In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: January 6, 2026

SHANNON JACKSON,	*	
	*	
Petitioner,	*	No. 17-538V
	*	
v.	*	Special Master Young
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	

William E. Cochran, Jr., Black McLaren, et al., PC, Memphis, TN, for Petitioner.
Catherine Elizabeth Stolar, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION ON ENTITLEMENT[1]

On April 17, 2017, Shannon Jackson ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program")[2] alleging that the influenza ("flu") vaccine Petitioner received on November 11, 2015, "resulted in the development of acute disseminated encephalomyelitis ("ADEM")." Pet. at 1, ECF No. 1. Petitioner later expanded her claim, requesting compensation "for a neuroinflammatory condition of the central nervous system [("CNS")], most likely myelitis, with aspects of transverse myelitis [("TM")], disseminated encephalomyelitis, and optic neuritis [("ON")] caused by a seasonal [flu] vaccination administered on November 10, 2015." Pet'r's Pre-Hearing Br. at 1, ECF No. 110.

[1] Because this Decision 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 Decision will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755 ("the Vaccine Act" or "Act"). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

A careful analysis and weighing of all the evidence and testimony presented in this case in accordance with the applicable legal standards[3] reveals that Petitioner has failed to provide preponderant evidence that the flu vaccine she received November 10, 2015, was the cause-in-fact of her injuries, specifically ADEM, or any neuroinflammatory condition of the CNS, including TM or neuromyelitis optica spectrum disorder ("NMOSD"). Accordingly, Petitioner is not entitled to an award of compensation.

I. Procedural History

On April 17, 2017, Petitioner filed her petition. Pet. Over the next five months, Petitioner filed 19 exhibits consisting of medical records, two affidavits, and a vaccine verification form. Pet'r's Exs. 1–9, ECF No. 9; Pet'r's Exs. 10–17, ECF No. 10; Pet'r's Ex. 18, ECF No. 14; Pet'r's Ex. 19, ECF No. 19. On January 16, 2018, Respondent filed his Rule 4(c) Report arguing compensation should be denied. Resp't's Rep., ECF No. 24.

I held a Rule 5 status conference on February 13, 2018, during which I ordered Petitioner to file an expert report. Min. Entry, docketed Feb. 13, 2018; ECF No. 25. Petitioner filed an expert report from Lawrence Steinman, M.D., Dr. Steinman's C.V., and medical literature on July 12, 2018. Pet'r's Exs. 25–33, ECF No. 29; Pet'r's Exs. 34–42, ECF No. 30; Pet'r's Exs. 43–51, ECF No. 31.

From February 21 to May 2, 2018, Petitioner filed additional medical records. Pet'r's Exs. 20–21, ECF No. 26; Pet'r's Exs. 22–24, ECF No. 27. Petitioner filed an amended expert report on July 20, 2018, which corrected the date of vaccination. Pet'r's Ex. 52, ECF No. 32. Respondent filed an expert report from Eric Lancaster, M.D., Ph.D., Dr. Lancaster's C.V., and medical literature on September 17, 2018. Resp't's Ex. A, ECF No. 34; Resp't's Ex. B, ECF No. 36.

On October 4, 2018, I granted Petitioner's motion to amend her petition, which she subsequently filed on October 15, 2018. Am. Pet., ECF No. 37. Petitioner filed a supplemental expert report from Dr. Steinman on November 19, 2018. Pet'r's Ex. 53, ECF No. 39. On December 21, 2018, Respondent filed a supplemental expert report from Dr. Lancaster. Resp't's Ex. C, ECF No. 45. Petitioner filed a second supplemental expert report from Dr. Steinman on January 18, 2019. Pet'r's Ex. 56, ECF No. 49.

On May 16, 2019, Petitioner filed her motion for interim attorneys' fees and costs, and Respondent filed his response on May 30, 2019. ECF Nos. 52, 54. Petitioner did not file a reply. On November 12, 2019, I issued a decision granting an award of interim attorneys' fees and costs. ECF No. 61.

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

On May 28, 2019, Respondent filed a second supplemental expert report from Dr. Lancaster. Resp't's Ex. D, ECF No. 53. Petitioner filed updated medical records on August 9, 2019, and a third supplemental expert report from Dr. Steinman on August 12, 2019. Pet'r's Ex. 59, ECF No. 56; Pet'r's Ex. 60, ECF No. 57. Petitioner filed additional updated medical records on September 20, 2019. Pet'r's Exs. 67–72, ECF No. 59. On January 22, 2020, Respondent filed a third supplemental expert report from Dr. Lancaster. Resp't's Ex. E, ECF No. 65.

Petitioner filed additional medical records on January 31, 2020, and June 4, 2020. Pet'r's Exs. 71–72, ECF No. 66, Pet'r's Ex. 73, ECF No. 68. On July 9, 2020, Petitioner filed a fourth supplemental expert report from Dr. Steinman. Pet'r's Ex. 74, ECF No. 69. Respondent filed a fourth supplemental expert report from Dr. Lancaster on October 2, 2020. Resp't's Ex. F, ECF No. 70. Petitioner filed a fifth supplemental expert report from Dr. Steinman on October 23, 2020. Pet'r's Ex. 75, ECF No. 71. Respondent filed a fifth supplemental expert report from Dr. Lancaster on January 7, 2021. Resp't's Ex. G, ECF No. 72.

On January 25, 2021, Petitioner filed additional medical records. Pet'r's Exs. 76–78, ECF No. 74. Petitioner filed a sixth supplemental expert report from Dr. Steinman on February 5, 2021. Pet'r's Ex. 79, ECF No. 79. And Petitioner again filed updated medical records on June 8, 2021. Pet'r's Ex. 80, ECF No. 76.

The case was referred to ADR on September 23, 2021. ECF No. 77. The case remained in ADR until May 11, 2022, when Respondent wished to continue to defend this case. ECF No. 83. During this time Petitioner continued to file medical records. Pet'r's Exs. 81–83, ECF No. 78; Pet'r's Exs. 84–85, ECF No. 80; Pet'r's Exs. 86–88, ECF No. 82.

On October 14, 2022, I scheduled this case for an entitlement hearing set to begin on January 25, 2023. ECF No. 85. On November 28, 2022, Petitioner filed a pre-hearing brief and a seventh supplemental expert report from Dr. Steinman. ECF No. 87; Pet'r's Ex. 95, ECF No. 88.

On January 9, 2023, I granted the parties' joint motion to continue the hearing because Petitioner was hospitalized and her condition, and its effect, if any, on the case was unknown. ECF Nos. 90–91. From April 21, 2023 to October 10, 2023, Petitioner filed updated medical records. Pet'r's Exs. 101–02, ECF No. 94; Pet'r's Ex. 103, ECF No. 97; Pet'r's Exs. 104–05, ECF No. 98; Pet'r's Exs. 106–07, ECF No. 99; Pet'r's Exs. 108–09, ECF No. 100. On December 23, 2024, Respondent filed a sixth supplemental expert report from Dr. Lancaster. Resp't's Ex. H, ECF No. 101. On January 5, 2024, I rescheduled the hearing for November 7, 2024. ECF No. 102.

Petitioner filed a second motion for interim attorneys' fees and costs on February 6, 2024, and Respondent filed a response on March 8, 2024. ECF Nos. 103–04. Petitioner did not file a reply. On February 28, 2025, I issued a second decision granting an award of interim attorneys' fees and costs. ECF No. 132.

Petitioner filed updated medical records on March 27, 2024, April 19, 2024, and June 12, 2024. Pet'r's Exs. 110–12, ECF, No. 105; Pet'r's Exs. 113–14, ECF No. 106; Pet'r's Ex. 115, ECF No. 108. On August 2, 2024, Petitioner filed her pre-hearing brief. Pet'r's Pre-Hearing Br. On September 24, 2024, Respondent filed a seventh supplemental expert report from Dr. Lancaster.

Resp't's Ex. I, ECF No. 112. On October 7, 2024, Petitioner filed updated medical records. Pet'r's Exs. 123–24, ECF No. 115. Respondent filed his pre-hearing brief on October 15, 2024. Resp't's Pre-Hearing Br., ECF No. 116. On November 5, 2024, Petitioner filed a pre-hearing reply brief. Pet'r's Pre-Hearing Reply, ECF. No. 126.

The entitlement hearing was held November 7 and November 8, 2024. Min. Entry, docketed Nov. 8, 2024. Petitioner filed a post-hearing brief on January 13, 2025. Pet'r's Post-Hearing Br., ECF No. 131. Respondent filed a post-hearing brief on March 28, 2025. Resp't's Post-Hearing Br., ECF No. 138. Petitioner filed a post-hearing reply brief on May 9, 2025. Pet'r's Post-Hearing Reply, ECF No. 140.

On August 8, 2025, and September 12, 2025, Petitioner filed over 4,000 pages of medical records. Pet'r's Ex. 132, ECF No. 142; Pet'r's Ex. 133, ECF No. 143. On October 31, 2025, I held a status conference to discuss the relevancy of the numerous records filed eleven months after the hearing and four months after post-hearing briefs. Min. Entry, docketed Oct. 31, 2025; ECF No. 145. After discussion, and in effort to resolve entitlement in the most efficient manner, I decided that the parties should simultaneously file expert reports assessing the newly filed records' relevancy to the case, application to the asserted biological mechanism, and the significance of the multiple sclerosis ("MS") diagnosis in the medical records. ECF No. 145. That same day, Petitioner filed additional medical records. Pet'r's Ex. 134, ECF No. 144. On December 12, 2025, Petitioner filed an eighth supplemental report from Dr. Steinman and Respondent filed an eighth supplemental report from Dr. Lancaster. Pet'r's Ex. 136, ECF No. 149; Resp't's Ex. O, ECF No. 147. This matter is now ripe for consideration.

II. Factual Background

A. Medical Records

Petitioner was thirty-five years old with medical history that included hypertension, obesity, and occasional smoking at the time of vaccination. Pet'r's Ex. 4 at 3. On January 17, 2015, Petitioner was seen in the emergency room ("ER") with complaints of chest and neck pain with "left arm numbness and tingling and pain radiating down to her thumb" since the prior evening. *Id.* at 3–4. After physical evaluation, Petitioner was assessed with cervical neuralgia, given steroids and pain medication, and advised to follow up with neurosurgery. *Id.* at 4.

On November 10, 2015, Petitioner saw her primary care physician ("PCP") for neck pain, constipation, and "excess sweating." Pet'r's Ex. 3 at 11. She was assessed with constipation and advised to increase her fiber intake. *Id.* at 12. Petitioner received a flu vaccination that day. *Id.*; Pet'r's Ex. 2 at 1. Nine days later, on November 19, 2015, Petitioner was seen at CareSpot Express Healthcare Clinic for bilateral posterior neck and upper back pain that had started three weeks earlier. Pet'r's Ex. 5 at 3. She described the pain as aching and aggravated by head rotation. Petitioner also reported that the pain sometimes "radiate[d] down into right shoulder [for two] weeks." *Id.* She denied injury or trauma to her neck and stated that she "saw her [PCP] about same problem [two] weeks ago [and] was told it was stress [and] tension." *Id.* Cervical spine X-rays and a neurological examination were normal. *Id.* at 5. Petitioner was treated onsite with a gluticosteroid and non-steroidal anti-inflammatory medication ("NSAID"). *Id.* She was discharged with a diagnosis of neck pain and was given prescriptions for baclofen and a Medrol dosepak. *Id.*

Petitioner presented to the ER on December 5, 2015, complaining of neck pain and "shooting pain up to the head" for three weeks. Pet'r's Ex. 3 at 86. She reported being seen by her PCP and urgent care for the same issues. *Id.* The pain was "constant and worsening," and her neck pain was exacerbated by movement. *Id.* Petitioner also reported associated symptoms of weakness in the lower extremities and sweating without fever. *Id.* at 86–87. Her PCP "told her it was due to constipation and [hypertension]." *Id.* at 87. A brain CT scan without contrast showed no acute intracranial findings. *Id.* at 90. Petitioner was assessed with acute headache and altered gait, and she was transferred from the ER to the observational unit in stable condition. *Id.* at 90–91. She was advised to follow up with her PCP in five to seven days. *Id.*

On admission to St. Francis Hospital, Petitioner's chief complaints were "[m]ultiple; complaint of headache, neck pain, dizziness, sweating from the inside, lower extremity pain, stiffness, weakness, and difficulty walking." Pet'r's. Ex. 3 at 78. Petitioner's initial evaluation in the ER was unremarkable, but she was "admitted per the [ER] physician's discretion for further evaluation." *Id.* On neurologic examination, Petitioner had weakness but was able to walk, was slightly dizzy, had normal strength and reflexes, and had no sensory deficit. *Id.* at 80. She had minimal edema in her extremities with "some local tenderness." *Id.* at 79. Petitioner was assessed with generalized pain and weakness with difficulty walking, otherwise non-specific, dizziness, and a probable "stress/anxiety reaction." *Id.* at 80. Her history of hypertension was also noted. *Id.* The admitting physician, Dr. Said Elias, saw Petitioner the next day, on December 6, 2015, and discussed the nonspecific nature of her multiple symptoms that did not point toward a specific diagnosis. *Id.* Dr. Elias "opted to treat [Petitioner] as outpatient with an anti-inflammatory medication and muscle relaxer with outpatient follow[-]up with her [PCP]." *Id.* She was advised to return to the ER for worsening symptoms and to follow up with her PCP for evaluation, consideration of an outpatient magnetic resonance imaging ("MRI"), and a neurology consultation. *Id.* at 81.

