In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: November 13, 2024

DONALD R. IZARD,	*	PUBLISHED
Petitioner,	*	No. 17-623V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Chronic Inflammatory Demyelinating Polyneuropathy ("CIDP"); Systemic Lupus Erythematosus ("SLE").
Respondent.	*	

Stacey Amanda Subryan-Gerber, Tiveron Law, PLLC, Amherst, NY, for Petitioner.
Sarah Christina Duncan, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

I. INTRODUCTION

On May 9, 2017, Donald R. Izard ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that he developed chronic inflammatory demyelinating polyneuropathy ("CIDP") as a result of an influenza ("flu") vaccine administered on October 14, 2015. Petition at Preamble (ECF No. 1). Respondent argued against

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it 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 Decision 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, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

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compensation, stating that this petition should be dismissed. Respondent's Report ("Resp. Rept.") at 1, 8 (ECF No. 12).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[3] the undersigned finds that Petitioner has failed to provide preponderant evidence that his flu vaccine caused him to develop CIDP, systemic lupus erythematosus ("SLE"), or any other injury. Thus, Petitioner has failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

II. ISSUES TO BE DECIDED

The parties stipulated that Petitioner received a flu vaccine on October 14, 2015. Joint Submission, filed May 23, 2023, at 1 (ECF No. 131). The parties also stipulated that Petitioner suffers from SLE. Id.

The parties dispute Petitioner's diagnosis of CIDP. Joint Submission at 1. Petitioner argues he had "asymptomatic [SLE] which was triggered by the flu vaccine and presented as CIDP." Pet. Motion for Ruling on the Record ("Pet. Mot."), filed Sept. 18, 2023, at 3 (ECF No. 151). "Petitioner[] concedes that a diagnosis of SLE is reasonable . . . [and] further asserts . . . that CIDP was brought on by the administration of the flu vaccine in October of 2015." Id. at 4. Respondent argues Petitioner has not established by preponderant evidence that he suffers from CIDP, and instead argues there is preponderant evidence supporting a diagnosis of "[SLE] with neuropathy as the presenting symptom." Resp. Response to Pet. Mot. ("Resp. Response"), filed Nov. 16, 2023, at 26-28 (ECF No. 156).

The onset of Petitioner's alleged vaccine injury is also in dispute as well as all three Althen prongs: (1) whether the flu vaccine can cause Petitioner's alleged injury, (2) whether Petitioner's condition was caused by his October 14, 2015 flu vaccine, and (3) whether the onset of Petitioner's condition began within a timeframe for which it is medically acceptable to infer causation-in-fact. Joint Submission at 1-2.

[3] While the undersigned has reviewed all of the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

III. BACKGROUND

A. Procedural History

Petitioner filed his petition on May 9, 2017, along with medical records and an affidavit.[4] Petition; Pet. Exhibit ("Exs.") 1-5. Respondent filed his Rule 4(c) report on January 3, 2018, "recommend[ing] that compensation in this case be denied." Resp. Rept. at 1.

Petitioner filed expert reports from Dr. Marcel Kinsbourne and Dr. Vera Byers on June 15, 2018. Pet. Exs. 14, 16. On October 31, 2018, Respondent filed expert reports from Dr. Vinay Chaudhry, Dr. Diane L. Kamen, and Dr. Ross M. Kedl. Resp. Exs. A, C, E. Petitioner filed an expert report from Dr. Rebecca M. Shepherd on March 18, 2019 and an expert report from Dr. Joseph A. Bellanti on August 6, 2019. Pet. Exs. 20, 27. Respondent filed supplemental expert reports from Dr. Chaudhry and Dr. Kedl on November 21, 2019 and a supplemental expert report from Dr. Kamen on December 5, 2019. Resp. Exs. G-I.

Thereafter, a status conference was held and an entitlement hearing was scheduled for March 2021. Order dated Jan. 24, 2020 (ECF No. 61); Prehearing Order dated Mar. 11, 2020 (ECF No. 66). In January 2021, Petitioner's counsel requested the entitlement hearing be continued, which the undersigned granted. Order Granting Motion to Continue Hearing dated Jan. 28, 2021 (ECF No. 71). The entitlement hearing was rescheduled for April 2022. Prehearing Order dated Aug. 3, 2021 (ECF No. 80). Prior to April 2022, Respondent filed an expert report from Dr. Harold Moses. Resp. Ex. J.

On April 20, 2022, the undersigned held a status conference pursuant to Respondent's request. Order dated Apr. 20, 2022, at 1 (ECF No. 107). On April 19, 2022, Petitioner filed approximately 2,000 pages of medical records, some of which were duplicative of previously filed medical records. Id. The entitlement hearing was scheduled for April 26-29, 2022. Id. Respondent requested a continuation of the hearing to review the recently filed records and potentially request outstanding medical records so that their experts could review the records. Id. The undersigned also noted a review of the newly filed records showed some records were obtained by Petitioner's Counsel's firm in 2019 but were not filed until April 19, 2022. Id. The undersigned granted Respondent's oral motion for a continuance. Id. The entitlement hearing was rescheduled for July 2023. Prehearing Order dated May 20, 2022 (ECF No. 113).

Over the next year, Petitioner continued to file medical records. Pet. Exs. 29-51. On June 23, 2023, Petitioner filed a status report, indicating that records remained outstanding and "request[ing] a short continuation to permit both Petitioner and Respondent to adequately prepare for the hearing." Pet. Status Rept., filed June 23, 2023 (ECF No. 142). On June 26, 2023, the parties filed a joint status report, indicating that they preferred to proceed with a ruling on the record instead of the entitlement hearing in July 2023. Joint Status Rept., filed June 26, 2023 (ECF No. 143). The entitlement hearing was cancelled, and the parties were given a

[4] Petitioner continued to file medical records and affidavits throughout litigation. Petitioner did not file an exhibit 6, which appears to be consolidated with exhibit 4. See Pet. Ex. 27 at 1.

briefing schedule. Order dated June 26, 2023 (ECF No. 144); Ruling on the Record Order dated Aug. 21, 2023 (ECF No. 148).

Petitioner filed his motion for a ruling on the record on September 18, 2023. Pet. Mot. Respondent filed his response on November 16, 2023, and Petitioner filed a reply on December 18, 2023. Resp. Response; Pet. Reply to Resp. Response ("Pet. Reply"), filed Dec. 18, 2023 (ECF No. 157).

This matter is now ripe for adjudication.

B. Medical Terminology

1. Chronic Inflammatory Demyelinating Polyneuropathy

CIDP is "characterized by progressive weakness and impaired sensory function in the legs and arms" due to "damage to the myelin sheath (the fatty covering that wraps around and protects nerve fibers) of the peripheral nerves." Pet. Ex. 22-h at 1;[5] see also Resp. Ex. A, Tab 8 at 1-2.[6] "It often presents with symptoms that include tingling or numbness (beginning in the toes and fingers), weakness of the arms and legs, loss of deep tendon reflexes (areflexia), fatigue, and abnormal sensations." Pet. Ex. 22-h at 1; see also Resp. Ex. E, Tab 5 at 3.[7] The weakness progressively increases for more than two months. Pet. Ex. 27, Tab 11 at 1;[8] Resp. Ex. A, Tab 8 at 2; Resp. Ex. E, Tab 5 at 3. Treatment can include corticosteroids (prednisone), plasmapheresis, and intravenous immunoglobulin ("IVIG"). Pet. Ex. 22-h at 1; Pet. Ex. 27, Tab 11 at 2, 9; Resp. Ex. A, Tab 8 at 2, 6; Resp. Ex. E, Tab 5 at 3.

2. Systemic Lupus Erythematosus

SLE is "a chronic, inflammatory, often febrile multisystemic disorder of connective tissue that proceeds through remissions and relapses" and "may be either acute or insidious in onset." Systemic Lupus Erythematosus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87476 (last visited Sept. 27, 2024). The etiology of SLE is unknown. Id. It is characterized by widespread inflammation and tissue

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[5] Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) Information Page, Nat'l Inst. Neurological Disorders & Stroke, http://www.ninds.nih.gov/disorders/cidp/cidp.htm (last modified June 1, 2016).

[6] Marinos C. Dalakas, Advances in the Diagnosis, Pathogenesis and Treatment of CIDP, 7 Nature Revs. 507 (2011). This article was also cited as Resp. Ex. E, Tab 2.

[7] Adi Hersalis Eldar & Joab Chapman, Guillain Barré Syndrome and Other Immune Mediated Neuropathies: Diagnosis and Classification, 13 Autoimmunity Revs. 525 (2014).

[8] Hubertus Köller et al., Chronic Inflammatory Demyelinating Polyneuropathy, 352 New Eng. J. Med. 1343 (2005).

damage thought to be caused by autoantibodies attacking self-cells. Pet. Ex. 23-e at 1;[9] Resp. Ex. H, Tab 8 at 1;[10] Resp. Ex. H, Tab 11 at 2.[11]

There are two classification criteria relevant to SLE discussed herein. The first, is the 1997 diagnostic criteria for SLE set forth by the American College of Rheumatology ("ACR"). See Pet. Ex. 23-a at 1;[12] Resp. Ex. I, Tab 1. Under the 1997 ACR criteria, a patient must meet at least four out of 11 criteria. Pet. Ex. 23-b at 2.[13] These criteria include malar rash, discoid rash, photosensitivity, oral ulcers, nonerosive arthritis, pleuritis or pericarditis, renal disorder, neurologic disorder (seizures or psychosis), hematologic disorder (e.g., hemolytic anemia),[14] immunologic disorder (positive anti-DNA, positive anti-Smith,[15] or positive antiphospholipid antibodies), and positive antinuclear antibodies ("ANA")[16] (at any point in time). Pet. Ex. 23-a

[9] Malgorzata J. Podolska et al., Inflammatory Etiopathogenesis of Systemic Lupus Erythematosus: An Update, 8 J. Inflammation Rsch. 161 (2015).

[10] Yafang Huang et al., Is Systemic Lupus Erythematosus Associated with a Declined Immunogenicity and Poor Safety of Influenza Vaccination? A Systematic Review and Meta-Analysis, 95 Medicine 1 (2016).

[11] Zhengfa Liao et al., Immunogenicity and Safety of Influenza Vaccination in Systemic Lupus Erythematosus Patients Compared with Healthy Controls: A Meta-Analysis, 11 PLoS One 1 (2016).

[12] This appears to be a handout made from information contained in Resp. Ex. I, Tab 1 (Marc C. Hochberg, Updating the American College of Rheumatology Revised Criteria for the Classification of Systemic Lupus Erythematosus, 40 Arthritis & Rheumatism 1725 (1997)).

[13] Felina Anić et al., New Classification Criteria for Systemic Lupus Erythematosus Correlated with Disease Activity, 55 Croatian Med. J. 514 (2014).

[14] Hemolytic anemia is "characterized by excessive hemolysis and impaired erythropoiesis. There are two major groups: the inherited anemias, which are generally due to intrinsic cell defects . . . and the acquired anemias, which are due to the actions of extrinsic agents such as infectious agents, poisons, physical trauma, or antibodies." Hemolytic Anemia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56147 (last visited Sept. 27, 2024).

[15] Anti-Smith antibody is "highly specific for SLE." 2 Bevra Hannahs Hahn & Betty P. Tsao, Pathogenesis of Systemic Lupus Erythematosus, in Kelly's Textbook of Rheumatology 1233, 1225 (Gary S. Firestein eds., 8th ed. 2009).

[16] Antinuclear antibodies, or ANA, are "antibodies directed against nuclear antigens; ones against a variety of different antigens are almost invariably found in [SLE]." Antinuclear Antibodies, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited Sept. 27, 2024).

at 1. Peripheral neuropathy was not expressly included as a condition that fulfilled the neurologic criteria. See id.

The 1997 ACR criteria were revised by the Systemic Lupus International Collaborating Clinics ("SLICC") in 2012. Pet. Ex. 20, Tab 3 at 1.[17] The newer criteria were thought to better "correlate with disease activity." Pet. Ex. 23-b at 1. Under the SLICC classification criteria, one is appropriately diagnosed with SLE if they satisfy "[four] of the clinical and immunologic criteria," including one of each category.[18] Id. at 5. The criteria do not need to present concurrently. Id.; see also Pet. Ex. 20, Tab 5 at 5-6.[19] The SLICC clinical criteria include inflammatory conditions from different connective tissue systems, including, but not limited to, skin (e.g., rashes, ulcers, nonscarring alopecia),[20] joints (e.g., synovitis), pulmonary (e.g., pleurisy, pleural effusion), cardiac (pericarditis), neurologic (e.g., peripheral neuropathy), and hematology (hemolytic anemia). Pet. Ex. 20, Tab 3 at 6 tbl.3. Immunologic criteria include, for example, elevated ANA, elevated anti-double-stranded DNA ("dsDNA") antibodies,[21] and the presence of anti-Smith antibodies. Id. Peripheral neuropathy is specifically noted as meeting the criteria for a neurological inflammatory condition. Id.

As acknowledged in the SLICC criteria, peripheral neuropathies, including CIDP, are associated with SLE. Pet. Ex. 20, Tab 3 at 6 tbl.3; see also Pet. Ex. 20, Tab 1 at 3;[22] Pet. Ex. 20,

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[17] Michelle Petri et al., Derivation and Validation of the Systemic Lupus International Collaborating Clinics Classification Criteria for Systemic Lupus Erythematosus, 64 Arthritis & Rheumatism 2677 (2012). This article was also cited as Pet. Ex. 23-d and Resp. Ex. C, Tab 1.

[18] For the complete list of clinical and immunologic criteria used in the SLICC classification system, see Pet. Ex. 20, Tab 3 at 6 tbl.3.

[19] Ester A.R. Hartman et al., Performance of the 2012 Systemic Lupus International Collaborating Clinics Classification Criteria Versus the 1997 American College of Rheumatology Classification Criteria in Adult and Juvenile Systemic Lupus Erythematosus: A Systematic Review and Meta-Analysis, 17 Autoimmunity Revs. 316 (2008).

[20] Alopecia is the "lack or loss of hair from skin areas where it normally is present." Alopecia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=1905 (last visited Sept. 27, 2024).

[21] Anti-dsDNA antibody is "a type of antinuclear antibody specific for double-stranded DNA, found in the serum of patients with [SLE]." Anti-dsDNA Antibody, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56790 (last visited Sept. 27, 2024).

[22] Philippe Hantson et al., Acute-Onset Chronic Inflammatory Demyelinating Polyneuropathy with Cranial Nerve Involvement, Dysautonomia, Respiratory Failure, and Autoantibodies, 41 Muscle & Nerve 423 (2010).

Tab 2 at 9;[23] Pet. Ex. 23-c at 2 Box 1;[24] Pet. Ex. 27, Tab 11 at 4 tbl.2; Resp. Ex. C, Tab 3 at 2.[25] CIDP is a "manifestation of SLE and can occur before, after, or simultaneously with the onset of SLE." Pet. Ex. 20, Tab 2 at 9; see also Pet. Ex. 20, Tab 1 at 4; Pet. Ex. 20, Tab 6 at 1;[26] Pet. Ex. 23-c at 3 ("In adults, approximately 28% to 40% of [neuropsychiatric SLE] manifestations develop before or around the time of the diagnosis of SLE."); Resp. Ex. C, Tab 3 at 2-3. "Pathologic findings of CIDP or CIDP-like polyneuropathies in SLE patients[] [] are highly variable." Pet. Ex. 20, Tab 2 at 8.

C. Factual History

1. Medical History[27]

Petitioner was sixty-six years old when he received a flu vaccine (Fluzone) on October 14, 2015.[28] Pet. Ex. 2 at 2; Pet. Ex. 3 at 7. He had a past medical history of depression and anxiety, prostate cancer (in remission), hypertension, mixed hyperlipidemia, bladder calculus, and iron deficiency anemia. Pet. Ex. 3 at 22-45; Pet. Ex. 26.

On January 26, 2016, more than three months post-vaccination, Petitioner presented to his primary care provider ("PCP"), Sukhwinder Kodial, M.D., for depression, panic anxiety syndrome, hypertension, and anemia. Pet. Ex. 3 at 22. Review of systems noted "t[i]n[g]ling in [his] hand[s] and feet with stress, no nocturnal symptoms, no weakness in [the] [upper extremities] and [lower extremities]." Id. at 25-26. Petitioner's neurological examination was normal, including his sensory examination and his balance and gait. Id. at 26.

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[23] Ernest R. Vina et al., Chronic Inflammatory Demyelinating Polyneuropathy in Patients with Systemic Lupus Erythematosus: Prognosis and Outcome, 35 Seminars Arthritis & Rheumatism 175 (2005). This article was also cited as Pet. Ex. 23-f and Resp. Ex. C, Tab 6.

[24] Eyal Muscal & Robin L. Brey, Neurologic Manifestations of Systemic Lupus Erythematosus in Children and Adults, 28 Neurologic Clinics 61 (2010).

[25] Hrudya Abraham et al., Chronic Inflammatory Demyelinating Polyneuropathy (CIDP): An Uncommon Manifestation of Systemic Lupus Erythematosus (SLE), 18 Am. J. Case Reps. 980 (2017).

[26] R. Jasmin et al., Successful Treatment of Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) in Systemic Lupus Erythematosus (SLE) with Oral Cyclophosphamide, 21 Lupus 1119 (2012).

[27] This summary of medical records is taken from Respondent's Response, as the undersigned finds Respondent provided an accurate representation of the records. See Resp. Response at 8-21. The summary has been edited by the undersigned.

[28] Petitioner had previously received flu vaccines on January 14, 2013 and September 8, 2013 without apparent adverse effects. Pet. Ex. 3 at 7, 31.

On January 28, 2016, Petitioner presented to his urologist regarding his history of prostate cancer. Pet. Ex. 10 at 16. No neurological symptoms were reported or noted. Id.

On March 1, 2016, Petitioner followed up with Dr. Kodial regarding depression, anxiety, anemia, hyperlipidemia, and hypertension. Pet. Ex. 3 at 15. No neurological symptoms were reported or noted, and Petitioner's physical examination was normal. Id. at 15-21.

On May 19, 2016, Petitioner returned to Dr. Kodial for depression with anxiety, anemia, mixed hyperlipidemia, as well as "[n]ew [o]nset" neuropathy. Pet. Ex. 3 at 9. Petitioner reported a two-month history of "balance disorder," but denied numbness in his hands and feet, headache, weakness, and vision problems. Id. at 10. Review of systems was positive for gait disturbance and difficulty falling sleep. Id. at 12. On examination, Petitioner had neurological sensory deficits, including "decreased position sense" in his lower extremities, and ataxia. Id. at 13. At this visit, Petitioner was noted to have uncontrolled iron deficiency amenia, and he was to continue taking an iron supplement. Id. at 9, 12-13. Dr. Kodial referred Petitioner to a neurologist. Id. at 9, 14. Bloodwork performed that day revealed an elevated erythrocyte sedimentation rate ("ESR")[29] of 39 (normal range 0-12). Pet. Ex. 25 at 7.

Petitioner presented to neurologist Bennett Myers, M.D. at DENT Neurologic Institute ("DENT"), on June 24, 2016, for "tingling in [his] hands and feet." Pet. Ex. 4 at 14. Petitioner reported that "in January of this year [he] developed tingling in his feet that he would notice with standing," with no antecedent illness or trauma. Id. He further reported imbalance in the shower when his eyes were closed, but that the symptoms were "tolerable." Id. Petitioner indicated that in April 2016, he "developed [a] tingling sensation in his hands that was constant" and "imbalance with walking." Id. He reported that around April 2016, he had a mildly elevated ESR of 39, he was prescribed prednisone 20 mg per day, and his symptoms resolved after two days.[30] Id. However, two weeks after he finished his prescription for prednisone (the day of this neurology visit), his symptoms returned. Id.

Past medical history was significant for onset of paresthesias in the feet in January 2016, and in the hands in April 2016, with associated imbalance. Pet. Ex. 4 at 14. On physical examination, Petitioner had normal strength measuring 5/5 bilaterally in the upper and lower

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[29] Erythrocyte sedimentation rate, or ESR, is "the rate at which erythrocytes precipitate out from a well-mixed specimen of venous blood . . . ; an increase in rate is usually due to elevated levels of plasma proteins, especially fibrinogen and immunoglobulins . . . . It is increased in monoclonal gammopathy, hypergammaglobulinemia due to inflammatory disease, hyperfibrinogenemia, active inflammatory disease, and anemia." Erythrocyte Sedimentation Rate, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=102146 (last visited Sept. 27, 2024).

