In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 22-322V

HEATHER PETERSON,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: October 30, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Edward Kraus, Kraus Law Group, Chicago, IL, for Petitioner.

Alexa Roggenkamp, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On March 24, 2022, Heather Peterson filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that an influenza "(flu") vaccine she received on March 26, 2019, caused her to develop transverse myelitis "(TM"), and then multiple sclerosis ("MS"). Petition (ECF No. 1) ("Pet.") at 3. She has since acknowledged that her TM was the presenting symptom of what was later properly diagnosed as MS. *See* Petitioner's Motion for Ruling on the Record and Memorandum in Support, dated Dec. 27, 2024 (ECF No. 36) ("Mot.") at 9–11.

Both parties have filed expert reports and have also briefed their positions for resolution of this matter on the basis of the written record. *See* Mot.; Respondent's Brief, dated Mar. 27, 2025

[1] Under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

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(ECF No. 38) ("Opp."); Petitioner's Reply, dated Apr. 28, 2025 (ECF No. 39) ("Reply"). For the reasons set forth below, I hereby deny entitlement. Petitioner has not established preponderantly that the flu vaccine can cause MS, or did so to her—and no new medical or scientific thinking exists on the topic that would better support this causal theory, which I have on many prior occasions found wanting.

I. Factual Background

Petitioner was born on November 25, 1986 (and was therefore 32 years old when she received the vaccine at issue). *See* Pet. at 1. She had a medical history significant for hypothyroidism, Hashimoto's Thyroiditis, and Vitamin D deficiency. Ex. 5 at 110; Ex. 1 at 9.

Vaccination and Initial Neurologic Symptoms

On March 26, 2019, Ms. Peterson went to her primary care physician ("PCP"), complaining of fatigue and flu symptoms. Ex. 7 at 64–66. She had a generally normal examination but was diagnosed with the flu. *Id*. Her PCP prescribed Tamiflu, and also administered a flu vaccine. *Id*.; Ex. 5 at 1. There is no evidence of any immediate temporal reaction to this vaccination.

Petitioner's affidavit states that three days later (March 29, 2019), she noted the development of numbness and tingling of fingertips after holding her infant son during a movie, but assumed it was transient. Affidavit, dated Mar. 25, 2022, filed as Ex. 14 (ECF No. 10-5) ("Peterson Aff."), at 2; Ex. 21 at 77. On the morning of the 30th, however, she awoke with progressive numbness and tingling from her left upper extremity. Peterson Aff. at 2.

Petitioner maintains (although it does not appear the records relevant to these visits were filed) that she subsequently sought chiropractic assistance three times in the days thereafter for her paresthesia concerns. Peterson Aff. at 2. Eventually, however, she felt the numbness spreading to the left side of her body, then to her right side, and went to the emergency department ("ED") at Northwest Community Healthcare on April 2, 2019, for treatment. *Id;* Ex. 8. at 7. But she was discharged after no explanation could be provided for her symptoms from the testing she received. Peterson Aff. at 2.

The next day (April 3, 2019—now a bit more than a week after vaccination), however, Ms. Peterson awoke with worsened symptoms, and she went back to the ED complaining of left arm numbness for the prior four days. Ex. 8 at 3–6. She reported that the numbness first ran from her fingers to her elbow, and the next day, the numbness progressed to her entire left arm and left side. *Id*. On exam, she had decreased sensation in her left arm, side, and leg. *Id*. Her lab results and head CT were normal, however. *Id*. The differential diagnosis included MS, and Petitioner was discharged home and advised to follow-up with neurology. *Id*.

Later that same day, Petitioner visited a different hospital's ED, again reporting left arm weakness and paresthesias beginning five days prior while attending a movie (as well as the fact that she had experienced an upper respiratory infection with rhinorrhea and congestion for a week around that same timeframe). Ex. 21 at 65–66. She denied other symptoms, some of which are often viewed as neurologic (such as bladder control or dizziness). *Id.* at 65. The differential diagnosis proposed the possibility of a lesion "likely at level of [cervical spine]," although no imaging had yet been performed, and also deemed MS to be a "significant consideration." *Id.* at 66. Ms. Peterson was subsequently admitted to the hospital. *Id*.

Hospitalization and Evaluation of Condition

While hospitalized, Petitioner was evaluated by neurologist Smriti Wagle, M.D. Ex. 21 at 100–02. On exam, her muscle strength was 4/5, and her deep tendon reflexes were 2/4. *Id*. An MRI of the cervical spine (with and without contrast) performed on April 3, 2019, revealed "slightly expansile oval T2 hyperintense lesion within the spinal cord posteriorly to the left of midline at L3 level there is slight enhancement. It measures about 13 x 7 x 5 mm in the craniocaudal, AP and transverse dimensions." *Id*. at 102, 105.

Lab results included elevated ANA and IgG antibody readings, among other things. Ex. 21 at 105, 131; Ex. 2 at 33, 59. However, eight oligoclonal bands (a well-accepted biomarker of MS) were seen in her cerebrospinal fluid ("CSF") testing performed on April 5th (although other signs of infection were not). Ex. 21 at 125–26 (deeming the oligoclonal bands "supportive of a diagnosis of multiple sclerosis in the appropriate clinical setting"). Petitioner received IV steroids and was discharged home on April 5, 2019. *Id.* at 105.

A few days later (April 8, 2019), Petitioner visited her PCP for follow-up of her left-sided numbness that she reported began on "3/29/19." Ex. 7 at 67–68. Her left-hand strength was 4/5, and she had decreased sensation and impaired coordination. *Id*. at 69. Her PCP diagnosed myelitis "possible [secondary to] flu vaccine, but cannot confirm. Could be autoimmune such as MS or TM." *Id*. Petitioner was advised to continue steroids and follow-up with neurology, occupational therapy ("OT"), and physical therapy ("PT"). *Id*.

On April 10, 2019, Ms. Peterson had her first PT/OT appointment. Ex. 4 at 111. At this evaluation, she displayed poor sensory motor control and weakness in her left upper extremity, and interference with her activities of daily living ("ADLs"). *Id*. Later that month, she saw again Dr. Wagle for follow-up. Ex. 6 at 77–80. She had now finished her course of steroids and reported improved feeling in her leg and better strength in her left arm. *Id*. On exam, she did not have tremor or dysmetria, had a normal gait, and could walk unassisted. *Id*. Dr. Wagle diagnosed acute TM, and referred Petitioner to an MS specialist. *Id*. at 79, 80.

On May 10, 2019, Petitioner was evaluated by MS specialist Wayne Rubinstein, M.D. Ex. 6 at 81–82. Dr. Rubenstein diagnosed petitioner with non-longitudinally extensive and non-necrotizing upper cervical TM, temporally related to the March receipt of a flu vaccine. *Id*. at 84. Dr. Rubinstein also noted, that "[t]he interval [between vaccination and onset] is short, however, at 4–7 days.," and that her CSF findings were "not specific" either for MS or "postvaccinal TM." *Id*.

Approximately two months later (July 9, 2019), Petitioner went back to Dr. Wagle and reported ongoing left palm numbness (although she had felt baseline strength since engaging in PT). Ex. 6 at 85–86. By early October, she had been discharged from PT/OT. Ex. 4 at 17–19. And when she saw Dr. Wagle again that October, she reported only some residual left-hand tingling and numbness in her left two fingers. Ex. 6 at 90–92. Repeat MRI imaging performed in November 2019 revealed normal results for her brain, but "pathologic intramedullary T2 signal abnormality within the cervical spinal cord, extending from C2-C3" for her cervical spine. Ex. 5 at 95–98.

Symptoms Relapse in 2020

For nearly a year, Ms. Peterson's condition was largely quiescent. But in October 2020, a repeat brain MRI showed "few small foci of demyelination as described above likely reflecting multiple sclerosis." Ex. 23 at 58. Around the same time, neurologist Elena Grebenciucova, M.D., opined Petitioner likely had some kind of central nervous system demyelinating disease—possibly MS depending on comparison of earlier MRI imaging. Ex. 9 at 17–19.

On October 16, 2020, Petitioner saw neurologist Thomas Shoemaker, M.D., for an initial consultation regarding suspected relapsing-remitting MS. Ex. 26 at 20–29. Her history was noted, as well as the fact that the October 2020 MRIs showed that the lesion in her cervical cord was stable, but that there was a new small foci in the brain. *Id*. Dr. Shoemaker diagnosed Petitioner with MS, opining that "[w]hile it is possible that her partial myelitis may have been provoked by the influenza vaccination, given the time course as well as the CSF findings[,] *this is unlikely*." *Id*. at 28 (emphasis added).

Toward the end of October 2020, Petitioner had a telehealth visit with Dr. Grebenciucova to discuss medications. Ex. 9 at 12–14. A year later, repeat brain and cervical spine MRIs were performed, and the cervical MRI revealed "[s]table high T2 signal abnormality within the upper posterior spinal cord suggesting multiple sclerosis" (Ex. 23 at 30), while the brain MRI showed "[s]ubtle focus of focal high T2 signal adjacent to the left lateral ventricular body posterior aspect is smaller/less conspicuous than previously" but "[n]o new abnormalities." *Id*. at 32. And in early November 2021, Petitioner had a follow-up visit with Dr. Rubinstein. Ex. 25 at 65–67. Her exam was essentially normal, and he assessed her with MS, "4/2019 cervical myelitis, temporally post

vaccinial [sic], with subsequent evolution of cerebral MRI abnormalities." *Id*. at 69. No additional relevant records have been filed.

II. Expert Opinions

A. *Petitioner's Expert – Dr. Carlo Tornatore*

Dr. Tornatore is a neurologist, and he prepared two written reports for Petitioner. Report, dated Nov. 29, 2023, filed as Ex. 29 (ECF No. 27-1) ("First Tornatore Rep."); Report, dated May 31, 2024, filed as Ex. 54 (ECF No. 33-1) ("Second Tornatore Rep.").

Dr. Tornatore graduated from Cornell University with a Bachelor of Arts and Sciences in Neurobiology, and attended Georgetown University Medical Center, where he received a Master of Science in Physiology. Curriculum Vitae, filed as Ex. 30 (ECF No. 27-2) ("Tornatore CV") at 2. He subsequently graduated from medical school at Georgetown University School of Medicine, completing a residency in the Department of Neurology at Georgetown University Hospital. *Id*. Dr. Tornatore also completed a fellowship in Molecular Virology at the National Institute of Health in Bethesda, Maryland. *Id*. He has published multiple articles addressing demyelinating disorders and their pathology. *Id*. at 8–13. Currently, Dr. Tornatore serves as a Professor and Chairman of the Department of Neurology at Georgetown University Medical Center, Chairman and Neurologist-in-Chief of the Department of Neurology at Medstar Georgetown University Hospital in Washington, D.C., and Medstar Health's Regional Director Neurology. *Id.* at 3.

