

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-591V

Filed: March 17, 2026

BRENDA BAUGHMAN,

Petitioner,

v.

SECRETARY OF HEALTH AND
HUMAN SERVICES,

Respondent.

Special Master Horner

Mark Theodore Sadaka, Law Offices of Sadaka Associates, LLC, Englewood, NJ, for petitioner.

Lauren Kells, U.S. Department of Justice, Washington, DC, for respondent.

DECISION¹

On April 25, 2018, petitioner, Brenda Baughman, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012).² (ECF No. 1.) Petitioner alleges that she suffered vaccine-induced polyneuropathy after receiving the pneumococcal (“Pneumovax 13”) and tetanus, diphtheria, and acellular pertussis (“Tdap”) vaccines on May 10, 2016. (*Id.*) For the reasons set forth below, I conclude that petitioner is *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations,

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

² Within this decision, all citation to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious or long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a “Table Injury.” That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the “Vaccine Injury Table,” corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1); § 300aa-11(c)(1)(C)(i); § 300aa-14(a).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient’s injury was “caused-in-fact” by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec’y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec’y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991). In this case, petitioner has not alleged an injury that appears on the Vaccine Injury Table. 42 C.F.R. § 100.3(a). Therefore, petitioner must demonstrate causation-in-fact.

The showing of causation-in-fact must satisfy the “preponderance of the evidence” standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); see also *Althen*, 418 F.3d at 1278-79; *Hines*, 940 F.2d at 1525. Under that standard, petitioner must show that it is “more probable than not” that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. She need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a “substantial factor” in causing the condition at issue and was a “but for” cause. *Shyface v. Sec’y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, petitioner must supply “proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury.” *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec’y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. *Id.*

A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the

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

Cases in the Vaccine Program are assigned to special masters who are responsible for “conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded.” Vaccine Rule 3(b)(1). Special masters must ensure each party has had a “full and fair opportunity” to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case, including having the discretion to decide cases without an evidentiary hearing. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1).

II. Procedural History

This case was initially assigned to another special master. (ECF No. 4.) After filing her petition, petitioner filed medical records and an affidavit. (ECF No. 7, 20; Exs. 1-14.) In February of 2019, petitioner confirmed she had filed all her medical records. (ECF No. 22.) Respondent filed his Rule 4(c) report, recommending against compensation in June of 2019 and petitioner was directed to file an expert report. (ECF No. 26.) Thereafter, on August 28, 2019, the case was reassigned to the undersigned. (ECF Nos. 29-30.)

In June of 2020, petitioner filed additional medical records and an expert report by neurologist Alberto Martinez-Arizala, M.D, along with supporting medical literature. (ECF Nos. 37-38; Exs. 15-27.) Respondent filed two responsive expert reports with supporting medical literature, one by neurologist Peter D. Donofrio, M.D. and another from immunologist Penelope A. Morel, M.D. (ECF Nos. 42-44; Exs. A-D.) In January of 2021, petitioner filed additional medical records. (ECF No. 46; Ex. 28.) Petitioner also filed a supplement expert report by Dr. Martinez-Arizala in April of 2021. (ECF Nos. 47-48; Exs. 30-41.) Thereafter, respondent filed responsive expert reports from Dr. Donofrio and Dr. Morel. (ECF No. 50; Exs. E-F.) On October 12, 2021, a Rule 5 status conference was held, which addressed multiple areas of inquiry not fully addressed by

the expert reports filed at that time. (ECF No. 55.) The parties subsequently filed supplemental reports responsive to that guidance between March and August of 2022. (ECF Nos. 63-64, 67; Exs. 42-73, G-H.)

A two-day entitlement hearing was held in October of 2024. (See Transcript of Proceedings (“Tr.”), at ECF Nos. 91-92.) Petitioner and the parties’ three experts testified. Shortly before the hearing, petitioner filed three additional medical articles (ECF No. 86; Exs. 74-76), which prompted an objection by respondent based on unfair surprise (Tr. 5-7, 249-51). Based on the nature of the literature, I declined to preclude the evidence but permitted respondent an opportunity to request additional proceedings at a later date. (*Id.*) During the hearing, I observed that both parties had presented literature that discussed the Institute of Medicine’s (IOM’s)³ 1994 report addressing adverse events associated with childhood vaccines. (*Id.* at 196-97.) Because I already had familiarity with that publication, as well as a later 2012 IOM report on the same subject matter, I filed the two publications as Court Exhibits I and II in the interest of completeness and permitted the parties an opportunity to supplement the record in response. (ECF No. 87; INSTITUTE OF MEDICINE, ADVERSE EVENTS ASSOCIATED WITH CHILDHOOD VACCINES: EVIDENCE BEARING ON CAUSALITY (Kathleen R. Stratton et al. eds., 1994) [hereinafter 1994 IOM report] (Ct. Ex. I); INSTITUTE OF MEDICINE, ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al. eds., 2012) [hereinafter 2012 IOM report] (Ct. Ex. II).) The parties subsequently confirmed they did not wish to supplement the record. (ECF Nos. 88, 93.)

Post-hearing briefs were filed between January and March of 2025. (ECF Nos. 95, 97-98.) Accordingly, this case is now ripe for resolution.

III. Factual History

a. Medical Records

Before she received the vaccine at issue in this case, petitioner had a history of osteoporosis and hyperlipidemia and saw her physician regularly for preventative care.

³ The Institute of Medicine (known as the National Academy of Medicine since 2015) is the medical arm of the National Academy of Sciences. The National Academy of Sciences (“NAS”) was created by Congress in 1863 to be an advisor to the federal government on scientific and technical matters (see An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863)), and the Institute of Medicine is an offshoot of the NAS established in 1970 to provide advice concerning medical issues. When Congress enacted the Vaccine Act in 1986, it directed that the IOM conduct studies concerning potential causal relationships between vaccines and illnesses. See § 300aa-1 note. Special masters have observed that the IOM employs a standard for finding causation that is higher than what is required by petitioner’s burden of proof. *E.g.*, *Raymo v. Sec’y of Health & Human Servs.*, No. 11-0654V, 2014 WL 1092274, at *21 n.39 (Fed. Cl. Spec. Mstr. Feb. 24, 2014). Accordingly, IOM reports and findings are typically approached with caution and generally not treated as dispositive. *Porter v. Sec’y of Health & Human Servs.*, 663 F.3d 1242, 1252 (Fed. Cir. 2011) (noting the special master’s comment that “IOM reports are favored, although not dispositive, in the Vaccine Program,” then affirming special master’s decision). However, numerous prior cases have demonstrated that special masters may account for IOM findings in reaching their decisions. See *Harris v. Sec’y of Health & Human Servs.*, No. 18-944V, 2023 WL 2583393, at *3 n.6 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (collecting cases).

(Ex. 2, pp. 27-35, 40-41; Ex. 6, pp. 5-7.) Additionally, in April of 2014, petitioner saw a chiropractor for left shoulder, elbow, and hand pain. (Ex. 8, pp. 1-14.) In May of 2015, petitioner underwent an EKG after reporting “episodes of racing heart.” (Ex. 2, pp. 36-38.) Petitioner returned to her chiropractor for her left shoulder, elbow, and arm in July of 2015. (Ex. 8, pp. 15-16.) She continued her chiropractic care throughout the rest of 2015 and into 2016, during which she reported bilateral lower back pain, right hip soreness, and right knee numbness, in addition to her original left shoulder and arm pain. (*Id.* at 18-34.) By May 5, 2016, petitioner reported that her pain was improving. (*Id.* at 33-34.) A urinalysis of May 2, 2016, showed trace amounts of blood, elevated white blood cells and bacteria, and the presence of leukocyte esterase and nitrites. (Ex. 2, pp. 50-51.) She had a follow up appointment with her primary care physician on May 10, 2016, during which the results of her urinalysis were noted; however, petitioner was asymptomatic, and her physician considered contamination as a potential explanation for these results. (*Id.* at 22.)

Petitioner received the Tdap vaccine at issue in this case, along with a pneumococcal vaccine, during her May 10, 2016 appointment. (Ex. 2, p. 25.) On May 23, 2016, petitioner reported worsening left sided thoracic and cervical pain, but her right sided lumbar/sacral/pelvis pain was improving. (Ex. 8, pp. 35-36.) She described her pain as “achy, dull, stiff,” and she associated her worsened symptoms with an “airplane ride” during the prior week. (*Id.* at 36.) Her cervical pain radiated to her left scapula and neck, and was associated with numbness in her bilateral scapula area, as well as moderate paraspinal spasms on the left side. (*Id.*) Her thoracic and lumbar pain was also associated with mild-to-moderate paraspinal spasms on the affected side. (*Id.*) Her left thoracic and cervical rotation was limited. (*Id.*) Petitioner reported no additional changes in her condition at her May 24, 2016 chiropractic appointment. (*Id.* at 37-38.)

On May 26, 2016, petitioner reported to the emergency department with bilateral leg pain that had been ongoing for five to seven days. (Ex. 6, p. 81.) Petitioner described cramping, tingling, and numbness. (*Id.*) She explained that her pain began after the flight from California about five days prior. (*Id.*) Petitioner underwent an ultrasound, which showed no deep vein thrombosis, bilaterally. (*Id.* at 86.) She was subsequently discharged in stable condition and with directions to follow up with her primary care provider. (*Id.* at 86-87.) Petitioner returned to her chiropractor on May 28, 2016, where she reported that her left thoracic pain and right lumbar/sacral/pelvis pain were increasing, though her cervical pain remained constant. (Ex. 8, p. 40.) She reported no additional changes during her May 29, 2019 appointment. (*Id.* at 41-42.)

Petitioner reported to the emergency department again on May 30, 2016, with bilateral leg pain for three days that waxed and waned. (Ex. 6, p. 88; Ex. 7, p. 7.) She explained that “she has had lower back pain for the last couple [of] weeks that has now radiated down her arms and legs bilaterally.” (Ex. 6, p. 88.) Petitioner explained that she had recently traveled by plane and was concerned about blood clots; however, her Doppler studies were negative. (Ex. 7, p. 7.) She noted numbness in her bilateral hands and “twitching” in her legs. (Ex. 6, p. 88.) Her physical exam revealed right

“lumbar paraspinal tenderness,” and that she was unable to raise her legs. (*Id.* at 90.) No motor sensory deficits were found; however, she did have paresthesias of the right leg below her knee. (*Id.* at 91.) Differential diagnoses included cauda equina syndrome, epidural abscess, vertebral fracture, degenerative disc disease, spondylosis, spondylolisthesis, osteoporosis, arthritis, ALS, spinal neoplasm, spinal cord infections, and muscle strains. (*Id.*) Petitioner was discharged in stable condition. (*Id.* at 92.) However, petitioner returned to the emergency department later that same day, explaining that she felt unsteady on her feet and that she had fallen. (*Id.* at 94.) She reported a “rash on her torso, generalized weakness, and tingling in her hands and legs.” (*Id.*) Petitioner underwent an MRI of the lumbar spine that revealed

1. Mild S-shaped scoliosis of the lumbar spine with mild to moderate multilevel degenerative changes resulting in multilevel foraminal stenosis, worse at L4-L5 where it is moderate, and minimal to mild canal stenosis, worst at L4-L5. Of note, the left L5 nerve root is impinged between the disc bulge and facet arthropathy.
2. No acute abnormalities in the lumbar spine.
3. Indeterminate lesion of L3, possibly atypical hemangioma, but if the patient has a known primary malignancy metastatic disease cannot be excluded. Consider postcontrast imaging on a nonemergent basis and comparison with any prior studies would also be helpful.

(Ex. 7, p. 26.) Her diagnoses included acute dermatitis; osteoarthritis of lumbar spine, unspecified spinal osteoarthritis complication status; and acute lumbar radiculopathy. (*Id.* at 16.) The next day, petitioner was seen by her primary care physician for a follow up on her leg and back pain and rash. (Ex. 2, p. 17.) She reported that her condition began as tingling and numbness in her feet that impacted her balance. (*Id.*) She again reported that her symptoms began after her trip. (*Id.*) Her primary care physician also reported that she was taking Fosamax for her osteoporosis, hormone replacement therapy for her menopausal symptoms, and fish oil and red yeast rice for her hyperlipidemia. (*Id.* at 17-18.)