Petitioner saw her PCP on December 9, 2015, for complaints of continued cervical pain and left leg heaviness. Pet'r's Ex. 3 at 9. Her history indicated that "one month ago," she woke up with constant neck pain, and started having left leg heaviness and pain two weeks later. *Id.* She also reported fatigue, dizziness, headaches, and left leg weakness. *Id.* On examination, she was tachycardic. *Id.* She could not lift her left leg and had calf tenderness. *Id.* at 10. Petitioner was referred to the ER for an ultrasound and doppler examination per deep vein thrombosis ("DVT") protocol. *Id.*

Petitioner was seen in the ER at Baptist Memorial Hospital–Memphis ("Baptist Hospital") on December 10, 2015. Pet'r's. Ex. 4 at 35, 37. She reported that she had left leg numbness beginning two weeks earlier, associated with "mild intermittent chest tightness, back of neck shooting pain radiating to [the] head, [headache] (5/10 pain level), body stiffness, [bilateral lower extremity] weakness, dizziness, arm tingling, upper back pain, and [left] toe numbness." *Id.* at 37. Petitioner denied similar symptoms in the past. *Id.* She told treaters that she thought "the pain may be due to Mirena [(a hormone-releasing IUD)] and had it removed [three days prior] on Monday." *Id.* She described the severity of her symptoms as moderate and continuous. *Id.* Petitioner was admitted to the hospital in stable condition with diagnoses of "[w]eakness of the left lower extremities probable cervical myelopathy, [c]onstipation, [u]rinary retention, and [h]ypertension." *Id.* at 40. Her admission record diagnoses were "[h]eache disorder, [c]hest pain at rest, [and w]eakness of left lower extremity." *Id.* at 46. Petitioner noted that she had suffered from a viral

illness approximately one week earlier, followed by feeling weak and difficulty lifting her left leg. *Id.* Petitioner reported a family history of lupus and sarcoidosis. *Id.* A brain MRI revealed multiple T2 FLAIR abnormalities involving the upper cervical spine, brainstem, and white matter, with the differential diagnosis including a demyelinating process, metabolic process, or infectious/inflammatory etiology. *Id.* at 49. A chest computed tomography ("CT") scan was negative for a pulmonary embolism; however, a renal cyst and left renal stone were seen. *Id.* at 49–50.

Neurologist, Dr. Kumar completed a neurology consultation with Petitioner, on December 11, 2015, for weakness of the lower extremity. Pet'r's Ex. 4 at 54–55. Petitioner reported that one month earlier, she began to have spasms in her neck with pain that spread to the upper and lower part of her neck and suboccipital region bilaterally. *Id.* at 55–56. Two weeks later, she began to have left leg and foot numbness that spread to her right leg and foot. *Id.* She developed progressive stiffness and weakness of the lower extremities that impaired her ability to walk unassisted in the few days leading up to her consultation. *Id.* She subsequently developed issues with bladder control and constipation for one week. *Id.* at 56. Petitioner further described "paresthesias, double vision, [and] swallowing difficulty," along with bilateral weakness and numbness in her "distal aspect of the hands." *Id.* On examination, Petitioner's neck flexion was restricted, and she had pain in the posterior of her neck "with worsening of tingling in the lower extremities." *Id.* at 57.

Dr. Kumar's impression was "progressive cervical myelopathy with bilateral lower and mild upper extremity weakness, spasticity, and bowel and bladder incontinence." Pet'r's Ex. 4 at 55. The etiology of Petitioner's symptoms was unclear; an MRI of the cervical spine was ordered. *Id.* A review of the brain MRI scan showed changes that were concerning for demyelination. *Id.* Given Petitioner's mother's history of sarcoidosis, that condition remained a possibility. *Id.* Because Petitioner's symptoms were progressive, treatment with solumedrol was initiated, along with Baclofen to address her spasticity. *Id.*

Over the course of her hospitalization, Petitioner had repeat brain MRIs to rule out MS and sarcoidosis. Pet'r's Ex. 4 at 42. An additional consultation was done on December 13, 2015, by Dr. Arnautovic, following the MRI results "consistent with [TM]." *Id.* at 53. Petitioner reported to him that "she recently had the flu vaccine," and also "mention[ed] possible viral infection" a week prior. *Id.* "Since then, she ha[d] not [been] able to lift her lower extremity extensively." *Id.* Petitioner's social history indicated "[s]he [was] a current every day smoker[, and] use[d] alcohol occasionally." *Id.*

A thoracic spine MRI with and without contrast also done on December 14, 2015, revealed "extensive abnormal signal and enhancement" throughout the thoracic cord. Pet'r's Ex. 4 at 42–43. The findings included extensive abnormal brain signal with prominent involvement of the corticospinal tracts and middle cerebellar peduncles, unchanged in comparison with the December 11, 2015 MRI. *Id.* Abnormal enhancement was also observed. *Id.* at 43. A demyelinating process was possible. *Id.*

During a follow-up with Dr. Kumar on December 16, 2015, Petitioner indicated improvement on steroids with "almost normal" arms and no neck pain or headaches. Pet'r's Ex. 4 at 64. She continued to have lower extremity weakness, difficulty ambulating, and lack of bladder and bowel control. *Id.* Dr. Kumar assessed Petitioner with cervical myelopathy and an extensive

spinal cord lesion. *Id.* at 65. He noted that the "[e]xact etiology of this is unclear" and considered ADEM, NMO, and MS. *Id.* Petitioner again mentioned "that she had a flu shot [a] few weeks prior to onset of her neurological symptoms." *Id.* A spinal fluid analysis was markedly abnormal with elevated protein. *Id.* Several tests, including NMO antibodies and cerebrospinal fluid cytology, were pending. *Id.* However, Petitioner was improving on steroids and discharged with diagnoses of lower left extremity weakness with "probable cervical myelopathy," constipation, urinary retention, and hypertension. *Id.* at 40. She was prescribed oral steroids with a 10 mg taper every other day. *Id.* A follow-up visit was planned for four to six weeks, and therapy was recommended. *Id.*

Petitioner returned to St. Francis Hospital ER on January 25, 2016, with complaints of numbness of her toes, headache, and neck pain. Pet'r's. Ex. 3 at 40. Patient reported history noted that Petitioner was diagnosed with MS on December 10, 2015. *Id.* She stated that her steroid treatment provided some relief, and she did not follow up with Dr. Kumar due to scheduling issues. *Id.* Once the steroid treatment ended, Petitioner stated that her symptoms returned. *Id.* She "[c]omplained of l[eft] side headache and b[ilateral] l[ower] e[xtremity] weakness, tremors, and urinary urgency (but not able to produce urine)." *Id.* Petitioner stated that she "was in pain secondary to her elevated [blood pressure]." *Id.* Additionally, she had numbness and tingling in the toes of both feet and used a cane to ambulate. *Id.* A physical examination was significant for lower extremity weakness and urinalysis was positive for infection. *Id.* at 42. Petitioner was diagnosed with hypertensive urgency, a urinary tract infection ("UTI"), and weakness of the left lower extremity. *Id.* She was discharged home in stable condition with instructions to follow up with her PCP. *Id.*

On February 1, 2016, Petitioner presented to her PCP for bilateral paresthesias in her toes, bilateral weakness in her legs, headaches, and fatigue. Pet'r's. Ex. 3 at 7. She walked with a slow gait and was "unable to mount the exam table." *Id.* at 8. Petitioner was assessed with weakness in both legs, bilateral toe numbness and paresthesias, hypertension, probable MS, headache, and allergic rhinitis. *Id.* Petitioner was advised to keep her appointment with Dr. Kumar scheduled for the following day and to avoid ibuprofen. *Id.* At her follow-up visit with Dr. Kumar on February 2, 2016, Petitioner reported a recent increase in weakness in the right upper and lower extremities and received another course of steroids. Pet'r's Ex. 8 at 6. She was continuing to take steroids without any improvement in her symptoms. *Id.* She denied headache, nausea, vomiting, and fever, and confirmed profuse sweating in her hands and lower extremities. *Id.* She also had stiffness and weakness of both upper extremities and was unable to control her bowel and bladder. *Id.* Petitioner required assistance with ambulation due to stiffness and weakness in her lower extremities. *Id.*

Petitioner's physical examination revealed bilateral increased tone in upper and lower extremities, severe bilateral spasticity, profuse palm sweating, decreased hip strength, bilateral, brisk, deep tendon reflexes, and impaired gait. Pet'r's Ex. 8 at 8. Lab tests were negative for NMO. *Id.* at 6. Dr. Kumar's impression was "[c]ervical myelopathy due to infectious [ADEM;] however, [MS could]not be completely excluded." *Id.* at 9. He also diagnosed Petitioner with spastic quadriparesis due to cervical myelopathy. *Id.* The plan was to continue steroids to treat her increased weakness and increase the dosage of baclofen to treat spasticity. *Id.* Brain and cervical spine MRIs were ordered. *Id.* Petitioner told Dr. Kumar about her November 2015 flu vaccine and asked about the potential cause of her neurological symptoms. *Id.* Dr. Kumar indicated that he could not "be 100% certain what may be the etiology of [Petitioner's] symptoms, but it could be

post vaccinal in origin since she did not have any viral infection prior to onset of her symptoms." *Id.*

Petitioner followed up with her PCP on February 17, 2016, and reported the ADEM diagnosis. Pet'r's Ex. 3 at 5. Her PCP noted that Dr. Kumar "was not completely convinced [Petitioner] [did not] have [MS] (although her MS panel wasn't diagnostic of MS)." *Id.*

On July 28, 2016, Petitioner was seen by Dr. Kumar to follow up on her "progressive" ADEM. Pet'r's Ex. 8 at 11. Petitioner relayed that she "did not notice any improvement other than few times" from the steroids. *Id.* She explained that "she [was] getting weaker in her lower extremities and [] developed numbness in the occipital and posterior aspect of the neck area and also she fe[lt] she [was] having muffling of sounds from both ears." *Id.* She had been given Neurontin for numbness, but did not like how she felt when taking it. *Id.* She also continued to have problems voiding and constipation, severe weakness in her lower extremities, and mild weakness in the upper extremities. *Id.* Dr. Kumar's impression was that while Petitioner's condition was previously thought to be ADEM, but it was "unusual that her symptoms [were] progressively getting worse, which initially responded to IV steroid and oral steroid." *Id.* at 13. Because her symptoms were progressing, hospitalization for MRIs and treatment with higher dose steroids and possibly plasma exchange were considered. *Id.* Petitioner presented to the Baptist Hospital ER later that day with generalized weakness and a worsening, unstable gait. Pet'r's Ex. 4 at 458. She was admitted for further evaluation and treatment. *Id.* at 462.