First Report

Dr. Tornatore's first report began with an overview of Petitioner's medical history. First Tornatore Rep. at 2–9. But he highlighted a few aspects of that history that he considered particularly significant to his opinion. He noted, for example, that Ms. Peterson had a prior history of an autoimmune condition, suggesting "a propensity for immune over-reactivity." *Id.* at 9. She had received the flu vaccine at issue on the same day she sought treatment for a possible wild viral infection, and Dr. Tornatore deemed this "unusual" and "not the standard of care for treatment of an acute influenza infection." *Id.* at 3. He otherwise noted that within a week of vaccination, Petitioner's differential diagnosis already included MS as a possibility, although TM was also proposed. *Id.* at 4. While by the summer of 2019 some treaters were opining that her presentation was not likely MS (and that it could be instead attributed to a post-vaccination reaction), as of the fall of 2020 her diagnosis was ultimately best characterized as MS, given subsequent imaging and corroborative CSF findings. *Id.* at 9.

Petitioner's treatment records clearly embrace MS as the ultimately proper diagnosis, and Dr. Tornatore agreed it was a proper diagnosis as well, based on the totality of the record (and in

particular the conclusions of her treaters). First Tornatore Rep. at 9. He even noted that Petitioner had tested positive for one known MS biomarker, oligoclonal bands, after CSF testing performed on April 5, 2019, and he deemed it likely they arose in the context of the initially-observed lesion, since they "are not found in asymptomatic individuals without MRI findings." *Id.* at 14.

Dr. Tornatore's report nevertheless characterized Petitioner's condition as vaccine-caused "TM/CIS," and he labored hard to isolate his causation analysis to that early aspect of Petitioner's medical history. First Tornatore Rep. at 9, 29. He defined TM to be a rare syndrome featuring spinal cord inflammation, usually occurring "across the width of the spinal cord," that clinically manifests with "varying degrees of weakness, sensory alterations and autonomic dysfunction." *Id.* at 10. TM also involves destruction of the nerve myelin sheath, resulting in nerve signal communication disruption. It is known to be caused by "post-infectious or post-vaccine autoimmune phenomenon," although Dr. Tornatore also allowed that TM is associated with MS itself. *Id.* at 11, 15; "Transverse Myelitis" – National Institute of Neurologic Disorders and Stroke (https://www.ninds.nih.gov/health-information/disorders/transverse-myelitis), filed as Ex. 31 (ECF No. 28-1) at 2 (including MS as a condition that "appear[s] to cause transverse myelitis"). He even stated that some authority now supported the view that this kind of condition might be more generally referred to as "myelitis," without regard to the positional location of the inflammation." K. Blackburn & B. Greenberg, *Revisiting Transverse Myelitis: Moving Toward a New Nomenclature,* 11 Frontiers. Neurol. 1 (2020), filed as Ex. 32 (ECF No. 28-2) at 2 (observing that not all myelopathies are attributable to inflammation, but can have other mechanisms or etiologies). TM has, he emphasized, been credibly associated with vaccinations, even if the link lacks firm epidemiologic proof. First Tornatore Rep. at 15.

Another diagnostic classification that Dr. Tornatore felt was applicable to Petitioner was "CIS," or "clinically isolated syndrome." First Tornatore Rep. at 11–12. He characterized CIS as featuring a single, monophasic occurrence of a CNS demyelinating event comparable to an MS flare, but occurring "in a patient not known to have [MS]." *Id.* at 12 (quoting A. Thompson et al., *Diagnosis of Multiple Sclerosis: 2017 Revisions of the McDonald Criteria,* 17 Lancet Neurol. 162-73 (2018), filed as Ex. 33 (ECF No. 28-3) ("Thompson") at 2 Panel 1). But "if a patient is subsequently diagnosed with [MS], the clinically isolated syndrome was the patient's first attack." Thompson at 2 Panel 1. Ms. Peterson, Dr. Tornatore noted, in fact could not yet have been diagnosed with MS (even if it could be suspected, as treaters seem to have surmised) because she could not demonstrate her satisfaction of the "dissemination in time or space" MS criterion—that she developed CNS lesions in different locations and at different times. First Tornatore Rep. at 12, 13. (As discussed in greater detail below, Dr. Tornatore's characterization of CIS as a component of an MS diagnosis (when the facts prove to support the latter) rather than as a free-standing separate occurrence, is an important concession in evaluating causation in this matter).

Ms. Peterson, Dr. Tornatore opined, demonstrated a single lesion on her cervical spine that was enhancing[3] when viewed by MRI in early April 2019, and which Dr. Tornatore deemed likely to have been no less than two to three weeks old (meaning it could have predated vaccination). First Tornatore Rep. at 13–14. However (and based on the contention that Petitioner's symptoms started on "5/8/2019"—something wholly unsupported by the medical record),[4] he deemed it reasonable to "surmise that the inflammatory process in the spinal cord started on or around 3/31/2019, which would correlate nicely with the 4/4/2019 MRI." *Id.* at 14. Of course—given Dr. Tornatore's assertion that the lesion could have been at least two weeks old, it would still predate vaccination (and hence could not be assumed to have caused its development).

From the foregoing, Dr. Tornatore attempted to construct a causation theory that would connect Petitioner's receipt of the flu vaccine to what transpired not long after. The immune system's built-in capacity to avoid autoimmune attack, he noted, could break down (and hence promote such cross-reactive damage). First Tornatore Rep. at 16. Vaccines provoke an immune response comparable to an infection. *Id.* Thus, vaccines (he reasoned transitively) are as capable of provoking autoimmune disease as wild infections, and he outlined any number of mechanisms by which this was possible. *Id.* at 16, (listing antigenic mimicry between foreign and self-antigens and "epitope spreading" (in which a response to foreign antigen causes nonspecific reactions by other immune cells), among other things), 18–22.

The wild flu virus could specifically, Dr. Tornatore contended, provoke just such a response, and he cited an item of literature in support. First Tornatore Rep. at 17–18; S. Markovic-Plese et al., *High Level of Cross-Reactivity in Influenza Virus Hemagluttinin-Specific CD4+ T-Cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis,* 169 J. Neuroimm. 31 (2005), filed as Ex. 43 (ECF No. 29-4) ("Markovic-Plese"). Thus, "T cells that recognized influenza antigens found in the influenza vaccine can likewise recognize CNS antigens," making a "downstream autoimmune response" possible. First Tornatore Rep. at 18.

Markovic-Plese, however, does not support causation as firmly as Dr. Tornatore suggests. In it, researchers derived a clone of a "T helper" cell (a kind of T cell that aids B cells in their production of antibodies in the adaptive immune response to foreign pathogens) from the serum of an existing MS patient who was then also experiencing an influenza A infection, using it *in vitro*

[3] *See Robinson v. Sec'y of Health & Hum. Servs.*, No. 14-952V, 2021 WL 2371721, at *16 (Fed. Cl. Spec. Mstr. Apr. 12, 2021) ("[b]efore a patient has an MRI with contrast, they are injected intravenously with a contrast dye called gadolinium. In a healthy person, or in an MS patient not having a flare, that dye should remain in the bloodstream. However, if an MS patient is symptomatic, the gadolinium will leak into the brain through an opening in the blood-brain barrier and will show as an enhancing image on MRI"). Thus, imaging that reveals an "enhancing" lesion evidences active disease process.

[4] It is possible that the reference to a May 2019 onset is a typographical error. Regardless, it lacks any record support. Why did Petitioner even seek medical intervention in April 2019, if not for treatment the symptoms later deemed to be an initial presentation of MS? Dr. Tornatore also does not later attempt to distinguish these initial symptoms from Petitioner's MS diagnosis.

to compare cross-reactive potential with different peptides relating to myelin basic protein—a "putative autoantigen in MS." Markovic-Plese at 32. And Markovic-Plese's authors did in fact identify mimicking peptide sequences that were also cross-reactive. *Id.* at 37. Of course, the authors aimed not to identify possible causes of MS, but instead to learn about ways to *improve* vaccine function in order to "provide a longer-lasting protection against viral infections and possibly decrease their effect on initiation of relapses in MS." *Id.* Moreover, the article says nothing about the capacity of a nonadjuvanted and inactivated flu vaccine to cause disease in this manner—and did not even test what the actual target antigen in MS would be.

Another item of literature referenced by Dr. Tornatore as evidence of a direct association between vaccination and MS is also far less supportive of his theory than he allows. *See* First Tornatore Rep. at 16–17; A. Langer-Gould et al., *Vaccines and the Risk of Multiple Sclerosis and Other Central Nervous System Demyelinating Diseases,* 71 JAMA Neurol. 12:1506-13 (Oct. 2014), filed as Ex. 42 (ECF No. 29-3) ("Langer-Gould"). Langer-Gould was an epidemiologic, case-controlled study of 780 vaccinated individuals diagnosed with MS or a different CNS-oriented demyelinating disease, comparing them to 3,885 control subjects, with a primary focus on the risk from the Hepatitis B or HPV vaccines (although its subjects had also received the flu vaccine). Langer-Gould at 1508, 1509. Dr. Tornatore emphasized Langer-Gould's finding that in fact there was a statistically-significant increased risk of CNS onset within 30 days of receipt of any vaccine for the studied group—consistent with the idea that in individuals with "subclinical autoimmunity," a vaccine might act as a "pro-inflammatory cofactor"—in much the same way an intercurrent infection might prompt an MS relapse/flare. First Tornatore Rep. at 16–17; Langer-Gould at 1512.