Petitioner had a chiropractic appointment on June 6, 2016, during which she reported her left sided cervical pain remained with moderate left cervical paraspinal spasms and moderate decreased left rotation and bending. (Ex. 8, p. 44.) Additionally, she reported that her bilateral lumbar pain was getting worse, that she was having left thoracic paraspinal spasms, and that her right bending lumbar motion was slightly decreased. (*Id.*) Her thoracic pain was improving. (*Id.*) The next day, petitioner returned to the chiropractor and reported the same symptoms. (*Id.* at 46.) Additionally, she brought her MRI and reviewed it with her chiropractor. (*Id.*)

On June 9, 2016, petitioner had an orthopedic spine consultation with Dr. Gordon Yee. (Ex. 4, pp. 43-44.) Dr. Yee recorded petitioner’s chief complaint as “[l]ow back pain, bilateral lower extremity pain and tingling.” (*Id.* at 43.) Petitioner explained that she has suffered from mild lower back pain for five years; however, three weeks ago, her “back pain worsened and she had onset of bilateral lower extremity symptoms.” (*Id.*) She reported that her increased pain was associated with “constant tingling in both

feet diffusely” and localized to her central lower lumbar spine and bilateral calves. (*Id.*) Dr. Yee observed mild lumbar scoliosis, decreased lumbar range of motion, and “mild tenderness over the lumbar spine posteriorly.” (*Id.*) Petitioner’s neurological examination showed “positive Trendelenburg test with mild weakness of left hip abductors,” but normal strength in all other lower extremity muscle groups. (*Id.*) Dr. Yee reviewed petitioner’s MRI. (*Id.*) Petitioner was assessed with bilateral back and lower extremity pain and tingling, lumbar degeneration, mild scoliosis, mild to moderate L4-5 spinal stenosis, and an indeterminate bone marrow lesion at L3 vertebral body. (*Id.* at 44.) Dr. Yee referred petitioner to physical therapy and prescribed hydrocodone. (*Id.*) Petitioner had her initial physical therapy consultation on June 13, 2016. (*Id.* at 60.) At this initial consultation, petitioner reported that she was doing yard work and cleaning in California and started experiencing discomfort on the flight home. (*Id.*) The day after her flight, petitioner “woke up with significant pain with numbness/tingling in her [bilateral] arms and legs.” (*Id.*) Petitioner’s physical exam noted that petitioner’s posture was “[v]ariable; slouched, forward flexed posture throughout most of examination,” and her range of motion tests revealed “[i]ncreased [bilateral lower extremity] symptoms with forward flexion; increased lumbar symptoms with flexion.” (*Id.*) On the right side, her hip flexion, knee flexion, knee extension, and ankle dorsiflexion were recorded as 4+/5 and her ankle plantarflexion was recorded as 5/5. (*Id.* at 61.) On the left, her hip flexion, knee flexion, knee extension, and ankle dorsiflexion were 4+/5, and her ankle plantarflexion was 4-/5. (*Id.*) Her Lasegue’s straight leg raise test was positive on both sides. (*Id.*)

Petitioner underwent another MRI on June 15, 2016, that revealed “[n]o abnormal enhancement within the lumbar spine specifically no evidence of enhancement corresponding to previous described indeterminate L3 lesion likely representing atypical hemangioma,” and “[m]ild periarticular and endplate reactive enhancement most prominent at L4-L5 endplate and L2-L3 facet articulations.” (Ex. 7, p. 33.) Petitioner also underwent an x-ray of her lumbar spine on June 21, 2016, which found “degenerative disk narrowing at L2-3, L3-4 and L4-5,” with the “most severe narrowing at L4-5,” and “asymmetric disk narrowing at L4-5 on the left and mild L4-5 retrolisthesis.” (Ex. 4, p. 1.) Additionally, the x-ray identified mild lumbar scoliosis. (*Id.*)

Petitioner had a follow up appointment with Dr. Yee on June 21, 2016. (Ex. 4, pp. 26-27.) Dr. Yee noted that petitioner’s pain had continued to worsen. (*Id.* at 26.) Petitioner explained that her current symptoms were lower back pain, bilateral lower leg pain, and constant tingling in her feet. (*Id.*) Her neurological exam revealed “weakness in tibialis anterior bilaterally and [extensor hallucis longus] bilaterally all measuring grade 4/5 strength,” along with “questionably decreased effort on motor testing.” (*Id.*) Dr. Yee reviewed petitioner’s MRI and x-rays. (*Id.*) Petitioner was assessed with lower back pain, “bilateral extremity pain and tingling,” bilateral possible mild weakness in her tibialis anterior and extensor hallucis longus, lumbar degeneration and mild scoliosis, “[m]ild to moderate L4-5 spinal stenosis,” and “[n]o enhancement with MRI contrast at the L3 bone marrow lesion, most likely atypical hemangioma.” (*Id.* at 27.) Dr. Yee ordered an electromyography and nerve conduction study (EMG/NCS) and prescribed Hydrocodone. (*Id.*) Petitioner had a chiropractic appointment on June 22, 2016, where

she reported that her left thoracic pain was improving, there was no change to her left cervical pain, and her bilateral lumbar/sacral/pelvis pain was getting worse. (Ex. 8, p. 48.) Petitioner presented to the emergency department on June 29, 2016, complaining of pain “all over.” (Ex. 6, p. 96.) She reported that her symptoms began six weeks prior, while on a road trip with her family. (*Id.*) She described “generalized pain and muscle aches,” for which she was taking ibuprofen and oxycodone. (*Id.*) Her physical exam revealed “1-2mm scattered erythematous lesions that are nonpalpable,” which petitioner reported had been there for six weeks. (*Id.* at 98.)

On July 1, 2016, petitioner underwent an EMG/NCS of her lower extremities, with Dr. Paulo Cancado. (Ex. 1, pp. 1-4.) The study showed evidence of an “[o]ngoing partial denervation of the bilateral medial gastrocnemius and anterior tibialis muscles, consistent with bilateral ongoing L4 and/or L5 and S1 radiculopathies,” or a moderate to severe “axonal sensory and an axonal and demyelinating motor polyneuropathy.” (*Id.* at 1.) Dr. Cancado noted that he preferred the latter diagnosis. (*Id.* at 8.) Additionally, the study showed “[p]rolongation of the bilateral H-reflex latencies, consistent with bilateral S1 radiculopathies or a peripheral neuropathy.” (*Id.* at 1.) During her consultation, Dr. Cancado recorded that petitioner had been experiencing pain, numbness, and paresthesias in her distal lower extremities and hands for six to seven weeks. (*Id.* at 5.) Her pain had been associated with a rash affecting the trunk and neck area. (*Id.*) Petitioner’s motor strength was recorded as “4/5 throughout, for exception of ankle dorsiflexion which were 3+/5 bilaterally,” and her “[d]eep tendon reflexes were trac[able] bilaterally symmetrically throughout and not obtainable [in the] ankles.” (*Id.* at 7.) Additionally, Dr. Cancado noted that petitioner had bilateral hand numbness and paresthesias and considered several differential diagnoses including “peripheral neuropathy and entrapment neuropathies such as carpal tunnel syndrome and ulnar neuropathy at the elbows.” (*Id.* at 7-8.)

Petitioner underwent a separate EMG/NCS for the numbness and paresthesias in her upper extremities on July 2, 2016. (Ex. 1, pp. 9-12.) This study found a moderate to severe bilateral “entrapment of the [m]edian nerves at the wrists, consistent with [b]ilateral [c]arpal [t]unnel [s]yndromes,” and “[a]n axonal and demyelinating sensorimotor polyneuropathy.” (*Id.* at 9.) Additionally, there was no evidence “of an [u]lnar neuropathy at the elbows,” nor did the study “suggest a cervical radiculopathy (at least motor) bilaterally.” (*Id.*) Dr. Cancado ordered blood tests for “potential causes of peripheral neuropathy.” (*Id.* at 14.) On July 7, 2016, petitioner underwent a brain CT, which showed “[n]o acute sinusitis,” and instead only “[m]inimal chronic inflammatory change in the left sphenoid sinus.” (Ex. 14, p. 1.) The study was otherwise normal. (*Id.*)

Petitioner had a follow up appointment with Dr. Cancado on July 11, 2016, for “recent onset of peripheral neuropathy.” (Ex. 1, p. 69.) He noted that petitioner’s EMG/NCS showed “axonal sensory and an axonal and demyelinating motor polyneuropathy” in her lower extremities, and “polyneuropathy as well as bilateral carpal tunnel syndromes” in her upper extremities. (*Id.*) He noted that petitioner had been taking Oxycodone and Naproxen and was recently started on Amitriptyline. (*Id.*)

Petitioner reported that she “continu[ed] to have tingling and pain affecting both distal lower extremities as well as tingling and stiffness in the hands.” (*Id.*) Dr. Cancado described petitioner’s lab work as “unremarkable.” (*Id.*) Dr. Cancado’s impression included polyneuropathy of unclear etiology, lumbosacral spondylosis, and bilateral carpal tunnel syndromes. (*Id.* at 70.) On July 21, 2016, petitioner had a follow up appointment with Dr. Yee for lower back pain, bilateral pain and tingling in her lower extremities, “[p]ossible mild weakness in tibialis anterior bilaterally and EHL bilaterally on previous examination,” and mild to moderate spinal stenosis at L4-5. (Ex. 4, p. 41.) Petitioner reported that she had undergone blood tests and had been referred to a specialist. (*Id.*) Petitioner’s exam revealed increased weakness in her lower extremities, and “[m]otor strength in [her] tibialis anterior, EHL, and peronei all measure grade 3/5 strength bilaterally.” (*Id.*) Dr. Yee reviewed petitioner’s EMG/NCS findings and assessed petitioner with demyelinating polyneuropathy, tingling in her hands and feet, weakness in her lower extremities, asymptomatic mild to moderate spinal stenosis at L4-5, “mild lumbar scoliosis and multilevel lumbar degeneration,” and bilateral carpal tunnel syndrome. (*Id.* at 41-42.) Petitioner’s antibody tests were negative, and her additional bloodwork was normal. (Ex. 1, pp. 16-21.)

Petitioner had her next follow up appointment with Dr. Cancado on July 28, 2016, for “polyneuropathy and bilateral carpal tunnel syndromes.” (Ex. 1, p. 72.) Petitioner reported that her pain continued, particularly in her lower extremities, impacting her ability to walk. (*Id.* at 72.) Her neurological exam revealed “unsteadiness on her feet,” and Dr. Cancado observed that petitioner was “unable to walk on her heels and toes.” (*Id.* at 73.) She was able to stand on her tiptoes, but not on her ankles. (*Id.*) Dr. Cancado’s impression was polyneuropathy of unclear etiology and bilateral carpal tunnel syndromes. (*Id.*) Petitioner remained on Fosamax and Amitriptyline. (*Id.* at 74.)

Petitioner began physical therapy on August 8, 2016. (Ex. 5, p. 222.) It was recommended that petitioner attend physical therapy two times a week for 10 weeks. (*Id.* at 222-23.) Petitioner attended physical therapy throughout August of 2016. (*Id.* at 207-08, 211-20.) Petitioner had a follow up appointment with Dr. Cancado on August 29, 2016, for “polyneuropathy of unclear etiology and bilateral carpal tunnel syndromes.” (Ex. 1, p. 75.) Although physical therapy had been partially helpful, petitioner continued to experience significant pain. (*Id.*) On exam, she continued to have “some degree of imbalance,” and she could not walk on her heels but could stand on her tiptoes. (*Id.* at 76.)

Petitioner continued to attend physical therapy throughout September of 2016. (Ex. 5, pp. 185-203.) On September 6, 2016, petitioner reported improvement, particularly in her right foot and left hand. (*Id.* at 202.) On September 16, 2016, petitioner had primary care appointment, during which it was noted that petitioner was in the care of Dr. Cancado and had been diagnosed with sensorimotor neuropathy of unknown etiology. (Ex. 2, p. 11.) Petitioner explained that her leg and back pain, tingling, and numbness, along with a rash, had begun after a trip to California. (*Id.*) It was noted that petitioner was on Fosamax for osteoporosis and hormone replacement therapy for menopausal symptoms. (*Id.* at 12.) Petitioner’s hyperlipidemia was also

noted. (*Id.*) Petitioner's physical exam revealed "[n]o bipedal edema, no cyanosis, no clubbing," and "peripheral pulses intact." (*Id.* at 14.) However, petitioner's straight leg raising test was positive bilaterally. (*Id.*) Her primary care provider's impression was "[l]eft hydronephrosis without hydroureter versus multiple parapelvic cysts in the left renal central sinus complex" and "[m]ultilevel degenerative facet joint arthropathy in the lumbar spine, along with moderate degenerative disc disease," as well as a retroverted uterus and "[b]ilateral moderate upper pulmonary lobe pleural and parenchymal scarring," particularly on the right. (*Id.* at 11-12.) The assessment was sensorimotor polyneuropathy of unknown cause, which had been progressing over the past few months. (*Id.* at 14.)

