a diagnosis. (*Id.* (citing Van den Bergh et al., *supra*, at Ex. Q, p. 6, tbl.2).) The fact that petitioner's nerve conduction study demonstrated prolonged F-waves greater or equal to 20% of the upper limit of normal for her left median and ulnar nerves is sufficient to be diagnostic of CIDP. (*Id.* (citing Ex. 10, p. 10; Van den Bergh et al., *supra*, at Ex. Q, p. 6, tbl.2).) Additionally, petitioner's elevated CSF protein and response to immunotherapy are further supportive of the CIDP diagnosis. (*Id*.)

Further, Dr. Chen seeks to rule out other explanations for petitioner's initial symptoms of numbness, tingling, and fatigue. First, she does not agree that petitioner suffered diabetic neuropathy. (Ex. 67, pp. 3-4.) Without diagnostic testing (such as skin or nerve biopsy or EMG), it is not possible to be more specific than to conclude that petitioner's numbness and tingling were consistent with peripheral neuropathy. (Ex. 131, p. 3.) However, Dr. Chen stressed that petitioner's sensory symptoms did not correlate to her glycemic control, which would ordinarily be expected. (*Id*. at 2-3.) That is, while petitioner had documented high A1C of 8.3 in March of 2015, she was not experiencing sensory symptoms at that time. (*Id*. at 2 (citing Ex. 16, p. 78).) Prior to onset of numbness and tingling, petitioner's A1C had improved, documented as 6.5 as of September 15, 2015. (*Id*. (citing Ex. 16, p. 146).) An exception would be treatment-induced neuropathy, which can occur from rapid and sudden control of diabetes. (*Id*. at 2-3 (citing Hideyuki Sasaki et al., *Spectrum of Diabetic Neuropathies*, 11 DIABETOLOGY INT'L 87 (2020) (Ex. 134)).) This was expressed as a concern by Dr. Papsdorf (Ex. 9, p. 6); however, Dr. Chen opines that this was not petitioner's presentation. (Ex. 67, p. 3.) Additionally, petitioner's nerve conduction studies showed conduction velocity slowing without a drop in amplitude in the left ulnar motor conduction, as well as F-wave latency delay that was not mild, which she indicates is not consistent with axonal neuropathy as should be present if petitioner was suffering diabetic neuropathy. (*Id.* at 3-4 (citing Ex. 7, p. 4; Ex. 10, pp. 10, 12; Bruce Perkins & Vera Bril, *Electrophysiologic Testing in Diabetic Neuropathy*, 126 HANDBOOK CLINICAL NEUROLOGY 235 (2014) (Ex. 72)).) In contrast, Dr. Chen stresses that petitioner's numbness and tingling are also consistent with CIDP and that petitioner's May 24, 2016 EMG included evidence of demyelination consistent with CIDP that predated the onset of her symptom of weakness. (Ex. 131, p. 3 (citing Ex. 7, pp. 1-4).)

Petitioner's symptoms also cannot be explained by vitamin deficiency, because petitioner had a normal methylmalonic acid (MMA) level. (Ex. 67, p. 4 (citing Ex. 9, p.

4).) An elevated MMA level would be necessary before one could conclude that a low normal Vitamin B level could be indicative of a functional deficiency. (*Id*. (citing Sally P. Stabler, *Vitamin B_{12} Deficiency*, 368 N. ENG. J. MED. 149 (2013) (Ex. U, p. 5)).) Additionally, petitioner had normal vitamin D levels and no evidence of myopathy on electrodiagnostic study. (*Id.* (citing Ex. 16, p. 144; Ex. 10, p. 9).) Therefore, there is no evidence she had any vitamin D-related muscular disorder. (*Id.*) Dr. Chen also disagrees that obstructive sleep apnea explains petitioner's fatigue. She observes that petitioner was diagnosed with obstructive sleep apnea in April of 2014 whereas her reported fatigue did not begin until the autumn of 2015, more than a year later. (*Id*. (citing Ex. 16, p. 34).) Moreover, petitioner's fatigue occurred at a time when she was losing weight and using a CPAP machine to treat her sleep apnea, suggesting that her fatigue was not responsive to treatment for her sleep apnea. (*Id.*)

Prompted to discuss whether diabetes can cause CIDP, Dr. Chen initially remarked that there is "not much" support for this in the relevant medical literature, stressing that CIDP is immune mediated, whereas diabetes is primarily metabolic and vascular. (Ex. 131, pp. 1-2 (citing Amanda C. Peltier & Peter R. Donofrio, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Bench to Bedside*, 32 SEMINARS NEUROLOGY 187 (2012) (Ex. 132)).) However, she acknowledged that at least some cohort studies support an increased risk of CIDP among diabetic patients. (*Id.* at 2 (citing Yan Chen & Xiangqi Tang, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy in Association with Concomitant Disease: Identification and Management*, 13 FRONTIERS IMMUNOLOGY 890 (2022) (Ex. 133); J. Bradley Layton et al., *Intravenous Immunoglobulin Initiation in Patients with Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A US Claims-Based Cohort Study*, 12 NEUROLOGY & THERAPY 1171 (2023) (Ex. 135)).) On that point, she noted that it is hypothesized that diabetes can cause injury to the nerve and thereby expose neuronal antigens to the immune system, leading to an autoreactive immune response. (*Id.* (citing Chen & Tang, *supra*, at Ex. 133).) She considers this hypothesis to be unsupported and the epidemiology to be unconvincing. Thus, she considers the idea that diabetes can lead to increased risk of CIDP to be "controversial." (*Id.*)