Langer-Gould, however, not only reached a different ultimate determination, but is not even all that supportive of the secondary conclusion that has in other cases[5] resulted in its embrace as supportive of MS causation. Its authors' primary conclusion was that there was "no long-term association of vaccines with an increased risk of MS"—contrary to Dr. Tornatore's theory writ large. Langer-Gould at 1512. At the same time (and more consistent with the causation theory embraced in this case), however, Langer-Gould did observe "a short-term increase in risk after vaccination of any type" in a younger population, seemingly supportive of what is alleged herein. *Id.*

But Langer-Gould's authors actually qualified significantly this aspect of their study's findings. They noted that because *overall* there was no greater exposure risk to MS from vaccination over time, "[t]his argues against causality because the risk in the vaccinated group

[5] *See, e.g.*, *Doles v. Sec'y of Health & Hum. Servs.*, 2025 WL 1177875, at *8 (Fed. Cir. Apr. 23, 2025). In *Doles,* the Federal Circuit found that Langer-Gould's determination of a heightened risk of MS symptoms in a specific sample of patients and a narrow timeframe could reasonably be given evidentiary weight by a special master in finding a vaccine had significantly aggravated a claimant's MS, even if the study did not reach statistically significant conclusions as to the association.

should remain elevated *regardless of whether the time window between exposure and clinical disease expression is defined as 15 days or 3 years*." Langer-Gould at 1512 (emphasis added). So what, then, did their findings about risk in a shorter timeframe post-vaccination actually suggest? Langer-Gould deemed those results to be consistent with the fact that proinflammatory immune stimulation was as much of a risk for *existing* MS patients (who often experience flares due to a variety of environmental factors) as those who may be experiencing "subclinical autoimmunity," causing a hastening of "symptom onset." *Id.* In other words, the findings showed that individuals who were *already experiencing MS* in some form might see their disease manifest sooner in the wake of vaccination, or experience a flare—but the vaccine had not *caused* the underlying subclinical condition (and more than the cause of a flare would provide an etiologic explanation for a person's MS in the first place). *Id.* ("vaccines (like infections) may accelerate the transition from subclinical to overt autoimmunity *in patients with existing disease*") (emphasis added)).[6]

The occurrence of just one vaccination event, moreover, could in turn stimulate a "progressive inflammatory disorder," in Dr. Tornatore's opinion. First Tornatore Rep. at 22–25. For support, Dr. Tornatore referenced an article involving "the marmoset model of MS." *Id.* at 22; B. t'Hart, *Experimental Autoimmune Encephalomyelitis in the Common Marmoset: A Translationally Relevant Model for the Cause and Course of Multiple Sclerosis,* 6 Primate Biol. 17 (2019), filed as Ex. 50 (ECF No. 30-2) (the "Marmoset Paper"). Dr. Tornatore maintained that this article highlighted (via animal model experiments involving "experimental autoimmune encephalomyelitis," or "EAE"—a known scientific animal model for MS) one particular experiment, in which animals immunized with mimics for a particular nerve myelin component (myelin oligodendrocyte glycoprotein, or "MOG") go on to develop MS-like CNS lesions, and in turn a progressive disease course comparable to human MS. First Tornatore Rep. at 22–25.

Dr. Tornatore found significant that this kind of experiment discussed in the Marmoset Paper revealed that "one need not have a humoral response against MOG to have an immune response (cellular in nature)." First Tornatore Rep. at 25.[7] And he noted that, in fact, molecular amino acid sequence homology can be shown between hemagglutinin in the flu vaccine and MOG peptides (based on Dr. Tornatore's own "BLAST" search).[8] Thus, it was scientifically conceivable

[6] Langer-Gould actually noted (in an earlier section of the article discussing existing literature on the topic of vaccine causation) that studies involving the HPV vaccine that seemed to support an association with CNS demyelination had observed in many of their studied subjects that they "had symptoms at the time of vaccination"—and thus the vaccines had only hastened "the transition from subclinical to clinical disease"—not caused it. Langer-Gould at 1507.

[7] Humoral means "pertaining to elements dissolved in the blood or body fluids, e.g., humoral immunity from antibodies in the blood as opposed to cellular immunity." *Humoral*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=23202&searchterm=humoral (last visited Oct. 30, 2025).

[8] Basic Local Alignment Search Tool ("BLAST") is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Oct. 30, 2025). A BLAST search involves review of an

not only that the flu vaccine could result in this specific kind of MS-associated damage, but also that it might do so independent of the creation of antibodies against this specific CNS-associated nerve tissue component.

A careful reading of the Marmoset Paper, however, reveals that it stands for far less (and even contradicts the concept of vaccine causation of MS). For starters, the main thrust of the article was its author's *defense* of this particular form of animal EAE study as scientifically worthwhile, despite objections to the ethics of such animal model research. Marmoset Paper at 19. With that goal, the article reviews numerous published marmoset-model studies involving MS, emphasizing the benefits of their findings in increasing medical science's comprehension of MS from many angles. The Marmoset Paper acknowledged, however, that questions have been raised about how relevant its findings actually are to human MS (although the paper's author is clearly in the camp of favoring animal model EAE experiments for the study of MS). *Id.* at 22–23.

Yet the Marmoset Paper also makes numerous statements regarding MS's likely pathogenesis that run counter to a causal foundation in Petitioner's case: that administration of a *single dose* of flu vaccine could begin a disease process culminating in MS. For example, it notes that some marmoset-specific experiments shed light on the possibility that "MS *might not be elicited by infection . . ., but by primary injury inside the central nervous system*." Marmoset Paper at 18 (emphasis added). It later notes that this possibility could be characterized as the "inside-out paradigm," in which lesions within the CNS drive disease processes due to a "physiological response to sustained excess antigen turnover in diseased tissues (the primary lesion)." *Id.* at 22; *see also id.* at 21 ("[t]he pathological hallmark of MS and the most likely cause of the accumulating neurologic deficits is the lesion").

The Marmoset Paper acknowledges uncertainty as to whether this paradigm has been proved over an "outside-in" scheme (where "MS is triggered by an external factor"—here, a vaccine), but its author clearly seems to *disfavor* the latter. Marmoset Paper at 31. In fact, its author writes that "data obtained in the marmoset EAE model are *strongly supportive* for the inside-out paradigm." *Id.* at 22 (emphasis added). Thus, although the Marmoset Paper *does* reference a number of MS-relevant studies, and some could be reasonably invoked to show how a foreign antigen like a vaccine component might have sufficient homologic similarity with nerve-related tissues for an autoimmune cross-reaction to occur, they are referenced to bulwark the model's value—*not* because the author believes this is how MS likely occurs. This calls into question the fundamental assumption of Dr. Tornatore's theory—that a single vaccination could cause a resulting pathogenic cascade that fundamentally occurs in the CNS. And the Marmoset Paper even expressly states that vaccination does not cause MS. *Id.* at 29 ("[t]he consideration that MS is

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online database to "compare[ ] nucleotide and protein sequences, to search for a homology between the … vaccine and [the body's myelin basic protein]." *Montgomery v. Sec'y of Health & Hum. Servs.*, No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019).

obviously not elicited by injection of an antigen/adjuvant formulation but develops spontaneously" points toward the need for research about how a CNS lesion might result in myelin damage that is reflected by clinical symptoms) (emphasis added).[9]

Dr. Tornatore further sought to establish how evidence of the existence of oligoclonal bands (a known biomarker for MS, and here seen in Petitioner's CSF testing in April 2019—at the time she was hospitalized) was supportive of a vaccine-encouraged disease process. First Tornatore Rep. at 14. He deemed it likely that their emergence coincided with the lesions then observed on MRI, but identified the bands as potential evidence for *peripherally-introduced* immune activity. C. Beseler et al., *The Complex Relationship Between Oligoclonal Bands, Lymphocytes in the Cerebrospinal Fluid, and Immunoglobulin G Antibodies in Multiple Sclerosis: Indication of Serum Contribution,* 12 PLOS ONE 10:1 (Oct. 2017) (https://doi.org/10.1371/journal.pone.0186842), filed as Ex. 36 (ECF No. 28-6) ("Beseler").

Beseler (drawing upon other studies supporting a "strong connection between IgG [antibodies] in the peripheral blood and that in the CNS of MS patients") sought to evaluate the serum and CSF findings in a sample of 115 subjects, 91 of which had been diagnosed with MS. Beseler at 2. The authors concluded, among other things, that there were nonlinear but "complex" associations between the number of oligoclonal bands and CSF IgG, on the one hand, and serum IgG. *Id.* at 4. But they also proposed that their data raised a question as to how "intrathecal IgG" (meaning in the CSF) could possibly be produced given the amounts they saw, and they tentatively proposed that this was due to blood serum-derived IgG. *Id.* at 9.

Beseler's authors only advanced this concept as a hypothesis, and they referenced a fairly-old article (published in 1971—but not apparently filed in this case) as "compelling" support for evidence of "antibody-mediated demyelination in MS." Beseler at 11. Beseler certainly does not stand for the conclusion that oligoclonal bands come from outside the CNS. But Dr. Tornatore deemed it further proof that "an inflammatory event in the periphery could trigger an immune response that ultimately will result in a CNS inflammatory event"—and here, that peripheral event could be vaccine-mediated. First Tornatore Rep. at 14. (This contention did not, however, explain (a) why it should be assumed one event—peripheral inflammation—would have temporal priority over a CNS event, as opposed to something beginning in the CNS that later shows up in the

[9] In addition, Dr. Tornatore's first report referenced unfiled experiments or studies discussed in the Marmoset Paper, as well as their data. *See, e.g.*, First Tornatore Rep. at 23 (reproducing a portion of "Figure 2" of Marmoset Paper (p. 19)). This Figure seems itself to have been derived from a different item of literature referenced in the Marmoset Paper but published in 1998. Marmoset Paper at 18 (referencing 't Hart et al., *Histopathological Characterization of Magnetic Resonance Imaging- Detectable Brain White Matter Lesions in a Primate Model of Multiple Sclerosis: A Correlative Study in the Experimental Autoimmune Encephalomyelitis Model in Common Marmosets (Callithrix jacchus)*, 153 Am. J. Pathol. 649–63 (1998) (*see* Marmoset Paper at 57 (bibliography)). But the underlying article *itself* does not appear to have been filed in this case.

periphery, or (b) how the oligoclonal bands—which are understood to be evidence of CNS harm, get into the CNS in the first place.[10]

The remainder of Dr. Tornatore's report involved his review of the other causation prongs. *See generally* First Tornatore Rep. at 25–29. With respect to the "did cause" prong, Dr. Tornatore maintained that the record established a "logical sequence of cause and effect" from vaccination to onset. *Id.* at 25. Thus, Petitioner received the vaccine while (likely) ill with a wild flu infection; those two factors worked synergistically together; this causes an autoimmune response; and the autoimmune reaction led to sensory symptoms several days later. *Id.* at 25–26. But other than the circumstances of Petitioner's vaccination and the post-vaccination onset of her first neurologic-like symptoms, Dr. Tornatore pointed to no other record proof supporting the conclusion that the flu vaccine she received was likely causal of her MS. And he also did not explain how he differentiated vaccine-caused TM from the circumstances in which initially-diagnosed TM actually reflected a first instance of MS (as is clearly so in this case).