On September 21, 2016, petitioner presented to Nurse Practitioner ("NP") Caitlin Shino for a neuromuscular evaluation of her bilateral foot tingling and gait imbalance. (Ex. 3, pp. 12-15.) It was noted that petitioner developed "back pain and tingling in her toes and finger tips as well as a diffuse rash of 'red dots' across her abdomen" within ten days after she received a Tdap and pneumococcal vaccination. (*Id.* at 12.) Petitioner's examination revealed an "abnormal tandem gait," that is "off balance with [asymmetric] steppage," specifically due to the left foot inverting. (*Id.* at 13-14.) Her upper extremity strength was intact, and in her lower extremities, her proximal muscle groups were at maximal strength. (*Id.* at 14.) However, her ankle dorsiflexion was 5-/4+, inversion was 5-/5-, eversion was 5-/3, and great toe extension was 3/3. (*Id.*) The record notes that "Dr. Carlson was able to come in and see [petitioner]; he reviewed the entire history and performed an independent examination. He formulates the following impression and plan: . . . [a]cute onset distal [asymmetric] non-length dependent polyneuropathy most likely etiology is an acute autoimmune neuropathy associated with tetanus vaccination." (*Id.*) Additionally, it was noted that "post-vaccinal neuropathies can develop often related to a leukocytoclastic vasculitis," and that petitioner "had a truncal rash associated with peripheral nerve symptomatology early in her presentation which is likely a dermatologic comorbid condition." (*Id.*) Alternative diagnoses, such as Lyme disease or sarcoidosis, were considered and ruled out. (*Id.*)

Petitioner continued her physical therapy throughout October 2016. (Ex. 5, pp. 161-84.) She was reevaluated on October 6, 2016 and reported improvement. (*Id.* at 175.) Petitioner had a follow up appointment with Dr. Cancado on October 31, 2016. (Ex. 1, p. 86.) He noted that her neuromuscular specialist had diagnosed her with post vaccine polyneuropathy. (*Id.* at 87.) A neurological exam noted that petitioner's balance had improved and that she was able to stand on her tiptoes; however, she was still unable to walk or stand on her heels. (*Id.*)

On November 4, 2016, petitioner presented to a new primary care provider. (Ex. 2, pp. 8-10.) She reported that she had developed a rash and bilateral tingling and pain in her feet after receiving a Tdap vaccine on May 10, 2016. (*Id.* at 8.) It was further noted that petitioner had been diagnosed with polyneuropathy that was likely caused by her Tdap vaccine. (*Id.* at 8.) It was observed that petitioner had experienced mild improvement in her neuropathic symptoms. (*Id.* at 10.) Petitioner continued to pursue physical therapy throughout November 2016. (Ex. 5, pp. 149-60.) She was reevaluated

on November 7, 2016, and she continued to report improvement. (*Id.* at 159.) On November 11, 2016, petitioner was reevaluated by NP Shino for “distal asymmetric non-length dependent polyneuropathy most likely an acute autoimmune neuropathy associated with tetanus vaccination.” (Ex. 3, p. 4.) Petitioner reported that her balance and walking had improved; however, she continued to have some numbness, tingling, and occasional pain in her feet. (*Id.*) Petitioner’s labs were negative for infection or inflammation. (*Id.*) Petitioner’s physical exam revealed ankle dorsiflexion at 5-/4+, inversion at 5/5, eversion at 5/5-, and great toe extension at 4-/4-. (*Id.* at 5.) Additionally, petitioner’s Achilles reflexes were absent. (*Id.*) Petitioner’s distal power and gait dynamics had improved. (*Id.*)

Petitioner continued physical therapy throughout December 2016 and January 2017. (Ex. 5, pp. 102-48.) Petitioner was reevaluated on December 5, 2016, during which she reported that she felt like her improvement was beginning to plateau. (*Id.* at 140.) Petitioner was reevaluated again on January 10, 2017, and reported additional improvement, explaining that she had “less sensation of ridges on the bottom of her feet,” but she continued “to have pain that radiate[d] across the dorsum of her foot and up, around the lateral ankle.” (*Id.* at 112.) Petitioner had a follow up appointment with Dr. Cancado on January 26, 2017, for “peripheral neuropathy possibly related to a tetanus shot.” (Ex. 1, p. 92.) He noted that petitioner’s condition was improving. (*Id.*) Her neurological exam revealed that petitioner was able to walk on her tiptoes and that her gait had improved; however, she was unable to walk or stand on her heels. (*Id.* at 93.)

Petitioner had a follow up appointment with her primary care physician on February 8, 2017, during which she reported chronic fatigue and foot pain associated with her neuropathy. (Ex. 2, p. 5.) It was also noted that petitioner was taking Alendronate for her osteoporosis. (*Id.*) Petitioner continued her physical therapy throughout February and March of 2017. (Ex. 5, pp. 57-101.) She was reevaluated on February 6, 2017, where she reported that her leg pain had “eased slightly.” (*Id.* at 98.) She was reevaluated again on March 3, 2017, where she reported a sharp pain in her ankle/foot and an increased sensation of stiffness. (*Id.* at 75.)

On March 18, 2017, petitioner returned for a neuromuscular evaluation of her “distal asymmetric non-length dependent polyneuropathy most likely an acute autoimmune neuropathy associated with tetanus vaccination.” (Ex. 3, p. 1.) Petitioner reported improvement in her balance and speed of walking; however, she continued to complain of pain, numbness, and tingling in her feet. (*Id.*) It was noted that petitioner’s HTLV-1, CRP, and ANCA labs were negative. (*Id.*) Her physical examination showed normal upper extremity strength and that her “[p]roximal muscle groups in lower extremities remain intact.” (*Id.* at 2.) Her ankle dorsiflexion had improved to 5/5- and her great toe extension had improved to 5-/4+. (*Id.*) Her Achilles reflexes were still absent, and her gait was “mildly off balance with poor heel strike bilaterally.” (*Id.*)

On May 4, 2017, petitioner had a follow up appointment with Dr. Cancado. (Ex. 1, p. 95.) He reported that petitioner’s symptoms were slowly improving. (*Id.*)

Petitioner was able to walk on her heels and tiptoes, but was unable to “do the tandem gait.” (*Id.* at 96.) During a follow up primary care appointment on May 11, 2017, petitioner reported that she was taking less of her pain medication, but her feet remained stiff and swollen. (Ex. 2, p. 1.) It was noted that petitioner’s osteoporosis score had worsened. (*Id.*) Petitioner underwent an echocardiogram on May 30, 2017, which indicated a heart murmur. (*Id.* at 56-58; Ex. 7, pp. 39-42.)

On September 15, 2017, petitioner sought a second opinion on her polyneuropathy from Dr. Michael Turner. (Ex. 6, p. 102.) Dr. Turner reported that petitioner had “ongoing symptoms from an immune mediated polyneuropathy triggered by tetanus vaccination in May 2016.” (*Id.*) Petitioner reported continued pain, numbness, and paresthesias. (*Id.*) Her neurological exam revealed that she had some numbness in her feet. (*Id.* at 103.) Dr. Turner recorded his impression as “[i]mmune mediated polyneuropathy secondary to tetanus vaccination.” (*Id.*) He started her on glutathione and superoxide dismutase, and he recommended a detox massage and sauna use, along with continued vibration plate therapy. (*Id.* at 104.) Petitioner had a follow up appointment with Dr. Cancado on November 2, 2017, during which he noted that petitioner’s symptoms were improving, and petitioner was continued on Amitriptyline. (Ex. 12, pp. 1-2.) During this visit, petitioner was able to walk on her heels and tiptoes, as well as “the tandem gait with minimal difficulty.” (*Id.* at 2.) Additionally, Dr. Cancado reported that petitioner walked with “mild unsteadiness.” (*Id.*)

Petitioner’s next follow up appointment with Dr. Cancado was on March 1, 2018. (Ex. 12, p. 4.) Dr. Cancado recorded that while petitioner’s condition had significantly improved, she experienced residual cramps in the arches and ankles,” which he suggested could be attributed to stopping Amitriptyline. (*Id.* at 4-5.) Petitioner’s neurological exam showed Tinel’s sign in both ankles and “decreased pinprick in the plantar surfaces of both feet.” (*Id.* at 5.) Dr. Cancado considered peripheral neuropathy and tarsal tunnel syndromes as possible differential diagnoses for petitioner’s continuing symptoms. (*Id.*) On September 10, 2018, petitioner had another follow up appointment with Dr. Cancado, during which it was noted that petitioner had noticed improvement in her symptoms after restarting Amitriptyline for her peripheral neuropathy. (*Id.* at 7.) Her neurological exam noted that she was able to walk on her heels and her toes and that she had no significant ataxia. (*Id.* at 8.) Petitioner was continued on Amitriptyline. (*Id.*)

In 2019, petitioner had two follow up appointments with Dr. Cancado, one on March 11, 2019, and another on September 30, 2019. (Ex. 15.) During the March 11, 2019 appointment, petitioner’s symptoms were improving, and she remained on Amitriptyline. (*Id.* at 1.) Petitioner was able to walk on both her heels and her tiptoes. (*Id.* at 2.) During her September 30, 2019 appointment, petitioner reported continued improvement on Amitriptyline and “no weakness or significant gait disturbance.” (*Id.* at 4.) She remained able to walk on her heels and tiptoes. (*Id.* at 5.)

Petitioner continued with physical therapy from April of 2017 until January of 2019, during which she continued to report pain and stiffness in her feet that would flare occasionally. (Ex. 5, pp. 1-56; Ex. 13.) On April 28, 2020, petitioner was evaluated by a

chiropractor for bilateral foot pain. (Ex. 28, p. 51.) Petitioner reported that she had been diagnosed with peripheral neuropathy “due to what is suspected as a vaccine-related injury.” (*Id.*) She reported a “dull, achy, and burning pain in her feet that is consistent and constant.” (*Id.*) Her physical exam revealed a 32% total reduced range of motion in her left ankle and a 45% total reduced range of motion in her right ankle. (*Id.* at 51-52.) Her neurological exam revealed weakness in the nerve root on the motor exam and reflex exam. (*Id.* at 52.) Her diagnoses were “[s]egmental and somatic dysfunction of lower extremity,” and pain in her left and right ankle joints. (*Id.* at 53.) She continued to see a chiropractor throughout the rest of 2020 for bilateral foot pain, weakness, and stiffness. (Ex. 28.)

b. Testimony

Petitioner submitted an affidavit and testified at the hearing. (Ex. 11; Tr. 9-45.) Petitioner testified that she received the Prevnar 13 and Tdap vaccines on May 10, 2016. (Ex. 11, ¶ 4.) She explained that, after a few days, she broke out into a rash. (*Id.* ¶ 5.) Roughly 10 days after receiving the vaccine, petitioner developed joint pain, numbness and tingling in her hands and feet, difficulty sleep, and “a general sensation of not feeling well.” (*Id.* ¶ 6.) Although she has recovered some of her function, she still experiences some pain and flare ups when she is on her feet for an extended period of time. (*Id.* ¶¶ 26-28.) Petitioner explained that, before she received the tetanus vaccine, she was involved with her grandchildren and her church, and she “loved piecing and quilting quilts for foster children.” (*Id.* ¶ 32.) However, after she received the tetanus vaccine, she had trouble “walking, climbing stairs, bending over to pick up things, lifting small items, sitting, sleeping, and paying attention because the pain was so distracting.” (*Id.* ¶ 33.) Petitioner still experiences “cramps and pain in [her] feet and ankles,” and she continues to undergo physical therapy, take supplements, and follow up with her physicians. (*Id.* ¶ 37.)

IV. Summary of Expert Opinions

a. Petitioner’s Neurologist, Alberto A. Martinez-Arizala, M.D.

Petitioner’s expert, Dr. Alberto A. Martinez-Arizala, submitted three expert reports in this case and testified at the entitlement hearing. (Exs. 16, 29, 42; Tr. 46-108, 239-52.) He was proffered without objection as an expert in neurology.⁴ (Tr. 51.) Dr.

⁴ Dr. Alberto A. Martinez-Arizala received his bachelor’s degree from Florida State University and his medical degree from the University of Miami School of Medicine. (Ex. 17, p. 1.) He completed an internship in categorical medicine at Walter Reed Army Medical Center and a residency in neurology at Letterman Army Medical Center. (*Id.*) He is board certified in neurology. (*Id.* at 2.) Dr. Martinez-Arizala currently works as the Director of the Spine Division and a professor of clinical neurology, neurosurgery, and physical therapy in the Department of Neurology at the University of Miami. (*Id.*; Ex. 16, p. 1.) He is also the principal investigator for the Miami Project to Cure Paralysis at the University of Miami. (Ex. 17, p. 2.) He was also the Chief of Spinal Cord Injury Service at the Miami VA Health Care System, where he treated patients with spinal cord disorders, multiple sclerosis, and peripheral neuropathies resulting in severe paralysis. (Ex. 16, p. 1.) He has published eight books and monographs, over 30 juried or refereed journal articles and exhibitions, and over 50 other works, publications, and abstracts. (Ex. 17, pp. 3-10.)

Martinez-Arizala opined that petitioner suffered an autoimmune polyneuropathy (Ex. 16, p. 5; Tr. 52) and, more specifically, based on her electrodiagnostic study of July 1, 2016, a moderate to severe axonal sensory and axonal and demyelinating motor neuropathy, which he described as “mostly” axonal. (Tr. 59-60, 68 (discussing Ex. 1, p. 1).) He further opined that petitioner’s neuropathy, the onset of which began about two weeks post-vaccination, can be associated with her tetanus vaccination, which he asserts is consistent with the opinion of the treating physician, Dr. Carlson. (Ex. 16, pp. 5, 7; Tr. 55, 71-72, 83-84.)