[30] Petitioner's medication list with Dr. Kodial on July 5, 2016 indicated that Petitioner had started prednisone on May 24 and June 3, 2016, but it is not clear who prescribed the medication or what prompted the prescription. Pet. Ex. 3 at 4. Petitioner's Walgreens records indicate only that Petitioner was being prescribed prednisone by Dr. Myers's office beginning June 24, 2016. Pet. Ex. 2 at 3-4.

extremities, absent reflexes throughout, a moderate wide-based gait with mild imbalance, a positive Romberg test, the ability to toe walk but inability to tandem walk, and normal sensation except absent vibration in his left toes and right knee. Id. at 16. Assessment was polyneuropathy. Id. Dr. Myers opined that Petitioner's history and examination were "strongly suggestive of a polyneuropathy" with a "possible inflammatory etiology" given his "[d]ramatic response to [p]rednisone." Id. Dr. Myers ordered an electromyography ("EMG")/nerve conduction study ("NCS") to evaluate for demyelination and conduction block and bloodwork to evaluate for an inflammatory disorder, paraproteinemia, and vitamin deficiency. Id. He noted that if Petitioner's EMG showed "clear demyelination or conduction block," he would likely resume prednisone daily "for a presumptive diagnosis of CIDP." Id. Petitioner's bloodwork that day revealed an elevated ANA of 1:1280 (normal range < 1:40),[31] elevated dsDNA of 183 (normal range 0-99), positive anti-Smith antibodies, and elevated ESR of 42. Pet. Ex. 25 at 6-7.

An EMG performed the same day, June 24, on Petitioner's right arm and leg revealed "complex abnormalities with unusual features . . . [confined] to the upper limb sparing legs." Pet. Ex. 4 at 18-19. The study was "[m]ost consistent with an acute/subacute non-length dependent motor predominant peripheral polyneuropathy with demyelination, only affecting the upper limb," and a right ulnar neuropathy at the elbow. Id. at 19. An EMG/NCS of Petitioner's right leg was "virtually normal" with "no EMG evidence to suggest a length dependent peripheral polyneuropathy or a right lumbosacral radiculopathy." Id.

On July 5, 2016, Petitioner presented to Dr. Kodial for an annual examination. Pet. Ex. 3 at 1. Dr. Kodial noted that Petitioner's neuropathy was stable on prednisone. Id.

On July 20, 2016, Petitioner returned to DENT and saw Amelia Smith, certified physician assistant ("PA-C"), for a follow-up examination. Pet. Ex. 4 at 11. PA-C Smith noted Petitioner's recent EMG and elevated ANA, dsDNA, and ESR. Id. Petitioner reported significant improvement since starting prednisone 20 mg daily, with his foot paresthesias completely resolved, and only mild paresthesias of the finger tips. Id. His balance had also improved, but he still felt unsteady at times and had some difficulty with fine motor skills. Id. He was working to schedule a rheumatology appointment. Id. Petitioner's physical examination revealed 5/5 strength bilaterally in all extremities, absent reflexes, negative Romberg test, intact vibration sense, a steady gait with a normal pace, and moderate imbalance on tandem walk. Id. at 13. PA-C Smith diagnosed Petitioner with polyneuropathy, with findings "indicative of an underlying inflammatory polyneuropathy" and bloodwork "indicative of an underlying rheumatologic disorder." Id. Petitioner's case was discussed with Dr. Myers, who wanted to keep Petitioner on daily prednisone and "treat him as presumed CIDP until he [saw] rheumatology." Id.

[31] This was noted to be a pattern found in patients with SLE. Pet. Ex. 25 at 35.

On July 28, 2016, Petitioner followed up with urology. Pet. Ex. 10 at 11-14. On the patient information form, Petitioner wrote "Flu vaccine CIDP" under allergies. Id. at 14. Petitioner reported that he had recently been diagnosed with CIDP.[32] Id. at 11.

At a September 6, 2016 follow-up with PA-C Smith at DENT, Petitioner reported that his foot paresthesias had resolved and his balance and mobility had improved. Pet. Ex. 4 at 8. His finger paresthesias, however, had increased and was now constant but "manageable." Id. He was taking prednisone 20 mg daily and had gained eight pounds. Id. Petitioner still had not seen a rheumatologist. Id. His examination remained mostly unchanged from July 20, 2016, except for only mild, as opposed to moderate, imbalance with tandem walking. Id. at 9. Assessment remained polyneuropathy. Id. at 10. PA-C Smith stated "[they] discussed that [Petitioner] does not have a clear-cut case of CIDP but [they would] be treating it as such based on symptoms and findings on EMG." Id. PA-C Smith noted Petitioner "is likely suffering from an underlying inflammatory polyneuropathy given the abnormalities on his rheumatologic testing." Id. Dr. Myers recommended decreasing Petitioner's prednisone dose to 15 mg and a trial of IVIG for four days. Id.

At a follow-up appointment on November 9, 2016 with Dr. Myers at DENT, Petitioner reported some improvement since September. Pet. Ex. 4 at 4. Petitioner still had paresthesias in his hands "most though not all of the time," but not in his feet. Id. He was taking prednisone 15 mg per day but had not had IVIG because his insurance company did not approve it. Id. at 4, 6. Physical examination now revealed 1+ patellar, biceps, triceps, and brachioradialis reflexes and absent ankle jerks. Id. at 6. Dr. Myers noted Petitioner had improved and assessment was inflammatory polyneuropathy responsive to prednisone. Id. Given his improvement, Dr. Myers decided to hold off appealing the insurance decision on IVIG and decreased Petitioner's prednisone dosage to 10 mg daily. Id.

On December 5, 2016, Petitioner presented to rheumatologist Harbrinder Sandhu, M.D. Pet. Ex. 11 at 10-13. Petitioner reported that in December 2015, "he started to have tingling in his feet subsequently going into his hands, [and] the symptoms got so bad that he was having [a] hard time doing his [activities of daily living,] he was getting balance problems, [and] he [was] getting weakness in his hands and feet." Id. at 10. Dr. Sandhu diagnosed Petitioner with SLE using the SLICC criteria, noting his positive ANA, positive Smith antibody, positive dsDNA, and peripheral neuropathy. Id. at 12. Dr. Sandhu noted that "[they] discussed that in [Petitioner's] case[,] the peripheral neuropathy is a presenting symptom." Id. He further noted that Petitioner also had alopecia but questioned whether it was due to medication. Id. Dr. Sandhu recommended that Petitioner start hydroxychloroquine (Plaquenil)[33] and see an ophthalmologist. Id. Dr. Sandhu indicated he would work with Dr. Myers to decrease

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[32] Petitioner continued to follow up with urology over the years, where he continued to report that he had been diagnosed with polyneuropathy or CIDP, was taking prednisone, and list the flu vaccine as an allergy. See Pet. Ex. 10 at 4-10; Pet. Ex. 36.

[33] Plaquenil is administered to treat symptoms associated with lupus/SLE. Hydroxychloroquine (Plaquenil): Benefits, Side Effects, and Dosing, Lupus Found. Am., https://www.lupus.org/resources/drug-spotlight-on-hydroxychloroquine (last visited Sept. 27, 2024).

Petitioner's prednisone dosage, but if Petitioner's symptoms continued to relapse, they would consider treatment with Imuran,[34] CellCept,[35] or possibly IVIG. Id. Given Petitioner's history of prostate cancer, Dr. Sandhu wanted to avoid immunosuppressive therapy and try IVIG first. Id. Bloodwork drawn at this appointment again revealed a positive ANA of 1:2560[36] and a high positive dsDNA antibody of 193 unit/mL (normal range 0-99), but negative Smith antibody. Id. at 3-4, 16.

Petitioner returned to Dr. Sandhu on December 19, 2016. Pet. Ex. 11 at 14. He reported numbness and tingling in his hands and feet off and on, but stated that he was "markedly improved from before" and his only side effect of prednisone was weight gain. Id. Petitioner wanted to hold off on seeing an ophthalmologist and did not want to start Plaquenil. Id. at 15. Assessment remained SLE. Id.

On February 8, 2017, Petitioner returned to Dr. Myers. Pet. Ex. 4 at 1. Petitioner reported feeling overall "a little worse," which he attributed to lowering his prednisone dose. Id. The tingling in his hands was more prominent, but it had not recurred in his feet. Id. Petitioner indicated that a rheumatologist had raised the question of SLE, with Plaquenil suggested as a treatment option. Id. Petitioner's physical examination revealed decreased finger extension strength and absent reflexes. Id. at 3. Dr. Myers noted that "[b]ased on the ongoing symptoms with some recent worsening, I have diagnosed him with CIDP." Id. He again recommended IVIG, but noted that Petitioner would need to be cleared by his PCP due to shortness of breath with exertion before beginning IVIG. Id. Dr. Myers recommended holding off on Plaquenil at that time because Petitioner did not have "typical symptoms of [SLE]," despite his "markedly positive ANA." Id. Petitioner was directed to maintain his current prednisone dose, with an attempt to wean if they could adequately control Petitioner's symptoms. Id.

Petitioner presented to a cardiologist on February 14, 2017, regarding "shortness of breath with moderate exertion" for "the last few months." Pet. Ex. 5 at 3. He was noted to have a history of peripheral neuropathy with numbness and tingling in his arms and legs. Id. A stress echocardiogram revealed "[p]oor functional capacity" and "[m]oderate to severe pulmonary hypertension with an estimated [pulmonary artery] systolic pressure of 70 mmHg." Id. at 1-2. The cardiologist noted that a referral to a pulmonologist might be warranted. Id. at 4.

On April 25, 2017, Petitioner presented to pulmonologist William Gibbons, M.D., regarding shortness of breath during cold weather and exertion. Pet. Ex. 8 at 12. Petitioner

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[34] Imuran (azathioprine) is a treatment used in a number of autoimmune disorders. Azathioprine, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=5231 (last visited Sept. 27, 2024).

[35] CellCept (mycophenolate mofetil) is "an immunosuppressive agent used in conjunction with cyclosporine and corticosteroids to prevent rejection of allogeneic renal, hepatic, and cardiac transplants." Mycophenolate Mofetil, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=32633 (last visited Sept. 27, 2024).

[36] Again, this was noted to be a pattern found in patients with SLE. Pet. Ex. 25 at 18.

"report[ed] a history of demyelinating polyneuropathy . . . diagnosed in early 2016" and that "he [was] being evaluated for [SLE]." Id. Petitioner was diagnosed with seasonal exertional dyspnea with possible asthma. Id. at 17. Pulmonary functions tests were ordered, and Petitioner was prescribed an inhaler. Id. Dr. Gibbons noted that Petitioner had a history of positive ANA and dsDNA antibodies and demyelinating polyneuropathy, was on prednisone, and had "several features of SLE." Id. Dr. Gibbons noted Petitioner was "an excellent candidate for an annual flu vaccine" for health maintenance.[37] Id. Later that day, Petitioner saw Dr. Kodial regarding his chronic conditions. Id. at 19-20. Dr. Kodial documented that Petitioner had stable CIDP and was taking prednisone. Id. Petitioner reported neuropathy symptoms in feet were improved, but he still had symptoms in his hands. Id. at 20.

On May 10, 2017, Petitioner returned to DENT and saw Tanya Geist, RPA-C. Pet. Ex. 7 at 1. He was taking prednisone 10 mg daily and continued to have tingling of the fourth and fifth digits of each hand and ongoing weakness in his left hand. Id. Petitioner also reported shortness of breath with exertion. Id. RPA-C Geist noted that based on Petitioner's positive ANA, Smith antibody, and dsDNA and peripheral neuropathy, "he was diagnosed with [SLE] with the presenting symptom of peripheral neuropathy." Id. Plaquenil was again recommended but declined. Id. The plan was to avoid immunosuppressive therapy due to his history of prostate cancer. Id. RPA-C Geist noted that pulmonary function tests and a repeat echocardiogram were scheduled. Id. On examination, Petitioner's reflexes remained absent, and he had slight finger weakness (4+/5 ulnar innervated intrinsics). Id. at 3. Assessment was CIDP and SLE, and the plan was to continue prednisone with the intent to begin IVIG pending pulmonary clearance. Id.

Petitioner followed up with rheumatologist Dr. Sandhu on May 16, 2017. Pet. Ex. 8 at 6. Petitioner reported marked improvement with the numbness and tingling in his hands and feet on prednisone despite side effects of weight gain and cushingoid facial appearance. Id. Assessment remained SLE. Id. at 7. Petitioner again declined Plaquenil. Id.

On August 1, 2017, Petitioner followed up with Dr. Gibbons, reporting improvement of his dyspnea with albuterol. Pet. Ex. 8 at 2. He was still taking prednisone 10 mg daily for his "polyneuropathy demyelinating disease." Id. Dr. Gibbons continued to prescribe an inhaler and noted that Petitioner likely had some non-atopic asthma. Id. at 4. He also reiterated that Petitioner "has several features of SLE." Id. Dr. Gibbons recommended that Petitioner receive a flu vaccine that autumn. Id.

On August 18, 2017, Petitioner followed up with Dr. Sandhu for "[SLE]-induced peripheral neuropathy." Pet. Ex. 11 at 20. Petitioner complained of shortness of breath and tingling in fingers, with pain at 1/10. Id. He had marked improvement of numbness and tingling in feet. Id. Assessment remained SLE. Id. at 21. Additional blood work was ordered and revealed a negative Smith antibody and high positive dsDNA antibody of 318 unit/mL (normal range 0-99). Id. at 3, 21.

[37] It does not appear that Petitioner received the flu vaccine at that appointment, although it was recommended.

Petitioner presented to a gastroenterologist on September 8, 2017 for evaluation of anemia. Pet. Ex. 30 at 3-6. Petitioner reported that he had been on iron supplements for four years, but he had cut back, after which his bloodwork showed changes requiring him to restart iron two months earlier. Id. at 3. The gastroenterologist noted Petitioner had microcytic anemia with normal iron levels. Id. at 5. He was unsure of the source of Petitioner's anemia and recommended a colonoscopy and upper endoscopy to rule out atrophic gastritis. Id. The procedures did not reveal the source of the anemia. Id. at 7-10, 12-19.

Petitioner received four days of IVIG beginning October 9, 2017. Pet. Ex. 44 at 1-4. On November 8, 2017, Petitioner followed up with RPA-C Geist. Id. at 6. Petitioner had previously contacted the office on September 25 to report "worsening tingling of the hands [and] tingling of the feet extending up to the knees with imbalance." Id. At this visit, he reported "his symptoms have gotten more intense over the last month," he fell once due to his imbalance, and his weakness in his hands and arms had continued. Id. Petitioner indicated the exacerbation of his symptoms began following his gastroenterology procedures. Id. RPA-C Geist noted that Petitioner was being followed by Dr. Sandhu for SLE and was undergoing additional hematology testing. Id. Physical examination revealed mildly decreased strength in upper extremities and absent reflexes. Id. at 8. Petitioner ambulated with a moderately wide-based gait and a mildly unsteady gait pattern. Id. Assessment remained CIDP and SLE. Id. Petitioner had no clear improvement after his first round of IVIG. Id. An updated EMG was recommended. Id.

A repeat EMG was done on November 17, 2017 and revealed "a definite EMG deterioration" in his right upper extremity since his June 2016 EMG. Pet. Ex. 24 at 1-3. Petitioner's median sensory amplitude was now absent, and he now had no ulnar sensory response. Id. at 3. The interpreting neurologist noted deterioration in the right upper extremity with a "a markedly increased percentage of polyphasic motor units in virtually all muscles of the right upper limb, interestingly sparing the deltoid muscle." Id.

Petitioner again received IVIG on November 9 and 10, 2017. Pet. Ex. 44 at 10-11.

On November 20, 2017, Petitioner arrived at the hospital by ambulance with a right ankle trimalleolar fracture. Pet. Ex. 31 at 10-12, 14-17. Petitioner reported that he walked with a cane and had unstable balance lately due to his neuropathy. Pet. Ex. 9 at 5. He underwent ankle surgery and was discharged to the rehabilitation unit on November 24, 2017, where he remained until December 7, 2017. Id. at 6, 281. At the rehabilitation unit, his chief complaint was "chronic bilateral hand numbness and tingling." Id. at 282. He "report[ed] [a] CIDP diagnosis approximately [one] year ago, etiology suspected flu shot." Id. at 281. Petitioner advised that he was transitioned from prednisone to IVIG "once symptoms began involving his hands." Id. at 281-82. He further stated that his symptoms were "slightly better since starting IVIG in October." Id. at 285. Petitioner received additional IVIG on December 8 and 9, 2017. Pet. Ex. 44 at 12-13.

Following his discharge, Petitioner received physical therapy ("PT") and occupational therapy ("OT") at home between December 10, 2017 and January 2, 2018. Pet. Ex. 32 at 193-301. On January 3, 2018, PA-C Geist noted that Petitioner had experienced clinical deterioration due to his recent fall. Pet. Ex. 44 at 9. Petitioner's "[v]isting home physical therapist

confirm[ed] [Petitioner's] clinical deterioration, with no improvement following IVIG infusions, overall decline in ability to walk, decline in self-care." Id. She noted that insurance had declined Rituxan,[38] and he was advised to go to the emergency department. Id. Petitioner was admitted to the hospital from January 3 to 8, 2018, due to increasing upper extremity weakness and paresthesia and an inability to walk due to his recent ankle fracture. Pet. Ex. 40 at 56. Petitioner reported "he ha[d] not noticed much improvement from the IVIG." Id. Petitioner received a five-day course of IVIG. Id. He was transferred to the medical rehabilitation unit on January 8 where he remained until January 26, 2018. Id. at 1180. He received intensive PT and OT during this time. Id. at 1181. He was also noted to have chronic anemia with normal iron levels but low hemoglobin levels. Id. at 1181-82.

Between January 26 and February 22, 2018, Petitioner was admitted to a skilled nursing facility. See Pet. Ex. 38. Dr. Myers saw Petitioner during his admission on February 9 and recommended that Petitioner stop IVIG, continue prednisone, and start Rituxan as soon as possible. Id. at 87. Beginning on February 11, 2018, Petitioner was noted to have yellowing eyes, followed by yellowing of his face. Id. at 39, 102-03. On February 22, he complained of right-sided abdominal pain. Id. at 3. He was sent to the emergency room for further evaluation. Id.

Petitioner was re-admitted to the hospital from February 23 to 28, 2018. Pet. Ex. 35 at 64. The consulting hematologist, Farid Berenji, M.D., noted that "[d]ue to the significant elevation in bilirubin and dropping hemoglobin, we have been asked to . . . rule out hemolytic anemia. . . . . [O]n [February 14,] a direct Coombs was positive.[39] [Petitioner reported] . . . episodes of hemolytic anemia in the past."[40] Id. at 102-06; Pet. Ex. 33 at 1-7. Dr. Berenji increased Petitioner's prednisone dose to 60 mg and recommended blood transfusions as needed and Rituxan if Petitioner did not respond to prednisone. Pet. Ex. 33 at 4; Pet. Ex. 35 at 106. Dr. Berenji noted that the working diagnosis was immune hemolytic anemia. Pet. Ex. 33 at 1; Pet. Ex. 35 at 103. Petitioner had his first Rituxan infusion on March 7, 2018. Pet. Ex. 44 at 29.

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[38] "Rituxan (rituximab) is a cancer medication used in combination with other cancer medicines to treat non-Hodgkin's lymphoma. Rituxan is also used in combination with another drug called methotrexate to treat symptoms of adult rheumatoid arthritis." Rituxan, RxList, https://www.rxlist.com/rituxan-drug.htm (last updated Dec. 16, 2021).

[39] A Coombs test, also called an antiglobulin test, is "a test for the presence of nonagglutinating antibodies against red blood cells, using antihuman globulin antibody to agglutinate cells coated with the nonagglutinating antibody. The direct antiglobulin test detects antibodies bound to circulating red cells in vivo. It is used in the evaluation of autoimmune and drug-induced immune hemolytic anemia and erythroblastosis fetalis." Antiglobulin Test, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=112414 (last visited Sept. 27, 2024).

[40] It is not clear when Petitioner had hemolytic anemia prior to this admission. Previous records refer to iron deficiency anemia. See, e.g., Pet. Ex. 3 at 21-22.