However, Dr. Chen also opines that, even if onset of petitioner's CIDP occurred "months" after her flu vaccination, it would still be plausible for her flu vaccine to have been the cause based on the above-discussed hypothesis. Specifically, she opines that

> Proponents of the view that diabetes can increase the risk of CIDP note that diabetes can injure nerves thereby exposing neuronal antigens to an immune system that previously were not exposed and hence increase the risk of autoimmunity. Notably, while CIDP is accepted to be an immune mediated disease, there is not evidence for a single immune process. The scientific literature points to a wide variety of immune processes at play for the pathology of CIDP and it's not clear which and if all of these processes are required. Furthermore, the onset of typical CIDP is expected to be on the chronic spectrum (symptoms lasting for at least 8 weeks) indicating that the underlying pathology of CIDP is protracted and may require

> repeated attacks on the peripheral nerves to result in the wide-spread injury of sensory or motor nerves in a sufficient number and distribution of nerves. The lack of the identification of an inciting event prior to the occurrence of CIDP further supports that the immune mediated attack on nerves may be a slow chronic process and hence supports the presentation of symptoms months out from the inciting event.

(Ex. 131, p. 4 (footnotes omitted) (citing Peltier & Donofrio, *supra*, at Ex. 132; Emily K. Mathey et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, 86 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 973 (2015) (Ex. 136)).)

Ultimately, though it is not her preferred explanation, Dr. Chen agrees that it is plausible that petitioner's initial symptoms of numbness and tingling represented diabetic neuropathy and that she then experienced later manifesting CIDP. (Ex. 131, p. 2.) In general, Dr. Chen approximates that "the likelihood of a diagnosis with both diabetic neuropathy and CIDP may be equivalent or 2-3 times more likely than an atypical presentation of CIDP." (*Id*. at 3.)

c. Peter Donofrio, M.D., for respondent[8]

Dr. Donofrio describes CIDP as chronic condition that presents either in a progressive stepwise fashion or in a relapsing-remitting pattern. (Ex. A, p. 9.) Although it is sometimes characterized as a chronic form of GBS, this is a misconception. (*Id*.) There are some overlapping clinical features, but they are two distinct conditions. (*Id*.) He disagrees that CIDP can be causally linked to flu vaccination, stressing the Institute of Medicine's 2012 conclusion that epidemiologic evidence is lacking and that mechanistic evidence, which was limited to case reports, was weak. (*Id*. at 10.) Whereas petitioner's experts indicate CIDP is under-diagnosed, Dr. Donofrio suggests that it is often *mis*diagnosed, indicating that nearly half (47%) of patients initially diagnosed with CIDP will fail to meet the minimum diagnostic criteria after specialist referral. (*Id*. at 9-10 (citing Jeffrey A. Allen & Richard A. Lewis, *CIDP Diagnostic Pitfalls*

[8] Dr. Donofrio received his medical degree from Ohio State University School of Medicine in 1975, before going on to complete a residency in internal medicine at Good Samaritan Hospital in Cincinnati, Ohio in 1978, a residency in neurology at the University of Michigan Medical Center in 1981, and a neuromuscular fellowship at the University of Michigan in 1982. (Ex. B, pp. 1-2.) While completing his fellowship, Dr. Donofrio worked as a neurology instructor at various institutions. (*Id.* at 3.) Thereafter, he worked as an assistant professor of neurology at the University of Michigan Medical Center and Veterans Administration Medical Center in Ann Arbor, Michigan. (*Id.*) In 1986, Dr. Donofrio accepted a position as an assistant professor of neurology at Wake Forest University School of Medicine, and he was eventually promoted to professor of neurology in 2005. (*Id.* at 2-3.) In 2006, Dr. Donofrio accepted a position as a professor of neurology at Vanderbilt University School of Medicine, where he is currently employed. (*Id.* at 2.) Dr. Donofrio is board certified in internal medicine, psychiatry and neurology, and electrodiagnostic medicine, and he maintains an active medical license in Ohio, Michigan, and Tennessee. (*Id.*) In his clinical capacity, he has evaluated a spectrum of neuropathies, including GBS and CIDP, and in his research capacity, he has published in the areas of GBS, CIDP, and other neuropathies, as well as reviewed literature and data on vaccine-related GBS, CIDP, and other neurologic conditions. (Ex. A, p. 1.) He has published several articles and book chapters, as well as a textbook title *The Textbook of Peripheral Neuropathy*, which was published in 2012. (*Id.*; Ex. B, pp. 12-22.)

and Perception of Treatment Benefit, 85 NEUROLOGY 498 (2015) (Ex. H; *see also* Ex. R); Kenneth C. Gorson & Clifton L. Gooch, *The (Mis)diagnosis of CIDP: The High Price of Missing the Mark*, 85 NEUROLOGY 488 (2015) (Ex. I)).) Dr. Donofrio suggests that "atypical" presentations are correlated with "misdiagnosis." (*Id*. at 10 (citing 2005 EFNS/PNS CIDP Guideline, *supra*, at Ex. D; 2010 EFNS/PNS CIDP Guidelines, *supra*, at Ex. E).)