Dr. Martinez-Arizala stressed that other possible causes of peripheral neuropathy, including infections, diabetes, vitamin deficiencies, and thyroid disorders, were ruled out in petitioner’s case. (Ex. 16, pp. 5, 7.) Contrary to Dr. Donofrio, he does not agree that petitioner was suffering from a urinary tract infection prior to onset of her condition.⁵ (Tr. 73, 90-91, 244-45.) Although he acknowledged that petitioner’s urinalysis had some markers of infection, he stressed that she was asymptomatic, never treated for a urinary tract infection, and that her urine sample was contaminated. (*Id.*) Dr. Martinez-Arizala acknowledged that petitioner’s tests for some autoimmune disorders were negative; however, he explained that “negative results exclude these specific diseases, but not all autoimmune diseases.” (Ex. 29, p. 1.) In addition, although petitioner’s lab results did not show any evidence of acute inflammation, the inflammatory markers CRP and ESR “are not usually elevated . . . in autoimmune neuropathies.” (*Id.* at 3 (citing Yosria A. Altaweel et al., *Correlative Study Between C-Reactive Protein, Clinical Severity, and Nerve Conduction Studies in Guillain-Barré Syndrome*, 54 EGYPTIAN J. NEUROLOGY PSYCHIATRY & NEUROSURGERY 1 (2018) (Ex. 38); Chetana Vaishnavi et al., *C-Reactive Protein in Patients with Guillain Barré Syndrome*, 57 INDIAN J. PATHOLOGY & MICROBIOLOGY 51 (2014) (Ex. 39)); Ex. 42, p. 1 (citing G. Sivaram Naik et al., *Anti-Ganglioside Antibodies Profile in Guillain-Barré Syndrome: Correlation with Clinical Features, Electrophysiological Pattern, and Outcome*, 65 NEUROLOGY INDIA 1001 (2017) (Ex. 43); Jiting Zhu et al., *Anti-Ganglioside Antibodies in Guillain-Barre Syndrome: A Novel Immunoblotting-Panel Assay*, 12 FRONTIERS NEUROLOGY 1 (2021) (Ex. 44)).) Dr. Martinez-Arizala also acknowledged that petitioner’s MRI scans did not show enhancing signals in the nerve roots; however, “autoimmune neuropathies can be associated with enhancement of the cauda equina nerves and roots on MRI, but this is not always present.” (Ex. 29, p. 1 (citing Kenneth C. Gorson et al., *Prospective Evaluation of MRI Lumbosacral Nerve Root Enhancement in Acute Guillain-Barré Syndrome*, 47 NEUROLOGY 813 (1996) (Ex. 34)).)

Further, Dr. Martinez-Arizala noted that, while the EMG data did suggest petitioner’s neuropathy was axonal, autoimmune peripheral neuropathies can be axonal. (Ex. 29, p. 2 (citing T.E. Feasby et al., *An Acute Axonal Form of Guillain-Barré Polyneuropathy*, 109 BRAIN 1115 (1986) (Ex. 35); R. Omdal et al., *Peripheral Neuropathy in Systemic Lupus Erythematosus – A Longitudinal Study*, 103 ACTA

⁵ Initially, Dr. Martinez-Arizala disputed that urinary tract infections have been shown to cause peripheral neuropathies. (Ex. 29, p. 2.) However, he ultimately conceded that it can happen, albeit rarely. (Ex. 42, p. 1; Tr. 91-92.)

NEUROLOGICA SCANDINAVICA 386 (2001) (Ex. 36); Lasse G. Gøransson et al., *Small-Diameter Nerve Fiber Neuropathy in Systemic Lupus Erythematosus*, 63 ARCHIVES NEUROLOGY 401 (2006) (Ex. 37)); Ex. 42, pp. 2-3.) As an example, Dr. Martinez-Arizala explained that the acute motor axonal neuropathy variant of GBS has been associated with infections, such as *Campylobacter jejuni* (“*C. jejuni*”), flu, SARS-CoV-2, and Hepatitis A, as well as the flu and COVID-19 vaccines. (Ex. 42, pp. 2-3 (citing Charlene Hafer-Macko et al., *Acute Motor Axonal Neuropathy: An Antibody-Mediated Attack on Axolemma*, 40 ANNALS NEUROLOGY 635 (1996) (Ex. 54); Marko Kutleša et al., *Acute Motor Axonal Neuropathy Associated with Pandemic H1N1 Influenza A Infection*, 13 NEUROCRITICAL CARE 98 (2010) (Ex. 55); Alireza Samadi et al., *A 30-Year-Old Man with Acute Motor Axonal Neuropathy Subtype of Guillain-Barré Syndrome Having Hepatitis A Viral Infection*, 11 MIDDLE E.J. DIGESTIVE DISEASES 110 (2019) (Ex. 56); Fabio Giuseppe Masuccio et al., *A Rare Case of Acute Motor Axonal Neuropathy and Myelitis Related to SARS-CoV-2 Infection*, 268 J. NEUROLOGY 2327 (2021) (Ex. 57); T. Bueso et al., *Guillain-Barre Syndrome and COVID-19: A Case Report*, 200 CLINICAL NEUROLOGY & NEUROSURGERY 106413 (2021) (Ex. 58); Nihal Akçay et al., *Axonal Guillain-Barre Syndrome Associated with SARS-CoV-2 Infection in a Child*, 93 J. MED. VIROLOGY 5599 (2021) (Ex. 59); Catherine Jordan et al., *Guillain-Barre Syndrome-Acute Motor Axonal Neuropathy as Neurologic Sequela in a COVID-19 Critically-Ill Patient: A Case Report*, 26 Respirology 177 (2021) (Ex. 60); Fulvio A. Scorza & Josef Finsterer, *Asymptomatic SARS-CoV-2 Infection Complicated by Acute, Motor and Sensory, Axonal Neuropathy (AMSAN)*, CLINICAL NEUROLOGY & NEUROSURGERY, Oct. 2021, at 1 (Ex. 61); Cristina Petrelli et al., *Acute Motor Axonal Neuropathy Related to COVID-19 Infection: A New Diagnostic Overview*, 22 J. CLINICAL NEUROMUSCULAR DISEASE 120 (2020) (Ex. 62); Nozomu Sato et al., *Acute Transverse Myelitis and Acute Motor Axonal Neuropathy Developed After Vaccinations Against Seasonal and 2009 A/H1N1 Influenza*, 50 INTERNAL MED. 503 (2011) (Ex. 63); Vikas Dalwadi et al., *Axonal-Variant Guillain-Barre Syndrome Temporally Associated with mRNA-Based Moderna SARS-CoV-2 Vaccine*, 13 CUREUS e18291 (2021) (Ex. 64); Antonina Luca et al., *Pure Sensitive Chronic Inflammatory Axonal Polyneuropathy Following Pfizer COVID-19 Vaccine*, 43 NEUROLOGICAL SCIS. 1431 (2022) (Ex. 65); Zachary P. Morehouse et al., *A Rare Variant of Guillain-Barre Syndrome Following Ad26.COV2.S Vaccination*, 13 CUREUS e18153 (2021) (Ex. 66); Nurhan Kaya Tutar et al., *A Variant of Guillain-Barre Syndrome After SARS-COV-2 Vaccination: AMSAN*, 74 IDEGGYOGY SZ. 286 (2021) (Ex. 67)).)

Dr. Martinez-Arizala also noted that petitioner was diagnosed with leukocytoclastic vasculitis, “an immune mediated vasculitis, which has been reported in association with influenza and tetanus vaccination.” (Ex. 29, p. 1 (citing Stella X. Chen & Philip R. Cohen, *Cutaneous Leukocytoclastic Vasculitis Following Influenza Vaccination in Older Adults: Report of Bullous Purpura in an Octogenarian After Influenza Vaccine Administration*, 10 CUREUS e2323 (2018) (Ex. 30); Seena Monjazeib et al., *A Case of Leukocytoclastic Vasculitis Following Influenza Vaccination*, 2 JAAD CASE REPS. 340 (2016) (Ex. 31); Sissi Cao & Dongmei Sun, *Leucocytoclastic Vasculitis Following Influenza Vaccination*, 2017 BMJ CASE REPS. J. 1 (Ex. 32); Liviana Da Dalt et al., *Henoch-Schönlein Purpura and Drug and Vaccine Use in Childhood: A Case-Control Study*, 42 ITALIAN J. PEDIATRICS 60 (2016) (Ex. 33)).) Though respondent’s

experts suggested that vasculitis is itself a known cause of neuropathies, Dr. Martinez-Arizala suggested that the leukocytoclastic vasculitis would be a separate, comorbid reaction to vaccination. (Ex. 42, pp. 3-4; Tr. 71-72, 92-93.) Moreover, he felt that the presence of the purported leukocytoclastic vasculitic event added further support to the notion that petitioner's post-vaccination clinical course was consistent with an immune-mediated process. (Tr. 63.) Skin biopsies are often used to confirm leukocytoclastic vasculitis, but Dr. Martinez-Arizala opined that "diagnosis can be strongly suggested by the history, physical examination, clinical course, and laboratory tests." (Ex. 42, p. 3.) Ultimately, however, he conceded that petitioner's rash "could have been anything," though he maintained that the timing at least raises a question as to whether it represented a post-vaccination vasculitis. (Tr. 252; *see also id.* at 71-72, 92-93.) Although respondent had raised that petitioner's neuropathy did not respond to the course of steroids she received for her rash, Dr. Martinez-Arizala explained that while steroids can be effective in treatment of autoimmune neuropathies, "they are not used for acute neuropathies." (Ex. 29, p. 3.)

Dr. Martinez-Arizala's theory of how vaccines cause autoimmune disorders is based on molecular mimicry, which is "where components of the vaccine possess sequence similarities between it and specific human proteins that results in the cross-activation of autoreactive T or B cells." (Ex. 16, p. 5.) He explained further that this cross reactivity "causes a reaction of the immune system against the pathogenic antigens that may harm the similar human proteins, essentially causing autoimmune disease." (*Id.* (citing Yahel Segal & Yehuda Shoenfeld, *Vaccine-Induced Autoimmunity: The Role of Molecular Mimicry and Immune Crossreaction*, 15 CELLULAR & MOLECULAR IMMUNOLOGY 586 (2018) (Ex. 18); Javaraiah Srinivasappa et al., *Molecular Mimicry: Frequency of Reactivity of Monoclonal Antiviral Antibodies with Normal Tissues*, 57 J. VIROLOGY 397 (1986) (Ex. 19)); *see also* Robert S. Fujinami & Michael B.A. Oldstone, *Amino Acid Homology Between the Encephalitogenic Site of Myelin Basic Protein and Virus: Mechanism for Autoimmunity*, 230 SCIENCE 1043 (1985) (Ex. 74); Anand M. Gautam et al., *A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis*, 176 J. EXPERIMENTAL MED. 605 (1992) (Ex. 75); Lawrence Steinman, *Autoimmune Disease*, 269 SCI. AM. 106 (1993) (Ex. 76).) Dr. Martinez-Arizala listed the increase in GBS following swine flu vaccination as an example of autoimmune peripheral neuropathy related to vaccination, where there is also additional evidence that molecular mimicry was the link between the disease and vaccination. (Ex. 16, p. 6 (citing Segal & Shoenfeld, *supra*, at Ex. 18; 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) (Ex. 21; *see also* Ex. 53)).)

As it pertains to the Tdap vaccine at issue in this case, Dr. Martinez-Arizala cited several case reports "of peripheral neuropathies related to the administration of the tetanus vaccine." (Ex. 16, p. 6 (citing Kannikar Kongbunkait et al., *Clinical Manifestations and Outcomes of Guillain-Barré Syndrome After Diphtheria and Tetanus Vaccine (dT) During a Diphtheria Outbreak in Thailand: A Case Series*, 19 NEUROLOGY ASIA 137 (2014) (Ex. 22) (reporting four cases of patients who developed GBS between

four weeks and four months after receiving the diphtheria and tetanus vaccine during a diphtheria outbreak in Thailand); Hussam Ammar, *Guillain-Barré Syndrome After Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccine: A Case Report*, 5 J. MED. CASE REPS. 502 (2011) (Ex. 23) (reporting one case of GBS just over one week following a tetanus toxoid, reduced diphtheria toxoid and acellular pertussis vaccine); W. Baust et al., *Peripheral Neuropathy After Administration of Tetanus Toxoid*, 222 J. NEUROLOGY 131 (1979) (Ex. 24) (reporting the case of a patient who developed peripheral neuropathy three weeks after receiving a tetanus toxoid vaccination); George I. Blumstein & Harold Kreithen, *Peripheral Neuropathy Following Tetanus Toxoid Administration*, 198 JAMA 1030 (1966) (Ex. 25) (reporting a case of a patient who developed a fever, rash, and motor and sensory paralysis within 24 hours of receiving a tetanus toxoid vaccine, and was eventually diagnosed with peripheral neuropathy); Leon Reinstein et al., *Peripheral Neuropathy After Multiple Tetanus Toxoid Injections*, 63 ARCHIVES PHYSICAL MED. & REHAB. 332 (1982) (Ex. 26) (reporting the case of a patient who received three tetanus toxoid vaccines within five months and developed occasional numbness of the feet 24 hours after the first shot, as well as sensorimotor polyneuropathy roughly six weeks after his final vaccination); Ute Quast et al., *Mono- and Polyneuritis After Tetanus Vaccination (1970-1977)*, 43 INT'L SYMP. IMMUNIZATION 25 (1979) (Ex. 27) (reporting 22 cases of neuritis between three hours and two weeks following tetanus toxoid vaccination)).)

Finally, with regard to timing, Dr. Martinez-Arizala explained that petitioner developed symptoms less than two weeks following her tetanus vaccination. (Ex. 16, p. 7.) Dr. Martinez-Arizala opines that this timeline “is quite consistent with the literature.” (*Id.*) He also opined that petitioner’s leukocytoclastic vasculitis developed 10 days after vaccination, which “fits what has been reported in the literature and the time it is generally accepted for the body to mount an abnormal immune response.” (Ex. 42, p. 4.)

b. Respondent’s Expert, Peter D. Donofrio, M.D.