Dr. Tornatore devoted a bit more effort to delineating the basis for his opinion that the timeframe from vaccination to onset was medically acceptable. First Tornatore Rep. at 26–29. He noted that it was difficult to identify "risk intervals," given the rarity of "vaccine-precipitated TM." *Id.* at 26. But (citing a publication by the Institutes of Medicine ("IOM")), he observed that the known timeframes for the adaptive immune response to a foreign antigenic stimulus provided a reasonable framework that could be relied upon in this context. *Id*; *Adverse Effects of Vaccines: Evidence and Causality*, Institute of Medicine (K. Stratton et al., eds. 2012), filed as Ex. 51 (ECF No. 30-3) ("2012 IOM Rep."), at 58. Here, an immune response could be evident within up to three days of vaccination—and an article specific to a different CNS-impacting demyelinating disease (acute disseminated encephalomyelitis, or "ADEM") established that a two-to-28-day timeframe was a likely risk interval post-vaccination. A. Rowhani-Rahbar et al., *Biologically Plausible and Evidence-Based Risk Intervals in Immunization Safety Research,* 31 Vaccine 271–77 (2012), filed as Ex. 52 (ECF No. 30-4) ("Rowhani-Rahbar"). And articles involving flu vaccine-associated peripheral neuropathies reached a comparable conclusion. L. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977,* 110 Am. J. Epid. 2:105-23 (1979), filed as Ex. 53 (ECF No. 30-5) ("Schonberger"). As a result, Petitioner's onset (which he proposed occurred four to nine days post-vaccination) fit well within these intervals. First Tornatore Rep. at 28.

[10] Beseler also acknowledged the significance of oligoclonal bands in MS—among other things, they may be explanatory of how a patient's MS progresses from an initial CIS. Beseler at 2 ("OCBs are associated with increased levels of disease activity and disability, a greater risk of second attack, the conversion from a clinically isolated syndrome (CIS) to early [relapsing-remitting MS], and greater brain atrophy").

Second Report

Dr. Tornatore's supplemental report grouped by topic his reactions to aspects of the report from Respondent's expert, Dr. Subramaniam Sriram. First, he questioned Dr. Sriram's opinion that Petitioner could not credibly have yielded CSF testing results suggestive of an immune reaction due to a peripherally-administered vaccine the week prior. Second Tornatore Rep. at 1–2. He reemphasized the fact that Ms. Peterson was vaccinated while likely ill with a respiratory infection (perhaps influenza), increasing the likelihood of an overactive immune reaction. *Id.* at 1. This was, he maintained, evidenced by both her slightly-elevated IgG and ANA levels, tested ten days post-vaccination, and was otherwise consistent with the timeframe for a vaccine-caused immune response. *Id.* at 1, 2. He also again argued (citing Beseler) that contrasting CSF evidence of normal-range IgG levels "strongly suggested that the [oligoclonal bands] seen in the CSF were *de novo*" (*Id.* at 2), and thus could have appeared *after* other instigating factors (here, vaccine-induced cross-reactive autoantibodies) responsible for the pathogenesis of MS. (This argument, it should be noted, is somewhat inconsistent with the idea that very-recent peripheral inflammation was seeping into the CSF (since it did not even show up in this testing), and does not otherwise negate the conclusion that tests demonstrating the presence of oligoclonal bands are evidence of a source *inside* the CNS as the reason for initial MS symptoms—a concept that articles like the Marmoset Paper readily concede have significant scientific support).

In challenging Dr. Sriram's contentions about the sequence of events leading to the appearance of the oligoclonal bands (a recognized biomarker for MS), Dr. Tornatore deemed Dr. Sriram "absolutely incorrect" in arguing that they can be present before radiologic evidence of CNS lesions is apparent. Second Tornatore Rep. at 2, 3. The article offered for this contention by Dr. Sriram revealed evidence of lesions on MRI in all four case subjects discussed, but without a history of neurologic symptoms. B. Hakiki et al, *"Subclinical MS": Follow-Up of Four Cases,* 15 Eur. J. Neurol. 858–61 (2008), filed as Ex. A Tab 4 (ECF No. 31-5) ("Hakiki"). In two of the cases, however, CSF testing *revealed* the presence of oligoclonal bands (and thus it cannot be said for those instances when the bands appeared in relationship to the radiologic evidence of lesions), while testing was not performed in a third—and hence only one case subject revealed no evidence of oligoclonal bands after imaging. Hakiki at 2–3. Hakiki's authors otherwise say nothing about when the bands likely appear in conjunction with lesion development.

Dr. Tornatore also endeavored to show, with ample citations to a number of scientific studies, that EAE model experiments had confirmed different aspects of his causation theory. *See generally* Second Tornatore Rep. at 7–10. Many of the studies he invoked were, however, more than 20 to 30 years old—raising the reasonable question why, if these articles so implicated a role for vaccination in sparking MS (or TM), there were not more recent studies confirming their purported findings. *See, e.g.*, D. Ziegler et al., *Experimental Allergic Neuritis-Like Disease in Rabbits After Injection with Influenza Vaccines Mixed with Gangliosides and Adjuvants,* 42 Infec.

& Immun. 2:824–30 (1983), filed as Ex. 63 (ECF No. 34-1) (research published 42 years ago); U. Jahnke, *Sequence Homology Between Certain Viral Proteins and Proteins Related to Encephalomyelitis and Neuritis,* 229 Science 282–84 (1985), filed as Ex. 64 (ECF No. 34-2) (research conducted more than 40 years ago).

One article that was more recently published, however, was offered by Dr. Tornatore to explain how the EAE model can be used "to induce oscillatory symptoms typical of the relapsing-remitting disease . . . , similar to that found in MS patients." C. Procaccini et al., *Animal Models of Multiple Sclerosis,* 759 Eur. J. Pharmac. 182–91 (2015), filed as Ex. 72 (ECF No. 33-9) ("Procaccini"). Like the Marmoset Paper, Procaccini is a review article mostly aiming at bulwarking the utility of different research models for the study of MS. Procaccini at 188. And it also notes the extent to which these models do not fully replicate MS—including the fact that one model discussed mimics MS through a "virus-induced pathology" (congruent with the contention here that vaccination could induce disease), even though in MS in humans, "persistent viral infection of the CNS has not been demonstrated." *Id.* at 187. But Procaccini does mention some animal models that have observed how MS-like disease could be induced by direct immunization with MBP-derived peptides or reactive T cell clones. *Id.* at 183–84. From this, Dr. Tornatore opined those studies about "longitudinally extensive TM" ("LETM")[11]—a form of TM he acknowledges was not descriptive of Petitioner's first MS incidence—provided evidence for how a flu vaccine might prompt disease. Second Tornatore Rep. at 8.

Research regarding TM, Dr. Tornatore maintained, provided helpful evidence even though MS is the injury in question. Again, referencing the Marmoset Paper, he opined that "a single antigen" that sparks one disease process (here, manifesting initially as what could be—but was not—a self-limiting case of TM) could, via *other* autoimmune mechanisms result in greater harm. Second Tornatore Rep. at 17. He specifically identified "epitope spreading"—a mechanism in which an initial immune response against a specific antigen expands over time to include responses against new epitopes on the same or entirely different ones, causing expansion of an autoimmune disease[12]—as a way that "multiple additional autoantigens can become targeted and persistent, leading to progressive disease." *Id.* But Dr. Tornatore offered no additional independent research or literature standing for the proposition that MS is thought to be propagated in this manner, and/or has the potential to become a secondary injury after the "first hit" of TM due to an initial cross-reaction followed by more widespread epitope spreading.

[11] Dr. Tornatore in fact included a chart setting forth the findings of several articles specifically looking at LETM. Second Tornatore Rep. at 4–5. I discuss some of these herein (although since MS is facially a distinguishable disease, I do not in detail review each article).

[12] *See Guzman v. Sec'y of Health & Hum. Servs.*, No. 15-736V, 2019 WL 2723392, at *26 (Fed. Cl. Spec. Mstr. May 14, 2019) (explaining that epitope spreading is "a process in which invading agents accelerate an ongoing autoimmune process by local activation of antigens presenting as a result of the existence of immune complexes.")

In so contending, Dr. Tornatore relied upon the notion that mimicry between flu vaccine antigens and peptide sequences in MBP was a likely mechanism for how injury occurred. Second Tornatore Rep. at 9–10. Indeed, he contended MBP was the target of attack in these cross-reactive instances, contrary to Dr. Sriram's arguments. *Id.* at 12–15. To support his argument, he cited yet another study more than 20 years old. B. Bielekova et al., *Encephalitogenic Potential of the Myelin Basic Protein Peptide (Amino Acids 83-99) in Multiple Sclerosis: Results of a Phase II Clinical Trial with an Altered Peptide Ligand,* 6 Nat. Med. 10: 1167–75 (Oct. 2000), filed as Ex. 67 (ECF No. 34-5) ("Bielekova"). Bielekova's authors sought to alter a specific peptide T-cell receptor in the MBP (for a group of eight patients *already* suffering from relapsing-remitting MS—and hence a distinguishable group from Petitioner), in order to suppress disease activity. Bielekova at 1167. They found instead, however, that three of the patients experienced exacerbations, and were able to identify two where the alterations were deemed to highlight the "encephalitogenic potential" of the peptide at issue. *Id.* at 1168, 1170, 1172

Dr. Tornatore opined that Bielekova's findings (notably, with respect to less than *half* of an eight-person sample) were highly significant, and he devoted two pages of his second report to reproducing charts, figures, and block quotes from the article. Second Tornatore Rep. at 13–16. He deemed it to constitute "strong evidence that immunization with peptides that bear resemblance to MBP can result in acute inflammatory events in the CNS." *Id.* at 16. But this contention greatly exaggerates Bielekova's findings. For one thing, Bielekova's authors were mainly focused on attempting to identify helpful immune therapies for treatment of MS, and hence did not aim to ascertain MS causes, making its overall focus distinguishable from what is at issue in this case. Bielekova at 1173. In addition, it involved a very small sample of patients who had MS—with no evidence provided by Dr. Tornatore of follow-up research confirming its findings, or more obviously extending them to other contexts. And Bielekova does not measure the impact of receipt of a *flu vaccine* on existing MS patients.

An even more compelling reason to give Bielekova less weight than Dr. Tornatore proposed lies in the fact that a core premise it embraces has not since been corroborated. Bielekova's authors (writing in 2000—thus 25 years ago), stated that the "current pathogenic concept of MS . . . assumes that autoreactive T cells are activated in susceptible individuals *most likely by cross-reactive foreign agents*." Bielekova at 1173.[13] This foundational premise is congruent with Dr. Tornatore's theory in this case, in which the "foreign agent" of vaccination is the instigating factor. And yet that premise has not been since confirmed by medical science—and indeed, articles like the Marmoset Paper *undermine* its reliability. Moreover, the experiment in

[13] Notably, Bielekova offers as a reference for this contention a foundational work (not filed in this case) about molecular mimicry and its capacity to cause production of cross-reacting antibodies capable of instigating an autoimmune disease. Bielekova at 1173 n.49 (referencing R. Fujinami & M. Oldstone, *Amino Acid Homology Between the Encephalitogenic Site of Myelin Basic Protein and Virus: Mechanism for Autoimmunity*, 230 Science 1043–45 (1985)). This evidence speaks more to the general reliability of molecular mimicry as a mechanistic concept than it supports the contention *in this case* that this is the relevant mechanism by which MS likely occurs.