On March 9, 2018, Petitioner presented to neurologist Nicholas Silvestri, M.D. Pet. Ex. 46 at 1-7. At that visit, Petitioner reported he received a flu vaccine in October 2015 and "[b]y Thanksgiving of that year he developed intense tingling in his hands and feet which progressively worsened." Id. at 1. He reported having been diagnosed with CIDP following an EMG/NCS in June 2016 and trying prednisone, IVIG, and Rituxan. Id. Dr. Silvestri indicated the increase in prednisone was to treat Petitioner's autoimmune hemolytic anemia ("AIHA").[41] Id. at 5. He opined that Petitioner's current symptoms, "particularly weakness[,] [were] likely due to deconditioning from his recent injury and prolonged hospitalization." Id. Dr. Silvestri advised Petitioner to continue Rituxan infusions and taper his prednisone dose at his hematologist's recommendation. Id. at 5. Petitioner followed up with Dr. Berenji on March 15, 2018. Pet. Ex. 33 at 21-23. Dr. Berenji noted that "the likely explanation for his anemia is immune hemolytic anemia," and that Rituxan "[was] also indicated for management of refractory [AIHA]." Id. at 21-23.

Petitioner received three additional Rituxan infusions in March 2018 and a blood transfusion in April 2018. Pet. Ex. 44 at 30-32; Pet. Ex. 33 at 26. He continued to follow up with Dr. Silvestri for his neuropathy and Dr. Berenji for his AIHA, he continued taking prednisone at varying dosages adjusted by his doctors depending on his symptoms and bloodwork, and he took gabapentin. See Pet. Ex. 33 at 29-249; Pet. Ex. 34 at 1-51; Pet. Ex. 46 at 8-25; Pet. Ex. 48 at 3-42.

In July 2018, Petitioner returned to rheumatology, where his diagnosis remained SLE "with autoimmune anemia and peripheral neuropathy with history of [CIDP]." Pet. Ex. 29 at 4-8. He was advised to continue Rituxan, as it "appear[ed] to be addressing his [CIDP], autoimmune anemia[,] and lupus." Id. at 7. In August 2018, Dr. Berenji recommended that Petitioner try low-dose Cytoxan[42] because he did not seem to be responding to Rituxan and tapering doses of prednisone, but Petitioner could not tolerate Cytoxan due to diarrhea. Pet. Ex. 33 at 43, 53.

On October 15, 2018, Petitioner presented for the first time to rheumatologist Amar Oza, M.D. Pet. Ex. 45 at 24-31. Dr. Oza's assessment was SLE. Id. at 30. He wrote, "Evaluating

[41] Autoimmune hemolytic anemia, or AIHA, is "any of a large group of anemias involving autoantibodies against red cell antigens. Those due to warm-reactive antibodies, . . . may be idiopathic or secondary to autoimmune diseases, hematologic neoplasms, viral infections, or immunodeficiency diseases, ... Those due to cold-reactive antibodies, . . . include cold agglutinin syndrome and paroxysmal cold hemoglobinuria . . . ." Autoimmune Hemolytic Anemia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56118 (last visited Sept. 27, 2024).

[42] Cytoxan (cyclophosphamide) is "a cytotoxic alkylating agent of the nitrogen mustard group, used as an antineoplastic, often in combination with other agents, for a wide variety of conditions" and is "also used as an immunosuppressive agent to prevent transplant rejection and in the treatment of certain diseases with abnormal immune function." Cyclophosphamide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=12166 (last visited Sept. 27, 2024).

[Petitioner] for the first time today, I suspect that SLE is likely involved in both his neurologic and hematologic disease." Id. He further noted that Petitioner "had blood work showing positive ANA and high titer positive dsDNA, which continue[d] to be positive despite treatment. With a report also of a prior Smith antibody positivity (not found on subsequent blood work), the latter [two] are quite specific for the presence of SLE." Id. He noted that while Petitioner "d[id] not have the classical clinical features of photosensitivity, facial rashes, mucositis, or kidney disease, the presence of his neurologic symptoms and even potentially serositis (with his pleural effusion) would be enough to meet both the traditional and 2012 SLICC classification criteria for [SLE]," including "immunologic (ANA, dsDNA, [S]mith), neurologic (CIDP), serositis (pleuritis), and hematologic (AIHA)." Id. Dr. Oza further documented that "[Petitioner] fe[lt] that all of his symptoms were subsequent to a flu vaccine that he took in 2015. Whether his neurologic/hematologic symptoms were secondary to the flu shot or secondary to [SLE], there is no evidence [he] had any symptoms or other reasons to suspect [SLE] before his flu shot." Id. Dr. Oza recommended Petitioner take Plaquenil for his SLE, so that his other medications might be tapered or reduced in frequency. Id.

In November 2018, Petitioner experienced an acute drop in platelets and was diagnosed with immune thrombocytopenic purpura. Pet. Ex. 33 at 79-82. Dr. Berenji recommended two days of IVIG and four weekly doses of Rituxan. Id. at 81. By January 2019, Petitioner's platelet count had returned to normal. Id. at 103-06.

In July 2019, Petitioner broke his left hip when he had a nightmare that he was falling, rolled in his sleep, awoke mid-fall, and hit the ground. Pet. Ex. 51-1 at 139-42, 181-85. Dr. Silvestri noted in November 2019 that Petitioner did not have a CIDP relapse after his surgical hip repair, "which suggest[ed] that it [was] [] under very good control." Pet. Ex. 48 at 3-7.

In May 2021, Dr. Silvestri noted that Petitioner had received two doses of the Covid vaccine without incident. Pet. Ex. 34 at 32.

Petitioner continued to follow up with Dr. Berenji (hematology) every one to two months and Dr. Silvestri (neurology) every six months in 2022 and 2023. See Pet. Exs. 52, 54. Between November 2022 and January 2023, Petitioner's hemoglobin levels dropped, and he completed four weekly doses of Rituxan, after which his numbers improved. Pet. Ex. 54 at 26-65.

No additional medical records are relevant.

2. Letter from Dr. Amar Oza

Rheumatologist, Dr. Oza, wrote a letter[43] stating Petitioner has "[CIDP], [AIHA], and [SLE], for which he is following with [rheumatology]." Pet. Ex. 19 at 1. Dr. Oza stated "[w]ith multiple bloodwork findings and his other conditions, it is my opinion that [SLE] is playing a role in his overall condition. It is my opinion that [Petitioner] did not have symptoms or signs of [SLE] before [] his flu vaccine in October of 2015." Id.

[43] This letter was not dated.

3. Affidavits

a. Petitioner

In Petitioner's first affidavit, he averred that approximately one month following his flu vaccine on October 14, 2015, he "began feeling tingling in [his] feet" that spread to his knees and led to difficulty walking. Pet. Ex. 1 at ¶¶ 5, 7. The tingling in his feet and imbalance led to falls in November 2015 and January 2016. Id. at ¶¶ 8-9. Thereafter, he presented to Dr. Kodial on January 26, 2016, for difficulty walking and tingling and numbness in his hands, knees, and feet, as well as fatigue, anxiety, and depression. Id. at ¶ 10. He experienced another fall in March 2016. Id. at ¶ 11. In April 2016, Petitioner's "tingling in [his] hands became constant" and he continued to have "severe imbalance with walking." Id. at ¶ 13. Thereafter, Dr. Kodial prescribed Petitioner prednisone and referred him to a neurologist at DENT. Id. at ¶¶ 14-15. Petitioner averred the prednisone relieved his symptoms of numbness and tingling in his hands and feet and returned when he stopped taking prednisone. Id. at ¶ 16.

In June 2016, Petitioner presented to neurologist Dr. Myers, who "concluded that [he] was likely suffering from polyneuropathy." Pet. Ex. 1 at ¶ 17. Dr. Myers observed Petitioner had absent reflexes, a moderate wide-based gait, and mild imbalance, and Petitioner was unable to tiptoe or walk with one foot in front of the other. Id. at ¶ 20. Based on his symptoms and June 2016 EMG, Petitioner averred that he has been treated for his CIDP at DENT. Id. at ¶¶ 21-26.

Petitioner began experiencing shortness of breath, which he asserted was "a symptom of CIDP," in the end of 2016. Pet. Ex. 1 at ¶ 27. Petitioner noted his testing with a cardiologist "confirm[ed] that [his] shortness of breath was an effect of the vaccine induced CIDP rather than an unidentified heart problem."[44] Id. at ¶¶ 28-30.

In February 2017, Petitioner's pain in his feet and ankles turned into "compete numbness," which progressed to his mid-calf by April 2017. Pet. Ex. 1 at ¶ 43.

As of April 12, 2017, the date on which Petitioner executed his first affidavit, Petitioner averred that he suffered from "symptoms of CIDP includ[ing] numbness, tingling, weakness, pain, loss of sensation, trouble walking, trouble with bowels or bladder, loss of reflexes, fatigue[,] and atrophy of muscles." Pet. Ex. 1 at ¶ 32. He also described "limited grip strength," numbness and stiffness that restricted fine motor skills in his fingers, constant pins and needles in his hands, and pain or dysesthesia in his lower extremities from his toes to knees that affected his sleep. Id. at ¶¶ 40-42, 44. Petitioner was also suffering from depression, anxiety, and weight gain. Id. at ¶¶ 33-34.

[44] Petitioner's cardiology records do not attribute his shortness of breath to his flu vaccination or to CIDP. See Pet. Ex. 5 at 4 (assessing Petitioner with "shortness of breath [that] may be pulmonary in origin").

Petitioner's second affidavit, executed on October 3, 2022, indicated that he did not see a pulmonologist after August 1, 2017 and he did not see a rheumatologist between August 18, 2017 and July 18, 2018 or after February 18, 2019.[45] Pet. Ex. 55 at ¶¶ 1-2.

In his third affidavit, executed on June 20, 2023, Petitioner noted he experienced "an increase in tingling throughout [his] body" in the beginning of 2023. Pet. Ex. 56 at ¶ 5. He increased his gabapentin dosage on recommendation of Dr. Silvestri, and due to this, he developed swelling in his left arm and feet. Id. at ¶¶ 6-7. Petitioner added that "[d]ue to [his] [CIDP], [he] ha[s] been experiencing a decrease in [his] hemoglobin levels, a byproduct of hemolytic anemia." Id. at ¶ 9. And "[d]ue to the substantial decrease in hemoglobin levels," he was given Rituxan infusions in January 2023. Id. at ¶ 10.

b. Marianne Izard

Marianne Izard is the wife of Petitioner. Pet. Ex. 12 at ¶ 3. Mrs. Izard stated Petitioner was "generally healthy, strong and athletic, and able to do most ordinary tasks and activities in an above average manner" throughout their relationship. Id. at ¶ 4. She explained that within three to four weeks of the October 2015 flu vaccination, Petitioner "started to feel numbness and tingling in his legs but had no reason to believe it was related to the vaccine." Id. at ¶¶ 6-7.

On Thanksgiving 2015, Mrs. Izard averred that Petitioner fell. Pet. Ex. 12 at ¶ 8. Mrs. Izard did not see the fall, but she heard it. Id. Petitioner told her "he felt numbness and tingling in his legs and his knees buckled and he just collapsed." Id. Petitioner further told her "he ha[d] been feeling numbness and tingling for a bit but did not want to bother [her] because [she] was being treated for problems with [her] knees." Id. She stated "[Petitioner] was hesitant to address his own issues because he was caring for [her] and transporting [her] around because of [her] knee problems." Id.

Mrs. Izard asserted that Petitioner fell again in January 2016 due to "the same sensations of tingling and numbness." Pet. Ex. 12 at ¶ 9. She also reported a third fall prior to Petitioner being seen at DENT. Id. at 10.

As of June 13, 2018, the date in which Mrs. Izard executed her affidavit, Petitioner's condition is "far worse," although here was "some improvement" with treatment. Pet. Ex. 12 at ¶ 11.

c. Joseph Welsh

Joseph Welsh is a family friend of Petitioner. Pet. Ex. 13 at ¶ 3. He stated that on Thanksgiving of 2015, he thought Petitioner "did not seem like himself." Id. at ¶ 4. He thought Petitioner "seemed off" and "appeared weaker than normal like when someone has a cold or just

[45] But see Pet. Ex. 56 at ¶ 3 (Petitioner's third affidavit stating he "ha[s] continued to treat with [his] rheumatologist after October 1, 2022"). Petitioner did not file updated rheumatology records although some remained outstanding and Petitioner confirmed medical records and the record were "complete." Pet. Status Rept., filed Aug. 17, 2023 (ECF No. 147).

feels down." Id. Petitioner told Mr. Welsh that he has been feeling weak and lethargic. Id. Mr. Welsh also noted that Petitioner fell that night, although he did not witness the fall. Id. Petitioner stated "he was in pain, and felt numbness and tingling in his legs before they 'just went out.'" Id. Mr. Welsh averred on June 14, 2018 that Petitioner has not been himself "[s]ince the 2015 Thanksgiving fall." Id. at ¶ 5. He felt that "because [he] see[s] [Petitioner] intermittently, [he is] able to recognize a [] decline in [Petitioner's] health as opposed to his immediate family who see[] him every day." Id.

d. Dr. Nicholas Silvestri

Dr. Silvestri executed an affidavit on March 11, 2019. Pet. Ex. 18 at 3. Dr. Silvestri is a board-certified neurologist with over ten years of clinical experience in neurology and neuromuscular medicine. Id. at ¶ 1.

Dr. Silvestri began treating Petitioner on March 9, 2018, two and one-half years after Petitioner received the flu shot at issue here. Pet. Ex. 18 at ¶ 3. His office has continued to see Petitioner at least once every three months since March 2018. Id. On initial examination, Dr. Silvestri diagnosed Petitioner with CIDP "in correlation with his reported symptoms of weakness and tingling in the arms and legs, absent deep tendon reflexes[,] and unsteady gait." Id. at ¶ 4. Since then, he has treated Petitioner with steroids (prednisone) and Rituximab infusions for CIDP, physical therapy, and gabapentin for tingling. Id. at ¶ 5.

"As reported by [Petitioner], and as documented in [Dr. Silvestri's] records, [Petitioner] began experiencing symptoms of CIDP in November of 2015," and not prior to the flu vaccine at issue. Pet. Ex. 18 at ¶ 6. On or around Petitioner's first visit with Dr. Silvestri on March 9, 2018, Dr. Silvestri learned of "a concurrent diagnosis of [SLE] by his rheumatologist, Dr. Sandhu." Id. at ¶ 7. Dr. Silvestri wrote "[i]t [was] [his] understanding that [Petitioner] was first evaluated by Dr. Sandhu upon [] recommendation of [] prior neurologist, Dr. M[]yers . . . , on or about December 5, 2016, on the basis of abnormal lab work, and the same symptoms suggestive of CIDP," including "bilateral tingling and weakness in the hands and feet as well as balance issues, which he began experiencing in November of 2015." Id. at ¶ 8.

Dr. Silvestri opined that based on his ten years of experience in neurology, "it is entirely possible for a patient to exhibit symptoms that meet criteria for more than one [] diagnosis, and it is entirely possible for a patient, such as [Petitioner], to exhibit symptoms that meet both the diagnostic criteria for both CIDP and SLE." Pet. Ex. 18 at ¶ 9. Furthermore, "SLE is known to occur in conjunction with peripheral neuropathy, including CIDP, and [he] believe[d] this to be the case with [Petitioner]." Id. at ¶ 10. He opined "vaccine administration can be related to the development of adverse effects including the development of certain diseases and/or chronic conditions, including CIDP and SLE." Id. at ¶ 11. Dr. Silvestri concluded "more likely than not . . . that [Petitioner] developed CIDP and SLE as a result of his [flu] vaccine administered on October 14, 2015" because "there is no other reasonable explanation for the development [of] these diagnoses and no other reasonable explanation for the onset of the conditions occurring immediately post-vaccine administration." Id. at ¶ 12.

D. Expert Reports

1. Petitioner's Expert, Dr. Marcel Kinsbourne[46]

a. Background and Qualifications

In 1955, Dr. Kinsbourne obtained his B.M., B.Ch. from Oxford University Medical School, and he completed postdoctoral training through 1964 in the United Kingdom. Pet. Ex. 15 at 1. Thereafter, he obtained board certification in neurology and licensing in the United States and Canada and worked as a professor at various teaching institutions. Id. at 2-3. Dr. Kinsbourne served on a number of editorial boards and authored or co-authored more than 400 publications. Id. at 4-39. Dr. Kinsbourne did not treat patients in a clinical setting after the 1990s.[47] Id. at 2-3.

b. Opinion

Dr. Kinsbourne opined Petitioner's flu vaccination caused him to develop CIDP. Pet. Ex. 14 at 6.

i. Diagnosis

Dr. Kinsbourne opined Petitioner "has a polyneuropathy[] . . . consistent with CIDP." Pet. Ex. 14 at 2. His opinion is based on the opinions of Petitioner's treating neurologists as well as clinical and EMG findings. Id. Additionally, Petitioner benefited from prednisone, which Dr. Kinsbourne noted is expected in CIDP patients. Id. And any delay in diagnosis or reporting of symptoms is typical in patients with CIDP. Id. at 2-3 (citing Pet. Ex. 22-d at 1 (finding "median disease duration at diagnosis of 10 months (range 2-64 months)")).[48]

Regarding SLE, Dr. Kinsbourne agreed that polyneuropathies are sometimes associated with SLE, however, he opined that it is uncommon and "hardly ever takes the form of CIDP." Pet. Ex. 14 at 5-6. Although Petitioner tested positive for two serological markers associated with SLE, Dr. Kinsbourne opined these markers can also be seen in healthy individuals. Id. For any further opinions regarding SLE, he relied on the report of Dr. Byers, and her opinion that Petitioner did not meet the diagnostic criteria for SLE.[49] Id. at 6.

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[46] Petitioner submitted one expert report from Dr. Kinsbourne, dated June 12, 2018. Pet. Ex. 14.

[47] Dr. Kinsbourne was a long time well respected expert in the Vaccine Program. He passed away in April 2024.

[48] R.S. Laughlin et al., Incidence and Prevalence of CIDP and the Association of Diabetes Mellitus, 73 Neurology 39 (2009).

[49] Because the parties stipulated that Petitioner has SLE, the undersigned does not discuss Dr. Kinsbourne's conflicting opinions in this regard. See Joint Submission at 1; Pet. Ex. 14 at 5-6.

ii. Causation

1. Althen Prong One

Dr. Kinsbourne opined the flu vaccine can cause CIDP via molecular mimicry when epitopes in the flu vaccine cross-react with epitopes on myelin in peripheral nerves. Pet. Ex. 14 at 4-6; see Pet. Ex. 22-e.[50]

Although Dr. Kinsbourne agreed Petitioner does not have Guillain-Barré syndrome ("GBS"), he relied on GBS literature regarding the mechanism of causation due to the similarities between GBS and CIDP and the fact that "[i]t is well accepted that [the flu] vaccine is an occasional cause or trigger of GBS." Pet. Ex. 14 at 2-5; see, e.g., Pet. Ex. 22-h at 1 ("CIDP is closely related to [GBS] and it is considered the chronic counterpart of that acute disease."). He explained that CIDP, like GBS, is immune-mediated and targets the myelin sheaths of peripheral nerves. Pet. Ex. 14 at 3. Additionally, he stated that both conditions present with numbness and weakness that progresses from distal to proximal, beginning in the hands and feet. Id.; see, e.g., Pet. Ex. 22-k at 1.[51]

In support of his opinions, Dr. Kinsbourne referenced several medical articles. Kaida et al.[52] discussed molecular mimicry in GBS, and the role of anti-ganglioside antibodies which are thought to attack gangliosides on peripheral nerve myelin and axons in GBS. Pet. Ex. 22-c at 1. Nachamkin et al.[53] also noted the association of anti-ganglioside antibodies with the development of GBS in humans. Pet. Ex. 22-g at 1. The authors conducted a study on mice and found flu vaccines contained structures that can induce anti-ganglioside antibodies after vaccination. Id. at 1, 7. Dr. Kinsbourne opined these studies confirmed flu vaccines induce anti-ganglioside antibodies. Pet. Ex. 14 at 5. He further asserted that "[s]ince CIDP also features anti-ganglioside antibodies, the cross-reaction with myelin in CIDP can be similarly explained." Id.

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[50] Silva Markovic-Plese et al., High Level of Cross-Reactivity in Influenza Virus Hemagglutinin-Specific CD4+ T-Cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis, 169 J. Neuroimmunology 31 (2005). This article was also cited as Resp. Ex. E, Tab 11.

[51] Pieter A. van Doorn et al., Clinical Feature, Pathogenesis, and Treatment of Guillain-Barré Syndrome, 7 Lancet Neurology 939 (2008).

[52] Kenichi Kaida et al., Antiganglioside Antibodies and Their Pathophysiological Effects on Guillain-Barré Syndrome and Related Disorders—A Review, 19 Glycobiology 676 (2009). This article was also cited as Resp. Ex. E, Tab 3.