Citing the EFNS/PNS diagnostic criteria, Dr. Donofrio opines that petitioner's presentation cannot support a "probable," let alone "definite" diagnosis of CIDP. (Ex. A, p. 9.) He opines that she does not satisfy the clinical criteria given that she did not have symmetrical proximal and distal weakness and sensory dysfunction of all extremities when her condition first presented. (*Id*. (citing 2010 EFNS/PNS CIDP Guidelines, *supra*, at Ex. E, p. 6, tbl.4).) Dr. Donofrio further noted that separate CIDP diagnostic criteria by Koski et al. require documented weakness in all four limbs with proximal weakness in at least one limb, which petitioner did not have. (*Id.* (citing C.L. Koski et al., *Derivation and Validation of Diagnostic Criteria for Chronic Inflammatory Demyelinating Polyneuropathy*, 277 J. NEUROLOGICAL SCIS. 1 (2009) (Ex. F)).) Dr. Donofrio also disagrees with Dr. Chen's assessment that petitioner's condition improved with IVIG treatment. (Ex. W, p. 3.) But in any event, he would not find such improvement to be diagnostically informative. (*Id.*; Ex. A, p. 10; Ex. N, p. 4 (citing Allen & Lewis, *supra*, at Ex. R).) Although petitioner's CSF protein level could be used to help support a CIDP diagnosis, this would also be a common finding in diabetes and diabetic neuropathy. (Ex. W, pp. 4-5.)

Dr. Donofrio further opines that petitioner's EMG and nerve conduction studies did not satisfy the criteria for a demyelinating neuropathy. Dr. Donofrio expresses that the electrodiagnostic criteria for CIDP are "strict and exact." (Ex. N, p. 4.) As noted above, Dr. Donofrio stresses that conduction velocities of less than 70% of the lower limit of normal in two or more motor nerves is *required* for a CIDP diagnosis. (*Id*.) However, none of petitioner's studies demonstrated this abnormality. (*Id*. (citing Ex. 7, pp. 1-5; Ex. 10, pp. 6-13; Ex. 30, p. 7).) Additionally, petitioner did not undergo additional testing, such as MRI to rule out spinal nerve root issues. (Ex. A, p. 9.) Although Dr. Chen suggested that petitioner demonstrated slowed conduction velocity in her left ulnar nerve as some evidence of demyelination, Dr. Donofrio counters that this result is only 1 m/s below the permitted lower limit and exceeds the lower limit once the result is corrected for temperature. (Ex. W, p. 2.) Thus, this finding is not evidence of demyelination. (*Id.*) Moreover, this particular finding is better explained as compression neuropathy secondary to her diabetes, especially because the finding was not symmetric (the opposite ulnar nerve study was normal). (*Id*.) Dr. Donofrio also charges that Dr. Chen misinterprets petitioner's F-wave data as evidence of demyelination. Whereas Dr. Chen simply observed prolonged F-wave latency greater than or equal to 20% of the upper limit for the left median nerve and the left ulnar nerve, Dr. Donofrio indicates that these findings must be assessed in the setting of the results of the left median and ulnar motor nerve distal latencies. (*Id*.) Because these motor nerve distal latencies were also prolonged, the prolonged F-wave data cited by Dr. Chen can be attributed to pathology at the wrist and forearm, rather than evidencing

proximal demyelination. (*Id*.) Even if Dr. Chen were correct, evidence of abnormality in only one nerve is only weak support for demyelination. (*Id.* at 3 (citing Van den Bergh et al., *supra*, at Ex. Q, p. 6, tbl.2).)

Dr. Donofrio stresses that, while petitioner did receive a CIDP diagnosis, other treating physicians were unwilling to reach that diagnosis, instead diagnosing peripheral neuropathy, insulin neuritis superimposed on diabetic neuropathy, and paresthesias. (Ex. A, p. 10 (citing Ex. 2, pp. 1-10; Ex. 10, pp. 1-5; Ex. 5, pp. 1-15).) He observes that Dr. Bittle, the neurologist that initially diagnosed CIDP, did not explain what diagnostic criteria were used to render the diagnosis and that preserved reflexes in the biceps and triceps were documented, which does not support a CIDP diagnosis in the context of petitioner's nerve conduction studies under the EFNS/PNS criteria. (Ex. N, p. 4 (discussing Ex. 4, pp. 1, 6).) By contrast, Dr. Papsdorf did not believe that petitioner's May and June nerve conduction studies demonstrated demyelination and instead opined that petitioner had diabetic neuropathy with superimposed insulin neuritis. (Ex. W, p. 3.) Dr. Donofrio stresses that petitioner's June NCS, which was never interpreted as being consistent with CIDP, showed low amplitude CMAPs, which are expected in an axonopathy. Thus, Dr. Donofrio opines that, in addition to being inconsistent with CIDP, is consistent with diabetic neuropathy. (*Id*.)

Dr. Donofrio explains that he maintains his diagnostic opinion after accounting for the finding of fact regarding symptom onset. (Ex. N, p. 3.) He also stresses that Dr. Chen did not cite any specific criteria for her diagnosis of CIDP. (*Id.*) He is not persuaded by Dr. Chen's citation to the case report by Bissay et al. (Ex. 62), contending that the failure of IVIG treatment in that case counsels against the CIDP diagnosis.[9] (*Id*.) He stresses that even more recent revised criteria by the European Academy of Neurology/Peripheral Nerve Society published in 2021, which accounts for variants beyond "typical" CIDP, still do not include fatigue as diagnostic of CIDP. (*Id.* at 3-4 (citing Van den Bergh et al., *supra*, at Ex. Q).) Moreover, whereas Dr. Chen identified numbness and weakness as the "core" features of CIDP, Dr. Donofrio counters that numbness is not a core feature of CIDP, but that the core features of CIDP should also include areflexia or hyporeflexia. (*Id*. at 4.) Dr. Donofrio disagrees that petitioner's presentation is consistent with any of the established "variants" of CIDP, which he identifies as distal CIDP, multifocal CIDP, focal CIDP, pure motor CIDP, and sensory CIDP. (Ex. W, p. 2.)