On July 29, 2016, Petitioner was evaluated by Dr. Feiyu Chen, a neurologist, who noted Petitioner's extensive workup, including numerous MRIs and spinal fluid analysis. Pet'r's Ex. 4 at 462. Dr. Chen believed that Petitioner's clinical picture "may [be] consistent with myelitis but not really look like multiple sclerosis [sic]." *Id.* Petitioner's condition "seem[ed] to be slowly getting worse in last few days." *Id.* at 462–63. She started on oral steroids the day before her evaluation, but "it [did] not do very much." *Id.* at 463. Petitioner reported persistent bowel and bladder incontinence, upper and lower extremity weakness, and headaches. *Id.* Dr. Chen assessed Petitioner with clinically worsening ADEM, inability to walk, impaired balance, ataxia, and overall weakness. *Id.* at 463–64. He ordered MRIs of the head and cervical and thoracic spine and treatment with high-dose steroids was initiated, and plasmapheresis was considered. *Id.* at 464. July 30, 2016 results from a brain MRI showed resolution of previous extensive hyperintensities with minimal residual small hyperintensities in the centrum semiovale and cortical spinal tracts. *Id.* at 456. There were no findings suggestive of an acute process. *Id.* An MRI of the cervical spine similarly showed significant improvement; the MRI of the thoracic spine showed persistent but improved cord hyperintensity without spinal cord enlargement or enhancement. *Id.* at 456–57. Petitioner was discharged on August 5, 2016, to HealthSouth rehabilitation facility with diagnoses of unstable gait and ADEM. *Id.* at 464–68. She was to continue with IV steroids for two weeks. *Id.*

At a follow-up appointment with Dr. Kumar on August 24, 2016, he reconsidered whether to treat Petitioner with either intravenous immunoglobulin ("IVIG") or plasma exchange therapy. Pet'r's Ex. 8 at 1. Because Petitioner developed complications from high-dose steroids, including acne and diabetes, and her condition had been getting progressively worse, plasma exchange therapy was recommended. *Id.* at 4. On August 26, 2016, Petitioner was readmitted to Baptist Hospital with complaints of exacerbation of her ADEM with pain and weakness in her right leg.

Pet'r's Ex. 4 at 674. Her admitting diagnoses were ADEM, steroid-induced hyperglycemia, and thrombocytopenia. *Id.* at 675, 679. Petitioner underwent a neurology consult by Dr. Ganta for ADEM with worsening leg weakness and paraplegia, and urinary incontinence. *Id.* at 701. Because her condition was worsening, Petitioner was treated with IVIG. *Id.* at 702. Dr. Ganta initiated treatment with Imuran, a steroid-sparing agent, and started Petitioner on a prednisone taper. *Id.*

Petitioner returned to inpatient rehabilitation therapy September 9 to September 22, 2016. Pet'r's Ex. 11 at 1502–15. She was discharged with a wheelchair, hospital bed, and commode, and plans for home health care. *Id.* at 1504. Her discharge diagnoses were ADEM, deep vein thrombophlebitis ("DVTP") treated with Lovenox, hypertension, neurogenic bowel and bladder, and paraplegia. *Id.* at 1516.

Petitioner saw Dr. Ganta in follow-up on February 6, 2017. Pet'r's Ex. 12 at 1. Since her hospitalization, she had leg spasms, persistent leg weakness, and urinary incontinence. *Id.* She continued to use a wheelchair for ambulation. *Id.* at 2. Her examination revealed lower extremity weakness and decreased reflexes. *Id.* Dr. Ganta's impression was myelitis of unclear etiology, possible ADEM, and paraplegia with urinary incontinence. *Id.* While the etiology was unclear, he felt it was definitely inflammatory given the elevated CSF protein. *Id.* He recommended continued treatment with Imuran and outpatient IVIG. *Id.* Petitioner continued her treatment for the next several years with intermittent improvement of her symptoms, but notes from a June 26, 2018 visit with her neurologist, Dr. Zhang revealed that she continued to require the use of a wheelchair. Pet'r's Ex. 59 at 30.

On March 21, 2019, Petitioner was seen by a new neurologist, Dr. Pillai, who assessed Petitioner with "CNS demyelinating disorders, [TM] and idiopathic [TM]." Pet'r's Ex. 29 at 24. Results from her January 6, 2019 brain MRI revealed "enhancement of the right occipital lesion as well as generalized supratentorial enhancement." *Id.* The spinal MRI revealed "[d]iffuse signal changes throughout the cervical and thoracic spinal cord, with a long segment enhancement in the posterior spinal cord from C7 to T8." *Id.* Dr. Pillai described Petitioner's "aggressive and relapsing course" and noted that "[s]he ha[d] relapsing [TM] or brain stem encephalitis." *Id.* "The findings suggest[ed] severe demyelinating disease such as ADEM or NMO[SD]". Pet'r's Ex. 59 at 24.

On April 29, 2019, Petitioner returned to Methodist Hospital ER for progressive weakness. Pet'r's Ex. 69 at 449. Her workup did not reveal any acute process and she was diagnosed with lower extremity weakness associated with previous TM and was discharged the same day. *Id.* On May 1, 2019, Petitioner saw Dr. Pillai for follow-up. Pet'r's Ex. 59 at 13. Dr. Pillai noted Petitioner's recent hospitalizations and persistent weakness and indicated the need to pursue more aggressive therapies, including IgG, plasmapheresis, and rituxin or Cytoxan. *Id.* at 13, 16. Diagnoses included Devic's Disease (NMO) and other inflammatory and immune myopathies, (not elsewhere classified). *Id.* at 13. Petitioner returned to Methodist Hospital ER on May 9, 2019, and was admitted for 10 days for progressive weakness. Pet'r's Ex. 69 at 549, 562. Upon discharge, she continued to see Dr. Pillai regularly through October of 2022. Pet'r's Ex. 59 at 9; Pet'r's Ex. 73 at 7, 10; Pet'r's Ex. 80 at 5; Pet'r's Ex. 85 at 3, 5, 9; Pet'r's Ex. 101 at 6, 10, 14. Petitioner began undergoing plasmapheresis two times per month in 2020, but showed little improvement. Pet'r's Ex. 80 at 5; Pet'r's Ex. 83 at 3. Dr. Pillai continued to note Petitioner's active problems as Devic's Disease (NMO) and other inflammatory and immune myopathies. Pet'r's Ex. 80 at 5.

On January 3, 2023, Petitioner was admitted to Baptist Memorial Hospital and reported a two-week history of progressive upper and lower extremity weakness, burning neck pain, and blurred vision. Pet'r's Ex. 102 at 57. A brain MRI showed worsening supratentorial white matter T2 hyperintensities with enhancement, and possible enhancement of the right optic nerve. *Id.* at 59. A cervical spine MRI showed extensive diffuse cervical and thoracic spinal cord abnormal signal with spinal cord expansion and peripheral enhancement. *Id.* at 60. On January 4, 2023, Petitioner saw neurologist Pawan Rawal for a consultation. *Id.* at 84. Dr. Rawal diagnosed Petitioner with NMO with relapse and ordered Solu-Medrol and a full course of plasmapheresis. *Id.* at 85. She was discharged on January 26, 2023, to acute rehabilitation with orders to receive rituximab on an outpatient basis. *Id.* at 14. Her discharge diagnoses included NMO with acute exacerbation, chronic paraplegia, and spasticity. *Id*. at 13. A follow-up note from a visit with Dr. Rawal on April 20, 2023, listed Petitioner's diagnosis as Ab negative NMO. Pet'r's Ex. 104 at 3. On April 23, 2023, Petitioner was admitted to Baptist Memorial Hospital for weakness in the upper extremities, flank pain, and head and neck pain. Pet'r's Ex. 108 at 588. A brain MRI completed on April 24, 2023, showed marked worsening T2/FLAIR hyperintensities involving the subcortical and deep supratentorial white matter and corresponding enhancement of the perivascular spaces. *Id*. at 657. Additionally, new focal T2/FLAIR hyperintensity was detected in the left optic nerve. *Id.* A cervical spine MRI showed mild degenerative disc disease, persistent T2 hyperintense signal and swelling of the spinal cord, and longitudinally extensive enhancement, greater in the dorsal cord and less in the ventral cord over the previous exam. *Id.* at 656. Petitioner received five doses of plasmapheresis and was discharged on May 7, 2023. *Id*. at 824.

Petitioner was readmitted to Baptist Memorial Hospital June 26 to July 5, 2023, for increased weakness and paresthesias. Pet'r's Ex. 108 at 11–12, 21, 42. She was later hospitalized multiple times in 2023 into 2024 for complications related to Methicillin-resistant Staphylococcus aureus ("MRSA"). Pet'r's Ex. 113 at 728, 2460–61; Pet'r's Ex. 114 at 19–21.

Petitioner was readmitted to the hospital on March 1, 2024, for hyperglycemia, fever, and chills, with concerns for possible recurrent infection. Pet'r's Ex. 115 at 182. Her present illness history noted "seronegative NMO/idiopathic acute [TM]/[ADEM]," neurogenic bladder, and stage IV decubitus ulcers. *Id.* A brain MRI showed internal development of findings concerning for encephalitis or multifocal infarction. *Id.* Petitioner was hospitalized again on April 6, 2024, for upper extremity pain and increasing upper extremity weakness as well as fevers and intermittent chills. Pet'r's Ex. 115 at 1903–04. An MRI of Petitioner's hips revealed septic arthritis and blood tests were positive for MRSA. *Id.* Her discharge documentation included a plan for "[MS] #NMO Seronegative." *Id*. at 186.

Hospitalizations from September 26 to October 11, 2024, and November 29 to December 3, 2024, for abscess infection and sepsis repeatedly listed MS as an active diagnosis in Petitioner's medical history. Pet'r's Ex. 133-1 at 19; Pet'r's Ex. 133-2 at 279–86. Petitioner continued to receive aggressive treatment for severe complications due to her condition, including drug-resistant UTIs, ischemic stroke, and evidence of enhancing brain lesions. Pet'r's Ex. 133-2 at 878–1000. Her treaters struggled to definitively diagnose her condition from the differential list demyelinating conditions. *See id.*

On June 24, 2025, Dr. Priscilla Lao evaluated Petitioner in the ophthalmology clinic. Pet'r's Ex. 134 at 24–31. Petitioner reported one week of left more than right sided vision loss. *Id.*

Aquaporin-4 ("AQP4") and myelin oligodendrocyte glycoprotein ("MOG") tests were conducted with negative results. *Id.* Dr. Lao observed that Petitioner's condition was "highly concerning for NMO" and recommended that she present to the ER. *Id.* Petitioner was readmitted to the hospital for pulse steroids and PLEX with neurology consultation. *Id.* On June 26, 2025, Dr. Lao and Dr. Karen Park performed an ophthalmology consultation and confirmed a diagnosis of NMO. *Id.* Petitioner's presentation of vision loss in both eyes was thought to be consistent with ON and concerning for NMO. *Id.* Petitioner's condition continued to progress, and Dr. Petrinjac-Nenadic noted that her diagnosis had evolved to likely primary progressive MS with quadriparesis and bilateral optic neuritis as of July 2025, ten years post vaccination. Pet'r's Ex. 133-4 at 575.

B. Petitioner's Statements

1. Affidavit

On April 26, 2017, Petitioner filed an affidavit that briefly described her medical history and alleged injury. Pet'r's Ex. 1. She noted that she did not have a pre-vaccination history of ADEM, but her impending divorce caused "occasional neck pain related to that stress and tension." *Id.* at ¶ 5. Petitioner described a different, burning pain in her head and neck within several weeks following her flu vaccination. *Id.* at ¶ 6. She also "began having pain and weakness in [her] legs which led to difficulty walking." *Id.* Petitioner denied that she had a viral infection or that she told any provider of such in December of 2015. *Id.* at ¶ 8. At the time of the writing, Petitioner suffered from continued numbness and tingling in her legs and feet, hand weakness, and short-term memory loss. *Id.* at ¶ 9.