Bielekova involved direct transmission of peptides, not induction of cross-reactive antibodies. It is thus a category error to treat as interchangeable "immunization" with direct injection of peptides for a specific purpose and receipt of inactivated flu antigens contained in a vaccine. At bottom, Bielekova may suggest that MBP is the situs for *further* nerve damage in existing MS patients, but it does not establish that damage *begins* here—let alone due to cross-reactivity from antibodies generated by a vaccine.

Dr. Tornatore also endeavored to supplement his arguments about a vaccine-induced cross-reactive process in other ways. For example, he contended that a number of articles establish that flu vaccine antigens have been associated with antibodies discovered in the serum of patients with LETM. Second Tornatore Rep. at 6; N. Nakamura et al., *Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases,* 42 Intern. Med. 2:191–94 (Feb. 2003), filed as Ex. 59 (ECF No. 33-6) ("Nakamura"). Nakamura, however, is only a case study report, and identified two instances in which *distinguishable* CNS-impacting neurologic conditions (TM and acute disseminated encephalomyelitis) were observed after receipt of a flu vaccine—and to the extent the article discusses MS, it notes that the greater risk of the impact of a flu *infection* warrants vaccination. *Id.* at 194. Another article (specifically considering the distinguishable peripheral neuropathy of Guillain-Barré syndrome) also showed how flu vaccine-derived antibodies likely relevant to the disease process in question could arise. I. Nachamkin et al., *Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome,* 198 J. Infec. Dis. 15:226–33 (July 2008), filed as Ex. 61 (ECF No. 33-8). Of course, proof that the flu vaccine can lead to a different disease is not particularly robust evidence that it will do so for other, different diseases—and this is true even if the diseases both involve nerve demyelination.

The post-vaccination timeframe in which Petitioner's neurologic symptoms manifested was, Dr. Tornatore reiterated, consistent with reliable science on the subject, despite Dr. Sriram's arguments to the contrary. In so arguing, Dr. Tornatore contended that Dr. Sriram had misinterpreted a chart from Rowhani-Rahbar. Second Tornatore Rep. at 16–17; Rowhani-Rahbar at 273 Fig. 2. Dr. Sriram had contended that the chart demonstrated that an autoimmune process driven by vaccination was most likely to occur no sooner than one week post-vaccination (and hence faster than what Petitioner experienced). But Dr. Tornatore noted that the dates where mean values, allowing for the possibility of a shorter onset. Second Tornatore Rep. at 17. This argument may be correct, but it does not rebut the likelihood that "more often than not" onset would occur in a longer timeframe than the one at issue in this case.

Dr. Tornatore otherwise repeated arguments comparable to those he advances in other cases where he serves as an expert (arguments that I have repeatedly deemed wanting in persuasiveness and reliability in many prior decisions). He contended that the rarity of vaccine injuries rendered epidemiologic evidence a probative nullity in Program cases (Second Tornatore

Rep. at 3)—an argument that essentially advances a *legal opinion* about what evidence bears on a vaccine injury claim that not only exceeds his competence as a medical expert, but is wrong (from the standpoint of the Program's consistent treatment of such evidence as relevant).[14] He then went on to argue that due to injury rarity, case reports of TM post-vaccination warrant special emphasis, citing a number as highly relevant and persuasive. *Id.* at 4–6.

Dr. Tornatore also performed his own "in silica" peptide homology sequence review, comparing an amino acid peptide string in MBP with influenza A, and observing a homologic sequence that he noted "has been found to be encephalitogenic" in an animal study performed nearly 40 years ago. Second Tornatore Rep. at 11, 12; N. Potter et al., *Immunochemical Specificity of Antisera Raised Against the Encephalitogenic Peptide SH624, Residues 59-74 of the Myelin Basic Protein,* 12 Neurochem. Rs. 1:9–14 (1987), filed as Ex 66 (ECF No. 34-4) ("Potter"). I have, however, noted repeatedly in prior decision that showings of "naked" homology do not appreciably advance the contention that mimicry between vaccine antigenic components and a self-tissue establishes a likely mechanism for an autoimmune disease.[15] And Potter (like other experiments discussed in other articles filed in this case) involves direct "immunization" with MBP peptides, distinguishable from the effect of a vaccine (where the possibility of a comparable cross-reaction is lower—and impact more likely muted).

B. *Respondent's Expert – Dr. Subramaniam Sriram*

Dr. Sriram is also a neurologist, and he prepared a single written report. Report, dated Mar. 15, 2024, filed as Ex. A (ECF No. 31-1) ("Sriram Rep.").

Dr. Sriram received a Bachelor of Medicine and a Bachelor of Surgery from the University of Madras in Madras, India. Curriculum Vitae, filed as Ex. B (ECF No. 32-1) ("Sriram CV") at 1. He then served as an intern and resident at Wayne State University and completed a residency in neurology at Stanford University, where he also served as chief resident and eventually completed a post-doctoral fellowship in neuroimmunology. *Id*. He is board-certified in both neurology and internal medicine. *Id*. He also holds academic positions as a professor of experimental neurology and therapeutics as well as an associate professor in molecular biology and immunology. *Id.* 2. Dr. Sriram is heavily involved in clinical work as he directs the Multiple Sclerosis Clinic at Vanderbilt University Medical Center where he sees roughly 1450 patients a year. Sriram Rep at 1. In addition, he runs a basic science laboratory looking at pathways that promote neurologic repair.

[14] *See Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009) ("[a]lthough . . . a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the special master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury").

[15] *See Schultz v. Sec'y of Health & Hum. Servs.*, No. 16-539V, 2020 WL 1039161 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) ("[m]ere demonstration of theoretical homology alone, based on computer-driven searches involving databases of amino acid sequences, does not carry the day.").

https://www.vumc.org/neurology/person/subramaniam-sriram-mbbs (last visited Oct. 30, 2025). In addition, Dr. Sriram has published numerous articles on various aspects of clinical and immune mediated diseases of the nervous system. Sriram CV at 9–21; Sriram Rep. at 1.

Like Dr. Tornatore, Dr. Sriram's expert report began with a review of Petitioner's relevant medical history. Sriram Rep. at 2–4. He also commented on MS, defining it as a chronic CNS inflammatory disease involving demyelination. *Id.* at 4. MS usually presents clinically with symptoms of "dysfunction of either optic nerves, brainstem, or spinal cord," followed by relapse symptoms which can vary in their severity. *Id.* Its diagnosis requires evidence of lesions in the brain or spinal cord "disseminated in space and time" (meaning appearing in different places and at different times). *Id.* at 4, 5. Although MS is understood to likely be autoimmune in its mechanism, no specific antigenic autoantibody responsible for it has been identified, and it has no known cause. *Id.* at 5.

Acute TM, by contrast, is restricted to the spinal cord, and involves evidence of different kinds and levels of neurologic dysfunction, typically below the level of the lesion. Sriram Rep. at 6. "Incomplete" forms of TM usually involve asymmetrical deficits "with variable degree of paralysis of arms and legs" plus autonomic dysfunction. *Id.* at 7. And this form of TM is very often seen as an MS relapse. *Id.*

Dr. Sriram deemed Petitioner's MS diagnosis (which he clarified to properly be relapsing-remitting MS)[16] to have full record support. Sriram Rep. at 5. He agreed that as of Petitioner's initial early-April presentation, her exam work-up was enough to establish CIS, and there was proof of *one* lesion. *Id.* But as her symptoms evolved, "it is clear that her initial presentation of myelitis was in fact the first episode of MS." *Id.* He felt that her initial symptoms (predominantly left-sided tingling and numbness that later spread on that side, coupled with a cervical (meaning close to the neck) lesion) reflected the kind of incomplete myelitis that would characterize an MS initial flare. *Id.* at 2–3, 7.

Unlike Dr. Tornatore, however, Dr. Sriram did not consider Petitioner's initial CIS presentation (which hinted at MS, even if that diagnosis could not then have been formally applied) to have any analytic importance with respect to the causation issues raised by this case. As he observed, "TM is the most common initial presentation of MS," but "it does not follow that since Ms. Peterson did not meet the criteria of MS at onset of her symptoms," the initial TM-like symptoms could be evaluated in isolation as a separate injury (even if in theory it prompted what followed). *Id.* at 9. Rather, "she had MS from the start"—not TM that "then developed into MS."

[16] Relapsing-remitting Multiple Sclerosis ("RRMS") is the most common course of MS. It "can be characterized as either active (with relapses and/or evidence of new MRI activity over a specified period of time) or not active, as well as worsening (a confirmed increase in disability following a relapse) or not worsening." *Relapsing-Remitting Multiple Sclerosis (RRMS)*, National Multiple Sclerosis Society, https://www.nationalmssociety.org/understanding-ms/what-is-ms/types-of-ms/relapse-remitting-ms (last visited Oct. 30, 2025).

Id. Accordingly, "[t]he question here is not the relationship between Flu vaccine and TM/CIS but its relationship to MS," and literature specific to TM was irrelevant for that purpose. *Id.* at 10.

The flu vaccine was not likely capable of causing MS, Dr. Sriram contended. Sriram Rep. at 8–9. He noted that MS patients are "highly studied," and the causes for flares/exacerbations have been looked at extensively—yet vaccines have not been deemed associated. *Id.* at 8; F. DeStefano et al., *Vaccinations and Risk of Central Nervous System Demyelinating Disease in Adults,* 60 Arch. Neurol. 504–09 (Apr. 2003), filed as Ex. A-2 (ECF No. 31-3) ("DeStefano") (case-control study comparing instances of MS or optic neuritis in 450 subjects (compared to 950 healthy controls) found no increased risk of onset or recurrence/flares after receipt of a number of vaccines, including flu vaccine).

More recent studies arrived at similar conclusions. A. Hapfelmeier et al., *A Large Case-Control Study on Vaccination as Risk Factor for Multiple Sclerosis,* 93 Am. Ac. Neurol. 9:e908–16 (2019), filed as Ex. A-5 (ECF No. 31-6) ("Hapfelmeier"), at e909, 914–15 (case control study of German population of more than 12,000 MS patients compared to over 200,000 controls; over a five-year period, incidence of new-onset MS not greater for recipients of large number of vaccines, including flu, than control group). Indeed, Hapfelmeier tentatively proposed, given its findings, that vaccination might be protective against MS. Hapfelmeier at e915–16. And Dr. Sriram deemed these studies to confirm what neurology specialists already understood to be the case. If, he reasoned, medical science even suspected that an "aberrant immune response" due to vaccination could spark MS, it would be testing blood serum as well as CSF for proof when MS was suspected, but it does not. Sriram Rep. at 11–12 ("[w]e don't test patients for an immune response to MBP in either their blood or CSF").