According to Dr. Donofrio, there are a number of other potential explanations for petitioner's various symptoms suggested within the medical records. (Ex. N, p. 5.) Petitioner's fatigue, in particular, might be explained by documented attention deficit disorder, diabetes, atrial fibrillation, obesity, obstructive sleep apnea, low vitamin D, low vitamin B12, insomnia, acid reflux, elevated cholesterol, periodic limb movement during

[9] Notably, as explained above, Dr. Donofrio also conversely indicates that patients misdiagnosed with CIDP frequently report improvement with immunotherapy despite not actually having CIDP, suggesting that improvement after receiving IVIG does not necessarily support the diagnosis. (Ex. A, p. 10; Ex. N, p. 4 (citing Allen & Lewis, *supra*, at Ex. R).) Thus, Dr. Donofrio opines that, while CIDP should improve with IVIG treatment, such improvement is not specific to CIDP. (Ex. N, p. 4.)

sleep, anxiety and depression, and a thyroid nodule. (*Id*. (citing Ex. 16, pp. 20, 34, 51, 68, 77, 108, 169; Ex. 12, p. 11; Ex. 4, p. 3).) Dr. Donofrio also supports Dr. Papsdorf's diagnosis of insulin neuritis (*Id.* (citing Ex. 10, pp. 4-5)) and further stresses, contrary to Dr. Chen's assessment, that petitioner's A1C remained elevated after onset of her numbness and tingling. (*Id*. (citing Ex. 16, p. 69 (A1C of 8.3 as of March 13, 2015); Ex. 43, pp. 1-13 (A1C of 6.3 as of March 18, 2016); Ex. 10, p. 3 (A1C of 8.8 as of July 20, 2016)).) Numbness and tingling are common initial symptoms of diabetic neuropathy, which will be experienced by 40-50% of diabetics within 10 years of diagnosis. (*Id*. at 5-6 (citing Caitlin W. Hicks & Elizabeth Selvin, *Epidemiology of Peripheral Neuropathy and Lower Extremity Disease in Diabetes*, 19 CURRENT DIABETES REPS. 86 (2019) (Ex. V)).)

d. William Hawse, Ph.D., for respondent[10]

Dr. Hawse's report is limited to seeking to rebut the general theory of causation presented by Dr. Souayah. (Ex. DD.) Because the analysis that follows does not address general causation, it is not necessary to explain Dr. Hawse's opinion.

V. Party Contentions

Petitioner argues that her correct diagnosis is CIDP. (ECF No. 97, p. 6.) She stresses that, despite a "windy" road to that diagnosis, it was the clinical judgment of two neurologists. (*Id*.) In particular, petitioner argues that the fact that Dr. Papsdorf initiated IVIG treatment following petitioner's July 2016 electrodiagnostic study is evidence that she changed her diagnosis from diabetic neuropathy to CIDP. (*Id*. at 7-8.) Moreover, her expert explains that it is not unusual for CIDP patients to have difficulty in getting properly diagnosed. (*Id*. at 6.) Acknowledging the finding of fact that onset of weakness first occurred in July of 2016, petitioner argues that fatigue is a common presenting symptom of CIDP and that onset of weakness in CIDP can occur "several years" after the initial onset of other symptoms. (*Id*. at 7.) Therefore, petitioner argues that the onset of her CIDP occurred at the time she first experienced numbness and tingling, approximately five weeks post-vaccination. (*Id*. at 14.)

Petitioner cites numerous cases for the proposition that molecular mimicry has been accepted as a sound a reliable theory of causation for vaccine-related GBS. (ECF No. 97, pp. 8-9 (citing *Conte v. Sec'y of Health & Human Servs.*, No. 17-403V, 2020 WL 5743696, at *23 (Fed. Cl. Spec. Mstr. July 27, 2020); *Barone v. Sec'y of Health &*

[10] Dr. Hawse received his Ph.D. in biophysical chemistry from Johns Hopkins University in 2009, before going on to complete two postdoctoral fellowships, one in structural immunology at the University of Notre Dame in 2013 and another in immunology and signal transduction at the University of Pittsburgh in 2015. (Ex. OO, p. 1.) His research focused on the biochemical basis for T cell receptor cross-reactivity and CD4+ T cell differentiation and immune tolerance. (Ex. DD, p. 1.) From there, Dr. Hawse accepted a faculty position as a research assistant professor at the University of Pittsburgh. (Ex. OO, p. 1; Ex. DD, p. 1.) He was subsequently promoted to assistant professor of immunology in 2019. (Ex. OO, p. 1; Ex. DD, p. 1.) Dr. Hawse's background is in basic immunological mechanisms focused on adaptive immune response, and he has published nearly 30 peer-reviewed articles. (Ex. DD, p. 2; Ex. OO, pp. 2-5.)