[53] Irving Nachamkin et al., Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome, 198 J. Infectious Diseases 226 (2008). This article was also cited as Resp. Ex. E, Tab 9.

While Dr. Kinsbourne acknowledged "[t]here [are] no controlled epidemiological stud[ies] of CIDP . . . relative to vaccination risk," he cited several studies examining post-vaccination CIDP and/or relapse of CIDP. Pet. Ex. 14 at 4-5. In McCombe et al.,[54] the authors reported four of 92 patients with CIDP had received a vaccination (smallpox, polio, or tetanus) prior to disease onset. Pet. Ex. 22-f at 6 tbl.3. The flu vaccine was not one of the vaccines at issue. See id. at 1-12. The study was conducted in Australia, covering years 1968 to 1983. Id. at 2. It is not known whether the flu vaccine was routinely administered during those years, and if so, whether it was like the flu vaccine at issue here.

Pritchard et al.[55] examined the risk of relapse in patients with GBS and CIDP using patient completed questionnaires to members of a support group. Pet. Ex. 22-i at 1-2. Two of 46 (4.3%) patients with CIDP experienced a relapse following flu vaccination, and one had a simultaneous pneumococcus vaccination. Id. at 2. It does not appear that new-onset CIDP after vaccination was studied. See id. at 1-3. The authors acknowledged the limits of the study, including the fact that it utilized patient questionnaires and the response rate was low. Id. at 1, 3. The time frame of the study is not clear, and information about the type of flu vaccines administered to the patients in the study was not provided.

2. Althen Prongs Two and Three

Relying on Petitioner's affidavit, Dr. Kinsbourne opined Petitioner developed tingling in his feet, knees, and hands and difficulty maintaining his balance approximately one month following his October 14, 2015 flu vaccination. Pet. Ex. 14 at 1. Although there was a delay in reporting the symptoms to his PCP, Dr. Kinsbourne asserted that CIDP "onset is subtle and initially can easily be overlooked, misinterpreted[,] or forgotten." Id. at 2. Additionally, "many patients with CIDP, [like Petitioner,] cannot pinpoint the exact day of onset." Id.

Dr. Kinsbourne opined that an onset of less than six weeks following flu vaccination is "medically reasonable for neuroimmune polyneuropathies." Pet. Ex. 14 at 5.

2. Petitioner's Expert, Dr. Vera S. Byers[56]

a. Background and Qualifications

Dr. Byers works for a consulting company, Immunology Inc. Pet. Ex. 17 at 1. At Immunology Inc., she designs, supervises, and runs epidemiologic studies on populations

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[54] P.A. McCombe et al., Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical and Electrophysiological Study of 92 Cases, 10 Brain 1617 (1987). This article was also cited as Resp. Ex. E, Tab 7.

[55] J. Pritchard et al., Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy Following Immunisation, 73 J. Neurology Neurosurgery & Psychiatry 348 (2002).

[56] Petitioner submitted one expert report from Dr. Byers, dated June 14, 2018. Pet. Ex. 16.

exposed to carcinogenic environmental chemicals. Id. She is the principal medical witness in over 30 legals cases, involving over 3000 plaintiffs. Id. She is board certified in internal medicine and has a Ph.D. in basic immunology awarded in 1969 from the University of California, Los Angeles. Id. at 3-4. Dr. Byers received her M.D. from University of California, San Francisco ("UCSF") followed by a residency at UCSF. Id. at 4. She was an Adjunct Professor of Immunodermatology at UCSF from 1974 to 2008. Id. at 6. Dr. Byers has authored or co-authored over 100 journal articles regarding immunology and cancer research. Id. at 7-20. Her disease expertise includes some autoimmune diseases, such as rheumatoid arthritis and multiple sclerosis, but does not include CIDP or SLE. See id. at 1. She does not identify any specialized education, training, or experience in diagnosing or caring for patients with CIDP or SLE. See id. at 1-22.

b. Opinion

Dr. Byers focused her opinions on Petitioner's diagnosis, opining Petitioner has CIDP, not SLE. Pet. Ex. 16 at 2-3. Dr. Byers' report mainly discussed the reasons why she did not believe that Petitioner had SLE.[57] See id. Specifically, she opined that because Petitioner's anti-Smith testing was negative, and it was one of the three laboratory testing cited by Petitioner's treating physicians to support his SLE diagnosis, the SLE criteria fail. Id. at 2.

Like Dr. Kinsbourne, Dr. Byers opined it is rare for SLE to present as a neuropathy. Pet. Ex. 16 at 3. But see Pet. Ex. 20, Tab 2 at 1 ("CIDP is an uncommon, but not rare, manifestation of SLE."); Pet. Ex. 23-c at 2 Box 1 (noting peripheral neuropathies are associated with SLE). Dr. Byers cited Anic et al., a study that compared the SLICC and ACR criteria and found "SLICC neurological disorder was observed in 24% of [their] patients, while ACR neurological disorder in only 5%." Id. (citing Pet. Ex. 23-b at 4).

With regard to causation, Dr. Byers opined "[t]he strongest association between vaccines and CIDP is the fact that [GBS] following seasonal vaccines is a table injury in the vaccine court." Pet. Ex. 16 at 3. She added that "[m]ost neurologists believe the initial etiology of CIDP and GBS are the same." Id. For further discussion on causation, she deferred to Dr. Kinsbourne. Id.

3. Petitioner's Expert, Dr. Rebecca M. Shepherd[58]

a. Background and Qualifications

Dr. Shepherd is board certified in internal medicine and rheumatology. Pet. Ex. 20 at 1; Pet. Ex. 21 at 1. She obtained her M.D. from Vanderbilt School of Medicine, and she completed a residency in internal medicine, a clinical research fellowship in the Department of Bone and

[57] Because the parties stipulated that Petitioner has SLE, the undersigned does not discuss in detail Dr. Byers' opinions that are inconsistent with the stipulation. See Joint Submission at 1.

[58] Petitioner submitted one expert report from Dr. Shepherd, dated February 28, 2019. Pet. Ex. 20.

Mineral Research, and a fellowship in rheumatology at Washington University School of Medicine. Pet. Ex. 21 at 3. Dr. Shepherd currently is a Partner at Arthritis and Rheumatology Specialists, Chief of Rheumatology at Lancaster General Health Physicians/Penn Medicine, the Director of Osteoporosis Care at Lancaster General Health Physicians/Penn Medicine, and an instructor in the Family Practice Residency Program at Lancaster General Health. Id. at 1. She is "familiar with the diagnosis of [SLE] by virtue of [her] training, education, and experience." Pet. Ex. 20 at 1.

b. Opinion

Dr. Shepherd's expert report focused on Petitioner's diagnosis. Dr. Shepherd opined, "to a reasonable degree of medical certainty," Petitioner met the SLICC diagnostic criteria for SLE. Pet. Ex. 20 at 6. Dr. Shepherd explained that there are two sets of diagnostic criteria for SLE: ACR and SLICC. Id. at 5. The ACR criteria are more specific and used in clinical trials, while the SLICC criteria "were developed to address perceived flaws in the ACR[] criteria including the omission of many SLE neurologic manifestations." Id. (internal quotations omitted). Dr. Shepherd opined Petitioner did not meet the ACR criteria but met the SLICC criteria. Id. Specifically, Petitioner had a positive anti-Smith antibody, positive dsDNA, positive ANA, and neuropathy. Id.

Dr. Shepherd explained that neuropathy is a common manifestation of SLE. Pet. Ex. 20 at 5. Up to 30% of patients have a peripheral neuropathy confirmed on EMG/NCS, with the most common presentation being a slowly progressive sensory axonal neuropathy. Id.; see Pet. Ex. 20, Tab 4 at 1, 6;[59] Pet. Ex. 20, Tab 6 at 1-2.

According to Dr. Shepherd, SLE is also associated with CIDP. Pet. Ex. 20 at 6. She opined that Petitioner developed "CIDP as a presenting symptom of SLE." Id. Her opinion is based on Petitioner's symptoms, EMG/NCS, response to prednisone, and positive serologies for SLE. Id. "SLE is []associated with CIDP, which is characterized by subacute inflammatory demyelination of the large fibers which leads to both distal and proximal weakness." Id. CIDP has a progressive disease course and typically responds to treatment with prednisone due to its inflammatory nature. Id.

In support of her opinions, Dr. Shepherd cited medical literature about CIDP associated with SLE. Vina et al. studied six patients with CIDP and SLE and concluded CIDP was a "manifestation of SLE" that "can occur before, after, or simultaneously with the onset of SLE." Pet. Ex. 20, Tab 2 at 1, 9. The authors also conducted a literature review, identifying 13 patients with CIDP associated with SLE. Id. Hantson et al. described a case report of a 27-year-old woman with SLE-associated CIDP. Pet. Ex. 20, Tab 1 at 1, 4. Jasmin et al. reported a case of CIDP with underlying SLE in a 26-year-old male. Pet. Ex. 20, Tab 6 at 1. The authors explained that while "[p]eripheral neuropathy [was] a known manifestation of [SLE]," CIDP

[59] G.K. Bertsias et al., EULAR Recommendations for the Management of Systemic Lupus Erythematosus with Neuropsychiatric Manifestations: Report of a Task Force of the EULAR Standing Committee for Clinical Affairs, 69 Annals Rheumatic Diseases 2074 (2010).

with SLE was "uncommon." Id. Overall, all three articles focused on diagnosis and treatment protocols. They did not discuss vaccinations or causation.

4. Petitioner's Expert, Dr. Joseph A. Bellanti[60]

a. Background and Qualifications

Dr. Bellanti is board certified in pediatrics and allergy and immunology. Pet. Ex. 28 at 3. He received his M.D. from the University of Buffalo, after which he completed an internship at Millard Fillmore Hospital in Buffalo, New York and a pediatric residency at the Children's Hospital of Buffalo. Id. at 2. Dr. Bellanti completed a special NIH training in Immunology at the University of Florida and was a Research Virologist at Walter Reed Army Institute of Research. Id. He works as a Professor of Pediatrics and Microbiology-Immunology at Georgetown University School of Medicine, serves as Director of the International Center for Interdisciplinary Studies of Immunology at Georgetown University, and works as a Pediatrician at Georgetown University Hospital. Id. at 1-3. Dr. Bellanti has participated in numerous scientific and professional societies and committees and has authored or co-authored over 500 publications throughout his career. Id. at 3-7, 11-51.

b. Opinion

i. Diagnosis

Dr. Bellanti agreed that Petitioner had a "diagnosis of a peripheral neuropathy" that had been generally agreed upon by all of the experts. Pet. Ex. 27 at 4. In his opinion, it was "irrelevant" as to whether Petitioner's neuropathy was a presenting symptom of SLE or a separate condition (CIDP and SLE). Id. at 4, 6.

Overall, he classified Petitioner's condition as an "autoimmune disease" that first presented as an attack on Petitioner's peripheral nervous system followed by a second attack resulting in nonscarring alopecia. Pet. Ex. 27 at 4, 7. He determined "[Petitioner] developed CIDP that [] was subsequently diagnose[d] as SLE" and both conditions are part of Petitioner's overall autoimmune disorder. Id. at 4, 6. He opined Petitioner's diagnosis was SLE based on the fact that he had alopecia, elevated ANA, elevated anti-dsDNA, and positive anti-Smith antibodies. Id. at 4, 6-7.

Dr. Bellanti explained that peripheral nerves and nerve roots are targeted in CIDP, while the kidney, skin, joints, and central nervous system are targeted in SLE. Pet. Ex. 27 at 7. SLE has a variable presentation and the initial presenting evidence of organ injury is usually not the peripheral nervous system. Id. Nevertheless he agreed that literature "suggests the peripheral nervous system can [] be attacked as a part of [SLE] and can [] be the presenting symptom." Id. In support, he quoted Respondent's expert Dr. Kamen, who noted "[a]pproximately 10-20% of patients with SLE have peripheral nervous system involvement presenting with sensorimotor polyneuropathies, with less common syndromes involving acute [inflammatory demyelinating

[60] Petitioner submitted one expert report from Dr. Bellanti, dated August 5, 2019. Pet. Ex. 27.

polyneuropathy ("AIDP")] or [CIDP]." Id. (quoting Resp. Ex. C at 5 (citing Resp. Ex. C, Tab 3 at 2)). He added that Respondent's literature "specifically states[,] '[CIDP] is an uncommon manifestation of [SLE].'" Id. (quoting Resp. Ex. C, Tab 3 at 3).

ii. Causation

1. Althen Prong One

Dr. Bellanti opined the flu vaccine "can trigger GBS, chronic GBS, peripheral neuropathy[,] and SLE" in genetically predisposed individuals. Pet. Ex. 27 at 4. In general, he explained that "there are a number of antigens and excipients in the vaccin[e] that can prompt an autoimmune reaction or impact tolerance in a predisposed individual, all of which mediate the expression of autoimmune disease(s) by epigenetic mechanisms."[61] Id.

The development of autoimmune diseases, according to Dr. Bellanti, involves an "interplay between genetic factors, environmental triggers, and regulatory aberrations of the immune response," and "[t]hese factors lead eventually to the loss of self-tolerance." Pet. Ex. 27 at 6 (quoting Pet. Ex. 27, Tab 6 at 1).[62] He opined that both infections and vaccinations can lead to autoimmune diseases. Id. at 7, 9 (citing Pet. Ex. 27, Tab 12);[63] see also Pet. Ex. 27, Tab 4 at 23-24;[64] Pet. Ex. 27, Tab 9 at 2-5.[65] He explained that immune responses to infections or vaccinations are mediated by TH17 (pro-inflammatory) and Tregs (anti-inflammatory) responses. Pet. Ex. 27 at 5 (citing Pet. Ex. 27, Tab 3).[66] These responses are balanced in a normal healthy host; however, in a genetically predisposed host, Dr. Bellanti explained that the responses are imbalanced and can lead to the development of autoimmune diseases. Id.

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[61] Epigenetics "refers to the processes by which genotype (the genetic information contained within our DNA) brings about phenotype (the observable physical properties of an organism)." Pet. Ex. 27 at 4-5 (citing Pet. Ex. 27, Tab 2 (Joseph A. Bellanti, Genetics/Epigenetics/Allergy: The Gun Is Loaded . . . but What Pulls the Trigger?, 40 Allergy & Asthma Proc. 76 (2019))). For more on epigenetics, see Pet. Ex. 27 at 5.

[62] Immunology IV: Clinical Applications in Health and Disease (Joseph A. Bellanti et al. eds., 4th ed. 2012). Petitioner filed one page of this chapter.

[63] Ami Schattner, Consequences or Coincidence? The Occurrence, Pathogenesis and Significance of Autoimmune Manifestations After Viral Vaccines, 23 Vaccine 3876 (2005).

[64] Juan-Manuel Anaya et al., The Autoimmune Ecology, 7 Frontiers Immunology 1 (2016).

[65] Cezar Augusto Muniz Caldas & Jozélio Freire de Carvalho, The Role of Environmental Factors in the Pathogenesis of Non-Organ-Specific Autoimmune Disease, 26 Best Prac. & Rsch. Clinical Rheumatology 5 (2012).

[66] Andre Barkhordarian et al., Influenza 2009 Pandemic: Cellular Immune-Mediated Surveillance Modulated by TH17 & Tregs, 6 Bioinformation 29 (2011).

Dr. Bellanti proposed four mechanisms by which infection and vaccination can cause an autoimmune disease: molecular mimicry, polyclonal activation, "tissue damage caused by the infecting agent (or vaccine)," and bystander activation. Pet. Ex. 27 at 8-10.

First, he explained that molecular mimicry occurs when "an inciting factor, most frequently an infectious organism (or vaccine)[,] [] shares epitopes with the host's tissues[,] resulting in tissue damage by an immune response inadvertently directed against one's own body constituents." Pet. Ex. 27 at 8; see also Pet. Ex. 27, Tab 11 at 6 ("Molecular mimicry refers to a process in which the host generates an immune response to an inciting factor, most frequently an infectious organism that shares epitopes with the host's affected tissue."). In a review article on molecular mimicry and autoimmunity, Albert and Inman[67] explained that

> [a]ccording to [molecular mimicry,] a susceptible host acquires an infection with an agent that has antigens that are immunologically similar to the host antigens but differ sufficiently to induce an immune response when presented to T cells. As a result, the tolerance to autoantigens breaks down, and the pathogen-specific immune response that is generated cross-reacts with host structures to cause tissue damage and disease.

Pet. Ex. 27, Tab 13 at 1. Albert and Inman noted "[i]t is possible that vaccination against infectious diseases activates pathways of molecular mimicry in genetically susceptible hosts, and this may be the basis of adverse reactions to vaccines." Id. at 6.

Dr. Bellanti cited studies that suggested that molecular mimicry is the principal mechanism by which autoimmune disease can develop. Pet. Ex. 27 at 9 (citing, e.g., Pet. Ex. 27, Tab 14;[68] Pet. Ex. 27, Tab 15).[69] More specific to this case, he noted molecular mimicry causes GBS following *Campylobacter jejuni* infection, occurring in approximately one-third of GBS cases. Id. at 8. Dr. Bellanti also cited literature that discussed the association between the 1976 flu vaccine and GBS. Id. (citing Pet. Ex. 27, Tab 10).[70] For CIDP specifically, Dr. Bellanti noted viruses and vaccines have been proposed as triggers, with molecular mimicry proposed as

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[67] Lori J. Albert & Robert D. Inman, Molecular Mimicry and Autoimmunity, 341 New Eng. J. Med. 2068 (1999).

[68] Zi-Shan Zhao et al., Molecular Mimicry by Herpes Simplex Virus-Type 1: Autoimmune Disease After Viral Infection, 279 Science 1344 (1998).

[69] Julie K. Olson et al., A Virus-Induced Molecular Mimicry Model of Multiple Sclerosis, 108 J. Clinical Investigation 311 (2001).

[70] James S. Marks & Thomas J. Halpin, Guillain-Barré Syndrome in Recipients of A/New Jersey Influenza Vaccine, 243 JAMA 2490 (1980).

the causal mechanism. Id. at 9; see Pet. Ex. 27, Tab 16 at 1.[71] Dr. Bellanti did not discuss molecular mimicry in the context of SLE. See Pet. Ex. 27 at 8-9.

Next, he discussed other mechanisms that can lead to autoimmune disease: polyclonal activation, "tissue damage caused by the infecting agent (or vaccine)," and bystander activation. Pet. Ex. 27 at 9-10. Dr. Bellanti discussed each of these mechanisms generally, without providing specificity. See id.

For polyclonal activation, he explained that a vaccination or infection, such as Epstein-Barr virus ("EBV"), can induce a generalized stimulation of the immune system, which leads to autoantibodies attacking host organs. Pet. Ex. 27 at 9. He added that this process can occur without the stimulation of a response to a specific autoantigen. Id. He wrote SLE is thought to occur from polyclonal B cell activation. Id. Dr. Bellanti did not provide literature or other evidence in support of this opinion.

According to Dr. Bellanti, his third proposed mechanism, "tissue damage caused by the infecting agent (or vaccine)," leads to a "rais[ed] [] immune reaction to the 'newly exposed' or to the 'altered' autoantigens exposed on the tissues." Pet. Ex. 27 at 9. Dr. Bellanti did not discuss the mechanism further and did not file any supportive evidence or literature.

The last mechanism Dr. Bellanti proposed was bystander activation. Pet. Ex. 27 at 9. He wrote bystander activation occurs when microbial infection causes tissue damage, leading to the release of self-antigens that stimulate the innate immune response and activate self-antigen-expressing antigen presenting cells. Id. T-cell like receptors on antigen presenting cells then cause an "up-regulation of [major histocompatibility complex ("MHC")] and co-stimulatory molecule expression, leading to secretion of various cytokines, local inflammation[,] and recruitment of additional autoreactive lymphocytes." Id. Again, Dr. Bellanti did not provide any support literature for this mechanism, although some of his literature acknowledged bystander activation may be involved in the pathogenesis of autoimmune diseases. See id. at 9-10; Pet. Ex. 27, Tab 9 at 3 ("Molecular mimicry and/or bystander activation may be involved, much like the mechanisms which cause the autoimmune diseases associated with infectious agents.").

Regarding CIDP, Dr. Bellanti cited a case report of post-vaccination CIDP.[72] Brostoff et al. described a 74-year-old man with a 10-week history of progressive right-sided facial weakness, and then ascending weakness of his extremities with exertional shortness of breath.