Respondent’s first expert, Dr. Peter D. Donofrio, provided three expert reports and testified during the entitlement hearing. (Exs. A, E, G; Tr. 109-61.) During the hearing, he was proffered without objection as an expert in nerve and muscular disorders, as well as electrodiagnostic medicine.⁶ (Tr. 114.) Stressing that there are over 100 known causes of peripheral neuropathies (*Id.* at 115-16), Dr. Donofrio

⁶ Dr. Peter D. Donofrio received his medical degree from Ohio State University School of Medicine. (Ex. B, p. 1.) From there, he completed two residencies, one in internal medicine at Good Samaritan Hospital and another in neurology at the University of Michigan Medical Center. (*Id.* at 2.) Additionally, he completed a neuromuscular fellowship at the University of Michigan. (*Id.*) He is board certified in internal medicine, psychiatry and neurology, electro diagnostic medicine, and neuromuscular medicine. (*Id.*; Ex. A, p. 1.) He currently works as the Vice-Chair of Clinical Affairs in the Department of Neurology, as a professor of neurology, and as the Director of the Neuromuscular Division at Vanderbilt University School of Medicine. (Ex. B, p. 1; Ex. A, p. 1.) He has experience evaluating “a spectrum of neuropathies such as Guillain-Barre Syndrome (GBS) and CIDP and the related condition of Miller Fisher Syndrome (MFS).” (Ex. A, p. 1.) He has published over 100 journal articles, in addition to one textbook, and over 100 book chapters and abstracts. (Ex. B, pp. 13-32; Ex. I, pp. 13-32.)

highlighted the lack of laboratory support for an autoimmune etiology for petitioner's condition. (Ex. A, p. 7.)

Specifically, Dr. Donofrio noted that petitioner underwent testing for several autoimmune conditions and antibodies, none of which could be confirmed. (Ex. A, p. 7.) While Dr. Donofrio does acknowledge that "negative antibody studies do not rule out all autoimmune diseases," he still maintained that "[i]t is hard to justify the use of the term auto-immune without any supportive evidence." (Ex. E, p. 1.) Additionally, petitioner's MRI scans "did not show enhancing signals in the nerve roots, another abnormality that would support the concept of an auto-immune neuropathy," which can be a sign of autoimmune neuropathies, such as GBS, though he emphasizes that petitioner was not diagnosed with GBS. (Ex. A, pp. 7-8; Ex. E, p. 1.) He also noted that petitioner did not undergo a spinal tap or nerve biopsy, both of which would have aided in the diagnosis of an autoimmune neuropathy. (Ex. A, p. 7.) Dr. Donofrio further noted of petitioner's electrodiagnostic studies that, "[e]ven though the two sets of nerve conduction studies were described as demyelinating, analysis of the raw data suggests an axonal loss process rather than a demyelinating one." (*Id.* at 8 (citing 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) (Ex. A, Tab 1)).) Dr. Donofrio opined that this would leave an inflammatory autoimmune condition less likely, though he acknowledged that axonal neuropathies can be autoimmune. (Tr. 127-29; Ex. G, p. 1.) Dr. Donofrio also noted briefly that petitioner's leukocytoclastic vasculitis diagnosis was based on clinical features alone, while "[d]ocumentation of vasculitis of peripheral nerve would require a biopsy of an involved peripheral nerve." (Ex. E, p. 1; Ex. G, pp. 2-3.) Dr. Donofrio contends that because petitioner did not have a biopsy nor did she meet all the criteria for classification of hypersensitivity vasculitis (leukocytoclastic vasculitis), she did not have an autoimmune vasculitis. (Ex. G, p. 2.) He did acknowledge during the hearing, however, that a biopsy would not be a realistic expectation in clinical practice. (Tr. 145-46.)

Dr. Donofrio also explained that, if petitioner's condition is autoimmune, other possible explanations for petitioner's condition are a urinary tract infection that petitioner was diagnosed with on May 2, 2016, or a viral exanthem evidenced by petitioner's rash.⁷ (Ex. A, p. 8 (citing Ex. 4, p. 51).) He explained that "[t]he same hypothesis for

⁷ Dr. Donofrio's opinion on this point is harder to understand following the hearing. While he consistently distinguished the idea of holding an opinion that petitioner's neuropathy was an autoimmune reaction to an infection from the idea of holding the opinion that autoimmunity stemmed from her vaccination (Tr. 130-31), he also ultimately agreed that he believes a urinary tract infection more likely than not caused petitioner's neuropathy (*Id.* at 157-59), a conclusion which would also necessarily mean that an autoimmune etiology was more likely than not (*Id.* at 131 (agreeing that if the court accepts his testimony doubting the presence of autoimmunity, then the infection would be an unlikely cause)). Taking Dr. Donofrio's opinion as a whole (both his reports and his complete testimony), he seems to be opining that, even in the absence of clinical indicators of autoimmunity, patient history can still potentially be informative of the etiology of a polyneuropathy. However, based on this patient's history, her proposed

molecular mimicry would apply to the antigen of the agent causing the UTI.”⁸ (*Id.*) Dr. Donofrio also alternatively suggested that petitioner’s joint pain, back pain, swollen joints, stiffness, and sensory motor defects could be evidence of a rheumatologic condition, which have been associated with peripheral neuropathy. (*Id.* (citing Haatem Reda & Russell L. Chin, *Peripheral Neuropathies of Rheumatologic Disease and Gluten-Related Disorders*, 34 SEMINARS NEUROLOGY 413 (2014) (Ex. A, Tab 2)).)

Regarding petitioner’s theory, Dr. Donofrio noted that petitioner cited several pieces of medical literature on the association between the tetanus vaccine and GBS, and a single case report of radial nerve palsy and brachial plexopathy after tetanus vaccination. (Ex. A, p. 9.) He observed, however, that petitioner has not been diagnosed with GBS, and opined that “petitioner’s expert did not discuss a medical theory as to how the Tdap vaccine or the Prevnar vaccine or the combination of the two caused her polyneuropathy.” (*Id.*) He noted that the Institute of Medicine “does not have a section on tetanus vaccination and peripheral neuropathy.”⁹ (*Id.*) Dr. Donofrio also suggested that, because petitioner’s expert described petitioner’s vasculitis as “skin isolated small vessel vasculitis . . . not associated with peripheral neuropathies,” petitioner’s expert ruled out “any association of the petitioner’s polyneuropathy with the skin isolated vessel vasculitis as the polyneuropathy would represent a second organ system.” (Ex. G, p. 3.) Dr. Donofrio explained that, even if petitioner’s rash was temporally related to her vaccination, “petitioner could easily have developed a rash from other causes.” (*Id.*)

c. Respondent’s Expert, Penelope A. Morel, M.D.

Respondent’s second expert, Dr. Penelope Morel, submitted three expert reports and testified at the hearing in this case. (Exs. C, F, H; Tr. 164-238.) During the hearing

urinary tract infection, but not her vaccination, could be so informative. Under this logic, the presence of an identifiable autoimmune trigger contributes to the overall conclusion that autoimmunity is a likely etiology. Thus, even having concluded that a urinary tract infection is not preponderantly supported (see note 19, *infra*), I do not take Dr. Donofrio to be conceding that petitioner’s condition is autoimmune.

⁸ Urinary tract infections are commonly due to bacteria, which “have antigens that can lead to auto-immune responses.” (Ex. E, p. 1.) For example, “the most common precipitating infection for GBS is campylobacter jejuni, a bacterial infection of the gut.” (*Id.*) Dr. Donofrio stated that, because a urine culture was not ordered, he could not identify the cause of petitioner’s UTI and could not, therefore, “comment on antigens in the organism that may have precipitated an auto-immune process.” (Ex. G, pp. 1-2.) However, as further support of his conclusion that petitioner’s condition could have been caused by her UTI, he cited several pieces of medical literature. (*Id.* (citing Yoon-Sik Jo et al., *Recurrent Guillain-Barré Syndrome Following Urinary Tract Infection by Escherichia coli*, 33 J. KOREAN MED. SCI. e29 (2018) (Ex. G, Tab 2); Yuriko Kono et al., *Rapidly Progressive Guillain-Barré Syndrome Following Escherichia coli Infection*, 46 INTERNAL MED. 589 (2007) (Ex. G, Tab 3); Aaron Fischer & Juan Avila, *Guillain-Barré Syndrome Following an Extended-Spectrum Beta-Lactamase Escherichia coli Urinary Tract Infection: A Case Report*, 13 CUREUS e19673 (2021) (Ex. G, Tab 4); Wisit Cheungpasitporn et al., *Association Between Acute Urinary Retention and Guillain-Barré Syndrome*, 29 AM. J. EMERGENCY MED. 373 (2012) (Ex. G, Tab 5)).)

⁹ Referring to the IOM’s 2012 report later filed as Court Exhibit II.

she was proffered without objection as an excerpt in immunology.¹⁰ (Tr. 166.) Although she acknowledged that “several polyneuropathies have an immune etiology” (Ex. C, p. 3 (citing Marinos C. Dalakas, *Pathogenesis of Immune-Mediated Neuropathies*, 1852 *BIOCHIMICA ET BIOPHYSICA ACTA* 658 (2015) (Ex. C, Tab 1))), Dr. Morel opined that such conditions are well defined, and petitioner has not been diagnosed with any of them (Tr. 169-71).

Some immune mediated neuropathies, like GBS, are associated with “antibodies generated to neuronal structures,” while others, such as MS or CIDP, “are associated with immune cell infiltrates and histopathological analysis will reveal the accumulation of immune cells, such as T cells or macrophages, in the area of demyelination.” (Ex. C, p. 1 (citing Dalakas, *supra*, at Ex. C, Tab 1; John A. Goodfellow & Hugh J. Willison, *Antiganglioside, Antiganglioside-Complex, and Antiglycolipid-Complex Antibodies in Immune-Mediated Neuropathies*, 29 *CURRENT OP. NEUROLOGY* 572 (2016) (Ex. C, Tab 2); Hugh J. Willison et al., *Guillain-Barré Syndrome*, 388 *LANCET* 717 (2016) (Ex. C, Tab 3); Jolien Wolbert et al., *Deciphering Immune Mechanisms in Chronic Inflammatory Demyelinating Polyneuropathies*, 5 *JCI INSIGHT* e132411 (2020) (Ex. C, Tab 4)).) Additionally, some immune mediated neuropathies include vasculitis, during which “the inflammatory response is directed to the vessels that supply the nerves.” (*Id.* at 3 (citing Michael P. Collins & Robert D. Hadden, *The Nonsystemic Vasculitic Neuropathies*, 13 *NATURE* 302 (2017) (Ex. C, Tab 5); S.H.B. Hawke et al., *Vasculitic Neuropathy: A Clinical and Pathological Study*, 114 *BRAIN* 2175 (1991) (Ex. C, Tab 6); Linda R. Watkins & Steven F. Maier, *Neuropathic Pain: The Immune Connection*, 12 *PAIN*, March 2004 (Ex. C, Tab 7)).) Dr. Morel explained that, generally, immune polyneuropathies are “responsive to immune-modulating therapies,” and “will have some evidence of an aberrant immune response in the form of evidence of systemic inflammation, . . . the presence of circulating auto-antibodies[,] and/or immune infiltrates in the relevant regions.” (*Id.* (citing Wolbert et al., *supra*, at Ex. C, Tab 4; Helmar C. Lehmann et al., *Pathogenesis and Treatment of Immune-Mediated Neuropathies*, 2 *THERAPEUTIC ADVANCES NEUROLOGICAL DISORDERS* 261 (2009) (Ex. C, Tab 8)).)

In petitioner’s case, however, Dr. Morel explained that “potential causes of immune-mediated pain were investigated” yet “none of these tests supported the notion that her polyneuropathy was caused by the immune system.” (Ex. C, p. 3.) Specifically,

¹⁰ Dr. Penelope A. Morel received her Bachelor of Medicine degree from the University of Southampton Medical School in the United Kingdom in 1979 and her Doctor of Medicine degree from the University of Geneva in Switzerland in 1983. (Ex. D, p. 1.) She completed three fellowships, one at the Scripps Clinic and Research Foundation in 1985, one in immunology at Stanford University Medical Center in 1987, and one at the University of Pittsburgh Cancer Institute in 1989. (*Id.* at 2.) She currently works as an affiliate member at the Center for Vaccine Research and University of Pittsburgh Cancer Institute, as well as a professor in the Department of Immunology and the Division of Rheumatology at the University of Pittsburgh. (*Id.* at 2-3.) Her research focuses on the field of autoimmunity. (Ex. C, p. 1.) Specifically, she has “examined genetic predispositions in various human rheumatological conditions, focusing on HLA and FcR alleles,” and she has focused on “how autoimmunity is initiated and how to design therapies that would restore peripheral tolerance.” (*Id.*) She has published 70 refereed articles; 38 articles, chapters, and reviews; and 95 abstracts. (Ex. D, pp. 4-21; Ex. C, p. 1.)

[t]here was no evidence for any auto-antibody production and the CRP and ESR levels were consistently low. No biopsies were performed and so it is not possible to determine whether immune infiltrates were present. Electrophysiological tests were compatible with a combined sensory and motor demyelinating neuropathy. In addition, [petitioner] was extensively investigated for rheumatoid arthritis, fibromyalgia, systemic lupus erythematosus and other autoimmune disorders but all tests were negative.