Human Servs., No. 11-707V, 2014 WL 6834557, at *8-9 (Fed. Cl. Spec. Mstr. Nov. 12, 2024); *Koller v. Sec'y of Health & Human Servs.*, No. 16-439V, 2021 WL 5027947, at *18 (Fed. Cl. Spec. Mstr. Oct. 8, 2021); *Pierson v. Sec'y of Health & Human Servs.*, No. 17-1136V, 2022 WL 322836, at *31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Deshler v. Sec'y of Health & Human Servs.*, No. 16-1070V, 2020 WL 4593162, at *19 (Fed. Cl. Spec. Mstr. July 1, 2020); *Maloney v. Sec'y of Health & Human Servs.*, No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022)).) Petitioner further cites *Berg v. Secretary of Health & Human Services* to support the relevance of evidence pertaining to GBS in cases alleging CIDP. (*Id*. at 9-10 (quoting No. 16-650V, 2021 WL 6883495, at *44 (Fed. Cl. Spec. Mstr. Dec. 14, 2021)).) In addition to citing several case reports regarding CIDP arising after flu vaccination, Dr. Souayah explained the pathophysiologic similarities between GBS and CIDP as relating to post-infectious humeral and cellular dysfunction, including molecular mimicry and polyclonal activation. (*Id*. at 10-11 (citing Ex. 30, pp. 8, 12-13, 15).) Although petitioner acknowledges that "the degree of eventual T-cell dysfunction could be a differentiating factor," both GBS and CIDP are associated with antigen-presenting molecules from the HLA-DR/DQ system. (*Id*. at 11 (citing Ex. 30, p. 8).) Petitioner argues that she is not required to demonstrate "exactly which molecules reacted to the vaccination to incite her immune system's attack on her nerves." (*Id.*)

Petitioner contends that there is no causal relationship between diabetes and CIDP. (ECF No. 97, p. 12.) Thus, she argues that her flu vaccination is the only potential cause of her CIDP. (*Id*.) However, petitioner does acknowledge that diabetes may have played a contributory role in her development of CIDP. (*Id.*) Given the difficulties petitioner's treating physicians had in arriving at a diagnosis and pinpointing onset of petitioner's CIDP, the lack of any attribution of petitioner's condition to her flu vaccination should not be viewed as surprising. (*Id*.) Citing several prior cases, petitioner contends that the onset of her CIDP occurred within a timeframe from which a causal inference can be drawn based on the prior finding of fact. (*Id*. at 13-14 (citing *Nieves v. Sec'y of Health & Human Servs.*, No. 18-1602V, 2023 WL 3580148 (Fed. Cl. Spec. Mstr. Apr. 17, 2023), *mot. for rev. denied*, 167 Fed. Cl. 422 (2023); *Kelley v. Sec'y of Health & Human Servs.*, 68 Fed. Cl. 84, 102 (1005); *Daily v. Sec'y of Health & Human Servs.*, No. 07-173V, 2011 WL 2174535, at *9 (Fed. Cl. Spec. Mstr. May 11, 2011); *Patel v. Sec'y of Health & Human Servs.*, No. 16-848V, 2020 WL 2954950, at *18-21 (Fed. Cl. Spec. Mstr. May 1, 2020); *Strong v. Sec'y of Health & Human Servs.*, No. 15-1108V, 2018 WL 1125666, at *21 (Fed. Cl. Spec. Mstr. Jan. 12, 2018)).) In her motion, petitioner did not cite any medical evidence establishing on this record what the appropriate timeframe would be pursuant to the theory she advanced. (*Id*.) However, in her reply, petitioner clarified that she relies on Dr. Souayah's opinion that up to six weeks is the appropriate timeframe within which CIDP could develop post-vaccination. (ECF No. 100, p. 6 (citing Ex. 30, p. 7; P. A. McCombe et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical and Electrophysiological Study of 92 Cases*, 110 BRAIN 1617 (1987) (Ex. 79)).)[11]

[11] While petitioner is correct that Dr. Souayah noted a six-week onset period on page 7 of his report, he also separately identified an eight-week period on page 10 of his report, as noted above in the summary of Dr. Souayah's opinion.

Preferring his own expert's assessment, respondent argues first and foremost that petitioner never actually suffered CIDP. (ECF No. 98, pp. 24-31.) Instead, petitioner's entire clinical history is more likely explained by diabetic neuropathy. (*Id*. at 30-31.) Respondent argues this alone is fatal to petitioner's claim. (*Id*. at 31 (citing *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010); *Lombardi v. Sec'y of Health & Human Servs.*, 656 F.3d 1343, 1352-53 (Fed. Cir. 2011)).) Addressing an *Althen* analysis for CIDP *arguendo*, respondent contends that petitioner has presented no reliable evidence causally linking the flu vaccine to CIDP. (*Id*. at 32-39.) The fact that molecular mimicry explains some autoimmune diseases does not mean that it explains CIDP in particular, and it is insufficient for petitioner to merely rely on prior cases or analogy to GBS. (*Id*.) Respondent stresses the lack of any treating physician opinion supporting vaccine causation in this case and further suggests that petitioner's comorbidities cloud her attempted causal relationship. In particular, respondent urges that the numbness and tingling that began in November 2015, as determined by the prior fact finding, are best explained by diabetic neuropathy. (*Id*. at 40-41.) Moreover, respondent argues that there is a "strong" relationship between diabetes and CIDP that supports a casual inference between petitioner's diabetes and any CIDP in preference to any causal relationship between petitioner's flu vaccination and her alleged CIDP. (*Id*.) Respondent disagrees that onset of CIDP would have occurred five weeks post-vaccination as Dr. Chen opined; however, even if that were the onset, petitioner has not presented a medical opinion sufficient to establish the appropriate timeframe for the development of a post-vaccination CIDP. (*Id*. at 43-45.)