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[71] J.M. Brostoff et al., Post-Influenza Vaccine Chronic Inflammatory Demyelinating Polyneuropathy, 37 Age & Aging 229 (2008).

[72] Dr. Bellanti also cited Gable et al., who described two patients who developed distal acquired demyelinating symmetric ("DADS") neuropathy after vaccination. Pet. Ex. 27, Tab 17 at 1 (Karissa L. Gable et al., Distal Acquired Demyelinating Symmetric Neuropathy After Vaccination, 14 J. Clinical Neuromuscular Disease 117 (2013)). Unlike CIDP, DADS neuropathy "is characterized by distally predominant sensory symptoms with no or mild distal weakness." Id. However, the Petitioner did not have DADS, and the patient in Gable et al. did not have SLE.

Pet. Ex. 27, Tab 16 at 1. Onset of this progression begin two days following a flu vaccination. Id. The authors noted that CIDP "involves an autoimmune response against peripheral nerve myelin" and "[v]iruses and viral vaccines have been proposed as putative triggers in the pathogenesis of autoimmune disease with postulated mechanisms including antigen mimicry, triggering self-reactive T-cell clones, and cytokine upregulation that may induce aberrant MHC class II expression." Id. Because onset was soon after vaccination, the authors suggested the flu vaccine may have triggered the patient's CIDP. Id. The patient was not diagnosed with SLE. See id.

Lastly, in regard to SLE, Dr. Bellanti cited a 2012 article from Muniz Caldas and Freire de Carvalho. Pet. Ex. 27 at 7-8 (citing Pet. Ex. 27, Tab 9). Muniz Caldas and Freire de Carvalho stated "[t]here are almost 25 cases in the literature relating SLE and vaccination," citing an article that was not filed here. Pet. Ex. 27, Tab 9 at 3. Also, it does not appear that any of these cases occurred following a flu vaccination. Pet. Ex. 27 at 7-8; see Pet. Ex. 27, Tab 9 at 3-4. There was no discussion of the 25 cases of SLE. See Pet. Ex. 27, Tab 9 at 3-4. Nor were specific causal mechanisms described relative to SLE and vaccination. See id. Due to the lack of information, it is difficult to assess the accuracy of the statement.

Dr. Bellanti also cited Wang et al.,[73] a 2017 literature review and meta-analysis evaluating the relationship between vaccinations and risk of SLE and rheumatoid arthritis. Pet. Ex. 27, Tab 5 at 1. The authors reviewed 12 studies on SLE published between 2002 and 2016 and after analysis, they "suggested that vaccinations significantly increased [the] risk of SLE." Id. at 3, 4 tbl.1. Of the 12 studies, only one study examined SLE after vaccinations, which may have included the flu vaccine, in the United States. Id. at 4 tbl.1. The remaining 11 studies took place in other countries, with different vaccines, including different flu vaccines than the one administered to Petitioner. See id. The largest SLE study was conducted in Sweden. Id. The Swedish study assessed the risk of SLE associated with the Pandemrix vaccine between 2009 and 2010. Id. The Pandemrix vaccine was an adjuvanted H1N1 vaccine that was not licensed in the United States. See Historical Vaccine Concerns, Ctrs. for Disease Control & Prevention, July 31, 2024, https://www.cdc.gov/vaccine-safety/historical-concerns/index.html. Due to the differing vaccines included in the study, the findings of increased risk can not be extrapolated to the vaccine at issue here.[74] The authors did not discuss the mechanisms by which a vaccine can cause SLE.

2. Althen Prong Two

Dr. Bellanti opined that the flu vaccination was "the simplest explanation" for all of Petitioner's autoimmune diseases. Pet. Ex. 27 at 4. Specifically, Dr. Bellanti opined that Petitioner "developed CIDP that [] was subsequently diagnose[d] as SLE." Id. at 6. In Dr.

[73] Bin Wang et al., Vaccination and Risk of Systemic Lupus Erythematosus and Rheumatoid Arthritis: A Systematic Review and Meta-Analysis, 15 Autoimmunity Revs. 756 (2017).

[74] The Petitioner received the Fluzone vaccine in 2015. The parties did not file the package insert or other information about whether it contained an adjuvant. Thus, based on the filings, there is no evidence of an adjuvant in the Fluzone vaccine at issue.

Bellanti's opinion, Petitioner's illness was "due to genetic factors and the regulatory aberrations of his immune response." Id. He opined that the medical literature "provide[s] moderate evidence for vaccinations as risk factors of SLE." Id. at 7. And he added that that "[t]he onset of SLE . . . following vaccinations from a large number of case-reports or case-series further support[s] the causal role of vaccinations in the development of SLE." Id. Dr. Bellanti conceded that "the molecular mechanisms underlying the associations of vaccinations with SLE . . . are still not well defined." Id.

Dr. Bellanti explained that Petitioner's initial reaction to the flu vaccine (an attack on his peripheral nerves and nerve roots) was likely due to molecular mimicry, which was followed by a more systemic reaction likely due to polyclonal activation and bystander effect. Pet. Ex. 27 at 4, 8. He believed that the fact that Petitioner's injury "presented as an autoimmune attack of the myelin of the peripheral nerves point[ed] strongly to the flu vaccine as being the trigger in a genetically susceptible individual." Id. at 7.

According to Dr. Bellanti, the vaccine administered to Petitioner, Fluzone (high dose), contains a "number of antigens and excipients"[75] that can "prompt an autoimmune reaction or impact tolerance in a predisposed individual." Pet. Ex. 27 at 4. However, the parties did not file the package insert or other information to show the contents of the flu vaccine administered to Petitioner.

He opined that because the flu vaccine can cause both a peripheral neuropathy and SLE, it is "irrelevant" and "unimportant" to determine whether the neuropathy was a presenting symptom of SLE or whether Petitioner developed two separate conditions. Pet. Ex. 27 at 4, 6.

3. Althen Prong Three

Dr. Bellanti opined Petitioner "more likely than not" first exhibited symptoms of CIDP in mid-November 2015, within 42 days of vaccination, which is appropriate for vaccine causation. Pet. Ex. 27 at 6, 10. In support of an onset of mid-November 2015, he cited to Petitioner's affidavit reporting tingling and imbalance in November, leading to a fall. Id. at 10 (citing Pet. Ex. 1 at ¶¶ 7-8). Dr. Bellanti stated that elderly patients are not typically alarmed by such symptoms so as to prompt a doctor's visit or report such history. Id.

For support, he cited Brostoff et al., who reported an onset of CIDP two days post-flu vaccination. Pet. Ex. 27, Tab 16 at 1. Additionally, Gable et al. reported an onset of DADS neuropathy two weeks following a diphtheria-tetanus-pertussis vaccination and two months after a flu vaccination. Pet. Ex. 27, Tab 17 at 1, 3.

[75] Dr. Bellanti states that the high dose Fluzone vaccine contains excipients, including "egg protein, octylphenol ethoxylate (Triton X-100), sodium phosphate-buffered isotonic sodium chloride solutions, formaldehyde, and mf59 adjuvant." Pet. Ex. 27 at 4. While Dr. Bellanti stated that there are antigens and excipients in the vaccine that could "prompt and autoimmune reaction or impact tolerance in a predisposed individual," he did not identify any such component or explain how that component of the vaccine could cause SLE or CIDP.

Assuming Petitioner's onset was in January 2016, Dr. Bellanti opined that a causal relationship between the flu vaccine and Petitioner's injuries was "less likely, but still probable." Pet. Ex. 27 at 11. He cited to Schonberger et al.,[76] a study of GBS post-flu vaccine, which he maintained reported "an increased relative risk out as far as 10-12 weeks" post-vaccination.[77] Id. (citing Pet. Ex. 27, Tab 18). However, Schonberger et al. concluded that the increased risk of GBS was concentrated primarily within a five week time frame after vaccination, "although it lasted for approximately [nine] or 10 weeks." Pet. Ex. 27, Tab 18 at 1. But after ten weeks, "the relative risks no longer remained significantly different." Id. at 8. Schonberger et al. and Marks and Halpin reported that the onset of GBS peaked at two to three weeks post-flu vaccination. Id. at 6; Pet. Ex. 27, Tab 10 at 2.

5. Respondent's Expert, Dr. Vinay Chaudhry[78]

a. Background and Qualifications

Dr. Chaudhry is board certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. Resp. Ex. A at 1. He received his M.B. and B.S. in India and then completed an internship and various residencies and fellowships from 1980 to 1989. Resp. Ex. B at 2-3. He was a Professor of Neurology at Johns Hopkins University School of Medicine and the Co-Director of the Neurology EMG Laboratory at Johns Hopkins Hospital. Id. at 1, 3; Resp. Ex. A at 1. Dr. Chaudhry has an active clinical practice where he sees over 2,000 patients per year. Resp. Ex. A at 1. He has authored or co-authored over 200 publications. Resp. Ex. B at 3-20. "[He] [is] considered an expert in evaluation and treatment of patients with peripheral neuropathies." Resp. Ex. A at 1.

b. Opinion

Dr. Chaudhry opined Petitioner did not suffer from CIDP and his neuropathic symptoms were not caused by the flu vaccine. Resp. Ex. A at 11.

i. Diagnosis

Dr. Chaudhry addressed whether Petitioner suffered from CIDP and deferred to a rheumatologist for any discussion of SLE. Resp. Ex. A at 6.

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[76] Lawrence B. Schonberger et al., Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977, 110 Am. J. Epidemiology 105 (1979).

[77] Dr. Bellanti also noted another study, which he did not cite or file, "estimated that an elevated risk lasted for at least 26 weeks" post-vaccination. Pet. Ex. 27 at 11.

[78] Respondent submitted two expert reports from Dr. Chaudhry. Resp. Ex. A (dated October 25, 2018); Resp. Ex. G (dated November 13, 2019).

Dr. Chaudhry opined Petitioner did not have CIDP. Resp. Ex. A at 6-7, 11; Resp. Ex. G at 14-16, 21. He defined CIDP as "an acquired sensorimotor demyelinating polyneuropathy that is characterized clinically by progressive symmetric weakness of [more than] [eight] weeks duration and areflexia; electrophysiologically by features of demyelination including prolonged distal and F-wave latencies, reduced conduction velocity, and conduction block/temporal dispersion; and laboratory features of albuminocytological dissociation in cerebrospinal fluid." Resp. Ex. A at 6 (emphasis omitted) (citing Resp. Ex. A, Tab 1;[79] Resp. Ex. A, Tab 2).[80]

More specifically, Dr. Chaudhry explained Petitioner did not have typical CIDP, "characterized by progressive proximal and distal weakness and sensory dysfunction," because Petitioner did not have any documented weakness in 2016 and his sensory examination showed "patchy inconsistent findings." Resp. Ex. A at 6 (citing Resp. Ex. A, Tab 1 at 5 tbl.4). Dr. Chaudhry summarized all findings on physical examinations relating to documented weakness and noted weakness was present only on February 8 and May 10, 2017, when minor weakness in Petitioner's finger extension or intrinsics were documented on examination. Resp. Ex. G at 14-15. Similarly, Dr. Chaudhry summarized Petitioner's sensory symptoms and opined Petitioner, unlike CIDP patients, did not have paresthesias with symmetrical sensory loss to vibration or proprioception.[81] Id. at 15.

Additionally, Dr. Chaudhry explained Petitioner did not have atypical CIDP because he had a "relatively normal" sensory examination, his NCS did not show sensory abnormalities despite his sensory symptoms, and his EMG criteria were not fulfilled. Resp. Ex. A at 6 (citing Resp. Ex. A, Tab 1 at 3 tbl.1, 5 tbl.4). According to Dr. Chaudhry, a normal sensory NCS in the

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[79] P.Y.K. Van den Bergh et al., 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 — First Revision, 17 Eur. J. Neurology 356 (2010). It appears this article also issued in another journal. See Resp. Ex. G, Tab 8 (Joint Task Force of the EFNS and the PNS, 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 — First Revision, 15 J. Peripheral Nervous Sys. 1 (2010)). These guidelines for diagnosing CIDP "are commonly accepted consensus-derived criteria that capture both typical and atypical clinical variants of CIDP." Resp. Ex. A, Tab 13 at 1 (Jeffrey A. Allen et al., Challenges in the Diagnosis of Chronic Inflammatory Demyelinating Polyneuropathy, 8 Brain & Behav. e00932 (2018)).

[80] Richard A. Lewis, Chronic Inflammatory Demyelinating Polyneuropathy: Etiology, Clinical Features, and Diagnosis, UpToDate, https://www.uptodate.com/contents/chronic-inflammatory-demyelinating-polyneuropathy-etiology-clinical-features-and-diagnosis/print (last updated Aug. 29, 2018). An updated version was filed as Resp. Ex. G, Tab 7.

[81] Proprioception is perception mediated by proprioceptors or proprioceptive tissue." Proprioception, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=41270 (last visited Sept. 27, 2024).

legs is not a feature of CIDP, especially after the disease has been present for two years. Id. at 8. For electrodiagnostic testing, Dr. Chaudhry observed that Petitioner's distal latency was prolonged in only one nerve, reduction of conduction velocity was only noted at entrapment sites, there was no conduction block, durations of wave forms were not reported, and his F-waves were not tested. Id. at 6 (citing Resp. Ex. A, Tab 1 at 3 tbl.1).

Based on the findings, Dr. Chaudhry opined that Petitioner's EMG was "most consistent with an acute/subacute non-length dependent motor predominant peripheral polyneuropathy with demyelination[] only affecting the upper limb," not typical in CIDP patients. Resp. Ex. G at 16 (quoting Pet. Ex. 4 at 19). He added that in CIDP, an EMG should show abnormalities in the legs and arms. Id. And an abnormal EMG alone without weakness does not fulfill the criteria for a diagnosis of CIDP. Id. Further, Petitioner had findings on his EMG that are not seen in a neuropathy, or CIDP. Id.

According to Dr. Chaudhry, supportive criteria were also not present. Resp. Ex. A at 6-7. Petitioner did not have his CSF tested, nor were an MRI or nerve biopsy conducted. Id. (citing Resp. Ex. A, Tab 1 at 6 tbl.5); Resp. Ex. G at 16.

Further, Dr. Chaudhry explained that Petitioner's resolution of symptoms after taking prednisone 20 mg for two days does not occur in patients with CIDP. Resp. Ex. A at 7. He also suggested that insurance may have denied IVIG due to Petitioner's "questionable diagnosis" of CIDP. Id.; see also Resp. Ex. G at 16.

Lastly, Dr. Chaudhry took issue with Dr. Bellanti's opinions as to diagnosis, specifically that fact that he classified Petitioner's diagnosis generally as an "autoimmune disease." Resp. Ex. G at 11-12. He explained that there are more than 100 causes of peripheral neuropathy, and of these, only 15% are immune-mediated. Id. at 11. And immune neuropathies differ in presentation, pathogenesis, and response to treatment. Id. Therefore, it is inappropriate to suggest that all neuropathies are autoimmune in nature. Id. at 11-12.

Although Dr. Chaudhry agreed that the evidence shows the presence of a neuropathy, other etiologies and neuropathies were not excluded. Resp. Ex. A at 7. Citing to Köller et al., Dr. Chaudry emphasized that differential diagnoses of CIDP can include neuropathy associated with systemic inflammatory or immune-mediated diseases, including SLE. Resp. Ex. G at 20 (citing Pet. Ex. 27, Tab 11 at 4 tbl.2).

ii. Causation

1. Althen Prong One

Dr. Chaudhry opined "the [flu] vaccine is not causative for CIDP." Resp. Ex. A at 11.

First, Dr. Chaudhry addressed Dr. Kinsbourne's and Dr. Bellanti's comparison of GBS with CIDP. Dr. Chaudhry opined that the two illnesses differ "clinically, pathologically, in response to treatment, and prognosis." Resp. Ex. A at 8-11; see also Resp. Ex. G at 13-14. He summarized key differences between the two. Resp. Ex. A at 8-11; Resp. Ex. G at 13-14. CIDP

differs from GBS due to cranial nerve involvement, autonomic dysfunction, greater sensory involvement, and respiratory compromise. Resp. Ex. A at 8; see Resp. Ex. A, Tab 4 at 8.[82] The time frame from onset to peak symptoms is different, with GBS reaching peak in less than four weeks and CIDP more than eight weeks. Resp. Ex. A at 9; see Resp. Ex. A, Tab 4 at 1. Onset of CIDP is "typically less clear" than in GBS. Resp. Ex. A at 9; see Resp. Ex. A, Tab 4 at 1. CIDP and GBS also differ in clinical course: CIDP is chronic with a relapsing remitting clinical course versus GBS which has a monophasic clinical course. Resp. Ex. A at 9; see Resp. Ex. A, Tab 4 at 8 (noting the clinical course with CIDP has been "described as relapsing-remitting, steady progressive[,] or stepwise progressive").

Additionally, Dr. Chaudhry opined that the diseases have different pathology. Resp. Ex. A at 9; see Resp. Ex. A, Tab 4 at 9. Several antibodies and infections have been linked to GBS. Resp. Ex. A at 9. However, no specific antibody or infectious agent has been identified as a causative factor in CIDP. Id. (citing Resp. Ex. A, Tab 6 at 3 ("Although some patients have reported antecedent infections prior to onset of neurological symptoms neither the target(s) nor the trigger for the autoimmune response has been identified and no infectious agent has been consistently linked with initiation of disease.")).[83] Additionally, Dr. Chaudhry noted GBS has several variants, each with varying electrodiagnostic results (demyelinating, axonal, normal), pathophysiology, and response to treatment. Id. at 10. He also opined that CIDP has variants that differ in their pathogenesis. Id. Therefore, Dr. Chaudhry disagreed with the assertion that GBS and CIDP share the same pathogenesis. Id.

Dr. Chaudhry also noted treatment differs between GBS and CIDP. Resp. Ex. A at 9. IVIG or plasma exchange are used in GBS whereas steroids and other immune drugs are used in CIDP. Id.

Given the differences between GBS and CIDP, Dr. Chaudhry opined that "[t]hinking that GBS and CIDP are similar is being overly simplistic." Resp. Ex. A at 9 (citing Resp. Ex. A, Tab 4 at 8 ("CIDP is commonly considered to be a chronic form of GBS . . . . However, those conceptions may be overly simplistic.")); see also Resp. Ex. G at 13.

Next, Dr. Chaudhry disputed Dr. Kinsbourne's and Dr. Bellanti's assertions that molecular mimicry is the causal mechanism of CIDP. Resp. Ex. A at 10; Resp. Ex. G at 12-14. Dr. Chaudhry acknowledged molecular mimicry is a known mechanism for one form of GBS associated with *Campylobacter jejuni* infection; however, that infection did not occur here. Resp. Ex. A at 10-11; Resp. Ex. G at 11-12. Additionally, the flu virus and flu vaccine have not been shown to induce anti-ganglioside antibodies, despite Dr. Kinsbourne's assertion that their

[82] Eroboghene E. Ubogu, Inflammatory Neuropathies: Pathology, Molecular Markers and Targets for Specific Therapeutic Intervention, 130 Acta Neuropathologica 445 (2015). This article was also cited as Resp. Ex. G, Tab 1.

[83] Emily K. Mathey et al., Chronic Inflammatory Demyelinating Polyradiculoneuropathy: From Pathology to Phenotype, 86 J. Neurology Neurosurgery & Psychiatry 973 (2015). This article was also cited as Resp. Ex. G, Tab 3.

presence could be at play here. Resp. Ex. A at 11 (citing Resp. Ex. A, Tab 9;[84] Resp. Ex. A, Tab 10;[85] Resp. Ex. A, Tab 11).[86] Overall, Dr. Chaudhry opined Petitioner's experts provided no evidence to support molecular mimicry as a mechanism, or evidence that the flu vaccine can cause CIDP. Resp. Ex. A at 10-11; Resp. Ex. G at 13.

Dr. Chaudhry, quoting Ubogo, stated "[u]nlike AIDP, antecedent infection or trauma rarely precipitates CIDP, reducing the likelihood that molecular mimicry serves as a trigger to initiate aberrant tissue-specific pathogenic immune responses." Resp. Ex. A at 9 (quoting Resp. Ex. A, Tab 4 at 12). Similarly, a 2015 article by Dalakas[87] explained that "[i]n contrast to GBS where molecular mimicry with bacterial or viral antigens triggers the disease in some patient subsets, there is no convincing evidence that viral infections are antecedent events in CIDP." Id. (quoting Resp. Ex. A, Tab 5 at 5); see also Resp. Ex. A, Tab 8 at 4. And Mathey et al. acknowledged that "[a]lthough some patients have reported antecedent infections prior to onset of neurological symptoms neither the target(s) nor the trigger for the autoimmune response has been identified and no infectious agent has been consistently linked with initiation of [CIDP]." Resp. Ex. A at 9-10 (quoting Resp. Ex. A, Tab 6 at 3).