(*Id.*)

Dr. Morel explained that a biopsy would have been required for a definitive diagnosis, but one was not performed. Nonetheless, if petitioner was suffering from an immune mediated polyneuropathy, “there would have been evidence of inflammation, either locally or systemically,” resulting in elevated levels of CRP or ESR. (Ex. H, p. 1; Ex. F, p. 1.) While Dr. Morel acknowledged that these would not be elevated in patients with GBS, “[e]levated CRP and ESR are seen in situations of ongoing acute or chronic inflammation, such as chronic inflammatory demyelinating polyneuropathy, systemic autoimmune diseases, and other causes of immune-mediated polyneuropathy.” (Ex. F, p. 1.) Other possible signs of inflammation include elevated WBCs, presence of auto-antibodies, biopsy evidence of immune cell infiltration, and response of symptoms to anti-inflammatory treatment. (Ex. H, p. 1.) There is no evidence of any of these in petitioner’s records. (*Id.*) Therefore, without evidence of inflammation, it is “not possible to conclude that an autoimmune response was responsible for the polyneuropathy that she experienced.” (Ex. F, p. 1.) Therefore, Dr. Morel opined that petitioner suffered from idiopathic polyneuropathy. (Ex C, p. 4.)

Dr. Morel also noted that petitioner did develop “a rash at the onset of her illness that resolved following a short course of steroids.” (Ex. C, p. 3.) She explained that while petitioner’s rash resolved after the steroids, her neuropathy did not, “casting doubt on an inflammatory cause for her symptoms.” (*Id.* at 3-4.) She notes that petitioner’s expert opines that petitioner’s rash was caused by leukocytoclastic vasculitis and cited case reports in which Henoch-Schonlein purpura was preceded by a DTaP vaccination. (Ex. H, p. 1 (citing Sara De Nitto et al., *Henoch-Schonlein Purpura Following Anti Diphtheria, Tetanus, Acellular Pertussis and Inactivated Polio (DTaP-IPV) Vaccine: A Case Report*, 12 ANNALS CASE REPS. 264 (2019) (Ex. 69); Bindu Nair et al., *Leukocytoclastic Vasculitis Following DPT Vaccination*, 18 TROPICAL J. MED. RSCH. 1 (2015) (Ex. 70)).) However, Dr. Morel explained that these cases “were characterized by an increased WBC count, fever and evidence of inflammation.” (*Id.* at 1-2.) By contrast, she noted that petitioner’s rash “was described as acute dermatitis and did not appear to have the clinical characteristics of either [leukocytoclastic vasculitis] or [Henoch-Schonlein purpura], and was not associated with increased WBC count or fever.” (*Id.* at 2.) Dr. Morel noted further that, even after petitioner’s diagnosis of acute inflammatory polyneuropathy, petitioner “was not treated with the standard of care for such a condition.” (Ex. C, p. 4 (citing Wolbert et al., *supra*, at Ex. C, Tab 4; Lehmann et al., *supra*, at Ex. C, Tab 8); Ex. F, pp. 1-2.)

Regarding petitioner's theory of causation, Dr. Morel acknowledged that there are case reports linking the GBS to vaccination but opined that only a causal relationship with the flu vaccine has been documented. (Ex. C, p. 4 (citing 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) (Ex. C, Tab 9)).) Instead, Dr. Morel noted that a large study found no association between GBS and the tetanus vaccine. (*Id.* (citing Jessica Tuttle et al., *The Risk of Guillain-Barré Syndrome After Tetanus-Toxoid—Containing Vaccines in Adults and Children in the United States*, 87 AM. J. PUBLIC HEALTH 2045 (1997) (Ex. C, Tab 10)); Ex. F, p. 2.) Although there are case reports suggesting that GBS or CIDP followed tetanus vaccination, Dr. Morel explained that some of these case reports were reported prior to 1985 (Baust et al., *supra*, at Ex. 24; Blumstein & Kreithen, *supra*, at Ex. 25; Reinstein et al., *supra*, at Ex. 26; Quast et al., *supra*, at Ex. 27), and several described cases that occurred after multiple vaccinations (Baust et al., *supra*, at Ex. 24; Reinstein et al., *supra*, at Ex. 26). (Ex. C, p. 4.) She also noted that several cases included patients who were treated successfully with IVIG. (*Id.* (citing Kongbunkait et al., *supra*, at Ex. 22; Ammar, *supra*, at Ex. 23; Karissa L. Gable et al., *Distal Acquired Demyelinating Symmetric Neuropathy After Vaccination*, 14 J. CLINICAL NEUROMUSCULAR DISEASE 117 (2013) (Ex. C, Tab 11)).)

Both genetic and environmental factors, including viral infections, contribute to the development of autoimmunity. (Ex. C, p. 5 (citing Maria K. Smatti et al., *Viruses and Autoimmunity: A Review on the Potential Interaction and Molecular Mechanisms*, 11 VIRUSES 762 (2019) (Ex. C, Tab 12)).) Thus, Dr. Morel explained that “molecular mimicry has been around for a long time as a way to explain how infections may precipitate autoimmune diseases.” (*Id.*) In this context, “an appropriate immune response is mounted to a pathogen resulting in the generation of pathogen-specific T cells and antibodies,” which then “may cross react on self-proteins and thereby initiate autoimmunity.” (*Id.*) She explained that this hypothesis has been difficult to prove; however, “[o]ne of the best examples of molecular mimicry is GBS which is usually preceded by an infection of the respiratory or gastrointestinal tract,” which led “to the development of antibodies that target peripheral nerve gangliosides and lead to axonal damage and nerve conduction defects.” (*Id.* (citing Yhojan Rodríguez et al., *Guillain-Barré Syndrome, Transverse Myelitis and Infectious Diseases*, 15 CELLULAR & MOLECULAR IMMUNOLOGY 547 (2018) (Ex. C, Tab 13)).) This is the same theory by which the flu vaccine has been implicated in causing GBS. (*Id.* (citing Schonberger et al., *supra*, Ex. C, Tab 9)).)

Dr. Morel stressed that “[t]emporal association with vaccination does not prove that a vaccine caused a condition; evidence substantiating that an immune process is responsible for the disease should also be present.” (Ex. C, p. 5.) She opined that petitioner did not have any of this substantiating evidence, therefore “there appears to be very little evidence that the pain and neurological symptoms experienced by [petitioner] were immune mediated.” (*Id.*)

V. Analysis

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a “reputable medical theory,” showing that the subject vaccine can cause the type of injury alleged. *Pafford v. Sec’y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec’y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff’d*, 451 F.3d 1352 (Fed. Cir. 2006)). Such a theory need only be “legally probable, not medically or scientifically certain.” *Knudsen v. Sec’y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. See *Andreu v. Sec’y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, “[a] petitioner must provide a ‘reputable medical or scientific explanation’ for [the proposed causal] theory. While it does not require medical or scientific certainty, it must still be ‘sound and reliable.’” *Boatmon v. Sec’y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (citation omitted) (first quoting *Moberly v. Sec’y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010); then quoting *Knudsen*, 35 F.3d at 548-49).

In this case, petitioner contends that she suffered an autoimmune axonal polyneuropathy caused by her vaccination. (ECF No. 95, pp. 3-4.) In furtherance of that contention, her expert, Dr. Martinez-Arizala, advanced a general causation opinion based on the concept of molecular mimicry. (Ex. 16, p. 5; Tr. 57-58.) Petitioner argues that, because molecular mimicry is generally accepted as a mechanism of autoimmunity, most notably in the context of Guillain-Barré Syndrome, and because polyneuropathy can likewise be autoimmune, she has therefore satisfied her preponderant burden of proof. (ECF No. 95, pp. 4-5.) She argues that respondent’s experts rely on an “unattainably high standard of proof rather than acknowledging the broader context of the evidence.” (*Id.* at 3.) In particular, petitioner argues that she is not obligated to prove either an exact homology or a relevant host antigen to support her invocation of molecular mimicry, and that her only obligation is to present a “plausible, biologically tenable theory.” (ECF No. 98, p. 3.) Petitioner’s arguments relative to *Althen* prong one are not persuasive.

Of course, petitioner is not obligated to come forward with a specific biologic mechanism to meet her burden of proof. *Kottenstette v. Sec’y of Health & Human Servs.*, 861 F. App’x 433, 440-41 (Fed. Cir. June 15, 2021) (citing *Knudsen*, 35 F.3d at 549 and *Simanski v. Sec’y of Health & Human Servs.*, 671 F.3d 1368, 1384 (Fed. Cir. 2012)). However, because she does seek to meet her burden of proof under *Althen* prong one at least in part via presentation of such a mechanism – in this case, molecular mimicry – the soundness of that assertion must be assessed. *E.g.*, *Howard v. Sec’y of Health & Human Servs.*, No. 16-1592V, 2022 WL 4869354, at *24 (Fed. Cl. Spec. Mstr. Aug. 31, 2022) (“[P]etitioners are never required to establish [a]

mechanism—but they often attempt to do so, and therefore it is reasonable to evaluate their success in the effort.”), *mot. for rev. denied sub nom. Howard v. United States*, No. 16-1592V, 2023 WL 4117370 (Fed. Cl. May 18, 2023), *aff’d per curiam sub nom. Howard v. Sec’y of Health & Human Servs.*, No. 2023-1816, 2024 WL 2873301 (Fed. Cir. June 7, 2024). That is, although petitioners are not required to prove their theories in any one way, they must in all events support their proffered theory with sound and reliable scientific explanation, *Boatmon*, 941 F.3d at 1359, and provide evidence that establishes on balance that the vaccine at issue can more likely than not cause the injury at issue, *Cerrone v. Sec’y of Health & Human Servs.*, 146 F.4th 1113, 1120-23 (Fed. Cir. 2025).

As Dr. Martinez-Arizala explained, molecular mimicry is a concept whereby “components of the vaccine possess sequence similarities between it and specific human proteins that results in the cross activation of autoreactive T or B cells,” and that “[t]his immune cross reactivity causes a reaction of the immune system against the pathogenic antigens that may harm the similar human proteins, essentially causing autoimmune disease.” (Ex. 16, p. 5; see also Tr. 57-58.) Petitioner is correct that this is “a generally accepted scientific principle,” but “mere invocation of the scientific term does not carry a petitioner’s burden in a Program case.” *Deshler v. Sec’y of Health & Human Servs.*, No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec’y of Health & Human Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019)). Afterall, a “special master is entitled to require some indicia of reliability to support the assertion of the expert witness.” *Moberly*, 592 F.3d at 1324.

Petitioner provides no citation or explanation to support her suggestion that she can *reasonably* invoke molecular mimicry without any demonstration of either homology or a host antigen. (ECF No. 98, p. 3.) By contrast, Dr. Morel reasonably suggested that homology, though not enough on its own, is the fair starting point for assessing molecular mimicry. (Tr. 181-82.) This is consistent with prior program experience, which has stressed that even the identification of homology, which may be viewed merely as a threshold finding, “does not necessarily mean the similarity has significance to the immune system.” *Tullio v. Sec’y of Health & Human Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *aff’d*, 149 Fed. Cl. 448 (2020); see also *Caredio ex rel. D.C. v. Sec’y of Health & Human Servs.*, No. 17-0079V, 2021 WL 4100294, at *31 (Fed. Cl. Spec. Mstr. July 30, 2021) (“[D]emonstration of homology alone is not enough to establish a preponderant causation theory.” (emphasis omitted) (citing *Schultz v. Sec’y of Health & Human Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020))), *mot. for rev. denied*, No. 17-79V, 2021 WL 6058835 (Fed. Cl. Dec. 3, 2021). Thus, for example, in *Brayboy*, an omnibus proceeding addressing autoimmune premature ovarian insufficiency, the special master found it sufficient that the petitioners had, more robustly, “identified cross-reaction between components of the vaccine and proteins in the body that are directly responsible for the health and productivity of the organ at issue,” while also noting that further requiring additional steps, or insisting on direct, testable evidence would impermissibly heighten petitioner’s burden of proof. *Brayboy v. Sec’y of Health &*

Human Servs., No. 15-183V, 2021 WL 4453146, at *19 (Fed. Cl. Spec. Mstr. Aug. 30, 2021). Ultimately, when assessing theories based on molecular mimicry in light of petitioner's preponderant burden of proof, "[t]he line must be drawn somewhere between speculation and certainty." *Id.*

In this case, petitioner has not provided any evidence that suggests that the Tdap vaccine can cause axonal polyneuropathy via molecular mimicry. As respondent noted in his post-hearing brief (ECF No. 97, p. 14), Dr. Martinez-Arizala conceded during the hearing that he has neither proposed a homology that would support molecular mimicry nor identified a component of the vaccine that would cross-react within the body. (Tr. 103-04.) Instead, Dr. Martinez-Arizala sought to evidence the necessary connection circumstantially. However, while petitioners are permitted to rely on circumstantial evidence, *Althen*, 418 F.3d at 1280, prior petitioners who have successfully relied upon molecular mimicry have generally presented more substantial, and more persuasive, evidence, either direct or circumstantial, than is present in this case. *E.g.*, *Brayboy*, 2021 WL 4453146, at *19 (petitioner prevailed on molecular mimicry theory by presenting evidence of cross-reaction); *E.M. v. Sec'y of Health & Human Servs.*, No. 14-753V, 2021 WL 3477837, at *36-39 (Fed. Cl. Spec. Mstr. July 9, 2021) (molecular mimicry supported by proposed homology and identification of corresponding targets associated with the condition at issue); *Pierson v. Sec'y of Health & Human Servs.*, No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022) (petitioner prevailed on molecular mimicry theory with evidence circumstantially demonstrating a proposed homology, a relevant autoantibody, and cross-reactive potential); *Riese v. Sec'y of Health & Human Servs.*, No. 19-477V, 2025 WL 3463267, at *19-22 (Fed. Cl. Spec. Mstr. Nov. 3, 2025) (petitioner prevailed on molecular mimicry theory with demonstration of a proposed homology and evidence of a pathogenic antibody).