2. Testimony

Petitioner testified at the entitlement hearing and stated that she had no memory of an ER visit in January of 2015, during which she complained of neck pain. Tr. 23. She did remember speaking to her PCP in April of that same year "to find out what was going on with the neck pain and the headaches." Tr. 24. On November 10, 2015, Petitioner received her vaccination, and she testified that she did not remember anything else about that day nine years later. Tr. 25. Post vaccination, Petitioner remembered "the weakness in [her] legs and the headaches getting more intense and last the pain in [her] neck getting more intense." Tr. 26. She denied telling any treaters in November or December of 2015 that she had recently had a viral infection. Tr. 28. Petitioner stated, "I wouldn't know. How would I, you know, know if I had a viral infection?" *Id.* She stated that her treater, Dr. Kumar, "said at one point that [she], you know – the flu vaccine had given [her] ADEM, acute myelinating encephalitis – demyelinating encephalitis – myelitis." Tr. 28–29.

III. Experts

A. Expert Qualifications

1. Petitioner's Expert, Lawrence Steinman, M.D.

Dr. Steinman is a board-certified neurologist who has practiced adult and pediatric neurology for 40 years. Pet'r's. Ex. 52 at 1. He received his medical degree from Harvard University and subsequently completed an internship and residencies at Stanford University

Hospital. Pet'r's Ex. 26 at 1. He is currently a Professor at Stanford University in the Departments of Neurology and Neurological Science, and Pediatrics and Genetics. *Id.* He has "cared for hundreds of adults and children with various forms of neuroinflammatory diseases" including ADEM, NMO, TM, and MS. Pet'r's Ex. 52 at 1. Dr. Steinman has conducted research on and published numerous publications on neurology and the immune system. Pet'r's Ex. 26 at 4–40.

2. Respondent's Expert, Eric Lancaster, M.D., Ph.D.

Dr. Lancaster is a board-certified neurologist with certifications in electrodiagnostic medicine. Resp't's Ex. A at 1; Resp't's Ex. B at 2. He received his M.D. and Ph.D. from the University of Maryland School of Medicine. Resp't's Ex. A at 1; Resp't's Ex. B at 1. He subsequently completed a neurology residency and a neuromuscular fellowship at the University of Pennsylvania. Resp't's Ex. B at 1. He is currently an Assistant Professor of Neurology at the University of Pennsylvania. *Id.* His "research concerns autoimmune disorders characterized by antibodies," and he examines "autoantibodies to peripheral nerve proteins in autoimmune causes of peripheral neuropathy." *Id.* Dr. Lancaster has numerous publications on autoimmune neurological disorders. *Id.* at 4–6; Resp't's Ex. A at 1.

B. Expert Analysis

1. Expert Reports

a. Diagnosis

Petitioner's expert, Dr. Steinman, opined in his first report,[4] filed on July 12, 2018, that "[t]he [working] diagnosis here is a neuroinflammatory condition involving the central nervous system, most likely myelitis, with aspects of [TM] and disseminated encephalomyelitis." Pet'r's Ex. 25 at 9. He then provided the National Institute Neurological Disorders and Stroke ("NINDS") definitions of several such diseases, beginning with ADEM: "a brief but widespread attack of inflammation in the brain and spinal cord that damages myelin – the protective covering of nerve fibers" *Id.* The definition noted that the condition can follow infections or a measles, mumps, rubella vaccination. *Id.* He also included the clinical presentation: a rapid onset of "encephalitis-like symptoms such as fever, fatigue, headache, nausea and vomiting, and in the most severe cases, seizures and coma." *Id.* Additional symptoms include vision loss, weakness/paralysis, and impaired muscle movements. *Id.* at 10. The excerpt noted that ADEM can be misdiagnosed as MS, but explained that "unlike MS patients, persons with ADEM will have rapid onset of fever, a history of recent infection or immunization, and some degree of impairment of consciousness." *Id.* Additionally "ADEM usually consists of a single episode or attack of widespread myelin damage, while MS features many attacks over the course of time." *Id.*

Dr. Steinman next defined TM, also using NINDS authority, as "an inflammation of the spinal cord, a major part of the central nervous system." Pet'r's Ex. 25 at 10. Like ADEM, onset

[4] Dr. Steinman's first amended expert report is an exact duplicate of his first report and was filed on July 20, 2018. Pet'r's Ex. 52. Dr. Steinman replied to Dr. Lancaster's first expert report on November 19, 2018. Pet'r's Ex. 53. He noted his disagreement with many of Dr. Lancaster's contentions and ultimately wrote, "[m]y opinions in this case remain unchanged." *Id.* at 6.

may be sudden or develop over days or weeks, and "[s]ymptoms include pain, sensory problems, weakness in the legs and possibly the arms, and bladder and bowel problems." *Id.* at 11. Like ADEM, TM can "represent[] the first symptom of an autoimmune or immune-mediated disease such as [MS] or [NMO]." *Id.*

Respondent's expert, Dr. Lancaster, identified Petitioner's diagnosis as of September 15, 2018, as inflammation myelitis. Resp't's Ex. A at 7 (citing Pet'r's Ex. 24 at 5). He noted that myelitis is nonspecific for spinal cord disorders, and he further defined acute TM as "sudden injury to the spinal cord from a presumed inflammation mechanism." *Id.* at 7. Dr. Lancaster explained that when inflammation is detected by imaging and in spinal fluid, "the differential diagnosis is narrowed to infectious and autoimmune causes." *Id.* Dr. Lancaster noted that in Petitioner's case, onset was rapid and there "was no evidence of an infectious cause, despite appropriate investigations, and an autoimmune cause of Petitioner's spinal cord inflammation is highly likely." *Id.*

Dr. Lancaster noted that "[m]yelitis may be the presenting syndrome in [MS] or occur at any point during that disease process." Resp't's Ex. A at 7. However, he continued that "Petitioner never had evidence of inflammation in other brain regions or the optic nerves, so [MS] would not be the correct diagnosis in this case." *Id.* Dr. Lancaster added that Petitioner may still develop MS but it was unlikely. *Id.* He also briefly discussed NMO but opined that because Petitioner did not have AQP4 autoantibodies, that is "not the correct diagnosis in this case." *Id.* Similarly, Dr. Lancaster excluded ADEM as a possible diagnosis, because Petitioner did not meet the diagnostic criteria. *Id.* He defined ADEM as "[a]n acute inflammatory and multifocal disorder of the [CNS]" that is primarily seen in children. *Id.* Dr. Lancaster applied the Krupp et al.[5] criteria for pediatric cases of ADEM, MS, and NMO. Resp't's Ex. A, Tab 3. Krupp et al. characterized ADEM as heterogeneous and "best viewed as a 'syndrome' rather than a specific disorder.'" *Id.* at 2. A diagnosis requires all of the following: a) a polyfocal clinical CNS event with presumed inflammatory cause; b) encephalopathy that cannot be explained by fever; c) no progression three months post onset; and d) abnormal imaging. *Id.* Dr. Lancaster noted that "Petitioner did not have this required element" of encephalopathy (not explained by fever). Resp't's Ex. A at 7. Dr. Lancaster also discounted an ADEM diagnosis because "Petitioner showed clear clinical progression" of the disease for more than three months, despite being a monophasic condition. *Id.* He responded directly to Dr. Steinman: "we have spinal taps showing marked inflammation separated by [nine] months." *Id.* at 10. Krupp et al. determined that "[r]elapsing disease following ADEM that occurs beyond a second encephalopathic event is no longer consistent with multiphasic ADEM but rather indicates a chronic disorder, most often leading to the diagnosis of MS14 or NMO." Resp't's Ex. A, Tab 3 at 3. They "propose[d] that [pediatric] criteria for MS are met if after the initial ADEM a second clinical event meets the following three requirements: (a) is nonencephalopathic: (b) occurs three or more months after the incident neurologic illness: and (c) is associated with new MRI findings consistent with revised radiologic criteria for dissemination in space" and time. *Id.*

In response to Dr. Lancaster's criticisms, Dr. Steinman clarified that "[w]hether you want to call this "aspects of disseminated encephalomyelitis" or simply ADEM, is irrelevant." Pet'r's

[5] Lauren B. Krupp et al., *International Pediatric Multiple Sclerosis Study Group Criteria for Pediatric Multiple Sclerosis and Immune-Mediated Central Nervous System Demyelinating Disorders: Revisions to the 2007 Definitions*, 10 MULTIPLE SCLEROSIS J. 1261 (2013).

Ex. 56 at 2. He noted that ADEM was considered by Petitioner's treating neurologists that "may not have wanted to wait for 'alteration in the level of consciousness' and came in with the strong approved drugs including corticosteroids, azathioprine and IVIG." *Id.*

Following updated medical records, including a diagnosis of seronegative NMO by neurologist Dr. Rawal, Dr. Steinman filed a supplemental expert report. Pet'r's Ex. 106. He noted that the scheduled hearing had been postposed, partly due to the evolution of Petitioner's diagnosis and any potential impact on the applicability of the asserted causation theory. *Id.* at 1. Dr. Steinman stated simply: "I have reviewed the medical records obtained [as of August of 2023]. The medical records are clear that this is not NMO! AQP4 antibodies are negative." *Id.* In contrast, Dr. Lancaster agreed with Petitioner's updated diagnosis. Resp't's Ex. H at 3. He explained that not all NMOSD patients have AQP4 antibodies. *Id.* He referenced Wingerchuk et al.,[6] a 2015 paper that discussed the diagnostic criteria for AQP4-antibody-negative NMOSD. *Id.* Dr. Lancaster noted Petitioner's "objective evidence of a longitudinally extensive [TM], persisting and worsening over many months[;] evidence of bilateral optic nerve inflammation on her latest MRIs[; and] multiple attacks of worsening function attributed to spinal cord inflammation." *Id.* at 4.

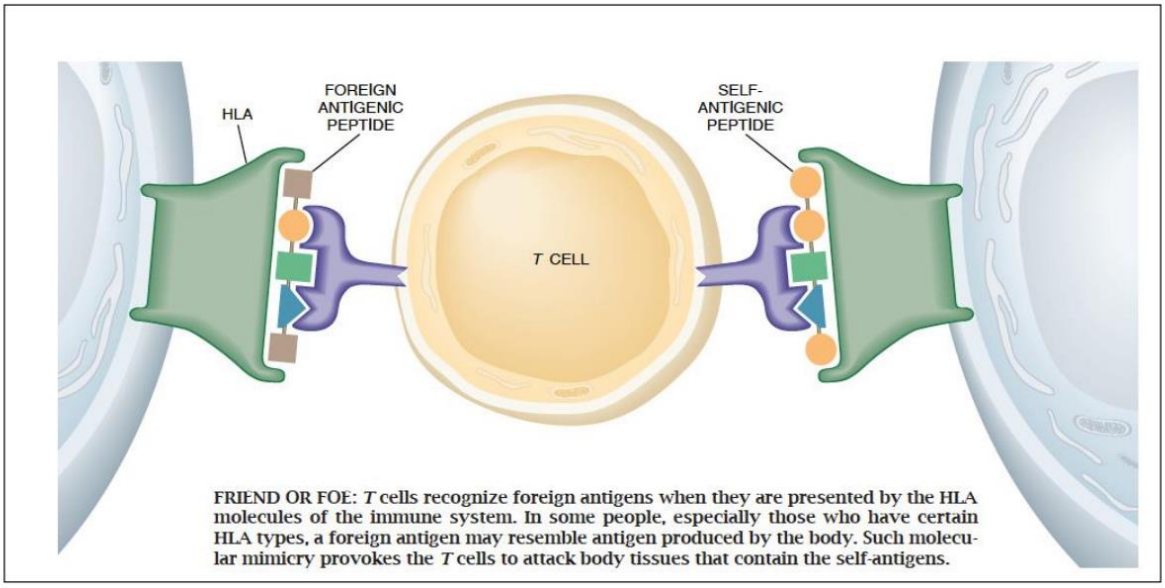
Dr. Steinman responded to Dr. Lancaster's argument that Petitioner was correctly diagnosed with NMOSD in another supplemental report. Pet'r's Ex. 116. He stated, "that the diagnosis of 'seronegative NMO' is the third most likely diagnosis here," following TM/ADEM and MOG, respectively. *Id.* at 1. Dr. Steinman criticized the treaters' decision to wait eight years in Petitioner's treatment "to do a MOG antibody test," arguing that the delay "may have ruined the chance to make a correct diagnosis of [MOG antibody-associated disease ("MOGAD")]." *Id.* Ultimately, his "opinions in this case remain unchanged since [his] first report in 2018." *Id.* at 9. Dr. Lancaster opined that "[i]t is illogical to reject a diagnosis for which Petitioner meets all criteria. It is even more illogical to invoke two other diagnoses for which Petitioner clearly does not meet the diagnostic criteria." Resp't's Ex. I at 2.

b. Causation

Dr. Steinman, relying on NINDS, noted that in most cases, TM is idiopathic. Pet'r's Ex. 25 at 11. However, he added there are immune system disorders that cause TM, including MS, NMOSD, "post-infectious or post-vaccine autoimmune phenomenon," and "an abnormal immune response to an underlying cancer." *Id.* at 12. Additionally, it can be unclear "whether direct viral infection [such as varicella zoster, herpes simplex, influenza, hepatitis B, mumps, pertussis, tetanus, diphtheria, measles, and rubella] or a post-infectious response to the infection causes the [TM]." *Id.*

In cases where there is vaccine causation, Dr. Steinman's "theory is based on the concept that components of the 2015 Fluzone vaccine triggered neuroinflammation in the central nervous system, characterized by aspects of ADEM and [TM]." Pet'r's Ex. 25 at 13. Relying on self-authored, peer-reviewed studies, Dr. Steinman described molecular mimicry by way of illustration. *Id.* at 14.