Other aspects of Dr. Tornatore's causation reasoning were called into question by Dr. Sriram. He noted that proposing a mimicry-driven autoantibody cross-reaction as the mechanistic driver of MS relied on the idea that "the autoantigen driving MS is a myelin antigen and very likely Myelin basic protein and the [flu vaccine] hemagglutinin is the antigenic culprit." Sriram Rep. at 11. But "there is very little evidence that MBP is the antigen in MS," adding that his own research into the topic had not identified an autoantibody to MBP as causal—let alone one produced in response to vaccination. *Id.* And he disputed that evidence from the Marmoset Paper about mimicry or cross-reactivity involving MOG proteins was relevant, noting that "MOG associated disease (MOGAD)[17] is not MS," and is instead treated as a distinguishable neurologic condition. *Id.*

[17] "Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD)" is defined as "an autoimmune condition where your immune system (antibodies) mistakenly attacks parts of you center nervous system. MOG is part of the protective cover that surrounds nerves (myelin) in your brain, spinal cord and eyes (optic nerves)." *Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD)*, Cleveland Clinic, https://my.clevelandclinic.org/health/diseases/myelin-oligodendrocyte-glycoprotein-antibody-disease-mogad (last visited Oct. 30, 2025).

Dr. Sriram also discussed what was the likely onset of Ms. Peterson's MS. Petitioner had tested positive for oligoclonal bands at her early April 2019 hospitalization. Sriram Rep. at 3, 5–6; Ex. 2 at 53. These bands reflect the existence of "antibodies present in the CSF but not in [blood] serum, indicating that they are made within the CFS." Sriram Rep. at 6. The fact that these bands were observed when MRI findings established the existence of a lesion, Dr. Sriram reasoned, was proof of "an ongoing immune process which predates receipt of the vaccine," since they were observed within approximately one week of vaccination—but would have likely taken a longer period of time to be produced. *Id.* at 7, 8. He deemed it "highly unlikely that the antibodies present in the CNS . . . developed in the periphery and moved to the CNS within seven days" of vaccination—and hence the flu vaccine could not have caused them to appear. *Id.* at 8.

In so arguing, Dr. Sriram disputed Dr. Tornatore's contention that because oligoclonal bands would generally not be found in asymptomatic individuals, "the MS had to begin at the onset of the initial symptoms." Sriram Rep. at 9. Treaters would not think to perform CSF testing *unless* a person's clinical presentation suggested the presence of a neurologic condition that could reflect MS. *Id.* Thus, the oligoclonal bands could certainly exist before onset of clinical symptoms. Dr. Sriram referenced Hakiki to show case report instances of what could be called "subclinical" MS, where (he contended) it was shown that "patients who had normal MRIs of their brains but spinal fluid studies showing positive oligoclonal bands and who on follow up went on to develop MS." *Id.*

In fact, the Hakiki case reports involved individuals whose imaging revealed unanticipated CNS lesions consistent with MS, but before clinical manifestations had occurred (although the imaging was performed because of nonspecific neurologic concerns (cervical trauma, headaches, tiredness, etc.). Hakiki at 859–60. And not all the case report subjects revealed the presence of oligoclonal bands. Thus, Dr. Sriram has misstated Hakiki's findings, as Dr. Tornatore alleges—although the article otherwise does not shed light on the question of what evidence of oligoclonal bands says about when a person's MS likely began. It also underscores the general notion that MS onset certainly *can* predate clinical symptoms—consistent with Dr. Sriram's more general argument.

Dr. Sriram also distinguished evidence, like Beseler, that Dr. Tornatore had relied upon to contend that different categories of immunoglobulins found in the CSF likely came from the serum—noting that in fact the issue really was where the *oligoclonal bands* came from. Sriram Rep. at 10. They were "present only in the CSF and not in serum," and hence they could reflect only "intrathecal synthesis" of immune cells reflecting inflammation in the CNS. Thus, their existence could not be assumed to be due to peripheral immune activity that had migrated to the CNS and instigated the disease process, as would be required under Dr. Tornatore's theory.

Dr. Sriram did not accept Dr. Tornatore's more general contentions of what a reasonable post-vaccination timeframe would be for vaccine-induced MS. Sriram Rep. at 12–14. To substantiate this argument, Dr. Tornatore had referenced Rowhani-Rahbar, which involved a risk interval for ADEM after infection, and hence was not precisely relevant to the context of purported MS due to vaccination. Rowhani-Rahbar at 273; Sriram Rep. at 12 ("we are not dealing with an infection but rather a vaccine, which unlike an active infection is not [a] proliferating and dividing organism"), 14. In addition, a closer reading of Rowhani-Rahbar suggested the true, greatest risk interval was no less than approximately six days—longer than Petitioner's post-vaccination onset. *Id.* at 13; Rowhani-Rahbar at Fig 2 at 273. And even the studies discussed in Rowhani-Rahbar involved longer risk intervals than the three to four days at issue in this matter. Sriram Rep. at 13–14; Rowhani-Rahbar at 275.

Thus, the kind of immune response that would need to occur before manifestation of clinical symptoms suggestive of a neurologic issue (consistent with Petitioner's presentation) would not occur in the short, post-vaccination timeframe at issue. Sriram Rep. at 14. Even the Marmoset Paper studies, which discussed various EAE studies, did not observe disease development (and only after immunizing the animal subjects with both a highly-powered adjuvant[18] designed to elicit observable changes, plus direct myelin antigens–not just vaccines that might indirectly elicit antibodies to those antigens) sooner than seven days later. Marmoset Paper at 23. And this was consistent with studies Dr. Sriram had conducted and published upon. C. Du et al., *Administration of Dehydroepiandrosterone Suppresses Experimental Allergic Encephalomyelitis in SJL/J Mice,* 167 J. Immunol. 12:7094–101 (2001), filed as Ex. A-3 (ECF No. 31-4), at 7098 Fig. 6 (animal study involving MS model; five days elapsed before subjects receiving MBP-primed immune cells manifested clinical symptoms). And none of this explained how oligoclonal bands would appear in CSF testing as early as they did in this case. Sriram Rep. at 14.

III. Procedural History

The Petition was filed in March 2022, and assigned to another special master in August of that same year, before being transferred to me in February 2023. After Respondent's Rule 4(c) Report contesting entitlement was filed in June 2023 (ECF No. 24), Petitioner filed Dr. Tornatore's first expert report that fall. The process of filing expert reports and other supportive items of medical literature was completed in July 2024, and I thereafter set a schedule for resolving the case

[18] "Freund Adjuvant" is defined as "a water-in-oil emulsion incorporating antigen, in the aqueous phase, into lightweight paraffin oil with the aid of an emulsifying agent. On injection, this mixture [] induces strong persistent antibody formation. The addition of killed, dried mycobacteria, e.g., Mycobacterium *butryicum*, to the oil phase (*Freund complete a.*) elicits cell-mediated immunity (delayed hypersensitivity), as well as humoral antibody formation." *Freund Adjuvant*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=55029 (last visited Oct. 30, 2025).

via ruling on the record. The parties have since filed briefs in support of their respective positions, and the matter is fully ripe for resolution.

IV. Parties' Arguments

Petitioner

Petitioner maintains all three prongs of the test for causation established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005). Br. at 9. But she frames her overall argument with a specific understanding of her diagnosis and medical history. *Id.* at 9–11. She emphasizes the fact that as of April 2019, she had been "diagnosed by two different neurologists" with TM or CIS, and that this was a proper understanding of the medical facts at that initial period of her treatment. *Id.* at 9, 10. TM, she emphasizes, is "understood" to be both a post-infectious or post-vaccine phenomenon, and that it is also on a "continuum" of demyelinating conditions. *Id.* at 10. Later on, and as her condition progressed, she was properly diagnosed with MS—but "her flu vaccination was not just the trigger of an acute demyelinating event, but also responsible for initiating what was subsequently understood/identified as a chronic demyelinating condition." *Id.* at 11.

Regarding the first, "can cause" *Althen* prong, Petitioner deems Dr. Tornatore's opinion to be sufficiently reputable to meet the preponderant evidentiary test. Br. at 11–12. He offered a mechanism of molecular mimicry between the flu vaccine antigens and nerve myelin, and even showed that the flu vaccine did possess sufficient amino acid sequential homology to MOG peptides for a cross reaction to be possible. *Id.* at 21. She also noted that many prior decisions in the Program have found "that it is medically plausible for MS to be caused by vaccinations in certain circumstances" (*Id.* at 24) and cited a number of such decisions. *Id.* at 22–23. (As noted below, however, it is legally erroneous to characterize a claimant's *Althen* prong one burden as requiring only a showing of plausibility).

Petitioner emphasized the recognized scientific validity of that theory—both generally and specifically as an explanation for some comparable demyelinating diseases. Br. at 12–15. Dr. Tornatore further provided specific evidence that this mechanism could explain how the flu vaccine can cause CNS demyelination akin to what is experienced in MS. *Id.* at 17–18 (referencing Markovic-Plese and Langer-Gould). An initial cross-reactive autoantibody attack triggered by vaccination could lead to a "progressive inflammatory disorder such as MS," as the different studies referenced in the Marmoset Paper (in particular involving MOG peptides) revealed. *Id.* at 19-20). And Bielekova revealed MS exacerbation could occur in patients exposed to an MBP peptide—suggesting that a chronic response was possible if antigens with homology to myelin, of the sort contained in the flu vaccine, were introduced to the immune system. *Id.* at 20–21.

With respect to the second, "did cause" prong, Petitioner emphasized that she both had a demonstrated autoimmune propensity pre-vaccination, and was also likely experiencing an infectious process at the time of vaccination, making it likely her immune system was "activated in a manner that would likely cause unbalanced synergistic local inflammation." Br. at 26. Her subsequent development of TM close-in-time to vaccination was evidence that this had occurred. *Id.* And her initial treaters in April 2019 allowed for the possibility that the vaccine explained her injury (although Petitioner cannot point to doctors who later made the MS diagnosis also expressing that tentative view). *Id.* at 27–28.

Finally, Petitioner contends that she can meet the third *Althen* prong governing timing. Her TM manifested within four days of vaccination, and eight days after vaccination enhancing lesions were revealed on MRI. In Dr. Tornatore's view, such a lesion would be *no more* than two to three weeks old—and therefore could have been generated at the time of vaccination or not long after. Br. at 29. Overall, a timeframe of CNS demyelination occurring within four to nine days post-vaccination was consistent with Dr. Tornatore's theory, and supported by independent items of literature like Rowhani-Rahbar. *Id.* at 29–30.