Dr. Martinez-Arizala first and foremost cited the established link between two particular flu vaccine formulations (the Swine flu and H1N1) and GBS as "the best example we have" of post-vaccinal molecular mimicry. (Tr. 58; Ex. 16, pp. 5-6.) However, Dr. Martinez-Arizala acknowledged that petitioner's own condition is not directly analogous to GBS. (Tr. 58-59; Ex. 29, p. 3.) Thus, petitioner's own condition is at a minimum one step removed from this body of evidence. As Dr. Morel reasonably explained, those peripheral neuropathies that are considered immune-mediated tend to be well-defined and have identified immune processes. (Tr. 170-71.) Accordingly, this weakens the strength of this evidence as applied to this case.¹¹ Without more, mere analogy to GBS is not enough to support invocation of molecular mimicry vis-à-vis other types of peripheral neuropathy. *Accord McGill v. Sec'y of Health & Human Servs.*, No. 15-1485V, 2023 WL 3813524, at *26-28 (Fed. Cl. Spec. Mstr. May 11, 2023) (finding GBS to be a "flawed" analogy relative to small fiber neuropathy and accepting respondent's expert's testimony that "molecular mimicry involves a specific interaction that is not generalizable across differing contexts" and that "molecular mimicry is not the

¹¹ To be clear, respondent's experts do agree that GBS can be at least somewhat informative of the potential causes of other peripheral neuropathies. (Tr. 208.) Thus, I do not conclude that the proffered evidence pertaining to the causes of GBS is entirely irrelevant.

cause of all autoimmune disease”); *Houston v. Sec’y of Health & Human Servs.*, No. 18-420V, 2021 WL 4259012, at *16-17 (Fed. Cl. Spec. Mstr. Aug. 19, 2021) (explaining that “the filed literature establishes that CIDP should not be viewed as merely a ‘chronic’ form of GBS, even though both are peripheral neuropathies involving nerve demyelination” and that “while there is evidence supporting molecular mimicry as a disease mechanism driving GBS, Dr. Callaghan explained that there is no comparable evidence associating molecular mimicry with CIDP”).

Additionally, while examples of other vaccines resulting in *presumed* molecular mimicry¹² may reasonably challenge Dr. Morel’s assertion that vaccine proteins should be viewed more skeptically than complete infectious organisms as vehicles for molecular mimicry (Tr. 180-81), they do nothing to implicate the specific components of the Tdap vaccine as potential molecular mimics. Instead, petitioner has presented literature specifically implicating structures in the *flu* vaccine with the induction of anti-ganglioside antibodies (Nachamkin et al., *supra*, at Ex. 53), and has further presented literature that in turn associates anti-ganglioside antibodies with the axonal variant of GBS (Hafer-Macko et al., *supra*, at Ex. 54). Petitioner has not presented any evidence purporting to demonstrate that the Tdap vaccine (or any component of the vaccine) elicits the anti-ganglioside antibodies underlying petitioner’s theory.

Indeed, none of the three articles Dr. Martinez-Arizala cited involving post-Tdap GBS implicated anti-ganglioside antibodies. (Kongbunkait et al., *supra*, at Ex. 22 (reporting four cases of GBS arising after diphtheria-tetanus vaccination, and noting that anti-gangliosides are “believed” to be an explanation for post-vaccination GBS, but providing no data to indicate that the case subjects had such antibodies); Ammar, *supra*, at Ex. 23 (single case report of post-Tdap GBS without any discussion of anti-ganglioside antibodies); Quast et al., *supra*, at Ex. 27 (reporting on 22 cases of various neuropathies following tetanus vaccination, including two cases diagnosed as GBS, with no discussion of anti-ganglioside antibodies).)

Potentially the strongest piece of evidence suggesting that tetanus-containing vaccines can cause GBS via molecular mimicry is the 1994 IOM report introduced into the record as Court Exhibit I. (1994 IOM report, *supra*, at Ct. Ex. I, pp. 65, 103-07); see note 3, *supra*.) However, this publication equates GBS with acute demyelinating polyneuritis (“AIDP”) whereas an axonal injury is at issue in this case. (1994 IOM report, *supra*, at Ct. Ex. I, p. 103.) Moreover, the 1994 IOM report does not invoke anti-ganglioside antibodies to explain the phenomenon of post-tetanus vaccine GBS. (*Id.* at 103-07.) Instead, the 1994 IOM report suggests that no specific antibody has been

¹² While molecular mimicry is often persuasively hypothesized as the mechanism by which some formulations of the flu vaccine cause GBS, this has not been definitively established. Assessments of this causal relationship generally place significant weight on epidemiologic correlation between GBS and the 1976 Swine flu vaccine juxtaposed with evidence showing the pathogenesis of GBS may involve molecular mimicry as separately demonstrated in the context of *C. jejuni* infection. (2012 IOM report, *supra*, at Ct. Ex. II, pp. 350-63; Schonberger et al., *supra*, at Ex. C, Tab 9.)

observed to occur in more than “a fraction of cases.”¹³ (*Id.* at 63.) To that point, Dr. Martinez-Arizala acknowledged that the anti-ganglioside antibodies he hypothesizes as the cause of petitioner’s condition are seen in only about half of GBS cases (56%). (Tr. 60-61.)

Dr. Martinez-Arizala’s opinion is otherwise supported only by a series of case reports generally seeking to implicate tetanus vaccines in the manifestation of various neuropathies. (Kongbunkait et al., *supra*, at Ex. 22; Ammar, *supra*, at Ex. 23; Baust et al., *supra*, at Ex. 24; Blunsetin & Kreithen, *supra*, at Ex. 25; Reinstein et al., *supra*, at Ex. 26; Quast et al., *supra*, at Ex. 27.) Petitioners in this program often highlight the usefulness of case reports in cases of rare diseases or unusual occurrences. *E.g.*, *Patton v. Sec’y of Health & Human Servs.*, 157 Fed. Cl. 159, 166-68 (2021). However, case reports “do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value,” even though they are not entirely devoid of evidentiary value. *Paluck ex rel. Paluck v. Sec’y of Health & Human Servs.*, 104 Fed. Cl. 457, 475 (2012) (quoting *Campbell v. Sec’y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011)). Based on my review of the specific case reports filed in this case, they are insufficient to carry petitioner’s burden of proof. Although the six publications cited by Dr. Martinez-Arizala account for 27 individual cases, they are not uniform in either clinical presentation or proposed theory of causation (where a theory is even presented). Ultimately, none of the publications provide any useful data beyond a purported temporal association. However, “single case reports of Disease X occurring after Factor Y . . . do not offer strong evidence that the *temporal* relationship is a *causal* one—the temporal relationship could be pure random chance.” *Crutchfield v. Sec’y of Health & Human Servs.*, No. 09-0039V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014), *aff’d*, 125 Fed. Cl. 251 (2014). In fact, Dr. Martinez-Arizala himself conceded that case reports at best “suggest” a potential causal link. (Tr. 94.)

Setting aside petitioner’s analogy to GBS, none of the four other publications petitioner presented regarding case reports of post tetanus-vaccination neuropathies even invoke molecular mimicry generally, let alone implicate anti-ganglioside antibodies

¹³ Regarding the separate and broader question of whether the Tdap vaccine can cause GBS at all, the 1994 IOM report must be weighed against the IOM’s later 2012 report, which did not maintain the conclusion that there is adequate evidence to implicate tetanus vaccines as a cause of GBS (2012 IOM report, *supra*, at Ct. Ex. II, pp. 586-87). *Compare Harris*, 2023 WL 2583393, at *24 (the undersigned explaining of the 1994 and 2012 IOM reports that “the substance of both reports should be weighed to assess their persuasive value rather than reflexively accepting the latest report to the exclusion of the earlier report”), with *Kaczeroswki v. Sec’y of Health & Human Servs.*, No. 21-758V, 2025 WL 2798865, at *34 (Fed. Cl. Spec. Mstr. Aug. 28, 2025) (another special master explaining the view that the 2012 report “supplanted” the prior report (emphasis omitted)). To be clear, as illustrated by *Harris*, *supra*, there are prior instances in which petitioners have succeeded in providing preponderant evidence that tetanus-diphtheria and acellular pertussis vaccines can cause GBS. However, that is not the issue addressed here. Petitioner is not arguing that her Tdap vaccine caused GBS, she is implicitly arguing that the fact that the Tdap vaccine can cause GBS represents some evidence supporting her invocation of the mechanism of molecular mimicry to axonal peripheral neuropathies broadly. However, for the reasons discussed herein, the evidence filed into this record does not meaningfully support that proposition regardless of whether one were to otherwise conclude that the Tdap vaccine can more likely than not cause GBS.

in particular. (Baust et al., *supra*, at Ex. 24; Blumstein & Kreithen, *supra*, at Ex. 25; Reinstein et al., *supra*, at Ex. 26; Quast et al., *supra*, at Ex. 27.) Instead, these reports, which include conditions affecting the area of the brachial plexus, cite hypersensitivity¹⁴ responses as a potential mechanism. (Baust et al., *supra*, at Ex. 24; Blumstein & Kreithen, *supra*, at Ex. 25.) This is also consistent with how the IOM's 1994 report, which concluded that the Tdap vaccine can cause brachial neuritis, addressed the issue. That report distinguished non-demyelinating neuropathies such as brachial neuritis from GBS and stressed that the etiology for such neuropathies remains unknown, though hypersensitivity is possible. (1994 IOM report, *supra*, at Ct. Ex. I, pp. 107-08.) And, importantly, Dr. Martinez-Arizala did not seek to substantiate hypersensitivity or any other mechanism that could explain how Tdap vaccination can cause brachial neuritis. In fact, when I asked Dr. Martinez-Arizala during the hearing to speak to the significance, if any, of the brachial neuritis case reports he had cited, he was unable to do so. (Tr. 75-77.) Moreover, in contrast to the present case and consistent with Dr. Morel's testimony, the 1994 IOM report stressed that, while the underlying pathogenesis is not well understood, the conclusion that the Tdap vaccine can cause brachial neuritis is at least partly supported by the fact that it is "a well-defined clinical syndrome," which helps support a reliance on case reporting. (1994 IOM report, *supra*, at Ct. Ex. I, p. 108; Tr. 170-71.)

Finally, I do agree with petitioner that there may be some instances in which respondent's experts sought in effect to elevate petitioner's burden of proof. In particular, Dr. Morel overstated the degree to which the Tuttle study can be said to exonerate the Tdap vaccine as a cause of GBS.¹⁵ However, this is of no moment. Petitioner's case does not fail under *Althen* prong one because respondent's experts disagree. She has failed to meet her *Althen* prong one burden of proof because she has herself failed to come forward with evidence to support her proffered theory by a preponderance of the evidence. *Accord Cerrone*, 146 F.4th at 1120-23. In sum, Dr. Martinez-Arizala's initial report treated the question of general causation very lightly, essentially identifying the concept of molecular mimicry broadly and citing the above-

¹⁴ Though both are immune-mediated mechanisms, molecular mimicry and hypersensitivity are distinct. *Compare* 2012 IOM report, *supra*, at Ct. Ex. II, pp. 93-95 (explaining hypersensitivity), *with id.* at 99-102 (explaining molecular mimicry).

¹⁵ The Federal Circuit has previously stressed that a petitioner is not obligated to prove a case with epidemiology. *Capizzano*, 440 F.3d at 1325. Yet, "[n]othing in *Althen* or *Capizzano* requires the Special Master to ignore probative epidemiological evidence that undermines petitioner's theory." *D'Tiole v. Sec'y of Health & Human Servs.*, 726 F. App'x 809, 811 (Fed. Cir. 2018). In this case, Dr. Morel testified that she based her ultimate opinion as to general causation on large-scale epidemiology and that the Tuttle study in particular is "quite convincing." (Tr. 226-28, 233.) She even went as far as to agree that the Tuttle study may be the "only" reason she disagrees that the Tdap vaccine can cause GBS. (*Id.* at 228-29.) However, in its 2012 report, the IOM declined to weigh the Tuttle study in its assessment of the available epidemiologic evidence regarding post-Tdap GBS because it used data from passive surveillance and lacked an unvaccinated comparison population, ultimately concluding that there is insufficient epidemiologic data to either accept or reject a causal relationship. (2012 IOM report, *supra*, at Ct. Ex. II, p. 586.) Accordingly, I do not find Dr. Morel's reliance on the Tuttle study to be persuasive, leaving her causal opinion as to post-Tdap GBS largely reliant on a lack of large scale epidemiology, which petitioner is not required to produce.

discussed case reports with little to nothing that would unite the case reports and the theory.¹⁶ (Ex. 16, pp. 5-7.) And, despite the question of general causation having been contested by respondent's experts, he did not revisit the question of whether the Tdap vaccine can cause peripheral neuropathies in either of his supplemental reports. (Exs. 29, 42.) It was not until presenting Dr. Martinez-Arizala's rebuttal testimony that petitioner sought to bolster petitioner's theory by discussing several articles pertaining to the validity of molecular mimicry as a concept and the degree of homology that can support its invocation. (Tr. 239-42; Fujinami & Oldstone, *supra*, at Ex. 74; Gautam et al., *supra*, at Ex. 75; Steinman, *supra*, at Ex. 76.) However, the issue here is not the general viability of molecular mimicry, it is whether it is reasonably applied to this combination of vaccination and injury. Moreover, parsing the degree of homology that could support molecular mimicry is essentially irrelevant where there has been no presentation of a proposed homology in the first place.