[6] Dean M. Wingerchuk et al. *International Consensus Diagnostic Criteria for Neuromyelitis Optica Spectrum Disorders*, 85 NEUROLOGY 177 (2015).



Id.

He asserted that just five amino acids in a peptide chain that share homology with a viral chain, even if non-consecutive, can induce experimental autoimmune encephalomyelitis ("EAE") in mice models. Pet'r's Ex. 25 at 15. The Gautam et al.[7] study "provided the first clear demonstration that EAE in mice can be induced by a cross-reactive viral peptide." Pet'r's Ex. 42 at 4. Specifically, a herpesvirus peptide that shares limited homology (a few as five amino acids) with a myelin basic protein ("MBP") peptide can stimulate T cell hybridomas and induce EAE. *Id.* at 1. A Journal of Immunology study Dr. Steinman referenced in his report showed that where "[five] of 11 amino acids were identical between the [varicella] virus and MBP, and only [three] were consecutive[, they] were able to induce encephalomyelitis with paralysis." Pet'r's Ex. 25 at 16 (citing Pet'r's Ex. 42). Dr. Steinman acknowledged that although animal models are not direct evidence of a specific homology that would induce "[TM] in Petitioner [and] trigger[] autoimmunity to myelin antigens[, t]hey set a minimal standard for what COULD cause a neuroinflammatory disease." *Id.* at 18 (emphasis in original). Dr. Steinman stated that there "are at least three known antigens that are attacked by the immune system in various forms of neuroinflammation," including AQP4 (seen in NMO), MBP, and MOG. *Id.* at 14. He noted that "[AQP4] antibodies were not detected in Petitioner." *Id.* Dr. Steinman searched for homologies between the A/California/7/2009 (H1N1) component of the 2015 Fluzone vaccine received by Petitioner, and MBP, using the resources of the National Institutes of Health ("NIH") and the National Library of Medicine. *Id.* at 19. He identified the sequence GTCYPGDFIDY with five identical amino acids out of 11 and the sequence GYAADLKS with five of eight identical amino acids, both of which he argued are "sufficient to trigger neuroinflammation in the central nervous system." *Id.* at 21. Dr. Steinman also searched for homologies "between MOG and the hemagglutinin of the H1N1 component of Petitioner's [flu] vaccine." *Id.* at 21. He asserted that

[7] Anand M. Gautam et al., *A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis*, 161 J. IMMUNOLOGY 60 (1998).

"MOG is associated with ADEM and myelitis." *Id.* He identified "[o]ne sequence TGMVDGWY [with six] of [eight] identical amino acids[, and argued t]his is sufficient to trigger neuroinflammation in the central nervous system." *Id.* at 22.

To further support his assertion that molecular "mimicry between a virus and MBP can lead to clinical paralysis," Dr. Steinman also discussed the Ufret-Vincenty et al.[8] paper. Pet'r's Ex. 25 at 25 (citing Pet'r's Ex. 46). The team "passively transferred T cells that crossed-reacted" in mice "primed with the papillomavirus peptide and stimulated in vitro with MBP," and they "developed severe relapsing–remitting EAE with an incidence of 100%" when adjuvants were used. *Id.* Dr. Steinman described "[t]he rarity of such reactions in humans" as reassuring for the continued use of vaccines. *Id.* Further, he acknowledged that "[t]he use of adjuvants in the laboratory allows for a much higher percentage of experimental animals getting sick[] and thus allows dissection of the pathogenic mechanism." *Id.*

Dr. Steinman also noted that molecular mimicry is widespread and "immunity to nervous system antigens like myelin is rather widespread in normal[, healthy, individuals.]" Pet'r's Ex. 25 at 27. Consequently, "immunity to myelin is necessary but not sufficient to get actual disease." *Id.* He asserted genetic and environmental factors are needed for pathogenesis. *Id.* "Nevertheless, molecular mimicry is a key mechanism in understanding how tolerance to 'self' structures like myelin and axonal proteins is broken." *Id.* at 27–28.

Dr. Lancaster argued that there is "no good evidence" that low-level homology can cause autoimmunity or pathogenesis. Resp't's Ex. A at 10. He noted that "[t]here are 20 amino acids in human proteins that can be assembled in any order." *Id.* While the odds of a series of five amino acids being compared is one in 3.2 million (20x20x20x20x20), in a protein that is 1,000 amino acids longs, there are one million comparisons to do. *Id.* "Suddenly, the likelihood of finding a match is much higher." *Id.* The likelihood is again increased when the comparison includes gaps in the series homology. *Id.* Dr. Lancaster asserted that this process is "totally useless for predicting actual autoimmune disease, because if it is valid then just about anything causes autoimmunity to just about everything." *Id.* The Silvanovich et al.[9] study, cited by Dr. Steinman and Dr. Lancaster, noted that a sliding scale window "searches for short amino acid sequence matches of eight amino acids or fewer to identify proteins as potential cross-reactive allergens is a product of chance and adds little value to allergy assessments for newly expressed proteins." Pet'r's Ex. 45 at 1. However, the same study also noted that searches performed using BLAST algorithms, like the one performed by Dr. Steinman, "rank the similarity of a query protein to its corresponding matches and provide a measure of the reliability of alignment," when used "to determine the potential for immunologically based cross-reactivity where IgE directed against a known allergen could bind to the protein and elicit a clinical reaction in sensitized individuals." *Id.* at 1, 7. Dr. Lancaster asserted that Silvanovich et al. warns not to oversimplify this point and narrows "how we might actually predict a match, which requires very large areas of significant homology – they give the example of 35% identity over at least 80 amino acids." Resp't's Ex. D at 2.

[8] Rafael L. Ufret-Vincenty et al., *In Vivo Survival of Viral Antigen-Specific T Cells that Induce Experimental Autoimmune Encephalomyelitis*, 188 J. EXPERIMENTAL MED. 1725 (1998).
[9] Andre Silvanovich et al., *The Value of Short Amino Acid Sequence Matches for Prediction of Protein Allergenicity*, 90 TOXICOLOGY SCIENCES 252 (2006).

Dr. Steinman's studies on molecular mimicry are not instructive in Dr. Lancaster's opinion, because "[t]hese experiments involve particular strains of rodents and powerful adjuvants, as well as one particular carefully selected sequence to stimulate a particular T cell response in the animals." Resp't's Ex. A at 10. Likewise, the Ufret-Vicenty et al. study is not applicable because the relevant viral protein that activated the T cells and was also recognized by MBP "is not present in the [flu] vaccine." *Id.* 11–12.

c. Timing

Dr. Steinman wrote that Petitioner was diagnosed with ADEM versus TM on December 10, 2015, "within a month[] of the [flu] vaccine on November 15, 2015." Pet'r's Ex. 25 at 28. He characterized the complaints between vaccination and diagnosis as "harbingers of the diagnoses." *Id.* To establish a timeline, Dr. Steinman referenced Petitioner's "first visit in the records to urgent care after the [flu] vaccine [which] was four days later on November 19, 2015." *Id.* at 28 (citing Pet'r's Ex. 5 at 3–5). He noted her report to treaters that she was "seen by her PCP for the same symptoms about two weeks earlier and was told that her symptoms were due to stress and tension." *Id.* Dr. Steinman was unable to determine whether, at that time, "any of these visits were actually manifestations of demyelinating disease or whether they represented cervical disc disease." *Id.* at 28. He asserted that regardless, the temporal relationship is appropriate for post-vaccination ADEM. *Id.* He cited Bennetto & Scolding[10] and argued "neuroinflammation in the [CNS] manifests as ADEM 'typically [one to] 14 days after non-neural vaccines, a week or less after the appearance of a rash in exanthematous illnesses, and [one to three] weeks (or more) after rabies inoculation.'" *Id.* (citing Pet'r's Ex. 65). He cautioned that Petitioner's symptoms of cervical spondylotic myelopathy should not be confused with her later diagnosis of ADEM or TM, despite their similarities in presentation. *Id.*

Dr. Lancaster opined that "[t]he first symptoms possibl[y] related to the present illness were reported on [January 17, 2015], when Petitioner was evaluated by Dr. Abbas Mirza in the ER at Baptist hospital." Resp't's Ex. A at 2. He listed these as "neck pain, left arm pain and left hand numbness," and noted that onset "was the prior night." *Id.* Dr. Lancaster acknowledged that "[t]he diagnosis at the time was cervical neuralgia," but asserted "[i]t is possible that this was the initial symptom of her spinal cord inflammation." *Id.* In respectful disagreement with Dr. Steinman, Dr. Lancaster opined that "Petitioner already had neck pain when she received the vaccine. It is highly improbable that this was a coincidence and not the presenting symptom of her myelitis." *Id.* at 10. Dr. Lancaster noted that the reason for Petitioner's medical visit on the date she received her vaccination was neck pain. *Id.* at 8. During her visit to urgent care on November 19, 2015 (post vaccination), her complaint of neck pain had an onset of three weeks. This would have preceded her vaccination. *Id.*

Dr. Steinman challenged Dr. Lancaster's contention that Petitioner's pre-vaccination neck pain was related to her myelitis. Pet'r's Ex. 56 at 3. He opined that "it would be most unusual for neck pain to persist from a time point [three] weeks before the November [19, 2015] visit[,] until [December 5, 2015,] when the first symptoms were noted." *Id.* He continued that even if "the neck

[10] L. Bennetto & N. Scolding, *Inflammatory/Post-Infectious Encephalomyelitis*, 75 J. NEUROLOGY NEUROSURGERY PSYCHIATRY 22 (2004).

pain [was] the earliest manifestation of a smoldering myelitis, if not for the vaccine on November 15, 2015, the severe neurologic deficits would never have occurred." *Id.* at 4.

2. Testimony

a. Diagnosis

Dr. Steinman's testimony remained consistent with the opinions expressed in his written reports. Dr. Steinman explained that the research on these conditions has "changed so much that getting hung up on any one paper or trying to put this diagnosis in any one box becomes an errand that is close to impossible, so [he chose] to say that the main issue in her life . . . is inflammation of the white matter, and[] call[ed] it myelitis." Tr. 43. When asked directly to name Petitioner's diagnosis, Dr. Steinman stated, "I think she suffers from an overall diagnosis that I would call myelitis. It has various aspects, including ADEM, which we've defined, and [TM], which we've defined. It also could include seronegative NMO, and it could also include MOGAD." Tr. 44. Acknowledging that he identified more than one condition, he further explained "the overlap is extensive between all three diagnoses." *Id.* Generally, "myelitis – the myeloid part is the white matter that surrounds the part of the nerve root that conducts electricity, insulation on a wire, and the -itis means inflammation." Tr. 45. Dr. Steinman then defined ADEM as "disease of the brain, . . . [a]nd there are disseminated, meaning in many places, areas of inflammation, caused by an immune attack against something in the brain." Tr. 50. He asserted that despite Dr. Lancaster's contention, "encephalopathy, meaning some change in the level of your consciousness," is not necessary for diagnosis. Tr. 51. Dr. Steinman further criticized Dr. Lancaster's need to "wait[] until every box in the pediatric criteria for ADEM had been ticked" at the risk of Petitioner's condition unnecessarily worsening. Tr. 52–53. He noted that her three treating neurologists likewise did not wait for a definitive diagnosis before beginning treatment for myelitis with ADEM as a differential. Tr. 53.