VI. Analysis

For purposes of this decision, I will assume *arguendo* that CIDP can be caused by the flu vaccine for purposes of *Althen* prong one. The question of whether CIDP can be caused by the flu vaccine remains unsettled. *Compare, e.g. Daily*, 2011 WL 2174535, at *6-8 (concluding that petitioner had substantiated, and respondent had not sufficiently rebutted, that the flu vaccine can cause CIDP via molecular mimicry based on analogy to GBS) *with Jacunski v. Sec'y of Health & Human Servs.*, No. 09-524V, 2014 WL 5168422, at *12, *16-17 (Fed. Cl. Spec. Mstr. Sept. 23, 2014) (concluding that petitioner had not preponderantly proven that the flu vaccine can cause or significant aggravate CIDP via molecular mimicry); *see also Nieves*, 2023 WL 3580148, at *36, *46 (explaining that whether the flu vaccine can cause CIDP is "an *extremely* close call" and that "overlap between GBS and CIDP cannot be employed as a shortcut to entitlement, simply because certain principles that have been preponderantly shown bearing on the flu vaccine-GBS connection (like the mechanism of molecular mimicry) could *plausibly* be extended" to flu vaccine-CIDP). However, this case has also presented significant questions regarding petitioner's own personal clinical history that are dispositive regardless of the question of general causation. That is, even if flu vaccine can cause CIDP, petitioner cannot meet her burden of proof under either *Althen* prongs two or three.

a. *Althen* prong two

The second *Althen* prong requires proof of a logical sequence of cause and effect showing that the vaccine was the reason for the injury, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1375-77 (Fed. Cir. 2009); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1326 (Fed. Cir. 2006); *Grant*, 956 F.2d at 1148. However, petitioner may support her claim by presenting either medical records or the opinion of a competent medical expert. § 300aa-13(a)(1). Medical records and/or statements of a treating physician 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. *See* § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) ("[T]here 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 noted above, I previously issued findings of fact in this case. Specifically, I found that "the record as a whole preponderates in favor of finding that petitioner experienced numbness and tingling in her extremities along with fatigue beginning in late November 2015; however, there is not preponderant evidence on the current record that she suffered weakness until July of 2016." (ECF No. 74, p. 16; 2021 WL 5905687, at *12.) Following review of the subsequently filed expert reports and the parties' briefing, these findings stand and are applied within this analysis. Left unaddressed by the prior fact finding, however, was the further question of "whether or how these symptoms, coupled with the overall medical course reflected in the treatment records, support petitioner's allegation of vaccine-caused CIDP." (ECF No. 74, p. 16; 2021 WL 5905687, at *12.)

Throughout the pendency of this case, Dr. Donofrio has been consistent on respondent's behalf in opining that petitioner's condition is explained exclusively by diabetic neuropathy, which he stresses is the most common form of peripheral neuropathy worldwide. However, if petitioner did suffer CIDP, then Dr. Donofrio would opine that petitioner initially suffered a months long course of diabetic neuropathy followed by a later onset of CIDP heralded by the onset of weakness, which he places in July based on the above-referenced finding of fact. (Ex. Y, pp. 3-4.) Dr. Chen agrees on petitioner's behalf that it is plausible that she suffered both diabetic neuropathy and a later manifesting CIDP, but she prefers to invoke an atypical course of CIDP to explain petitioner's entire clinical history. (Ex. 131, pp. 2-3.)

Contrary to either party's assessment, a review of petitioner's medical history confirms that she was diagnosed with *both* diabetic neuropathy and CIDP. *See Sword v. United States*, 44 Fed. Cl. 183, 187-89 (1999) (explaining of special masters that "as fact-finders, they may find that truth lies somewhere in between the opposing, uncompromising views of the partisan experts"). Regarding respondent's view, even if Dr. Donofrio were correct that petitioner's electrodiagnostic study is not diagnostic of

CIDP under stringent application of the relevant criteria, he stands alone in drawing that conclusion as a matter of clinical judgment. In addition to Dr. Chen, two treating neurologists (Drs. Papsdorf and Bittle) felt that petitioner's July 2016 electrodiagnostic study was at least "suggestive" of CIDP and Dr. Bittle explicitly reached the diagnosis of CIDP. (Ex. 10, p. 6; Ex. 4, p. 6.) Conversely, petitioner is not persuasive in contending that Dr. Papsdorf retracted her initial diagnoses of diabetic neuropathy and insulin neuritis. Although petitioner is correct to observe that Dr. Papsdorf initiated treatment for CIDP following the July 2016 electrodiagnostic study, Dr. Papsdorf explicitly indicated that the study had not ruled out diabetic amyotrophy because it was incomplete. (Ex. 19, p. 65.) Indeed, petitioner specifically testified that she switched to another neurologist because she was frustrated that Dr. Papsdorf remained insistent that her condition was related to her diabetes. (Tr. 63-64.)