Dr. Chaudhry opined that Dr. Bellanti also failed to provide evidence to support his posited mechanisms of polyclonal activation, epitope spreading, and bystander activation playing a role in the development of CIDP. Resp. Ex. G at 13. Dr. Chaudhry noted Dr. Bellanti discussed these mechanisms generally for the pathogenesis of all autoimmune disorders, and not CIDP or SLE specifically. Id.

In further support of his opinions, Dr. Chaudhry cited to the 2012 Institute of Medicine ("IOM") report that found the evidence "inadequate to accept or reject a causal relationship between [flu] vaccine and CIDP." Resp. Ex. A at 7 (quoting Resp. Ex. A, Tab 3 at 2).[88] The

[84] David J. Wang et al., No Evidence of a Link Between Influenza Vaccines and Guillain-Barre Syndrome-Associated Antiganglioside Antibodies, 6 Influenza & Other Respiratory Viruses 159 (2012).

[85] Valérie Sivadon-Tardy et al., Guillain-Barré Syndrome and Influenza Virus Infection, 48 Clinical Infectious Diseases 48 (2009).

[86] Ting Lei et al., Anti-Ganglioside Antibodies Were Not Detected in Human Subjects Infected with or Vaccinated Against 2009 Pandemic Influenza A (H1N1) Virus, 30 Vaccine 2605 (2012).

[87] Marinos C. Dalakas, Pathogenesis of Immune-Mediated Neuropathies, 1852 Biochimica Biophysica Acta 658 (2015). This article was also cited as Resp. Ex. G, Tab 2.

[88] Inst. of Med., Influenza Vaccine, in Adverse Effects of Vaccines: Evidence and Causality 293, 334-35 (Kathleen Stratton et al. eds., 2012). Respondent provided a prepublication copy of this book. Upon review of the publication, the quotations remained the same.

IOM examined five publications, three of which Petitioner's expert relied on,[89] reporting cases of CIDP post-flu vaccination and found "[e]vidence beyond a temporal relationship" between flu vaccination and the development of CIDP was lacking. Id. at 7, 10 (quoting Resp. Ex. A, Tab 3 at 1-2).

2. Althen Prong Two

Dr. Chaudhry opined that Petitioner's flu vaccine did not cause his neuropathic symptoms. Resp. Ex. A at 11; Resp. Ex. G at 21. Dr. Chaudhry detailed Petitioner's clinical course to support this conclusion. Resp. Ex. A at 6; Resp. Ex. G at 1-10. Following the flu vaccination on October 14, 2015, Petitioner saw health care providers on January 26, January 28, March 1, and May 19, 2016. Resp. Ex. A at 5-6. At each of these visits, Petitioner's treating physicians documented a normal neurological examination. Id. According to Dr. Chaudhry, Petitioner's first symptoms suggestive of a peripheral neuropathy (balance issues for two months) were reported on May 19, 2016. Id. at 6. Dr. Chaudhry noted that Petitioner may have developed tingling in his hands and feet in January 2016 which progressed to his hands in April 2016. Id. After two days of taking prednisone 20 mg, Petitioner's symptoms resolved. Id. Petitioner's examinations later showed positive Romberg, absent reflexes, and an abnormal EMG confirmed to the upper limbs. Id. Subsequent follow-up examinations showed normal strength on July 20, September 6, and November 9, 2016. Id. Petitioner was diagnosed with SLE using the SLICC criteria on December 5, 2016. Id. Follow-up appointments in 2017 revealed that Petitioner's symptoms in his legs resolved but he continued to have intermittent symptoms in his upper extremities. Id.

Dr. Chaudhry also took issue with literature cited by Petitioner's experts. See Resp. Ex. G at 16-21. Mainly, he noted most of the literature discussed conditions Petitioner did not have (GBS, CIDP), failed to discuss any association of the condition with vaccination or vaccinations in general, or did not have any relevance to this case. Id.

3. Althen Prong Three

Dr. Chaudhry opined the earliest contemporaneous record documented onset in January 2016, three months after Petitioner's flu vaccination on October 14, 2015. Resp. Ex. A at 7. He opined that this timing was not temporally associated with the vaccine. Id. at 11.

Petitioner's affidavit, relied on by Petitioner's experts, was executed in April 2017. See Pet. Ex. 1. In it, Petitioner averred that the onset of his symptoms was November 2015, inconsistent with his medical records. Resp. Ex. A at 8; Resp. Ex. G at 10-11. Petitioner's medical records first noted neurological symptoms of intermittent tingling in his hands and feet with stress on January 26, 2016. Resp. Ex. A at 8 (citing Pet. Ex. 3 at 23-25). At that visit, Petitioner's neurological examination and motor skills were normal. Id. Dr. Chaudhry opined

[89] See Pet. Ex. 22-i; Pet. Ex. 22-l (Claudia Vellozzi et al., Safety of Trivalent Inactivated Influenza Vaccines in Adults: Background for Pandemic Influenza Vaccine Safety Monitoring, 27 Vaccine 2114 (2009) (also cited as Resp. Ex. E, Tab 8); Pet. Ex. 27, Tab 16.

that Petitioner's normal examination ruled out a diagnosis of CIDP. Id. Additionally, there was no documentation of falls at this visit, despite Petitioner's affidavits asserting he experienced falls prior to this visit. Resp. Ex. G at 11.

Records from January 28 through March 1, 2016 also failed to mention neuropathic symptoms or falls. Resp. Ex. A at 8 (citing Pet. Ex. 10 at 16; Pet. Ex. 3 at 15-16); Resp. Ex. G at 11. Dr. Chaudhry opined that Petitioner's "first symptoms suggestive of a peripheral neuropathy" did not occur until May 19, 2016. Resp. Ex. A at 6, 8 (citing Pet. Ex. 3 at 9).

Dr. Chaudhry also noted that despite Dr. Bellanti's assertions that the medical records are inconsistent with the affidavits and therefore "clearly wrong," the medical records from various providers consistently document Petitioner's neurologic symptoms as beginning in January 2016 (tingling in hands and feet) and April 2016 (imbalance with walking). Resp. Ex. G at 11 (citing Pet. Ex. 3 at 10-11; Pet. Ex. 4 at 14).

6. Respondent's Expert, Dr. Diane Kamen[90]

a. Background and Qualifications

Dr. Kamen is a board-certified rheumatologist. Resp. Ex. C at 1. She obtained her M.D. from the University of Kansas School of Medicine in 1999, and completed an internal medicine internship and residency, M.S. in clinical research, and a rheumatology fellowship from 1999 to 2005 at Medical University of South Carolina in Charleston. Resp. Ex. D at 1. Since 2005, Dr. Kamen has held academic appointments at Medical University of South Carolina and hospital privileges at two hospitals in Charleston, South Carolina. Id. She is also the Director of Clinical Research in the Division of Rheumatology & Immunology at Medical University of South Carolina. Id. Dr. Kamen's "research focus is in environmental influences on the development of autoimmune disease and clinical trials for patients with [SLE]." Resp. Ex. C at 1. She is a member of the Lupus Foundation of America Medical-Scientific Advisory Council, the Lupus Nephritis Trials Network, and the SLICC group. Id. She has also led several large observational studies "investigating potential environmental and epigenetic triggers . . . and characterizing the natural history of [SLE]," with a specific focus on neurologic manifestations of SLE. Id. She has authored or co-authored over 130 publications throughout her career. Resp. Ex. D at 19-44.

b. Opinion

Dr. Kamen opined that more likely than not, Petitioner's neuropathy was not caused by his October 2015 flu vaccination and instead was a presenting feature of his SLE. Resp. Ex. C at 6; Resp. Ex. I at 1.

[90] Respondent submitted two expert reports from Dr. Kamen, dated October 29, 2018 and December 4, 2019. Resp. Exs. C, I. The undersigned finds that of all the experts involved in this matter, Dr. Kamen has the most training and expertise relevant to SLE.

i. Diagnosis

Dr. Kamen agreed Petitioner was correctly diagnosed with SLE by Dr. Sandhu in December 2016 based on Petitioner's steroid-responsive inflammatory polyneuropathy and positive serologies. Resp. Ex. C at 4.

Dr. Kamen opined Petitioner met the SLICC criteria, although it is not required for clinical diagnosis of SLE. Resp. Ex. C at 4. In her first expert report, she opined Petitioner met at least four of the SLICC criteria, two clinical criteria and two immunologic criteria: (1) peripheral neuropathy; (2) nonscarring alopecia; (3) elevated ANA; and (4) elevated anti-dsDNA. Id. at 5. In her second expert report, she opined Petitioner also had AIHA, satisfying a fifth criterion under the SLICC criteria and meeting the SLE criteria for all currently accepted criteria sets. Resp. Ex. I at 1 (citing Pet. Ex. 20, Tab 3; Resp. Ex. I, Tab 1; Resp. Ex. I, Tab 3).[91]

The first criterion was met by the fact that Petitioner had a peripheral neuropathy in the absence of other known causes such as primary vasculitis, infection, and diabetes mellitus. Resp. Ex. C at 5. Because Petitioner had a peripheral neuropathy that did not meet the criteria for CIDP and responded to corticosteroids, Dr. Kamen opined "the likelihood . . . of his neurologic abnormalities being secondary to SLE" was "even higher." Id.

For support, Dr. Kamen relied on Abraham et al. noting that "[a]pproximately 10-20% of patients with SLE have peripheral nervous system involvement presenting with sensorimotor polyneuropathies, with less common syndromes involving [AIDP] or [CIDP]." Resp. Ex. C at 5 (citing Resp. Ex. C, Tab 3 at 2). Abraham et al. also recognized that neurologic and psychiatric symptoms are reported in 10-80% of patients with SLE and included CIDP as a neurological manifestation. Resp. Ex. C, Tab 3 at 2. The authors added that "CIDP can occur before, after, or simultaneously with the onset of SLE." Id. at 3. Dr. Kamen also cited a 1999 article from the ACR that developed a set of case definitions for neuropsychiatric syndromes observed in SLE, including polyneuropathy and AIDP (GBS). Resp. Ex. C, Tab 2 at 4 tbl.2.[92]

Further, Dr. Kamen cited to literature, including Vina et al., discussed above by Dr. Shepherd, to support her opinion that CIDP-like syndromes are associated with SLE. Resp. Ex.

[91] Martin Aringer et al., 2019 European League Against Rheumatism/American College of Rheumatology Classification Criteria for Systemic Lupus Erythematosus, 78 Annals Rheumatic Diseases 1151 (2019).

[92] ACR Ad Hoc Committee on Neuropsychiatric Lupus Nomenclature, The American College of Rheumatology Nomenclature and Case Definitions for Neuropsychiatric Lupus Syndromes, 42 Arthritis & Rheumatism 599 (1999).

C at 5 (citing Pet. Ex. 20, Tab 2 at 1, 9; Resp. Ex. C, Tab 4;[93] Resp. Ex. C, Tab 5);[94] Resp. Ex. I at 1 (citing Resp. Ex. I, Tab 4).[95] Rechthand et al. discussed two patients who presented with predominant motor polyneuropathy suggestive of a demyelinating etiology. Resp. Ex. C, Tab 5 at 2. Both patients had SLE. Id. Six other cases of SLE with CIDP were discussed. Id. Neuropathy was the first clinical manifestation of SLE in three of the patients. Id. Rechthand et al. concluded that "a subacute predominately motor demyelinating polyneuropathy is associated with SLE and may be the presenting feature." Id. at 3. Steroids were beneficial in those patients. Id.

Another article referenced by Dr. Kamen, Hanly et al. (Dr. Kamen is a named author of the article), reported that 41% of the SLE patients had a peripheral neuropathy with a presentation similar to that experienced by the Petitioner. Resp. Ex. I at 1 (citing Resp. Ex. I, Tab 4). Hanly et al. examined the frequency, clinical characteristics, associations, and outcomes of different types of peripheral nervous system diseases in 1,827 patients with SLE. Resp. Ex. I, Tab 4 at 1. Of the 1,827 patients, 139 (7.6%) experienced a peripheral nervous system event, and 41% of these had peripheral neuropathy. Id. at 4, 9. The authors acknowledged that the peripheral neuropathy cases were frequently attributed to non-SLE causes, but due to the lack of diagnostic testing, it was not known whether the neuropathy was related to SLE. Id. at 9. Dr. Kamen explained that because the case definitions in the study "were strict about requiring both subjective and objective evidence and attribution to SLE, the 7.6% [was] likely an underestimation of patients with SLE presenting with peripheral nervous system disease." Resp. Ex. I at 1.

The second criterion was met by Petitioner's development of nonscarring alopecia in the absence of other causes such as alopecia areata, drugs, iron deficiency, and androgenic alopecia. Resp. Ex. C at 5. The third and fourth criteria were met with elevated ANA and anti-dsDNA testing. Id. Dr. Kamen disagreed with Dr. Kinsbourne that anti-dsDNA titers are elevated in healthy individuals. Id. at 6 (citing Resp. Ex. C, Tab 7 at 1 (noting "[a]ntibodies that recognize and bind to DNA (anti-DNA antibodies) are the serological hallmark of [SLE]")).[96] She added that "consistently high titers of anti-dsDNA antibodies [were] detected in [Petitioner's] blood during times of active inflammatory polyneuropathy," which she believed was "sufficiently convincing [evidence] that his neuropathy [was] SLE-related." Id. And fifth, she opined

[93] Robert P. Sedgwick & Karl O. Von Hagen, The Neurological Manifestations of Lupus Erythematosus and Periarteritis Nodosa, 13 Bull. L.A. Neurological Soc'y 129 (1948).

[94] Emanuel Rechthand et al., Chronic Demyelinating Polyneuropathy in Systemic Lupus Erythematosus, 34 Neurology 1375 (1984).

[95] John G. Hanly et al., Peripheral Nervous System Disease in Systemic Lupus Erythematosus: Results from an International Inception Cohort Study, 72 Arthritis & Rheumatology 67 (2020). Dr. Kamen provided the electronic publication version of this article that was released in 2019.

[96] David S. Pisetsky, Anti-DNA Antibodies—Quintessential Biomarkers of SLE, 12 Nature Revs. 102 (2016).

Petitioner's AIHA diagnosis satisfied the fifth SLICC criterion. Resp. Ex. I at 1. Even if he did not have AIHA, Dr. Kamen maintained that Petitioner still met the SLICC criteria. Id.

Dr. Kamen added that if Petitioner's anti-Smith antibody was positive, then this would satisfy another SLICC criterion. Resp. Ex. C at 5. Dr. Kamen acknowledged Petitioner's anti-Smith testing was initially positive, and then negative. Id. at 4. She explained "[i]t would be unusual for anti-Smith antibodies to turn negative in the absence of targeted/B-cell depleting immunosuppressive therapy, [which Petitioner was not receiving at the time,] so confirming that he had a truly positive anti-Smith antibody level historically is important to this case." Id. She agreed with Dr. Byers that Dr. Sandhu likely misread Petitioner's anti-Smith antibody results, but disagreed with Dr. Byers' conclusion that Petitioner failed to meet the SLICC criteria. Id. Dr. Kamen maintained that even without positive anti-Smith testing, Petitioner would still meet the SLICC criteria for SLE. Id. at 6.

ii. Althen Prong Three

Dr. Kamen observed that the affidavits place onset of neurologic symptoms at five to six weeks after Petitioner's October 14, 2015 flu vaccination, which is inconsistent with Petitioner's medical records. Resp. Ex. C at 5. She cited the medical record from January 26, 2016, when Petitioner first reported tingling in hand and feet with stress (and without weakness). Id. (citing Pet. Ex. 3 at 25). Four months later, on May 19, 2016, Petitioner reported neurologic symptoms present for two months,[97] which she noted would place onset in March 2016, more than four months after the flu vaccine in question. Id. (citing Pet. Ex. 3 at 9-10). Dr. Kamen opined that even if Petitioner's symptoms began in January 2016, an onset of over two months post-vaccination, the onset is inconsistent with the vaccination being linked to Petitioner's development of neuropathy or SLE. Id. at 5-6. Dr. Kamen further opined that Petitioner's neuropathy was the presenting symptom of his SLE. Id. at 6.

7. Respondent's Expert, Dr. Ross Kedl[98]

a. Background and Qualifications

Dr. Kedl received his B.S. in Biology and Ph.D. in Pathobiology from the University of Minnesota, after which he completed a postdoctoral fellowship. Resp. Ex. F at 1. Following his fellowship, he worked as a senior immunologist at 3M Pharmaceuticals in their Immune Response Modifier program. Id. at 2; Resp. Ex. E at 2. In 2004, Dr. Kedl joined the University of Colorado faculty where he remains a professor of Immunology. Resp. Ex. E at 1; Resp. Ex. F at 1. He "ha[s] maintained an NIH funded research program centered on the biology of vaccine adjuvants and their capacity to induce robust and enduring cellular immunity." Resp. Ex. E at 1. His grants have "focused on the mechanisms of action of adjuvant biology and T cell

[97] Dr. Kamen also noted that this was the first documentation of neurologic deficits or abnormalities on examination. Resp. Ex. C at 2 (citing Pet. Ex. 3 at 13).

[98] Respondent submitted two expert reports from Dr. Kedl, dated October 24, 2018 and November 15, 2019. Resp. Exs. E, H.

vaccinology," "the study of antigen inexperienced T cell memory subsets[,]" and "the role of lymphatic endothelial cells in the management of T cell memory after vaccination or viral challenge." Id. at 1-2. Throughout his career, Dr. Kedl has authored or co-authored over 75 publications. Resp. Ex. F at 11-17. Dr. Kell is not a medical doctor, and therefore, he does not diagnose or treat patients with CIDP or SLE.[99]

b. Opinion

Dr. Kedl opined "the preponderance of evidence does not support a vaccine-related cause for [Petitioner's] neuropathy." Resp. Ex. E at 8 (emphasis omitted); see also Resp. Ex. H at 6-7.

In his first report, Dr. Kedl addressed each aspect of Dr. Kinsbourne's theory and concluded (1) "GBS and CIDP are not the same disease[]" and (2) "[t]here is no accepted relationship between CIDP and vaccination." Resp. Ex. E at 4-7.

With regard to the relationship between GBS and CIDP, Dr. Kedl agreed with Dr. Kinsbourne that both GBS and CIDP are immune-mediated and primarily target the myelin sheaths of peripheral nerves. Resp. Ex. E at 4. However, Dr. Kedl, quoting a 2011 article from Dalakas, stated "CIDP differs from GBS [] by its time course, mode of evolution, prognosis, and responsiveness to steroids." Id. (quoting Resp. Ex. A, Tab 8 at 1).

Dr. Kedl explained it would be misleading to assert that both diseases respond to treatment with immunomodulating agents since CIDP is responsive to steroids and GBS is not. Resp. Ex. E at 4. He explained this "differential responsiveness to steroids between GBS and CIDP is a key identifier of different mechanistic underpinnings and is therefore strong evidence of systematic differences in their mechanisms of causation." Id.

Additionally, Dr. Kedl opined GBS and CIDP "must differ substantially in their mechanistic underpinnings . . . otherwise they would not differ so substantially in their time course, mode of evolution, and severity." Resp. Ex. E at 5. He criticized Dr. Kinsbourne's reliance on Kaida et al., which Dr. Kinsbourne cited to support the premise that anti-ganglioside antibodies are present in both GBS and CIDP. Id. (citing Pet. Ex. 14 at 5; Pet. Ex. 22-c at 1). Dr. Kedl noted, however, that only one ganglioside-specific target was identified for CIDP and it had no impact on the subjects. Id. (citing Resp. Ex. E, Tab 4 at 1 ("High titers of anti-GD3 antibody and the distortion of antibody recognition found in CIDP seem to have no immediate effect on electrophysiologic function in the [peripheral nervous system].")).[100] Additionally, Eldar and Chapman found "the majority of GBS patients (AIDP subtype) have no identified autoantibodies so the pathogenesis of the disease is still debated." Id. (quoting Resp. Ex. E, Tab

[99] Dr. Kedl offered opinions about alternate medical conditions that more likely caused Petitioner's peripheral neuropathy. See Resp. Ex. E at 7-8; Resp. Ex. H at 7. Because he is not a medical doctor, the undersigned affords little weight to these opinions and does not discuss them herein.