In addition to ADEM, Dr. Steinman discussed NMOSD because it is another overlapping syndrome. Tr. 53. He explained that "the optic nerve goes back to the back of your brain in the visual cortex, . . . [s]o you get inflammation of the optic nerve. And that's often a feature of the disease we call [NMO]." Tr. 53. He noted that the syndrome also includes cases where the antibodies associated with NMO, AQP4, are not detected, which Dr. Wingerchuk's 2015 article called seronegative NMO. Tr. 54–55. Lastly, Dr. Steinman included MOGAD as a part of "the NMO story." Tr. 55. He noted that Petitioner's treaters did consider ADEM and imaged Petitioner's brain. Tr. 59. Without definitive results, "they[were] treating the wide number of named diagnoses, and fortunately, the treatments are all very similar." Tr. 59–60. Dr. Steinman continued that TM could develop into ADEM or they could coexist. Tr. 60. "Sometimes you see [TM] without ADEM. Most of the cases I've seen of [TM] do not involve ADEM, . . . but a large number have evidence of both." Tr. 60.

Evidence of a particular subset of myelitis can sometimes be determined by the presence of antibodies. Tr. 62. Dr. Steinman noted that Petitioner was tested for AQP4 early on in her treatment, with negative results. Tr. 63. However, the testing "may have been negative because they gave corticosteroids which could have suppressed the measurement." *Id.* Dr. Steinman mentioned that later in 2024, Petitioner underwent additional testing for MOG, which was also

negative. Tr. 64. He acknowledged the lack of evidence in this case "for positive aquaporin test, a positive MOG test, and [that] they never actually tested for an immune response to myelin basic protein. So[,] Petitioner[is] left with a theory provided where all the suspects cannot be proven." *Id.*

Ultimately, Petitioner had CNS inflammation with brain and spinal cord lesions, "so ADEM is one of the viable diagnoses." Tr. 66. Dr. Steinman dismissed the focus on identifying a specific diagnosis, testifying that the experts in this case were "all tied up in knots worrying about these various categories which overlap anyway." *Id.* When asked specifically about MS, Dr. Steinman asserted that he "would not call [what Petitioner has] MS, but [he] can understand why somebody might want to add that to the already complex series of diagnoses." Tr. 72. He conceded that Petitioner met the 2015 diagnostic criteria seronegative NMO. Tr. 136. However, Dr. Steinman asserted that Petitioner has ADEM, in "agree[ment] with the treating doctors." Tr. 138. He did not require encephalopathy or "the rigidity of three months," and noted that although ADEM is monophasic, relapses occur in rare instances. *Id.* Petitioner had an acute, disseminated myelitis with "lesions in various places on MRI, including the spinal cord, but many places in the brain." Tr. 139. He concluded, "[t]herefore, ADEM." *Id.* He added that between his first-place diagnosis (ADEM/TM), second-place diagnosis (MOGAD), and third-place diagnosis (seronegative NMO), if "talking to residents in a conference, [he'd] say [w]hat difference does it make." Tr. 146. Dr. Steinman was unable to "give a simple answer in real time" as to why he ranked the differential diagnoses the way he did. *Id.* He stated he "can give a pretty good answer if [he] spent two minutes looking at [his] report." *Id.*

The following extended question and answer period occurred in response to follow-up about Dr. Steinman's diagnostic rankings.

> THE COURT: I believe that [Respondent's counsel] is asking you, because of your -- because of your expertise, to consolidate and answer the very specific question of the distinguishing factors. If they're not exactly the same, there has to be some degree of difference.
> THE WITNESS: Right.
> THE COURT: And what she's looking for and quite frankly the reason why I'm entertaining this long tangent is because I'm also interested in the difference between the three conditions. You say they are umbrellas, but they are not exactly the same. So we're not interested in the similarities. We're interested in the difference.
> THE WITNESS: Right. The differences are that the 2015 Wingerchuk criteria are outdated, now that we have a more complete understanding of the MOGAD . . .
> THE COURT: Outdated how? What's new?
> THE WITNESS: Well, in the 2015, sometimes –
> THE COURT: No, no. Don't tell me about the 2015. Tell me about what's new now . . .
> THE WITNESS: We know much more about MOGAD than we did in 2015.
> THE COURT: What do we know that's -- what do we know now that is relevant to you deciding between MOGAD and TM and ADEM? What is different? . . .

> THE WITNESS: There's a whole disease category called MOGAD that there wasn't in 2015 when Wingerchuk said some people with seronegative NMO have MOG antibodies. Now we put them in a different disease category called MOGAD. We don't call it seronegative NMO. We call it MOGAD.
> THE COURT: So it is your testimony now that MOGAD and seronegative NMO are the same thing.
> THE WITNESS: No. They're subtly different, . . .
> THE COURT: What makes them different, Dr. Steinman? What makes them different?
> THE WITNESS: That there's a new category to put ahead of seronegative NMO.
> THE COURT: So how do you decide whether to put someone in the seronegative NMO bucket? A patient comes to see you. How do you decide whether or not to put them in the MOGAD bucket or the seronegative NMO bucket? What's the difference?
> THE WITNESS: By measuring -- being able to measure MOG antibodies . . .
> THE COURT: So if they have MOG antibodies, they have MOGAD. If they don't have MOG antibodies, they have seronegative NMO?
> THE WITNESS: Yeah. In a way they have seronegative MOGAD.
> THE COURT: . . . So are you saying seronegative NMO and seronegative MOGAD are the same thing?
> THE WITNESS: I would move seronegative NMO to mean negative for aquaporin-4, and seronegative MOGAD to mean negative for MOG.
> THE COURT: Well, we don't have those antibodies in either -- either of those antibodies in this case. So all of that is irrelevant to this case.

Tr. 149–52.

Finally, Dr. Steinman summarized that he believed ADEM to be the accurate diagnosis. Tr. 153. He continued that there is an explanation for the negative MOG antibodies, but "they waited too long" to do the antibody testing for AQP4. *Id.* He placed MOGAD in second place, "because [he had] a better explanation for they that could have been negative. And by the way, [] better molecular mimic, E value .01. And number three, [he] put as seronegative [NMO]." *Id.*

Dr. Lancaster, conversely, testified that Petitioner's condition was most consistent with seronegative NMO. Tr. 196. He relied on "the fact that she developed a very severe longitudinally extensive [TM] that progressed well beyond the time that [idiopathic TM] could be correct. And then she subsequently developed evidence of optic neuritis." Tr. 196. Dr. Lancaster disagreed with Dr. Steinman's assertion that Petitioner did not have ON, referring to her "MRI studies showing evidence of inflammation in the optic nerve." Tr. 197. The lack of encephalitis ruled out the possibility that Petitioner suffered from ADEM for Dr. Lancaster. Tr. 199. He distinguished ADEM from NMO based on "the progression of the spinal cord lesions in NMO versus the time-limited progression in ADEM, . . . the effects on cognition in ADEM that are not generally seen in NMO," and the prominence of brain lesions seen in ADEM and brainstem, spinal cord, and optic nerve inflammation in NMO. Tr. 206. In Petitioner's case, Dr. Lancaster argued that her "years of progressions of severe inflammation" is more indicative of NMO. *Id.*

Next, Dr. Lancaster ruled out MOGAD as a differential diagnosis. He defined MOGAD as "the spectrum of beings that are found in association in patients with MOG antibodies," and asserted that he did not know what seronegative MOGAD is. Tr. 208. Unlike the specific antibody that characterizes MOGAD, "NMO is the clinical phenotype. It's what patients look like without specifying any particular antibody or immune mechanism." Tr. 209. Simply, NMO is "progressive myelitis with severe bilateral optic nerve involvement, [that is] not a part of ADEM, [and] it's beyond idiopathic [TM]." *Id.* Dr. Lancaster did not agree with Dr. Steinman's explanation of treatment and delayed testing for Petitioner's negative antibody panel. He testified that "it becomes quite unlikely in the context of still having clinical progression and attacks, because we're blaming an antibody for the disease that isn't there anymore." Tr. 212.

b. Causation

Dr. Steinman opined that "the [flu] vaccine had constituents that are a close -- we call them molecular mimics of proteins in the white matter that are attacked in various diseases, including ADEM and [TM]." Tr. 40. In the specific vaccine the Petitioner received, Dr. Steinman identified hemagglutinin and neuraminidase as the two components that "are of major interest." Tr. 47. Correspondingly, he identified three proteins in the myelin sheath involved in TM and ADEM specifically, MBP, AQP4, and MOG. *Id.* Post vaccination, Dr. Steinman explained, "these proteins are attacked by the immune system, and when those proteins are attacked, the myelin gets injured, gets inflamed, and does not do a very good job of conducting electricity." Tr. 48. He asserted that "[i]t does not make any difference" with respect to his causation theory whether the proper diagnosis is NMO, NMOSD, MOGAD, TM, or ADEM. Tr. 56.

On cross-examination, Dr. Steinman noted his use of the Immune Epitope Database to emphasize that he does a "hierarchical bioinformatic search" to find potential relevant homologies between peptide sequences. Tr. 103. When asked specifically to identify all the information he considered to conclude that, for example hemagglutinin and MBP shared sufficient homology to trigger neuroinflammation, he responded that he "can't be more specific than to say the totality of all the evidence." *Id.*

During a back-and-forth exchange with Respondent's counsel, Dr. Steinman was asked about the particulars of the methodology used by Gautam et al. to induce EAE in mice. He agreed that the researchers began with mice that possessed specific, known major histocompatibility complex molecules ("MHC")[11] that can bind to a specific MBP protein to cause EAE, because that match is required for molecular mimicry to occur. Tr. 154. Dr. Steinman also acknowledged additional controls within the study, including in vitro peptide binding compatibility testing, the use of an adjuvant, and the need for specific amino acids within the peptide chain to remain in nontransferable positions within the homologous sequence. Tr. 158–62. He explained that

[11] MHC is defined as a group of closely linked multiallelic genes located in a small region on one chromosome that determine the major allelic alloantigens that can stimulate an immune response, known as HLA molecules in humans. *See Major Histocompatibility Complex*, DORLAND'S ONLINE MED. DICTIONARY, https://www.dorlandsonline.com/dorland/definition?id=66341&searchterm=major+histocompatibility+complex (hereinafter, *Dorland's*); *see also Histocompatibility Antigens*, *Dorland's*. Alloantigen is defined as "an antigen present in allelic forms encoded at the same gene locus in different individuals of the same species." *Alloantigen*, *Dorland's*.

although a specific HLA complex with sequences that could cross react and lead to EAE in humans has not been identified, use of the Immune Epitope and Influenza Research Databases narrowed his BLAST search to relevant chains. Tr. 167. The BLAST searches conducted in this case on the entire protein did not reveal homologies within the amino-dominant epitope seen in a majority of humans. Tr. 168–69. Dr. Steinman argued that despite the risk of false positives based on homology, BLAST searches are "a good place to start." Tr. 173. "I'd rather cast a net that's wide and catch a false positive than miss a real positive." *Id.*

Dr. Steinman testified that his causation theory can explain any type of post-vaccination myelitis, including MS, and is defined by an appropriate temporal relationship (vaccination to symptom onset), BLAST search results, "useful" case reports (Karussis & Petrou),[12] and "helpful" medical literature (Jarius et al.).[13] Tr. 189. The Karussis & Petrou paper recorded 71 documented cases of CNS demyelinating diseases temporally associated with various vaccinations, including flu. Pet'r's Ex. 125 at 1. The authors highlighted a high percentage of flu vaccinees who presented "as optic neuritis and myelitis (with or without additional manifestations of ADEM), reminiscent to [NMO] (or, more generally, the NMO-spectrum of diseases)." *Id.* The paper discussed CNS autoimmunity and ADEM, "but only when the vaccine included Freund's adjuvant." *Id.* at 6. Ultimately, "the existing data from epidemiological studies argues against a clear causal relationship between vaccines in general, and MS or other demyelinating diseases and advocates in favor of the benefits of vaccinations versus the potential risks of CNS inflammation/demyelination." *Id.* at 7. The Jarius et al. article focused on defining NMOSD and noted that all conditions within the spectrum (MOG/MOGAD) rarely occur following vaccination. Pet'r's Ex. 127 at 8. The authors clarified that "[i]t is unclear whether infection or vaccination induces a de-novo autoimmune reaction in these patients (e.g., by molecular mimicry) or rather acts as a nonspecific inflammatory trigger causing clinical exacerbation in patients with pre-existing but previously clinically latent NMOSD." *Id.*

Both Drs. Steinman and Lancaster agreed that "you can theoretically have recognition of a peptide that is quite short, and that if you maintain just a few of the critical amino acids." Tr. 234. During his testimony, Dr. Lancaster added,

> Does that mean that just because you found a sequence in a vaccine protein and a sequence in a human protein that share just a few amino acids, that one is in any way likely to cause a clinically relevant autoimmune disease by stimulating an immune response against the other?