In rejecting petitioner's diagnosis of diabetic neuropathy, Dr. Chen relied in part on the fact that petitioner's July 2016 electrodiagnostic study did not demonstrate axonal nerve damage consistent with diabetic neuropathy. (Ex. 67, pp. 3-4.) However, this is not reliable in light of Dr. Papsdorf's explanation that the study was incomplete and unable to rule out diabetic amyotrophy. (Ex. 19, p. 65.) Moreover, whereas CIDP was not electrodiagnostically evidenced until petitioner's July 2016 study, Dr. Donofrio opined in agreement with Dr. Papsdorf's opinion that petitioner's earlier June 2016 nerve conduction study was consistent with axonal injury due to diabetic neuropathy. (Ex. W, p. 3.) Dr. Chen also doubted petitioner's diabetic neuropathy diagnosis because diabetic neuropathy is "correlated with the degree of glycemic control." (Ex. 131, p. 2 (citing Sasaki et al., *supra*, at Ex. 134).) She cited a September 15, 2015 A1C reading of 6.5, from which she observes "[i]t would be odd for her to have sensory symptoms of a diabetic neuropathy after she obtained glycemic control." (*Id.*) Dr. Donofrio observed, however, that subsequent A1C readings from March and July of 2016 showed that that it had become elevated again. (Ex. Y, p. 3 (citing Ex. 43, pp. 1-13; Ex. 10, p. 3).) Especially in light of Dr. Donofrio's observation, Dr. Chen is not persuasive in using a single A1C reading to extrapolate petitioner's degree of glycemic control over time.

Accordingly, the evidence preponderates in favor of a finding that petitioner suffered both diabetic neuropathy and later CIDP. In that regard, Dr. Donofrio noted that there is a known association between diabetes and CIDP, though he acknowledges that relevant studies have not been uniform in detecting an increased risk of CIDP among diabetics. (Ex. Y, pp. 1-2.) Dr. Chen considers the association "controversial," but likewise acknowledges that an increased risk of CIDP among diabetics has been detected epidemiologically. (Ex. 131, p. 2.) Although both experts stressed that association alone does not equate to a causal relationship, Dr. Chen further explained that the relevant literature hypothesizes that "diabetes causes injury to the nerve resulting in exposure of neuronal antigens that were previously hidden from the immune system . . . [which] may trigger autoreactive immune cells to recognize these self-antigens as foreign." (*Id.* (citing Chen & Tang, *supra*, at Ex. 133).) Dr. Donofrio agreed that this hypothesis "makes pathological and immunological sense." (Ex. Y, p. 3.) Such a hypothesis, especially when coupled with the evidence indicating an increased risk of

CIDP among diabetics, supports a direct causal relationship between diabetic nerve damage and later manifesting CIDP.[12]

Even if an atypical form of CIDP is *possible*, Dr. Chen is not persuasive in applying that concept to this case. Dr. Chen relies, in particular, on a case report by Bissay et al., reporting on a patient who was diagnosed with CIDP based on electrodiagnostic criteria despite his only symptom being fatigue. (Bissay et al., *supra*, at Ex. 62.) Even setting aside Dr. Donofrio's criticism of the Bissay subject's CIDP diagnosis (Ex. N, p. 3), the case report is not informative. As the case report authors note, fatigue is "a nonspecific symptom, and the differential diagnosis is extensive." (Bissay et al., *supra*, at Ex. 62, p. 1.) The fact that the Bissay subject's differential diagnosis was ultimately narrowed to CIDP does not mean that fatigue necessarily implicates CIDP. Rather, the Bissay subject was diagnosed based on electrodiagnostic evidence, not the presence of fatigue. Moreover, the Bissay subject's case was notable as potentially representative of a minimal form of CIDP because he lacked any clinical features of any other form of neuropathy. (*Id.* at 4.) Here, however, Dr. Donofrio explained that petitioner's May and June 2016 electrodiagnostic studies were more consistent with diabetic neuropathy than CIDP and her treating neurologist (Dr. Papsdorf) at the time felt that her differential diagnosis prior to onset of weakness favored diabetic neuropathy as an explanation for her presentation. Indeed, Dr. Donofrio observed that diabetic neuropathy can cause or worsen fatigue. (Ex. N, p. 5.) Additionally, a significant finding among patients purportedly experiencing so-called "minimal CIDP," in which fatigue and sensory symptoms are the primary presentation, is areflexia or hyporeflexia. (Bissay et al., *supra*, at Ex. 62, p. 3; Boukhris et al., *supra*, at Ex. 59, p. 6.) Dr. Donofrio likewise explained that this is a "core" feature of CIDP.[13] (Ex. N, pp. 3-4.) However, prior to June of 2016, petitioner did not have either areflexia or hyporeflexia. (*Compare* Ex. 5, p. 2 (emergency department record noting normal deep tendon reflexes on physical examination as of April 17, 2016), *with* Ex. 9, p. 2 (Dr. Schudy documenting reduced deep tendon reflexes as of June 30, 2016).) In citing the concept of "minimal CIDP," Dr. Chen does not adequately grapple with the fact that there was a change in petitioner's condition around June and July of 2016 wherein

[12] Dr. Chen did state that she felt the hypothesis is "purely hypothetical," but nonetheless sought to apply it as a means of opining that the flu vaccine could, when combined with pre-existing diabetic neuropathy, culminate in CIDP occurring "months" after vaccination. (*Compare* Ex. 131, p. 2, *with id.* at 4.) Notably, however, though demonstrating her willingness to accept a causal relationship between diabetes and CIDP, Dr. Chen did not adequately explain how this hypothesis would support the involvement of the flu vaccine in manifesting CIDP "months" after exposure. (*Id.* at 4.)