[100] Seigo Usuki et al., AIDP and CIDP Having Specific Antibodies to the Carbohydrate Epitope (–NeuAcα2–8NeuAcα2–3Galβ1–4Glc–) of Gangliosides, 232 J. Neurological Scis. 37 (2005).

5 at 2). Thus, Dr. Kedl opined the association of anti-ganglioside antibodies is tenuous and overlap between GBS and CIDP is "speculative and clinically unproven," and this "is hardly evidence for a common mechanism of causation." Id. Moreover, Dr. Kedl questioned the reliability and applicability of Comi.[101] Id. (citing Pet. Ex. 22-a). Dr. Kedl concluded Dr. Kinsbourne's arguments are "flawed" and his "selective utilization of [] aging literature" render his arguments to associate GBS and CIDP "unreliable." Id.

Next Dr. Kedl opined "[t]here is no accepted association between CIDP and vaccination." Resp. Ex. E at 5-7. He agreed with Dr. Kinsbourne that "[t]here is no controlled epidemiological study of CIDP . . . relative to vaccination risk." Id. at 5 (quoting Pet. Ex. 14 at 4). Dr. Kedl asserted that identifying a similarity between GBS and CIDP is a prerequisite to Dr. Kinsbourne's theory due to his reliance on studies of vaccination and GBS, which fails for the above-mentioned reasons. Id.

Dr. Kedl also explained Dr. Kinsbourne's literature does not support an association between CIDP and vaccination. Resp. Ex. E at 5-6. He criticized Dr. Kinsbourne's opinion that flu vaccines generally induce anti-ganglioside antibodies, finding it flawed in part due to Dr. Kinsbourne's reliance on Nachamkin et al. Id. at 6 (citing Pet. Ex. 22-g). Dr. Kedl criticized Nachamkin et al. because the study was on mice, not humans, and because the methodology lacks scientific rigor. Id. Dr. Kedl concluded "[a]ny connection between [flu] vaccination and CIDP lacks reliable epidemiological, medical[,] and scientific data." Id. at 8.

In his second expert report, Dr. Kedl addressed Dr. Bellanti's opinions and maintained "[t]here is no accepted association between flu vaccination and autoimmunity." Resp. Ex. H at 1-5. Like he did with Dr. Kinsbourne, Dr. Kedl criticized Dr. Bellanti's reliance on GBS. Id. at 4-5. He opined that Dr. Bellanti's "attempted association [with GBS] does not support the assertion that the flu vaccine can also cause all other known autoimmune conditions." Id.

Next, Dr. Kedl opined Dr. Bellanti provided no reliable evidence to support his assertion that the flu vaccine can trigger SLE. Resp. Ex. H at 3. He found fault with Dr. Bellanti's use of case reports noting that are "notoriously unreliable" because they "equate coincidence with causality." Id. Regarding Wang et al., Dr. Kedl noted that the SLE cases either involved a flu vaccine with an adjuvant, which he asserted was not applicable here, or the authors studied patients already diagnosed with SLE, and did not evaluate the risk of developing SLE. Id. (citing Pet. Ex. 27, Tab 5).

Dr. Kedl next concluded the flu vaccine did not exacerbate SLE. Resp. Ex. H at 4. He cited several supportive studies. Huang et al., for example, examined 15 studies, comprising 1,651 participants, comparing SLE patients with healthy controls. Resp. Ex. H, Tab 8 at 1-2. Meta-analysis showed "no significant difference in adverse event rates between SLE patients and healthy controls." Id. at 1, 6. Similarly, Liao et al. reviewed 18 studies with a total of 1,966 SLE patients. Resp. Ex. H, Tab 11 at 1. Of the 1,966 patients with SLE, 32 experienced a mild

[101] Cristoforo Comi, Fas-Mediated T-Cell Apoptosis in Chronic Inflammatory Demyelinating Polyneuropathy, 16 J. Peripheral Nervous Sys. 45 (2011). This article was also cited as Resp. Ex. E, Tab 6.

exacerbation of SLE. Id. at 2, 8, 11. Similarly, Mathian et al.[102] reviewed data on flu vaccinations in SLE patients and stated that "[n]o study has shown a warning about an increase in SLE activity following [flu] vaccines." Resp. Ex. H, Tab 9 at 4.

Dr. Kedl concluded[103] that "[a]ny connection between [flu] vaccination and SLE lacks reliable medical, scientific[,] and epidemiological support." Resp. Ex. H at 6.

8. Respondent's Expert, Dr. Harold Moses, Jr.[104]

a. Background and Qualifications

Dr. Moses is a board-certified neurologist who works as an attending physician and neurology professor at Vanderbilt University Medical Center. Resp. Ex. J at 1; Resp. Ex. K at 3. After he obtained his M.D. from University of North Carolina, Chapel Hill, he completed an internal medicine internship at University of North Carolina Hospitals, a neurology residency at Mayo Clinic in Minnesota, and a fellowship at Vanderbilt University Medical Center. Resp. Ex. K at 1. He has held academic appointments at Vanderbilt University Medical Center since 1999 and works as an attending physician in neurology and neuroimmunology in the Vanderbilt Hospital system. Id. at 2-3. As a clinical neurologist, he focuses on multiple sclerosis and other immune-related disorders of the central nervous system. Resp. Ex. J at 1. He has approximately 1,600 patients in his practice, 90% of whom have multiple sclerosis, and he is involved in numerous clinical trials over the past 20 years. Id.; Resp. Ex. K at 4-7. He has treated patients with CIDP in a hospital setting. Resp. Ex. J at 1.

Dr. Moses was retained by Respondent to replace Dr. Chaudhry due to his scheduling conflicts during pendency of this matter. Order dated Mar. 2, 2021 (ECF No. 73); Resp. Ex. J at 1.

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[102] Alexia Mathian et al., Lupus and Vaccinations, 30 Current Op. Rheumatology 465 (2018).

[103] For further evidence to support his assertion that there is no reliable association between the flu vaccine and SLE, Dr. Kedl cited the 2012 IOM report. Resp. Ex. H at 4 (citing Resp. Ex. L (Inst. of Med., supra note 88, at 373-79)). The IOM examined four studies to evaluate the risk of SLE after flu vaccination and found the evidence was "insufficient or absent to assess an association between [the flu] vaccine and onset of SLE." Resp. Ex. L at 3-5. However, these studies were not filed. For mechanistic evidence, the IOM identified nine publications and found the publications did not provide evidence beyond temporality. Id. at 5, 8. These studies were also not filed. The IOM concluded "[t]he evidence [was] inadequate to accept or reject a causal relationship between [the flu] vaccine and onset or exacerbation of SLE." Id. at 9. Because the underlying studies were not filed, the undersigned is not able to verify the accuracy of this information, and therefore, does not rely on the IOM's assessment in reaching her findings and decision.

[104] Respondent submitted one expert report from Dr. Moses, dated September 2, 2021. Resp. Ex. J.

b. Opinion

Dr. Moses summarized Dr. Chaudhry's expert reports. Resp. Ex. J at 1-2. Like Dr. Chaudhry, Dr. Moses opined Petitioner did not have CIDP. Id. at 2. He noted Petitioner did not have clinical or electrodiagnostic features consistent with CIDP, several diagnostic and supportive studies were not done, and Petitioner's response to a "relatively modest dose of prednisone" was "almost immediate[]." Id. Dr. Moses agreed Petitioner met the SLICC criteria for SLE based on his positive ANA, Smith antibody, and dsDNA and his peripheral neuropathy. Id.

Additionally, Dr. Moses agreed with Dr. Chaudhry's criticisms of Dr. Kinsbourne's and Dr. Bellanti's expert reports. Resp. Ex. J at 2.

Lastly, he noted Dr. Chaudhry's opinion that Petitioner's symptoms suggestive of a neuropathy began three months following his flu vaccination, and thus, Dr. Moses agreed with Dr. Chaudhry that Petitioner's symptoms were not temporally linked to his flu vaccine. Resp. Ex. J at 2. Dr. Moses, like Dr. Chaudhry, concluded there was no relationship between the flu vaccine and Petitioner's symptoms. Id.

IV. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove 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)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d

at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting 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))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell ex rel. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike 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 v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[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 v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not rigidly bound by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (noting Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(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 a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d. 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether a petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health &

Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

V. DIAGNOSIS ANALYSIS

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury[,]" determining facts relating to the claimed injury can be significant in a case where diagnosis is not clear. Id. Here, the parties dispute diagnosis, and so it is appropriate to first resolve that issue.

The undersigned finds Petitioner's correct diagnosis is SLE with neuropathy and/or CIDP as the presenting symptom and not a separate or distinct condition. There are several reasons for these findings.

First, the parties stipulated in their joint submission that Petitioner suffers from SLE. Joint Submission at 1.

Second, the parties' experts agree Petitioner suffers from SLE. Petitioner's expert Dr. Shepherd opined that Petitioner met the diagnostic criteria for SLE based on the SLICC. Pet. Ex. 20 at 6. Petitioner's expert Dr. Bellanti opined Petitioner was first diagnosed with "CIDP that [] was subsequently diagnose[d] as SLE." Pet. Ex. 27 at 4, 6. Respondent's experts Dr. Moses and Dr. Kamen opined Petitioner met the SLICC criteria for SLE and was correctly diagnosed with SLE. Resp. Ex. C at 6; Resp. Ex. J at 2.

Third, Petitioner's treating physicians diagnosed him with SLE, noting that his presenting symptom was peripheral neuropathy and/or CIDP. In December 2016, rheumatologist Dr. Sandhu diagnosed Petitioner with SLE using the SLICC criteria, documenting positive serologies and peripheral neuropathy. Dr. Sandhu determined that Petitioner's "peripheral neuropathy [was] a presenting symptom." Pet. Ex. 11 at 12. Dr. Sandhu's assessment remained SLE at all future visits. In October 2018, rheumatologist Dr. Oza also assessed Petitioner with SLE, opining that it likely explained both his neurologic and hematologic disease (AIHA). Pet. Ex. 45 at 30. Although Petitioner lacked the classical clinical features of SLE, Dr. Oza opined that Petitioner's neurologic symptoms and pulmonary findings satisfied both the ACR and SLICC diagnostic criteria: "immunologic (ANA, dsDNA, [S]mith), neurologic (CIDP), serositis (pleuritis), and hematologic (AIHA)." Id. Therefore, Petitioner's rheumatologists consistently diagnosed him with SLE, with peripheral neuropathy as a presenting symptom.

One of Petitioner's neurologists, Dr. Myers, opined in 2017 suggested that because Petitioner did not have typical symptoms of SLE, he did not have the illness. See Pet. Ex. 4 at 3. The undersigned does not find this statement persuasive because Dr. Myers is a neurologist, not a rheumatologist, and thus, his specialty is not SLE. "In weighing the persuasiveness of opinion testimony, special masters may consider the relative expertise of the witness." Koehn v. Sec'y of Health & Hum. Servs., No. 11-355V, 2013 WL 3214877, at *32 (Fed. Cl. Spec. Mstr. May 30, 2013), aff'd, 773 F.3d 1239 (Fed. Cir. 2014); see also Dwyer v. Sec'y of Health & Hum. Servs., No. 03-1202V, 2010 WL 892250, at *64 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (giving greater weight to M.D. epidemiologists' opinions on medical issues than to Ph.D. epidemiologist's opinion); Pafford, 451 F.3d at 1359 (affirming the special master's rejection of expert's testimony because he lacked proper qualifications in the specialty areas in which he testified).

Further, Dr. Myers acknowledged that Petitioner's bloodwork indicated he had an "underlying rheumatologic disorder." Pet. Ex. 4 at 11. Petitioner did not see a rheumatologist until December 2016, when he received the diagnosis of SLE. Thereafter, Petitioner continued to receive the diagnosis of SLE from his rheumatologists. The undersigned finds opinions after that time by neurologist Dr. Myers not conclusive as to Petitioner's rheumatology diagnosis of SLE.

The fourth reason the undersigned finds Petitioner's diagnosis is SLE is that the experts agree that this condition can present with peripheral neuropathy and/or CIDP. Dr. Kinsbourne noted polyneuropathies are sometimes associated with SLE. Pet. Ex. 14 at 5-6. Dr. Byers opined that while rare, SLE can present as a neuropathy and/or CIDP, and cited literature that supports this presentation. See, e.g., Pet. Ex. 23-c at 2 Box 1 (noting peripheral neuropathies are associated with SLE). But see Pet. Ex. 20, Tab 2 at 1 ("CIDP is an uncommon, but not rare, manifestation of SLE."). Dr. Shepherd opined that up to 30% of SLE patients have peripheral neuropathy confirmed by EMG/NCS. Pet. Ex. 20 at 5. She cited case reports showing peripheral neuropathy/CIDP is a manifestation of SLE. See Pet. Ex. 20, Tab 2 at 1, 9 ("CIDP is a[] . . . manifestation of SLE" that "can occur before, after, or simultaneously with the onset of SLE."); Pet. Ex. 20, Tab 1 at 4 (determining the patient "more likely had []CIDP related to SLE"); Pet. Ex. 20, Tab 6 at 1 (reporting a case of CIDP with underlying SLE). Dr. Bellanti agreed the literature showed that the peripheral nervous system is "attacked" in SLE and that neuropathy can be the presenting symptom. Pet. Ex. 27 at 7. And Dr. Kamen also cited literature that discussed neurological manifestations of SLE, including peripheral neuropathies like CIDP. See Pet. Ex. 20, Tab 2; Resp. Ex. C, Tab 4; Resp. Ex. C, Tab 5; Resp. Ex. I, Tab 4.

Given the agreement between the experts and the consistent and supportive medical literature, the undersigned finds that SLE can present as peripheral neuropathy and/or CIDP. The undersigned finds that it is not necessary to resolve the specific question of whether Petitioner has or had peripheral neuropathy versus CIDP. Regardless of the name of Petitioner's neuropathic condition, it emanated from his SLE. Petitioner did not have two distinct conditions, SLE and peripheral neuropathy/CIDP. Instead, the undersigned finds that Petitioner had one condition—SLE. Peripheral neuropathy/CIDP was a manifestation of his SLE.

Petitioner's expert Dr. Bellanti asserted that Petitioner had "CIDP that [] was subsequently diagnose[d] as SLE," and that both conditions are part of Petitioner's overall

autoimmune disorder. Pet. Ex. 27 at 4, 6. Thus, he suggests that Petitioner has two illnesses that fall under the umbrella of autoimmune disorders.

Although Petitioner's treating physicians suspected peripheral neuropathy/CIDP throughout 2016, once Petitioner obtained the necessary testing (EMG/NCS, blood work), his testing and clinical course were consistent with SLE with a neuropathy/CIDP as the presenting symptom. None of Petitioner's experts preponderantly explain why they believe Petitioner had a distinct case of peripheral neuropathy/CIDP separate from his SLE. In fact, Petitioner's expert Dr. Bellanti opined Petitioner's "CIDP . . . was subsequently diagnose[d] as SLE." Pet. Ex. 27 at 6. Thus, the undersigned finds that Petitioner's neurologic symptoms are part of his constellation of symptoms induced by his SLE and do not represent a separate or distinct condition.

In conclusion, based on the medical records, expert opinions, and medical literature, and for the above-mentioned reasons, the undersigned finds Petitioner's diagnosis was and is SLE that presented as peripheral neuropathy/CIDP.

VI. CAUSATION ANALYSIS

A. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

For the following reasons, the undersigned finds Petitioner has not provided preponderant evidence of a sound and reliable theory by which the flu vaccine can cause SLE, and therefore, Petitioner has failed to satisfy the first Althen prong.

First, only one of Petitioner's experts, Dr. Bellanti, discussed Althen prong one as it relates to SLE. Dr. Kinsbourne addressed Althen prong one only relative to CIDP, but not CIDP as a presenting symptom of SLE, and Dr. Byers and Dr. Shepherd did not address Althen prong one.

Dr. Bellanti opined the flu vaccine "can trigger . . . SLE" in genetically predisposed individuals and proposed four mechanisms by which vaccination can cause an autoimmune

disease: molecular mimicry, polyclonal activation, "tissue damage caused by the infecting agent (or vaccine)," and bystander activation. Pet. Ex. 27 at 4, 8-10. Most of his discussion related to molecular mimicry generally and how it relates to GBS, arguing the mechanism is also applicable to CIDP. However, Dr. Bellanti did not describe how the flu vaccination can cause SLE via molecular mimicry. Nor did he provide any literature or other evidence supporting a relationship between molecular mimicry and SLE. For his other proposed mechanisms, Dr. Bellanti failed to provide a supportive framework beyond simply asserting the theories generally.

Simply asserting a causal theory without context or a supportive factual framework based on medical literature, research, or other evidence is insufficient. Further, the causal theory must be specific to Petitioner's case. Moberly, 592 F.3d at 1322. Merely identifying a mechanism for a disease process without additional evidence specific to Petitioner's case is insufficient to preponderantly show causation. See Monzon v. Sec'y of Health & Hum. Servs., No. 17-1055V, 2021 WL 2711289, at *29 (Fed. Cl. Spec. Mstr. June 2, 2021); Baron v. Sec'y of Health & Hum. Servs., No. 14-341V, 2019 WL 2273484, at *17 (Fed. Cl. Spec. Mstr. Mar. 18, 2019); Duncan v. Sec'y of Health & Hum. Servs., No. 16-1367V, 2020 WL 6738228, at *11 (Fed. Cl. Spec. Mstr. Oct. 19, 2020), aff'd, 153 Fed. Cl. 642 (2021); Boatmon, 941 F.3d at 1360; LaLonde v. Sec'y of Health & Hum. Servs., 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing Moberly, 592 F.3d at 1322); W.C. v. Sec'y of Health & Hum. Servs., 704 F.3d 1352, 1356 (Fed. Cir. 2013).

Further, although molecular mimicry is an accepted scientific mechanism, generally opining that molecular mimicry is a causal theory, without more, is insufficient. See, e.g., Loyd ex rel. C.L. v. Sec'y of Health & Hum. Servs., No. 16-811V, 2021 WL 2708941, at *31 (Fed. Cl. Spec. Mstr. May 20, 2021) ("[T]hough molecular mimicry is a generally accepted scientific concept, and is frequently invoked in Program cases, the mere mention of it does not constitute satisfaction of the preponderant evidentiary standard. Rather, it must be shown that the mechanism likely does link the vaccine in question to the relevant injury." (internal citations omitted)); McKown v. Sec'y of Health & Hum. Servs., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (explaining that "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 omitted)); Sheets v. Sec'y of Health & Hum. Servs., No. 16-1173V, 2019 WL 2296212, at *17 (Fed. Cl. Spec. Mstr. Apr. 30, 2019) (determining Petitioner had not satisfied Althen prong one when he did not relate molecular mimicry "to either the vaccines in question or Petitioner's own specific condition").

CIDP has been alleged as a vaccine related injury post flu vaccination in Vaccine Program cases and in some of these, Petitioners have prevailed on Althen prong one. See, e.g., Nieves v. Sec'y of Health & Hum. Servs., No. 18-1602V, 2023 WL 3580148, at *36 (Fed. Cl. May 22, 2023) (noting prior cases have found petitioners provided preponderant evidence that the flu vaccine can cause CIDP), mot. for rev. den'd, 167 Fed. Cl. 422 (2023); see also, e.g., Strong v. Sec'y of Health & Hum. Servs., No. 15-1108V, 2018 WL 1125666, at *19-20 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (finding that the flu vaccine can cause CIDP, although denying entitlement for other reasons); Daily v. Sec'y of Health & Hum. Servs., No. 07-173V, 2011 WL 2174535, at *8 (Fed. Cl. Spec. Mstr. May 11, 2011).

This case, however, is distinguishable due to Petitioner's diagnosis of SLE. The cases set forth above do not address SLE associated CIDP, or CIDP as the presenting symptom of SLE. The literature herein states that "CIDP is a peripheral nervous system manifestation of SLE" that "can occur before, after, or simultaneously with the onset of SLE." Pet. Ex. 20, Tab 2 at 9. "Neuropsychiatric manifestations in SLE should be first evaluated and treated as in patients without SLE, and secondarily attributed to SLE and treated accordingly." Pet. Ex. 20, Tab 4 at 1. Therefore, the undersigned evaluates the causal theory here in the context of the umbrella diagnosis of SLE, and not as CIDP standing alone, or separate from the underlying illness at issue.