Tr. 234–35. Dr. Lancaster answered, "it's highly unlikely to be true or, as we said before, any vaccine causes dozens, hundreds, thousands of immune conditions." Tr. 235.

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[12] Dimitrios Karussis & Panayiota Petrou, *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, 13 AUTOIMMUNE REV. 215 (2014).

[13] Sven Jarius et al., *Update on the Diagnosis and Treatment of Neuromyelitis Optica Spectrum Disorders (NMOSD) – Revised Recommendations of the Neuromyelitis Optica Study Group (NEMOS). Part I: Diagnosis and Differential Diagnosis*, 270 J. NEUROLOGY 3341 (2023).

c. Timing

In his testimony, Dr. Steinman acknowledged that Petitioner had neck pain ten months pre vaccination. He reiterated that "[a]cute [TM] typically evolves 15 over days, and by definition, reaches its maximum deficit within 21 days." Tr. 58. He did "not think that the January neck pain ha[d] anything to do with the [TM]." Tr. 59. Dr. Steinman placed Petitioner's myelitis symptom onset at "the numbness that she was feeling in the legs [that] manifest[ed] in early December, so that would have been about three weeks" post vaccination. Tr. 91. This "timing for onset was consistent with what is known about neuroinflammatory conditions following infection or vaccination." Tr. 92. The Karussis & Petrou article noted that participants in their study averaged 14.2 days from vaccination to symptom onset with an outer maximum of five months. Pet'r's Ex. 125 at 1. Specifically following the flu vaccine, onset ranged from four days to 25 days. *Id.* at 4.

Dr. Steinman maintained that it was "coincidental" that Petitioner experienced neck pain ten months prior to her vaccination and the onset of her myelitis. Tr. 128. Reiterating that TM is an acute condition, he noted that the ten months is too long a period between symptoms and evidence of demyelination to be related. Tr. 124. However, Dr. Steinman testified that he could not say that Petitioner's November 10, 2015 vaccine, was not related to her neck pain that was reported on the same day, pre vaccination. Tr. 129.

Dr. Lancaster identified Petitioner's neck pain as her presenting symptom because neck pain is extremely common with these disorders. Tr. 215. He added that "the pain preceded the other neurologic symptoms, such as numbness, weakness, trouble controlling the bowel and bladder, gait problems, et cetera," with no other explanation. *Id.* Conceding that Petitioner's treaters initially attributed her neck pain to stress, Dr. Lancaster noted that neck pain is a common symptom that is related to NMO in one in a million cases. Tr. 216. However, in Petitioner's case, "the parts of the spinal cord that would correspond to neck pain, shoulder pain or pain going up the back of the head are visibly inflamed and enlarged, grossly swollen to the point where it was visible with swelling on an MRI scan." *Id.* Dr. Lancaster noted that Petitioner did not undergo imaging when she complained of neck pain in January of 2015, and as a result, we "don't know for sure what caused it at that time." Tr. 218.

By November of 2015, and specifically the date of Petitioner's vaccination, "it is extremely likely," according to Dr. Lancaster, "that the neck pain is due to the myelitis. This is because we could later clearly see the myelitis occurring." Tr. 218. He also noted that "the way [Petitioner's] clinical symptoms were described is a continuous evolution of that neck pain going on into December and then even subsequently during other attack[s] in the future." Tr. 219.

Dr. Lancaster also noted that in some cases of myelitis, a patient, even when seropositive, can remain asymptomatic for several years resulting in a delayed diagnosis. Tr. 222. He explained that this can occur in cases where MS follows exposure to the Epstein-Barr virus and noted that that "almost all patients with [MS] have been exposed to the Epstein-Barr virus at some point prior, often years or decades prior." *Id.*

3. Post Hearing

On December 12, 2025, Drs. Steinman and Lancaster submitted final reports to address the changes to Petitioner's diagnoses as noted in updated medical records that had not been filed prior to the hearing. Dr. Steinman acknowledged that "it is now reasonable to include MS as a diagnosis for [Petitioner] along with the other diagnoses included in [his previous] report[s]." Pet'r's Ex. 136 at 7. He noted Petitioner's ten years of suffering from her condition and how consequently, "[p]rogressive MS became the diagnosis favored by her treaters." *Id.* at 6. Dr. Steinman again listed the three most likely diagnoses for Petitioner as "(1) a neuroinflammatory condition involving the central nervous system, most likely myelitis, TM, disseminated encephalomyelitis, ON; (2) MOGAD; and (3) seronegative NMOSD." *Id.* Notwithstanding the additional medical records, he maintained that "[n]o matter the exact diagnosis, [Petitioner] has an autoimmune demyelinating condition of the [CNS]. Her immune system mistakenly attacked healthy myelin in her brain, spinal cord, and optic nerves." *Id.* at 7.

Dr. Lancaster briefly summarized Petitioner's updated medical records from 2024 and 2025. Resps't's Ex. O at 1–7. He reiterated his disagreement with Dr. Steinman's diagnosis rankings and noted that Petitioner's "MOG antibodies have now been measured multiple times, including during severe attacks. They simply were not present." *Id.* at 7. He continued that "Petitioner did not have encephalitis (altered mentation) early in the disease and had a progressive disease course over at least [five] years now – this is so far beyond the [three]-month window for progression that ADEM is simply not a reasonable diagnosis." *Id.* Dr. Lancaster cited the 2025 McDonald[14] MS criteria and noted that Petitioner has the requisite two out of three: "negative brain MRI at onset (check), more than [three] spinal segments involved by the myelitis (check) and AQP4 ab (absent)." *Id.* Her "multiple clinical attacks involving myelitis, optic neuritis, and brain lesions, along with the evidence of both active and old CNS lesions on MRI studies, meet these criteria." *Id.* at 8. However, he noted that even with this presentation, NMO is still viable and in his opinion, preferred over the primary progressive MS diagnosis raised by Petitioner's treaters. *Id.* In Petitioner's case, "there were discrete attacks and some improvement with immune therapy after attacks," including with respect to her ON. *Id.* The cycle of worsening and improvement is less akin to a more continuous, progressive MS. *Id.* Dr. Lancaster wrote that his "opinions regarding vaccine causation [were] unchanged." *Id.* at 9.

IV. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) that petitioner suffered an "off-Table injury," one not listed on the Table, as a result of receiving a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). In this case, Petitioner does not allege a Table injury and must prove that her injury was caused-in-fact by a Table vaccine.

[14] This article was not filed.

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. 42 U.S.C. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of the Dept. of Health & Hum. Servs*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). In *Broekelschen v. Sec'y of Health and Hum. Servs.*, the Federal Circuit recognized that in some circumstances, the special master may "first determine which injury was best supported by the evidence in the record before applying the *Althen* test." 618 F.3d 1339, 1346 (Fed. Cir. 2010). This principle also means that a petitioner must establish that the vaccinee suffers the injury allegedly linked to the vaccination. *Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343, 1353–54 (Fed. Cir. 2011).

The *Althen* test requires petitioners to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano*, 440 F.3d at 1326.

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. 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). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322 and *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592

F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357,1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. However, in the Vaccine Program, the *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."); *see also Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the

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

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

The *Daubert* factors are "meant to be helpful, not definitive." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 151 (1999). The factors do not "constitute 'a definitive checklist or test'" and may be applied differently depending on the facts of a particular case. *Id.* at 150 (quoting *Daubert*, 509 U.S. at 593).

"In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Daubert*, 509 U.S. at 590 (citation omitted). Thus, for Vaccine Act claims, 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. Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *24 (Fed. Cl. Spec. Mstr. Nov. 28, 2016) (stating that the Vaccine Act "require[s] a chain of reliable propositions supporting [a] petitioner's theory").

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278;

Capizzano, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. This is because "treating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records 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. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not "binding on the special master or court." 42 U.S.C. § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008); . Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also*

Grant, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Hum. Servs*., 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that she suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen*, 35 F.3d 543 at 547. In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Knudsen*, 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden then shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that the respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" 42 U.S.C. § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

V. Discussion

A. Diagnosis

While the specific iteration of Petitioner's condition is disputed among her treaters and the medical experts in this case, they all agree that she suffered from some type of inflammatory myelitis. In emergency care visits post vaccination, Petitioner's treaters considered various diagnoses including cervical myelopathy, ADEM, TM, NMOSD, and MS. It is clear from the medical record, the medical literature, and the reports of Drs. Steinman and Lancaster that all these demyelinating diseases overlap and can be difficult to differentiate. This is particularly true in the early stages, because for example, one distinguishing factor of ADEM is no progression of symptoms three months beyond onset. With some clinical presentations, it would be difficult to rule out ADEM until that time period has passed. Furthermore, the contemporaneous nature and need for accuracy make medical records persuasive evidence of the nature of Petitioner's condition. Petitioner's treaters, over a period of several years, admitted her to the ER for observation, conducted repeated laboratory and imaging tests, and referred her to several specialists in an attempt to define her condition. Her extensive workups revealed the difficulty identifying her specific injury, and her treaters appeared to carefully consider all conditions later asserted by Dr. Steinman and Dr. Lancaster.

ER records from late 2024 consistently characterize Petitioner's condition as a subtype of MS. Additionally, notes from an ophthalmology consult in June of 2025 include a confirmed diagnosis of NMO based on Petitioner's progressive bilateral vision loss. It is not the role of the special master to diagnose a petitioner, and in some cases, it may be impossible to definitively identify the exact condition that a petitioner is suffering from. *Astle v. Sec'y of Health & Hum.*

Servs., No. 14-369V, 2018 WL 2682974, at *19 (Fed. Cl. Spec. Mstr. May 15, 2018). The Federal Circuit explained that "[t]he function of a special master is not to diagnose vaccine-related injuries, but instead to determine based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [P]etitioner's injury." *Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343, 1351 (Fed. Cir. 2011) (internal quotation marks omitted) (quoting *Andreu*, 569 F.3d at 1382). However, any injury that Petitioner alleges must be specific enough for a causation theory to reasonably explain how an alleged vaccination leads to disease development. *See Broekelschen*, 618 F.3d at 1346 (citing *Kelley v. Sec'y of Health & Hum. Servs.*, 68 Fed. Cl. 84, 100-01 (2005) for the proposition that "the [P]etitioner [is] not required to categorize his injury where the two possible diagnoses [are] 'variants of the same disorder'").

Dr. Steinman initially agreed with Petitioner's treaters when she was first evaluated for a neurological condition and diagnosed with TM. In his written reports, Dr. Steinman described Petitioner's injury as TM and disseminated encephalomyelitis and later named ADEM as the most likely diagnosis. As Petitioner's symptoms continued and worsened, Dr. Steinman disagreed with the evolution of her treaters' assessments. He did not completely rule out NMOSD and noted that consideration of MS was reasonable. However, he maintained his opinion on TM/ADEM and asserted that whether he characterized Petitioner's condition as "containing aspects of disseminated encephalomyelitis or simply ADEM is irrelevant" to etiology and treatment. Pet'r's Ex. 56 at 2. Dr. Steinman was equivocal during the hearing when asked to identify Petitioner's diagnosis. He generally called her condition myelitis, "including ADEM, . . . [TM], [and] could include seronegative NMO and could also include MOGAD." Tr. 44. Dr. Steinman noted that the similarities between these diseases are evident in the medical records. However, he did not acknowledge that actual differences in disease progression could also affect pathogenesis. For example, ADEM is, by definition, acute (i.e., short term). However, Dr. Steinman dismissed the ADEM diagnostic criterion that symptoms should not exceed a three-month-progression. Further, Dr. Steinman minimized Petitioner's clear ON as a significant indicator for NMO without adequate explanation. Ultimately, he reiterated several times the specific diagnosis is not necessary to determine etiology and treatment. Dr. Steinman's discussion of diagnosis was overall vague and, by his own admission, of little consequence. Therefore, I did not find it particularly persuasive.