[13] Dr. Chen disputes the necessity of absent or reduced reflexes under the diagnostic criteria favored by Dr. Donofrio because that criteria allows that reflexes may be present among CIDP variants. (Ex. 67, p. 3 (citing Van den Bergh et al., *supra*, at Ex. Q, p. 4, tbl.1).) However, Dr. Chen overstates the significance of the CIDP variants. The Table that Dr. Chen cites first indicates that "typical" CIDP requires absent or reduced tendon reflexes in all limbs. (Van den Bergh et al., *supra*, at Ex. Q, p. 4, tbl.1.) The list of CIDP variants includes descriptions of various presentations, some of which do not affect all limbs. Regarding these variants, the criteria states "otherwise as in typical CIDP (tendon reflexes may be normal in unaffected limbs)." (*Id.*) Notably, the medical literature Dr. Chen herself cites for the concept of "minimal CIDP" notes of these patients that areflexia or hyporeflexia are the "most constant feature." (Boukhis et al., *supra*, at Ex. 59, p. 6.)

during the same period she saw onset of weakness, reduction in reflexes, and for the first time had an electrodiagnostic study "suggestive" of CIDP. Ultimately, notwithstanding her case-specific assessment, Dr. Chen agrees as a general matter that it would be 2-3 times more likely for petitioner to have had both diabetic neuropathy and CIDP as compared to the atypical presentation of CIDP that she proposes. (Ex. 131, p. 3.)

In sum, considering the record as a whole, the evidence preponderates in favor of a finding that petitioner's symptoms of numbness, tingling, and fatigue, which first arose approximately five weeks post-vaccination, are more likely explained by Dr. Papsdorf's initial diagnoses of diabetic neuropathy and/or insulin neuritis. Although petitioner was also later diagnosed with CIDP, there is not preponderant evidence that onset of that condition occurred prior to about June or July of 2016. Ultimately, the evidence weighs against any logical sequence of cause-and-effect implicating petitioner's flu vaccination as a cause of her CIDP for the following reasons:

- Although petitioner repeatedly reported her vaccine as a relevant part of her history, none of her physicians, including those that diagnosed CIDP, opined that her vaccination was a cause of her condition.

- Petitioner not only had a history of diabetes, which is associated with increased risk of CIDP, but was also suffering diabetic neuropathy, which the experts explain can help explain the development of CIDP. Even petitioner's own expert agrees that diabetes may have been at least a contributing factor in the development of her CIDP.

- A typical presentation of CIDP would arise with demonstrated weakness and reduced or absent reflexes, which in this case would place onset CIDP about 8-9 months post-vaccination. As discussed under *Althen* prong three, this timing of onset does not support a casual inference relative to petitioner's flu vaccination.

- In order to account for petitioner's own presentation – a months long course of numbness, tingling, pain, and fatigue only later followed by a separate onset of weakness – petitioner's expert suggests that she experienced an "atypical" or "minimal" form of CIDP. However, respondent's expert observes that petitioner's presentation does not fit any of the recognized variants of CIDP and none of petitioner's treating physicians diagnosed her as having any atypical presentation.

- Separate and apart from whether diabetes or diabetic neuropathy would be the cause of a subsequent presentation of CIDP, diabetic neuropathy would be sufficient to explain petitioner's symptoms of numbness, tingling, pain, and fatigue during the months prior to the onset of weakness, which is consistent with the treating neurologist's (Dr. Papsdorf's) impression and further undercuts any suspicion of an atypical form of CIDP in preference to a typical, but later manifesting, CIDP.

- Dr. Chen acknowledges that a presentation of diabetic neuropathy and subsequent CIDP would be 2-3 times more likely than an atypical presentation of CIDP.

Thus, considering the record as a whole, petitioner has not met her preponderant burden of proof under *Althen* prong two.

b. *Althen* prong three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. 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-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008).

Here, petitioner cites Dr. Souayah's opinion that CIDP can be causally related to a flu vaccination occurring up to six (or eight) weeks earlier. (ECF No. 100, p. 6 (citing Ex. 30, p. 7; McCombe et al., *supra*, at Ex. 79); *see also* Ex. 30, p. 10 (citing Gable et al., *supra*, at Ex. 111).) Thus, petitioner argues that she has met her burden of proof under *Althen* prong three based on the prior finding of fact, which found that petitioner experienced onset of numbness and tingling approximately five weeks post-vaccination. (ECF No. 97, p. 14.) However, for the reasons discussed above relative to *Althen* prong two, the onset of numbness and tingling is not indicative of the initial onset of CIDP in this case. Petitioner has not demonstrated that the onset of CIDP occurred within the six or eight weeks that Dr. Souayah identifies as the period within which a causal inference can be drawn.[14] Therefore, petitioner has not met her burden of proof under *Althen* prong three.

VII. Conclusion

There is no question that petitioner has suffered, and she has my sympathy. However, for all the reasons discussed above, I find that petitioner has not met her burden of proof in this case. Therefore, this case is dismissed.[15]

[14] Dr. Chen also opined that in the context of pre-existing diabetic neuropathy the flu vaccine could be the cause of CIDP manifesting "months" later. (Ex. 131, p. 4.) However, Dr. Chen did not adequately address the expected timing of this theory, stating only that the expected course would be "protracted." (*Id*.)

[15] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master