In Petitioner's Reply, she maintains that Respondent mischaracterizes her legal burden under *Althen*—arguing that Prong One only requires her to "provide a sound and reliable, plausible medical theory showing how the vaccine *can* cause the injury in question," not that she must "provide a theory that establishes, by preponderance, a true or correct explanation of how the vaccine *does* cause the injury." Reply at 2 (emphasis in original). Such a mischaracterization, explains Petition, is "clearly an elevation of [her] burden," and that where "some medically plausible theories may fall short in terms of how sound and reliable the Special Master deems them to be does not mean a petitioner must show by preponderant evidence that a theory is "true" or "correct" to prevail in an off-table claim. *Id.* at 4. Petitioner, however, emphasizes her understanding that her burden of proof is clearly one of preponderance of the evidence, and that she is in no way suggesting it is less than that. *Id.* at 6.

She also argues that she has provided more than a "bare assertion of molecular mimicry," and that proving that specific epitopes in the vaccine and the nervous system cross-react with one another" similarly elevates her burden of proof. *Id.* Instead, Petitioner contends that special masters are to consider the totality of the evidence when assessing a petitioner's theory—not whether a specific mechanism, including molecular mimicry, has been proven. *Id.* at 7. Despite Respondent's contention that the pathogenesis of MS is not clear or fully known, Petitioner nevertheless, maintains that the uncertainty regarding the pathogenesis of MS does not defeat her claim. *Id.* at 13. Rather, what is already known about the nature and pathogenesis of CNS demyelinating conditions and the immunological impact of the flu vaccine, is sufficient, according to Petitioner, to conclude that she has met her preponderant burden. Reply at 13.

Finally, Petitioner contends that her concurrent influenza infection at the time of her vaccination only strengthens her support needed to prove a logical sequence of cause and effect. *Id.* at 17. Specifically, Petitioner argues that her concurrent influenza infection "significantly augment[ed] the immune response to not only influenza antigens but also resulting in aberrant autoimmune response against CNS antigens." *Id.* Moreover, Respondent has not provided any evidence to the contrary, according to Petitioner. *Id.* She concludes her reply disagreeing with Respondent regarding a medically acceptable timeframe—noting that her initial TM/CIS symptoms and subsequent development of MS is consistent with her overall medical theory. *Id.* at 18.

Respondent

Respondent denies that Petitioner has preponderantly established any of the *Althen* causation test prongs. With respect to the first prong, Petitioner relies on a lesser "biologically plausible" standard of proof, when in fact this prong is subject to the same preponderance requirement that applies to the other two. Opp. at 10–11. And Petitioner did not otherwise meet the "can cause" prong with sufficient reliable evidence. As Dr. Sriram established, many large-scale epidemiologic studies have found no association between the flu vaccine and MS. *Id.* at 11 (citing Hapfelmeier, DeStefano). Articles like Langer-Gould were actually consistent in their conclusions. *Id.* at 11–12. And existing medical guidelines cited by Dr. Sriram supported the conclusion that the scientific community did not deem the flu vaccine to pose a risk to MS patients. *Id.* at 12.

Respondent deemed the components of Dr. Tornatore's opinion to be inadequately supported. His reference to molecular mimicry, for example, was too general (a criticism often directed against its invocation in other cases). Opp. at 12–13 (citations omitted). And where Dr. Tornatore tried to be more specific to the issues at hand when asserting molecular mimicry, he faltered. For example, much of his theory relied on attempting to show mimics between flu vaccine antigenic components and MBP—even though "there is very little evidence that MS is mediated by MBP." *Id.* at 13. Indeed, MS's pathogenesis remains "unclear," and therefore it could not be assumed to be driven by a vaccine as an environmental trigger and in the manner proposed by Dr. Tornatore. *Id.* at 13, 14. And the various items of literature offered to bulwark Dr. Tornatore's theory either over-relied on MBP associations (Markovic-Plese, Bielekova), did not actually show a pathogenic response to demonstrated mimicry (Markovic-Plese), or involved individuals who already had MS (Bielekova). *Id.* at 14–15. Thus, the showing of homology between putative flu vaccine antigenic components and MOG was not enough of a basis for the conclusion that a cross-reactive process driven by autoantibodies produced in response to vaccination could cause MS. *Id.* at 16.

The Marmoset Paper stood as no better evidence of a vaccine-MS association, Respondent contended. Opp. at 15–16. Dr. Tornatore identified within this paper the discussion of a different study, in which it was demonstrated that direct immunization of animal subjects with MOG-homologous peptides could result in EAE characterized by chronic demyelination. But not only was EAE not a particularly good match for MS (as Dr. Sriram established), but its focus on MOG made it even less applicable in this context, since autoimmune demyelinating diseases associated with MOG are distinguishable. *Id.* at 15–16.

The remaining two *Althen* prongs, Respondent argued, were also unmet. Although treaters had observed a temporal association in April 2019 between receipt of the flu vaccine and Petitioner's onset, the treater who later diagnosed her with MS in the fall of 2020 (Dr. Shoemaker) deemed her initial myelitis not to have been vaccine-associated, given the overall medical record course and evidence. Opp. at 16–17. And her onset was actually no more than three days post-vaccination—far too fast for an antibody-driven process due to vaccination to have occurred. *Id.* at 18. Evidence relied upon for the timeframe Petitioner favored, like Rowhani-Rahbar, involved different vaccines or CNS conditions. *Id.* at 18–19.

V. Applicable Law

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that her illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly*, 592 F.3d at 1321; *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[19] There is no Table claim for MS as an injury after the receipt of *any* covered vaccine—so such a claim can only sound in causation-in-fact.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. V. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard).

[19] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 418 F.3d at 1278: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs.*, 121 Fed. Cl. 230, 245 (2015), *vacated*, 844 F.3d 1363 (Fed. Cir. 2017).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Cerrone v. Sec'y of Health & Hum. Servs.,* 146 F.4th 1113, 1122 (Fed. Cir. 2025)*; Kalajdzic v. Sec'y of Health & Hum. Servs*., No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592

F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C.*, 704 F.3d at 1356 (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant

proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

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

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd sub nom. Rickett v. Sec'y of Health & Hum. Servs*., 468 F. Appx. 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993

F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.,* No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.,* 23 Cl. Ct. 726, 733 (1991), *aff'd,* 968 F.2d 1226 (Fed. Cir.), *cert. denied,* 506 U.S. 974, 113 S. Ct. 463, 121 L.Ed.2d 371 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

There are, however, situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("'[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent'") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *Lalonde v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Determining Matter on Record Rather Than at Hearing*

I have determined to resolve this case based on written submissions and evidentiary filings. My determination is consistent with the Vaccine Act and Rules, which not only contemplate but *encourage* special masters to decide petitions (or components of a claim) on the papers where (in the exercise of their discretion) they conclude that such a means of adjudication will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The Federal Circuit has affirmed this practice. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1365–66 (Fed. Cir. 2020). It simply is not the case that every Vaccine Act claim need be resolved by hearing—even where the petitioner explicitly so requests.

D. *Review of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. Appx. 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. MS and its Treatment in the Vaccine Program as a Putative Vaccine Injury

A. *Medical Characteristics of MS*

The parties agree that Petitioner was accurately diagnosed with relapsing-remitting MS, despite how treaters characterized her initial presentation. As noted in *P.M. v. Sec'y of Health & Hum. Servs.*, No. 16-949V, 2019 WL 5608859, at *21 (Fed. Cl. Spec. Mstr. Oct. 31, 2019):

> MS is a demyelinating CNS disease. *See Taylor v. Sec'y of Health & Human Servs.*, No. 13-700V, 2018 WL 2050857, at *21 (Fed. Cl. Spec. Mstr. Mar. 9, 2018). It likely has an autoimmune pathogenesis. *W.C. v. Sec'y of Health & Human Servs.,* No. 07-456V, 2011 WL 4537877, at *3 (Fed. Cl. Spec. Mstr. Feb. 22, 2011), *mot. for review den'd*, 100 Fed. Cl. 440 (2011), *aff'd,* 704 F.3d 1352 (Fed. Cir. 2013). Patients diagnosed with MS typically experience multiple episodes of CNS demyelination separated in time and

> space (meaning throughout the CNS), concurrent with a progressive, if remitting and relapsing, decline in their overall health course. *Taylor*, 2018 WL 2050857, at *21. An MRI can be used to corroborate the dissemination in space and time requirement, and often reveals old lesions as well as enhancing/new lesions. *Id.* Symptoms can include numbness or weakness in the body, loss of vision, tremors, unsteady gait, slurred speech, and dizziness. *Id.*

Significantly, medical science still does not understand *why* MS begins, or why its symptoms later recur—and no study has been generated supporting the proposition that a one-time neurologic "hit" can thereafter initiate what becomes a chronic injury (although the research into the MS-Epstein Barr virus ("EBV") connection is promising).[20] There are thus no widely-accepted known specific causes of MS—and no particular antibodies associated with it either. Yet many other demyelinating diseases—MOGAD, for example—*do* have associated antibodies, which at a minimum constitute biomarkers of the relevant disease even if medical science is not precisely confident that the antibodies in question drive it.

B. *Program Decisions Involving MS as Vaccine Injury*

Special masters have sometimes been persuaded by causation theories that certain vaccines could cause, or worsen, MS. *See, e.g.*, *Jane Doe v. Sec'y of Health & Hum. Servs*., No. 13-471V, 2023 WL 4741993, at *29 (Fed. Cl. Spec. Mstr. July 25, 2023) (hepatitis B vaccine significantly aggravated subclinical MS), *mot. for review den'd on other grounds*, 2023 WL 6474093 (Fed. Cl. Oct. 5, 2023); *Robinson v. Sec'y of Health & Hum. Servs*., No. 14-952V, 2021 WL 2371721, at *25 (Fed. Cl. Spec. Mstr. Apr. 12, 2021) (flu vaccine caused MS); *Hitt v. Sec'y of Health & Human Servs*., No. 15-1283V, 2020 WL 831822, at *9–10 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) (entitlement for petitioner based in part on finding that Respondent's expert had conceded the flu vaccine can cause MS); Br. at 22–23 (citations omitted I have not joined in this view, however—and the case *against* such a theory is far more scientifically reliable and persuasive (as discussed below).

In addition, certain CNS demyelinating diseases that manifest in an acute and monophasic manner, like TM or ADEM, have been reasonably associated with vaccines in many prior Program matters, based on sound medical science. MS is not the same, however. *Compare Raymo v. Sec'y of Health & Human Servs*., No. 11-654V, 2014 WL 1092274, at *23 (Fed. Cl. Spec. Mstr. Feb. 24, 2014) (finding causal relationship between flu vaccine and TM), *with Wei-Ti Chen v. Sec'y of Health & Human Servs*., No. 16-634V, 2019 WL 2121208, at *22 (Fed. Cl. Spec. Mstr. Apr. 19,

[20] Recent studies have posited an association between MS and an EBV infection. *See, e.g.*, *Jaye v. Sec'y of Health & Hum. Servs.*, No. 20-672V, 2024 WL 3691413, at *3–5, (Fed. Cl. Spec. Mstr. July 18, 2024). But claimants cannot persuasively invoke these studies in the context of *other* vaccines—especially since there is *no* EBV vaccine to begin with that could be analogized to any covered vaccine.