Dr. Lancaster also agreed with Petitioner's initial diagnosis of TM based on her presentation at that time. Similar to Dr. Steinman, Dr. Lancaster disagreed with Petitioner's treating neurologist that she currently suffers from MS, and he maintained his opinion more likely than not, the correct diagnosis remains seronegative NMOSD. Resp't's Ex. O at 9. Dr. Lancaster argued that a relapsing/remitting subtype "is less accurate" because of Petitioner's spinal cord and optic nerve lesions, and the extended scope and severity of her symptoms. *Id.* He continued that primary progressive MS is even less indicative here "due to the presence of clear discrete attacks with partial improvement after steroid therapy." *Id.* Dr. Lancaster agreed with treating ophthalmologists that Petitioner suffered from NMO, and he consistently maintained that seronegative NMO best matches the whole of Petitioner's clinical presentation. He noted that Petitioner has clear evidence of ON and acute myelitis involving lesions on more than three spinal segments. Even in his earlier reports, Dr. Lancaster never ruled out the possibility that Petitioner's condition may develop into MS. He also testified that NMO and MS may overlap in patients. This opinion is consistent with Petitioner's latest diagnosis, though Dr. Lancaster did not believe that

to be the case here. While I do not presume to determine Petitioner's diagnosis here, the totality of the record includes preponderant evidence that she suffered from NMOSD as diagnosed by Drs. Park and Lao and asserted by Dr. Lancaster. There is not preponderant evidence that Petitioner suffered from ADEM. It is not necessary to reach the question of whether Petitioner also suffers from MS. Petitioner does not propose a causation theory for vaccine-caused MS. Indeed Dr. Steinman argued that the exact diagnosis is irrelevant and that his theory is applicable to any demyelinating disease. Petitioner's causation theory is discussed further below in the *Althen* analysis.

B. *Althen* Prong One – Medical Theory

Dr. Steinman argued that components of the 2015 Fluzone vaccine triggered CNS inflammation by way of molecular mimicry. He provided a general explanation of molecular mimicry that applies to many types of cross-reactivity, benign and pathogenic. Indeed, Dr. Steinman agreed that molecular mimicry is a common occurrence in healthy individuals, and other factors are needed to for pathogenesis. The literature that Dr. Steinman cited in support of his theory established that EAE can be induced in mice due to a cross reaction with a viral peptide. However, the peptide chain was from herpesvirus and cross reacted with a MBP peptide in mice. Dr. Steinman did not identify the similarities between herpesvirus and the Fluzone vaccine, such that the reaction detailed in the study could be extrapolated to explain potential pathogenic cross reactivity between a vaccine for a different illness and disease in a human body. Dr. Steinman's response to this criticism was an acknowledgement that animal models are not direct evidence and an argument that the animal model can "set a minimal standard for what could cause a neuroinflammatory disease." Pet'r's Ex. 25 at 18. Dr. Steinman's self-described minimal standard requires additional support to meet the standard pursuant to *Althen*, specifically: can the vaccine at issue cause the type of injury alleged? Petitioner is not expected to provide evidence that is clear and convincing or to the degree of medical certainty. However, in the event that the theory proposed relies on extrapolation or analogy, the preponderant standard "require[s] a chain of reliable propositions supporting [a] petitioner's theory." *D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *24.

Dr. Steinman was asked directly to identify "the tools at hand that [he] use[d] to determine whether or not in general a vaccine can cause an injury." Tr. 103. His response was "I can't dissect it into I use it at each step." Tr. 104. He reiterated that he considered "[t]he totality of all the evidence," which he later explained included a filtered BLAST search, application of criteria from multiple studies that rely on homology of five amino acids in a sequence of 10/12, and review of the Immune Epitope Database for significant findings. Tr. 105–06. Dr. Steinman asserted that he identified potential sites for cross reactivity that shared homology of five peptide chains with Fluzone sequencing. In addition to MBP, Dr. Steinman identified MOG and AQP4 protein sequences as potential molecular mimics, but he focused on MBP for Petitioner's case. Dr. Steinman admitted that he was unable to find any evidence that the MBP sequences that he identified have cross reacted to cause neuroinflammation in any animal. Tr. 114. However, Dr. Steinman asserted that this theory was applicable to other vaccines (HPV) and any other types of myelitis (MS). Tr. 189–90. As I and other special masters have stated multiple times, and to Dr. Steinman in particular, molecular mimicry is a theory that has been accepted in the program as a causation theory for specific conditions following specific vaccinations. *See, e.g., R.G.C & S.S.C.*

v. Sec'y of Health & Hum. Servs., No. 18-1624V, 2025 WL 3142007 (Fed. Cl. Spec. Mstr. Aug. 28, 2025); *Anderson v. Sec'y of Health & Hum. Servs.*, No. 18-484V, 2024 WL 557052, at *30–32 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947, at *8–20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). It is not a one size fits all wherein a petitioner can simply identify an autoimmune disease, name molecular mimicry and assert that this theory applies regardless of vaccine or injury. That approach is particularly problematic in this case, because while vaccination involves a localized, acute introduction of an antigen to the body, MS and NMO are chronic conditions that involve sustained autoimmune dysfunction. It is unexplained and unclear how this initial cross reaction could evolve into an illness that can progress over a period of years or even decades. There may yet be such an explanation, but Dr. Steinman failed to meaningfully address this issue. As noted above, his circular and combative reasoning can make it difficult to assess whether his theory is sound or reliable. Petitioner is not expected to produce epidemiological studies or identify a specific mechanism with exact homology for cross reactivity. Petitioner is expected to provide some evidence that would establish causation based on the facts of her claim that is not so vague as to apply indiscriminately to every petition alleging some type of vaccine caused some type of myelitis. Petitioner has not provided preponderant evidence of a causation theory pursuant to *Althen* prong one.

C. *Althen* Prong Two – Actual Causation

Without a viable theory, it is impossible to determine if there is a logical sequence of cause and effect that is consistent with Petitioner's clinical presentation and diagnosis and vaccine causation. However, Petitioner must provide preponderant evidence that her vaccination was a substantial factor in causing her illness, and that she would not have developed her inflammatory myelitis in the absence of her Fluzone vaccination. Such supporting evidence is usually found in Petitioner's medical records, but Petitioner is not expected to produce opinions on causation from treaters. In fact, treaters are often silent in the medical records about the etiology of neurological conditions. Many of these diseases commonly alleged in the Program are usually idiopathic, slowly progressive, or are multifactorial; particularly for treaters, causation can be impossible to determine. Additionally, treaters often do not have the time or specialty to contemplate pathogenesis when treating a seriously ill patient.

In the present case, Petitioner was seen by a variety of medical professionals, including ER physicians, neurologists, and other specialists to understand the nature of her disease. Despite her concerns, and reported history of a recent vaccination, Petitioner's cervical myelopathy diagnosis with differentials of ADEM, NMO, and MS, included the annotation, "exact etiology of this is unclear." Pet'r's Ex. 4 at 65. Other forms of evidence that have previously provided support of a pathological response to vaccination include a hypersensitivity or localized allergic response, encephalitis, or fever/flu-like symptoms. Notably, Petitioner's medical records indicate that she complained of a possible viral illness at the beginning of December, approximately three weeks post vaccination. She subsequently denied this and testified that the medical records noted this complaint in error. Petitioner did not otherwise report any immediate adverse reaction to the vaccination and identified her IUD as a potential cause of some of her viral symptoms when asked. After a review of the complete medical record, filed statements, and testimony, there is not preponderant evidence that the vaccine Petitioner received caused her alleged injuries. Petitioner

has not met her burden with respect to *Althen* prong two. As discussed below, the evidence of the timing and presentation of her symptoms is also inconsistent with actual causation.

D. *Althen* Prong Three – Temporal Relationship

A proximate temporal relationship is evidence that can support causation. Conversely, evidence of relevant symptoms that precede vaccination is persuasive evidence that can weigh heavily against a petitioner's claim. In his written report, Dr. Steinman focused on Petitioner's symptoms that developed at and around the time of her November 2015 vaccination. He cited the Bennetto & Scolding study that argued neuroinflammation in the CNS manifests as ADEM typically one to 14 days after non-neural vaccines. Pet'r's Ex. 25 at 28. Dr. Steinman did not put this timeframe in the context of molecular mimicry, which usually carries a three-to-42-day temporal window. During his testimony, Dr. Steinman opined that Petitioner's symptom onset was later, in early December, three weeks post vaccination. This timing is more in line with his proposed theory and the four-to-25-day range articulated in the Karussis & Petrou article that discussed neuroinflammation following flu vaccination. Pet'r's Ex. 125 at 4. Dr. Steinman admitted that Petitioner reported neck pain on November 10, 2015, prior to her vaccination. However, he likened this complaint to her January 2015 complaints and insisted that it was completely unrelated to Petitioner's post-vaccination pain, reported days later. Dr. Steinman was asked a hypothetical: "if I come to see you[,] complain of neck pain and then I get a flu shot and it turns out that that neck pain was the onset of my demyelination? -- then the flu shot wouldn't have caused it, just based on the timing -- correct?" Tr. 132. He responded that it was impossible to answer that question, because the facts in this case included a similar complaint of pain nine months earlier without myelitis. Tr. 133. Dr. Steinman asserted that given the totality of the circumstances, Petitioner could have had neck pain that was not myelitis, because she had pre-existing cervical neuralgia.

Dr. Steinman's argument belies Petitioner's own words to her treaters. Petitioner first complained of chest and neck pain with left arm numbness, tingling, and radiating pain over nine months prior to her vaccination on January 17, 2015. She was diagnosed with cervical neuralgia, and Dr. Steinman has consistently argued that those symptoms are distinguishable in time and substance from her myelitis post vaccination. Holding as true Dr. Steinman's distinction of Petitioner's January 2015 pain due to remoteness, Petitioner again complained of neck pain on November 10, 2015, prior to her vaccination but on the same day. Nine days later, she reported a two-week history of radiating neck and shoulder pain, during a visit to an express care clinic on November 19, 2015. Petitioner was then seen by her PCP with similar issues of radiating neck pain and reported that she had previously been seen for the same issues by urgent care. Petitioner complained of neck pain and shooting head pain on December 5, 2015, in the ER. On December 11, 2015, Petitioner reported a one-month-history of neck spasms that had been followed, two weeks later, by left leg and foot numbness. She had also begun to experience parathesias and double vision. In her multiple visits to health care providers, Petitioner repeatedly described the pain as continuous, reoccurring, and similar. She complained that prior treaters had dismissed her pain as stress-related, and it had continued. Petitioner did not believe that her November 2015 pain and her December 2015 onward pain was different, and she conflated the symptoms to her treaters at that time. At this point Petitioner's doctors were concerned about demyelination. Indeed, her contemporaneous medical records consistently place her myelitis symptom onset at or slightly

before her vaccination. Accordingly, there is preponderant evidence that Petitioner experienced neck pain that continued and progressed as a manifestation of myelitis at least as early as November 10, 2015, immediately prior to her vaccination. Petitioner has not provided preponderant evidence of an appropriate temporal relationship for vaccine causation, and she has not met her burden pursuant to *Althen* prong three.

VI. Conclusion

After a careful review of the record, Petitioner has failed to provide preponderant evidence that her November 10, 2015 flu vaccine caused her injuries, specifically ADEM, or any neuroinflammatory condition of the CNS, including TM or NMOSD. Accordingly, Petitioner's claim is **DENIED**. 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). [15]

IT IS SO ORDERED.

s/Herbrina D.S. Young
Herbrina D.S. Young
Special Master

[15] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.