In light of all of the above, and considering the record as a whole, petitioner has not preponderantly demonstrated that the Tdap vaccine can cause autoimmune axonal polyneuropathy.

b. *Althen* prongs two and three

Because I have concluded that petitioner has not preponderantly demonstrated that the Tdap vaccine can cause autoimmune axonal peripheral neuropathy, it is not necessary to address in detail whether the vaccine did so in this particular case. Given the outcome regarding *Althen* prong one, by definition it likely did not. Thus, I address *Althen* prongs two and three only briefly. *Trollinger v. Sec'y of Health & Human Servs.*, 167 Fed. Cl. 127, 142 (2023) (affirming the Chief Special Master's dismissal based on a dispositive finding that petitioner had not satisfied *Althen* prong one). In particular, I note out of an abundance of caution that the evidence relevant to *Althen* prong two is not so robust as to otherwise influence the analysis under *Althen* prong one. See *Patton*, 157 Fed. Cl. at 169 (finding treating physician opinions relevant to assessing the reliability of the expert's theory of general causation).

The third *Althen* prong asks whether the timing of injury in this specific case aligns with what would be expected under the general theory presented under *Althen*

¹⁶ In the post-hearing briefing, petitioner's counsel suggested that "[t]his case would have been settled when I started practicing in this program." (ECF No. 95, p. 3.) It is important to note, however, that even accepting counsel's representation at face value, the history of reasoned decisions in the program should not have given petitioner any comfort that her proffered theory would be viewed as a settled matter. Cases alleging that the Tdap vaccines have caused neuropathies have resulted in mixed outcomes. Compare, e.g., *Swaiss v. Sec'y of Health & Human Servs.*, No. 15-286V, 2019 WL 6520791 (Fed. Cl. Spec. Mstr. Nov. 4, 2019) (finding a petitioner entitled to compensation for small fiber neuropathy caused by a Tdap vaccine in part based on the assumption it constitutes a variant of GBS), with *K.A. v. Sec'y of Health & Human Servs.*, No. 16-989V, 2022 WL 20213037 (Fed. Cl. Spec. Mstr. Apr. 18, 2022) (denying entitlement, distinguishing *Swaiss*, and explaining why a history of settlement of cases alleging Tdap-caused GBS do not suggest the causal relationship is settled); see also *Lapierre v. Sec'y of Health & Human Servs.*, No. 17-227V, 2019 WL 6490730 (Fed. Cl. Spec. Mstr. Oct. 18, 2019) (finding a lack of preponderant evidence that the Tdap vaccine can cause an unspecified form of sensory neuropathy).

prong one. The second *Althen* prong requires preponderant proof of a logical sequence of cause and effect, which is usually supported by facts derived from petitioner's medical records.¹⁷ *Althen*, 418 F.3d 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. While the opinions of treating physicians are often favored, *Capizzano*, 440 F.3d at 1326, a petitioner may support a cause-in-fact claim through presentation of either medical records or an expert medical opinion. See § 300aa-13(a).

Here, there is no apparent dispute that petitioner's neuropathy arose about two-weeks post-vaccination as petitioner argues. (Tr. 116.) Moreover, had petitioner preponderantly established her theory of causation, the notion that molecular mimicry could manifest a peripheral neuropathy within that timeframe would not necessarily be controversial. Afterall, the Table Injury of post-flu vaccine GBS carries a causal presumption if onset occurs between 3-42 days post-vaccination. 42 C.F.R. § 100.3(a). Respondent contests that petitioner has met her burden of proof under *Althen* prong three, but only to the extent she has not met her burden under *Althen* prong one. He does not argue that the timing is otherwise confounding. (ECF No. 97, pp. 22-23.)

Nonetheless, temporal association alone is not enough to satisfy petitioner's burden of proof. See, e.g., *Veryzer v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 344, 356 (2011) (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory causally connecting [the] vaccine and injury"), *aff'd per curiam sub nom. Veryzer v. United States*, 475 F. App'x 765 (Fed. Cir. 2012); *A.Y. v. Sec'y of Health & Human Servs.*, 152 Fed. Cl. 588, 595 (2021); *Forrest v. Sec'y of Health & Human Servs.*, No. 10-032V, 2017 WL 4053241, at *18 (Fed. Cl. Spec. Mstr. Aug. 10, 2017); *Cozart v. Sec'y of Health & Human Servs.*, No. 00-590V, 2015 WL 6746616, at *18 (Fed. Cl. Spec. Mstr. Oct. 15, 2015), *mot. for rev. denied*, 126 Fed. Cl. 488 (2016); *Crosby v. Sec'y of Health & Human Servs.*, No. 08-799V, 2012 WL 13036266, at *37 (Fed. Cl. Spec. Mstr. June 20, 2012). Afterall, *Althen* prong two is not without meaning.¹⁸ *Capizzano*, 440 F.3d at 1326-27.

¹⁷ Medical records are generally viewed as trustworthy evidence. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). These records are generally contemporaneous to the medical events and "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." *Id.* However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) (reasoning that "nothing . . . mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted").

¹⁸ The *Capizzano* Court described the circumstances in which *Althen* prong two may be a stumbling block as follows:

There may well be a circumstance where it is found that a vaccine *can* cause the injury at issue and where the injury was temporally proximate to the vaccination, but it is illogical to conclude that the injury was actually caused by the vaccine. A claimant could satisfy the first and third prongs without satisfying the second prong when medical records and medical opinions do not suggest that the vaccine caused the injury, or where the probability

Regarding *Althen* prong two, the critical issue is that the evidence does not preponderate in favor of a finding that petitioner's own polyneuropathy is autoimmune. Although each parties' neurology expert was willing to identify a potential autoimmune trigger (the vaccine for Dr. Martinez-Arizala and a urinary tract infection for Dr. Donofrio¹⁹), Dr. Donofrio ultimately explained that it is "always the case" that polyneuropathy is not in and of itself suspicious for autoimmunity. (Tr. 157-58.) Both parties' neurology experts agreed that autoimmunity is only one of numerous potential causes of polyneuropathy. (*Id.* at 52-53, 115-16.) And Dr. Donofrio further explained that, even as some of the most common causes of neuropathy are not at issue, such as alcohol abuse, vitamin deficiency, or diabetes, there are over 100 known causes of peripheral neuropathy and it is not unusual for the etiology of a patient's polyneuropathy to remain unknown even after a thorough work up. (*Id.* at 115-16, 141-42, 157-58.)

In this particular case, respondent's experts have additionally noted several aspects of petitioner's clinical presentation that do not readily support the conclusion that her polyneuropathy was autoimmune. Specifically: testing for inflammatory markers (CRP and ESR) did not show evidence of an inflammatory process (Ex. C, p. 3; Ex. F, p. 1); an extensive panel testing for autoimmune conditions and antibodies²⁰ was completely negative (Tr. 118-21); spinal MRI was negative for nerve root enhancement²¹ (*Id.* at 121-26); and the neuropathy was axonal, rather than demyelinating (*Id.* at 129). On petitioner's behalf, Dr. Martinez-Arizala, opined that none of these considerations rule out an autoimmune neuropathy (*Id.* at 59-63, 70, 86-87, 246-48) and respondent's expert, Dr. Donofrio, likewise conceded as much (*Id.* at 140-46). However, Dr. Martinez-Arizala did acknowledge that all of the tests performed in petitioner's case were negative for the cause of her neuropathy. (*Id.* at 64.)

of coincidence or another cause prevents the claimant from proving that the vaccine caused the injury by preponderant evidence.

440 F.3d at 1327.

¹⁹ Notably, neither of these proposed autoimmune triggers is persuasive for opposite reasons. Although the fact of petitioner's vaccination is not disputed, its role as an initiator of an autoimmune neuropathy is not preponderantly supported for the reasons discussed under *Althen* prong one. Conversely, Dr. Martinez-Arizala agreed on petitioner's behalf that a urinary tract infection can cause neuropathy; however, he was nonetheless persuasive in explaining why it is unlikely that petitioner suffered a urinary tract infection during the relevant period. (Tr. 90-91, 244-45.)

²⁰ During the hearing, petitioner's counsel made a point of stressing on cross-examination that petitioner's testing included only one of multiple anti-ganglioside antibodies that have been associated with GBS. (Tr. 144-45, 208-15.) Dr. Morel suggested, however, that the one antibody that was tested was the most likely antibody to be implicated. (*Id.* at 210-15.) While petitioner is correct that the antibody testing that was performed is not definitive with respect to the absence of any and all anti-ganglioside antibodies, the assertion that petitioner had any of those other anti-ganglioside antibodies remains speculative.

²¹ The significance of this finding is not necessarily clear with respect to petitioner's first MRI because it was performed without a contrast medium. (Tr. 147-48.) However, Dr. Donofrio also opined that petitioner's second MRI, which did use a contrast medium, was also performed at a time when it would have been expected to detect nerve root enhancement. (*Id.* at 125-26, 154.)

Without treating any one of the above-cited factors as dispositive, they do reveal a significant absence of any affirmative evidence of autoimmunity despite relevant diagnostics having been performed. *Accord Baldwin v. Sec’y of Health & Human Servs.*, 151 Fed. Cl. 431, 447 (2020) (explaining with regard to *Althen* prong two that “[p]etitioner was required to prove by a preponderance of the evidence that one of her expert’s theories actually occurred . . . [but] [p]etitioner did not identify anything in her medical records to support any of her experts’ various theories by a preponderance of the evidence”). Although Dr. Martinez-Arizala contended that an autoimmune neuropathy can be diagnosed based on clinical presentation in the absence of positive lab results (Tr. 70), he also explained that all neuropathies basically have the same manifestations (*Id.* at 64). The only affirmative clinical evidence Dr. Martinez-Arizala cited to support an autoimmune process was petitioner’s rash, which both he and petitioner’s treating physician (Dr. Carlson) assumed to be a vasculitis (*Id.* at 63, 71-72; Ex. 1, p. 83); however, Dr. Donofrio explained that “it’s really unclear why she had a rash. And rashes can be very difficult to associate with a patient’s illness.” (Tr. 133.) Thus, Dr. Martinez-Arizala ultimately conceded of the rash that it “could have been anything.” (*Id.* at 252.)

Indeed, petitioner’s polyneuropathy was noted to have an unclear etiology throughout much of her treatment history. As among her treating physicians, petitioner cites only Dr. Carlson as opining that her condition was vaccine-caused. (ECF No. 95, p. 6.) Dr. Carlson opined of petitioner’s polyneuropathy that the

most likely etiology is an acute autoimmune neuropathy associated with tetanus vaccination. Rare post-vaccinal neuropathies can develop often related to a leukocytoclastic vasculitis. It is intriguing that this patient had a truncal rash associated with peripheral nerve symptomatology early in her presentation which is likely a dermatologic comorbid condition.

(Ex. 1, p. 83.) However, this opinion is not strong evidence. First, it appears to be premised on the notion that petitioner suffered a leukocytoclastic vasculitis, but such an etiology for petitioner’s rash is not preponderantly established. (Dr. Carlson was aware of the rash by history only. (*Id.* at 81.)) Second, it appears to have been only a tentative assessment insofar as the assessment goes on to state that “[w]e will proceed with further testing to assess for inflammation, autoimmune disease or infection not already assessed.” (*Id.* at 83.)

Ultimately then, petitioner is left only to stress the fact that her neuropathy first arose post-vaccination and that many alternative causes were ruled out. (ECF No. 95, p. 6; ECF No. 98, pp. 3-4; Tr. 59, 63.) However, the Federal Circuit has explained that, “[a]lthough probative, neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation.” *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149); *Hibbard v. Sec’y of Health & Human Servs.*, 698 F.3d 1355, 1364-65 (Fed. Cir. 2012) (holding the special master did

not err in resolving the case pursuant to *Althen* prong two when respondent conceded that petitioner met *Althen* prong three).

For all these reasons, petitioner has not met her burden of proof with respect to specific causation under *Althen* prong two.

VI. Conclusion

Although petitioner has my sympathy for the pain and discomfort she has endured, for all the reasons discussed above, I find that she has not met her burden of proof. Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioner is not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

s/Daniel T. Horner

Daniel T. Horner
Special Master