Second, Petitioner's experts did not provide any foundational evidence, studies, or medical literature to show how a flu vaccine can cause SLE. Dr. Bellanti's literature pertaining to SLE either did not discuss or did not report an association with the flu vaccine. Muniz Caldas and Freire de Carvalho noted 25 cases in the literature relating to SLE and vaccination, but it does not appear that any of these cases dealt with the flu vaccine. Nor did Muniz Caldas and Freire de Carvalho discuss mechanisms by which a vaccine can cause SLE. Wang et al. is not applicable because the largest study, which provided the majority of data, examined the effects of the Pandemrix vaccine, which was not given here. Pet. Ex. 27, Tab 5 at 4 tbl.1. Further, Wang et al. did not discuss mechanisms by which vaccines—particularly the flu vaccine—can cause SLE.[105]

Therefore, the undersigned does not find Petitioner's literature persuasive. "An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of Health & Human Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280). Here, Dr. Bellanti failed to explain this literature, or how data from unrelated vaccines could be extrapolated to the flu vaccine at issue here. See K.O., 2016 WL 7634491, at *12 (finding the case reports offered by Petitioner had even less value than case reports do generally because they reported a sequence in which a vaccine, but not the vaccine at issue, preceded the onset of the injury at issue (citing Campbell v. Sec'y of Health & Hum. Servs., 97 Fed. Cl. 650, 668 (2011))); Crosby v. Sec'y of Health & Hum. Servs., No. 18-1478V, 2021 WL 3464125, at *9 (Fed. Cl. Spec. Mstr. July 22, 2021) (declining to give substantial weight to an article because it was on a different vaccine than the one at issue making reasoning difficult); see also Deshler v. Sec'y of Health & Hum. Servs., No. 16-1070V, 2020 WL 4593162, at *19-21 (Fed. Cl. Spec. Mstr. July 1, 2020) (declining to attribute case reports on the flu vaccine to pneumococcal vaccines); McDonald v. Sec'y of Health & Hum. Servs., No. 15-612V, 2023 WL 2387844, at *23 (Fed. Cl. Spec. Mstr. Mar. 7, 2023).

As explained by authors of various studies filed in this case, the vaccines studied in patients with SLE "are very heterogeneous" and include "mono or bivalent vaccine[s] against pandemic[s] [in] 1976 and 2009, seasonal trivalent vaccine[s,] and [a] few [] with [] adjuvants." Resp. Ex. H, Tab 9 at 4. Although the studies involved different vaccines, generally they have

[105] For additional issues with Wang, et al., see Chambers v. Sec'y of Health & Hum. Servs., No. 19-140V, 2022 WL 3369332, at *24 (Fed. Cl. Spec. Mstr. July 22, 2022).

failed to show an increase is disease activity in patients with SLE. Id.; see also Resp. Ex. H, Tab 8; Resp. Ex. A, Tab 11.

Lastly, although decisions of other special masters are not binding, the undersigned finds two Vaccine Program cases instructive, and agrees with the reasoning of her colleagues. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

In Andrews, the petitioner received flu and pneumonia vaccines on August 2, 2014. Andrews v. Sec'y of Health & Hum. Servs., No. 16-196V, 2021 WL 5755328, at *1 (Fed. Cl. Spec. Mstr. Oct. 21, 2021). The next day, she went to an emergency room complaining of fever, chills, sweats, weakness, and muscle aches. Id. *2. She had a moderately painful skin rash on her arm. Id. Laboratory studies revealed an elevated WBC count. Id. The following day, she went to her PCP, who diagnosed a vaccination side effect and ordered a Medrol dose-pack. Id. Subsequently, petitioner had chest pain, dizziness, and high blood pressure. Id. at *3. About six months later, in March 2015, Petitioner reported joint and muscle pain. Id. Bloodwork revealed an elevated C reactive protein with normal ESR and ANA. Id. at *3-4. In June 2015, ten months after vaccination, she saw a rheumatologist and reported a history of diffuse body aches and a red rash on her checks. Id. at *4. She was diagnosed with "probable" fibromyalgia. Id. The special master found that the petitioner did not establish by preponderant evidence that she had SLE. Id. at *16-18. The special master further analyzed causation using the Althen prongs and concluded that the petitioner presented "very thin evidence" as to prong one. Id. at *18. Petitioner's expert asserted the theory of molecular mimicry, relying on articles about rheumatoid arthritis. Id. at *18-19. The special master noted that the expert failed to present "a reasoned explanation" to explain how the articles supported the theory, stating that "[s]pecial masters are not required to accept the *ipse dixit* of program experts." Id. The same is true here. In this case, Dr. Bellanti similarly failed to provide reasoned explanations of his theory and literature.

In the second case, the Chief Special Master in Chambers found the petitioner did not provide preponderant evidence of a theory by which the flu and/or Tdap vaccines[106] can cause SLE. Chambers, 2022 WL 3369332, at *23-25. In Chambers, the petitioner had pre-existing pulmonary embolism, right low back and buttock pain, characterized as numbness, that radiated to her right thigh and knee, that was attributed to moderate spondylosis of the lumbar spine and joint dysfunction, piriformis syndrome, and sacroiliitis. Id. at *1-2. She received the vaccines at issue October 7, 2016. Id. Twelve days later, she reported body aches/pain, low grade fevers, fatigue, red and irritated eyes, hip pain, and episodes of heart racing. Id. at * 2. In November 2016, she saw a rheumatologist who noted that previous laboratory studies revealed a positive ANA. Id. at *3. She was ultimately diagnosed with SLE. Id. at *4-5. Molecular mimicry was advanced by the petitioner's expert as the causal mechanism, and again, medical literature related to rheumatoid arthritis was used to support the theory. Id. at *23-25. There were a number of problems identified by the Chief Special Master related to the study by Wang et al.

[106] Although the petitioner in Chambers received other vaccinations at the same time as her flu vaccine, her expert's focus during the hearing was on the flu vaccine. See Chambers, 2022 WL 3369332, at *9 n.12.

and the studies it relied upon. Id. at *24. The Chief Special Master concluded that reliance on cases about rheumatoid arthritis were insufficient, and that petitioner had not shown the vaccines at issue could cause SLE. Id. at *23-25.

Overall, the undersigned finds that here, Petitioner's theories are unsupported by medical or scientific facts, research, or any other reliable evidence. Moreover, these theories are speculative and/or conclusory in nature. When evaluating whether petitioners have carried their burden of proof, special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. See id.

In summary, Petitioner has failed to offer a sound and reliable medical theory in support of his claim. Therefore, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to the first Althen prong.

B. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical 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 trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Since Petitioner failed to prove Althen prong one, it follows that he cannot prove Althen prong two. However, even if Petitioner had proven Althen prong one, the undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause-and-effect

showing the October 14, 2015 flu vaccine to be the cause of Petitioner's SLE presenting with neuropathy/CIDP. See Althen, 418 F.3d at 1278.

First, Petitioner's clinical course is not consistent with a vaccine-related condition. Petitioner received the flu vaccine on October 14, 2015. More than three months later, on January 26, 2016, Petitioner saw his PCP. Review of symptoms documented tingling in his hands and feet without weakness and his neurological examination was normal. Petitioner returned to his PCP on March 1, 2016. No neurological symptoms were reported or noted, and Petitioner's physical examination was normal. On May 19, 2016, more than six months after his flu vaccine, Petitioner returned to his PCP with "[n]ew [o]nset" neuropathy. Pet. Ex. 3 at 9. Petitioner reported a two-month history of "balance disorder," but denied numbness in his hands and feet. Id. at 10. Under review of systems, Petitioner was positive for gait disturbance. Neurological examination revealed neurological sensory deficits, including "decreased position sense" in his lower extremities and ataxia. Id. at 13.

This summary demonstrates that Petitioner was seen by his PCP approximately two months and five months after vaccination. At both visits, Petitioners neurological examinations were normal. Although subjective tingling was reported in January 2016, it did not warrant referral to a neurologist or further workup at that time. It was not until May 2016 that Petitioner demonstrated any neurological deficit that warranted a neurology referral.

Petitioner argues he had "asymptomatic [SLE] which was triggered by the flu vaccine and presented as CIDP." Pet. Mot. at 3. And that "CIDP was brought on by the administration of the flu vaccine in October of 2015." Id. at 4. However, even if the tingling described in January heralded the onset of neuropathy/CIDP, the presenting symptom of his SLE, it occurred more than two months after vaccination, too remote to be consistent with vaccine causation, as described in more detail in the following section.

Additionally, Petitioner and Dr. Bellanti provided no evidence to show how the flu vaccine Petitioner received in October 2015 caused him to develop SLE. Opining "the simplest explanation for all of [Petitioner's] [autoimmune diseases] was the flu vaccine he received" does not meet the preponderance standard. Pet. Ex. 27 at 4. This statement is conclusory and without support. See Kreizenbeck, 2018 WL 3679843, at *31 (noting "conclusory expert statements that are not themselves backed up with reliable scientific support" are consistently rejected).

Although Petitioner's treating physician Dr. Silvestri offered an affidavit in support of Petitioner's claim, the undersigned does not find it persuasive. Dr. Silvestri, one of Petitioner's treating neurologists, executed an affidavit in March 2019. In it, he opined "vaccine administration can be related to the development of adverse effects including the development of certain diseases and/or chronic conditions, including CIDP and SLE." Pet. Ex. 18 at ¶ 11. Dr. Silvestri concluded that "it is more likely than not . . . that [Petitioner] developed CIDP and SLE as a result of his [flu] vaccine administered on October 14, 2015" because "there is no other reasonable explanation for the development [of] these diagnoses and no other reasonable explanation for the onset of the conditions occurring immediately post-vaccine administration." Id. at ¶ 12.

The undersigned does not find this affidavit persuasive for several reasons. First, the affidavit was executed in March 2019, more than one year after Dr. Silvestri started treating Petitioner, almost two years after the petition was filed, and more than three years after Petitioner developed neuropathic symptoms. See Zumwalt v. Sec'y of Health & Hum. Servs., No. 16-994V, 2019 WL 1953739, at *19 (Fed. Cl. Spec. Mstr. Mar. 21, 2019) (rejecting opinion from a treating provider when he presented an opinion two-and-one-half years after treatment and after litigation was initiated), mot. for rev. den'd, 146 Fed. Cl. 525 (2019).

Second, Dr. Silvestri's affidavit is inconsistent with his own medical records. None of Dr. Silvestri's records state that he believed that the flu vaccine caused Petitioner's neurologic symptoms. See Vergara ex rel. J.A.V. v. Sec'y of Health & Hum. Servs., No. 08-882V, 2014 WL 2795491, at *4 (Fed. Cl. Spec. Mstr. May 15, 2014) ("Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recorded in later medical histories, affidavits, or trial testimony."); Campbell, 69 Fed. Cl. at 779 ("It is, of course, true that where later testimony conflicts with earlier contemporaneous documents, courts generally give the contemporaneous documentation more weight."); Ricci v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 385, 391 (2011) ("Medical records from years later, merely chronicling a timeline between vaccination and injury, are not worthy of the same consideration as contemporaneous records.").

Lastly, Dr. Silvestri provided no evidence to support vaccine causation other than his conclusory statement. Special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck, 2018 WL 3679843, at *31. The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas, 2019 WL 2509626, at *19 (quoting Moberly, 592 F.3d at 1315).

Petitioner also offered a statement made in the medical records and a letter by Dr. Oza to support his petition. See Pet. Ex. 45 at 30; Pet. Ex. 19. Dr. Oza wrote, "Whether [Petitioner's] neurologic/hematologic symptoms were secondary to the flu shot or secondary to [SLE], there is no evidence [he] had any symptoms or other reasons to suspect [SLE] before his flu shot." Pet. Ex. 45 at 30. Dr. Oza made a statement in a letter he wrote on behalf of Petitioner. See Pet. Ex. 19 at 1. The undersigned does not find Dr. Oza's statements to be persuasive evidence of causation because Dr. Oza does not take a position on the question of vaccine causation. Instead, his statements address the question of whether Petitioner had symptoms prior to vaccination. As the evidence shows, there were no symptoms until approximately three months after vaccination.

Petitioner offers no contemporaneous medical records with opinions by his treating physicians in support of vaccine causation.

Generally, treating physician statements are typically "favored" 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.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280). However, no treating physician's views bind the special master, per se; rather, their views are carefully considered and evaluated. § 13(b)(1); Snyder, 88 Fed. Cl. at 746 n.67. "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." Welch v. Sec'y of Health & Hum. Servs., No. 18-494V, 2019 WL 3494360, at *8 (Fed. Cl. Spec. Mstr. July 2, 2019). An opinion by a treating physician that is not supported by a factual basis or other evidence is conclusory in nature. See Robertson v. Sec'y of Health & Hum. Servs., No. 18-554V, 2022 WL 17484980, at *17 (Fed. Cl. Spec. Mstr. Dec. 7, 2022); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010). Furthermore, "[a] treating physician's recognition of a temporal relationship does not advance the analysis of causation." Isaac v. Sec'y of Health & Hum. Servs., No. 08-601V, 2012 WL 3609993, at *26 (Fed. Cl. Spec. Mstr. July 30, 2012).

Accordingly, the undersigned finds that Petitioner has not satisfied his burden under Althen prong two.

C. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also be consistent with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn, 773 F.3d at 1243; Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358.

Here, the experts disagree as to Petitioner's onset, with Petitioner's experts placing onset in mid-November 2016, one month after vaccination, and Respondent's experts placing onset no earlier than January 2016, more than three months post-vaccination.

To summarize, Petitioner's experts Dr. Kinsbourne and Dr. Bellanti provided opinions as to onset. Dr. Kinsbourne, relying on Petitioner's affidavit, opined Petitioner's onset was approximately one month after his October 14, 2015 vaccination, and this onset of less than six weeks following flu vaccination is "medically reasonable for neuroimmune polyneuropathies." Pet. Ex. 14 at 1, 5.

Dr. Bellanti opined Petitioner "more likely than not" first exhibited symptoms of CIDP in mid-November 2015, within 42 days of vaccination, which is appropriate for vaccine causation. Pet. Ex. 27 at 6, 10. Like Dr. Kinsbourne, he relied on Petitioner's affidavit. Dr. Bellanti added that if Petitioner's onset was in January 2016, and not mid-November 2015, then "a causal relationship" was "less likely, but still probable." Id. at 11. For support, he cited to Schonberger et al., who reported an increased risk of GBS post-flu vaccination up to nine or ten weeks post-vaccination. Here, nine or ten weeks post-flu vaccine would place onset in mid-December 2015. Dr. Bellanti provided no literature or other evidence to support a finding that onset in January 2016 would be appropriate given the theory of molecular mimicry.

On the other hand, Respondent's experts opine that onset was no earlier than January 2016, which was not medically acceptable. Dr. Chaudhry relied on Petitioner's most contemporaneous-in-time records to opine that Petitioner's neurologic symptoms began in January 2016. He was not persuaded by Petitioner's affidavit that onset was in November 2015 because this date is inconsistent with contemporaneous medical records. Further, Petitioner executed his first affidavit in April 2017, one-and-one-half years after vaccination. Dr. Moses, like Dr. Chaudhry, concluded Petitioner's symptoms suggestive of a neuropathy began three months following his flu vaccination and agreed that Petitioner's symptoms were not temporally linked to his flu vaccine. Dr. Kamen also noted the affidavits were inconsistent with Petitioner's medical records. She opined that assuming Petitioner's symptoms began in January 2016, an onset of over two months post-vaccination is inconsistent with the vaccination being linked to Petitioner's development of SLE.

Here, Petitioner received his flu vaccine on October 14, 2015. The first neurological abnormality was documented on January 26, 2016, when he presented to his PCP, Dr. Kodial, and review of systems stated he had tingling in his hands and feet. Pet. Ex. 3 at 25-26. Neurologic examination was normal. In addition, when Dr. Myers saw Petitioner on June 24, 2016, he wrote that Petitioner reported tingling in his feet that began "in January of this year." Pet. Ex. 4 at 14.

Petitioner's affidavits, as well as the affidavits from Mrs. Izard and Mr. Welsh, all document that Petitioner fell Thanksgiving 2015 and other neuropathic symptoms occurred around this time frame. However, none of Petitioner's contemporaneous medical records document any fall or neuropathic symptoms before January 2016. The affidavits were executed later in time, in April 2017 and June 2018, years after vaccination.

Because Petitioner's affidavits are inconsistent with and contradicted by the contemporaneous medical records, the undersigned find it reasonable to give greater weight to the contemporaneous medical records. See Cucuras, 993 F.2d at 1528 (noting that "the Supreme Court counsels that oral testimony in conflict with contemporaneous documentary evidence deserves little weight"); Doe/70 v. Sec'y of Health & Hum. Servs., 95 Fed. Cl. 598, 608 (2010); Stevens v. Sec'y of Health & Hum. Servs., No. 90-221V, 1990 WL 608693, at *3 (Cl. Ct. Spec. Mstr. Dec. 21, 1990) (noting that "clear, cogent, and consistent testimony can overcome such missing or contradictory medical records"); Vergara, 2014 WL 2795491, at *4 ("Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recorded in later medical histories, affidavits, or trial testimony."). This finding also extends to the lay witness affidavits. Other special masters have been faced with similar situations and found the contemporaneous medical records more persuasive than the affidavits of lay witnesses. See, e.g., Rote v. Sec'y of Health & Hum. Servs., No. 90-036V, 1992 WL 165970, *5 (Cl. Ct. Spec. Mstr. July 1, 1992) (finding the lay witness testimony insufficient to overcome the weight of the contemporaneous medical records); Bergman v. Sec'y of Health & Hum. Servs., No. 90-1252V, 1992 WL 78671, *4 (Cl. Ct. Spec. Mstr. Mar. 31, 1992) (same); Daiza v. Sec'y of Health & Hum. Servs., No. 90-1188V, 1992 WL 59709, *4 (Cl. Ct. Spec. Mstr. Mar. 5, 1992) (same).

Thus, based on the most contemporaneous medical records, the undersigned finds Petitioner's onset of neurologic symptoms associated with his SLE occurred no earlier than January 2016, approximately three months after his October 14, 2015 flu vaccination.

Additionally, the undersigned finds this three-month period is not a medically acceptable time frame for the posited theory of molecular mimicry, or for any other proffered theory relevant to a post-vaccination autoimmune illness. Petitioner's experts provided no literature or evidence to suggest a flu vaccine can cause an SLE-associated neuropathy more than three months following a flu vaccination. Petitioner's expert Dr. Bellanti opined an onset in January 2016 would make "a causal relationship between the [flu] vaccine and [Petitioner's] injuries [] less likely, but still probable." Pet. Ex. 27 at 11. However, he provided no literature to support an onset of over three months is medically appropriate. Therefore, the undersigned finds Dr. Bellanti's opinion that a causal relationship between Petitioner's October 2015 flu vaccine and January 2016 onset is "less likely, but still probable" conclusory in nature, without basis, and not persuasive. See Kreizenbeck, 2018 WL 3679843, at *31 (noting "conclusory expert statements that are not themselves backed up with reliable scientific support" are consistently rejected).

Furthermore, even if the undersigned were to compare Petitioner's case of SLE with neuropathy as a presenting symptom with cases of GBS in the Vaccine Program like Petitioner's experts suggest, this three-month time frame would still not be medically acceptable. See, e.g., Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting two months is the longest reasonable timeframe for a flu/GBS injury); De La Cruz v. Sec'y of Health & Hum. Servs., No. 17-783V, 2018 WL 945834, at *1 (Fed. Cl. Spec. Mstr. Jan. 23, 2013) (finding an onset of GBS more than two months after flu vaccination not compensable); Aguayo v. Sec'y of Health & Hum. Servs., No. 12-563V, 2013 WL 441013, at *3 (Fed. Cl. Spec. Mstr. Jan. 15, 2013) (rejecting an onset of three-and-one-half months in a flu/GBS case); Corder v. Sec'y of Health & Hum. Servs., No. 08-228V, 2011 WL 2469736, at *27-29 (Fed. Cl. Spec. Mstr. May 31, 2011) (finding petitioner failed to prove that the flu vaccine can cause GBS four months after vaccination).

Lastly, even assuming that Petitioner's injury was CIDP, onset is too long after vaccination to be temporally related according to the relevant medical literature. Brostoff et al. reported an onset of CIDP two days post-flu vaccination. Pet. Ex. 27, Tab 16 at 1.

Therefore, the undersigned finds the temporal association is not appropriate given the mechanism of injury. Petitioner has failed to satisfy the third Althen prong.

VII. CONCLUSION

The undersigned extends her sympathy to Petitioner for all that he has suffered. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination he received caused him to develop

SLE presenting as neuropathy/CIDP. Therefore, Petitioner is not entitled to compensation and the petition must be dismissed.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master