2019) (determining there was insufficient evidence provided to support a causal connection between the flu vaccine and petitioner's subsequent development of neuromyelitis optica spectrum disorder, which is chronic and relapsing/remitting, like MS).

There are several compelling reasons for treating MS differently from ADEM or TM, despite their overlapping features. MS is *chronic and persistent*, and often can be subclinical for lengthy periods of time. And its symptomatic flares (common to relapsing/remitting MS) can be triggered by a wide array of external environmental factors. *L.Z. v. Sec'y of Health & Hum. Servs.*, No. 14-920V, 2018 WL 5784525, at *6 (Fed. Cl. Spec. Mstr. Aug. 24, 2018). But the trigger for such a flare, whatever its nature, cannot be said to have "started" the underlying disease process *ab initio.*

By contrast, more self-limiting and monophasic neurologic injuries are understood to occasionally be triggered by the single impact of an external stimulus, such as a wild infection—and hence reasoning that the same kind of monophasic illness could also begin with the single instance of vaccine exposure has far greater power. But no infectious cause for MS has yet been identified (except for the nascent EBV research). In addition (and as noted in my discussion of the Marmoset Paper), it is not at all clear whether MS is a disease that occurs from the "outside-in"—driven by external stimuli to the immune system that works its way inward, to the spine and brain. Marmoset Paper at 22. It may well be the case that lesions within the CNS are responsible for the subsequent symptoms—and that their generation does not occur due to an external/environmental trigger.

Because of this distinction, it is scientifically unreliable to apply the analytic framework relevant to how some vaccines may cause acute, self-limiting demyelinating nerve injuries to a chronic, often-insidiously-developing disease like MS. *Samuels v. Sec'y of Health & Hum. Servs.*, No. 17-071V, 2020 WL 2954953, at *18–19 (Fed. Cl. Spec. Mstr. May 1, 2020) (finding petitioner's actual injury was MS, an illness far less associated with vaccination than one-time acute CNS demyelinating events like ADEM); *Pek v. Sec'y of Health & Hum. Servs.*, No. 16-0736V, 2020 WL 1062959, at *17 (Fed. Cl. Spec. Mstr. Jan. 31, 2020) (determining that evidence and expert reports did not provide sufficient proof that a progressive, chronic demyelinating condition like MS could be initiated by the flu and Tdap vaccines); *Wei-Ti Chen,* 2019 WL 2121208, at *22; *Hunt v. Sec'y of Health & Human Servs.*, No. 12-232V, 2015 WL 1263356, *15 (Fed. Cl. Spec. Mstr. Feb. 23, 2015) (denying entitlement where MS was the alleged injury, but the literature offered only discussed a causal relationship between vaccines and ADEM).

Relying upon such reasoning, I have repeatedly rejected causation theories that many different covered vaccines are capable of causing (or worsening) MS. *See, e.g.*, *Garris v. Sec'y of Health & Hum. Servs.*, No. 22-1354V, 2025 WL 2401999 (Fed. Cl. Spec. Mstr. June 20, 2025) (hepatitis B vaccine not shown to be causal of MS); *Porch v. Sec'y of Health & Hum. Servs*., No.

17-802V, 2023 WL 21875 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (MMR vaccine did not cause MS); *P.M.*, 2019 WL 5608859 (flu vaccine not shown to be capable of worsening MS); *Samuels*, 2020 WL 2954953, at *19–21 (Tdap vaccine not established to be causal of MS); *Pek*, 2020 WL 1062959, at *16–17 (flu and Tdap vaccines not causal of MS); *P.M.,* 2019 WL 5608859, at *22–27 (flu vaccine not shown to be able to worsen MS or to have done so to specific claimant); *L.Z.*, 2018 WL 5784525 (flu vaccine and MS). Although other special masters may have been more solicitous of this causation theory, I am not required to endorse their determinations (any more than they were obligated to accept my reasoning). And while the existence of contrary decisions seem to suggest that the relevant science is equivocal on the issue, I have found (based upon my own carefully-considered reasoned decisions) that it *unquestionably is not*.

II. Petitioner's Initial Presentation of TM/CIS Does not Define her Proper Diagnosis

Petitioner's causation theory is based in part on an inconvenient fact, and one she endeavors through her expert to evade. As evidenced by the totality of the record, she was properly diagnosed with MS—and it is just as clear that her MS first manifested with symptoms that *could have been* a single and monophasic instance of TM—but unfortunately proved not to be. In addition, there is far better medical and scientific support for a single instance of TM to be caused by an environmental trigger akin to vaccination (since infections are known to lead to CNS demyelination), than with respect to MS.

What to do? Dr. Tornatore's solution was to focus upon Petitioner's medical history in the fairly immediate post-vaccination timeframe (March-April 2019), rather than place those presenting neurologic symptoms in the context of her ultimate (and evidentiarily-supported) MS diagnosis. He thus emphasizes the fact that Petitioner's admittedly close-to-vaccination presentation would be consistent with TM and CIS—had Petitioner *not* also gone on to meet the other MS criteria. Of course, *that is in fact what happened*—and literature filed by Petitioner in this case clearly establishes that TM can be an initial presenting clinical manifestation of MS.

It is not difficult to discern *why* Petitioner would want to frame her claim around the initial diagnosis of TM—an oft-compensated injury that is far better known to be post-infectious (and even post-vaccinal) than MS. But the evidence does not allow the conclusion that Petitioner's TM was *unrelated* to her later MS diagnosis. Rather, applicable independent evidence strongly supports the conclusion that a person who first experiences a myelitis-like presentation, coupled with proof of a lesion characteristic of TM, but who later meets the diagnostic criteria for MS, *suffered from MS*—not two separate diseases. And this record establishes that as time progressed, Petitioner's treaters (taking into account the entirety of her medical history) noted that she had not experienced a single isolated event of TM. Indeed—there were signs early on, recognized by treaters, that Ms. Peterson might actually have MS. Ex. 26 at 21.

It cannot be assumed that simply because a vaccine might be shown capable of triggering a single, acute instance of a CNS-impacting demyelinating disease, like TM, that this *in turn* means that MS presenting with TM is *also* necessarily vaccine-caused. When MS is later diagnosed, an initial flare that is TM-like is almost always understood to be *part* of the MS disease process. The MS diagnosis is not deemed incidental or secondary to the initial TM diagnosis, or something that could be avoided with proper treatment—it *subsumes* it. Thus, Petitioner's initial TM-like symptoms cannot credibly be viewed in isolation in this case. And it does not matter if treaters initially viewed a first presenting neuropathic symptom as TM alone—since they usually do so without knowing enough of a patient's total presentation to understand the true picture.

Relying on such logic, I have dismissed several cases where MS proves to be the evidentiarily-supported diagnosis, even though the petitioner's *initial* symptoms seemed to first-responding treaters to reflect TM, ADEM, or a comparable acute demyelinating neuropathy. *See, e.g.*, *Morgan v. Sec'y of Health & Human Servs*., No. 15-1137V, 2019 WL 7498665, at *16 (Fed. Cl. Spec. Mstr. Dec. 4, 2019), *mot. for review den'd*, 148 Fed. Cl. 454 (2020), *aff'd*, 850 F. App'x 775 (Fed. Cir. 2021); *Caruso v. Sec'y of Health & Human Servs.*, No. 15-200V, 2017 WL 5381154, at *12–13 (Fed. Cl. Spec. Mstr. Oct. 18, 2017), *mot. for review den'd*, 137 Fed. Cl. 386 (2018). Other special masters have also so concluded. *See, e.g.*, *Juranek v. Sec'y of Health & Hum. Servs.*, No. 19-226V, 2025 WL 399501, at *33–34 (Fed. Cl. Spec. Mstr. Jan. 8, 2025) (flu vaccine not shown to be capable of aggravating MS). It was not established in such cases that reasoning specific to how a vaccine might cause a monophasic, one-time CNS injury applied to a larger-scale, chronic injury.

Taking all of the foregoing into account, I do *not* find on the basis of this record that Petitioner's initial, TM-like presentation can be cabined off, for causation purposes, from her later MS diagnosis. *MS is the injury at issue,* despite the nature of Petitioner's initial symptoms—and therefore the claim must be analyzed as one seeking to prove that the flu vaccine could cause MS, and did so here. That is the evidence-based, medically-appropriate way to evaluate the claim, as persuasively recognized by Dr. Sriram. *See* Sriram Rep. at 9.

III. Petitioner Has Not Carried Her *Althen* Burden of Proof

I find none of the *Althen* prongs satisfied in this case. I address them in the order consistent with how they are presented in the *Althen* decision.

A. *Prong One*

1. The Evidence Does not Support the Conclusion that MS Is Understood to Evolve From TM

At its core, Petitioner's causation theory operates in part according to this syllogism: (a) the flu vaccine can cause TM; (b) TM is often a presenting symptom of MS; (c) therefore, the flu vaccine can cause MS. *See, e.g.*, Br. at 25 ("if the evidence supports a finding that TM can be triggered by a vaccine, the same evidence can support a finding that MS can be triggered by a vaccine—the pathogenesis and features of the initial injury is the same—whether it ultimately results in an acute or chronic condition depends on host factors"). But this reasoning is ultimately fallacious. Indeed, the fact that Petitioner was accurately diagnosed with MS weighs *heavily* against the conclusion that her receipt of a flu vaccine could explain everything that came after.

As discussed above, MS (which *has no known trigger*—unlike TM in some cases) is a different entity from TM, even if the two overlap. MS is not understood to be post-infectious in the way TM is. And as evidence Petitioner offered shows, it may actually begin *entirely* within the CNS, with outward clinical manifestations downstream from the appearance of lesions (rather than something impacting the periphery and subsequently invading the CNS, as is true of many viral or bacterial infectious processes). *See, e.g.*, Marmoset Study at 22. It is reasonable to conclude that when an individual presents with myelitis-like symptoms that could constitute TM or something comparable, but then goes on to meet the other clinical and testing criteria for MS, *the MS is the cause of* the TM.

It is certainly *plausible* (although that is not the standard of proof applicable to this claim—as noted above and below) that a petitioner might be able to show (via reliable independent medical or scientific evidence) that vaccine-caused instances of TM "set up" a person to later experience MS—comparable to what Vaccine Act claimants accomplish in other kinds of cases.[21] If vaccines can trigger CNS demyelination of one sort, they could theoretically be responsible for causing

[21] There are, in fact, other kinds of Program claims where petitioners can preponderantly show that an initial vaccine injury has a greater, secondary/collateral health impact. Thus, in many seizure disorder cases, petitioners have succeeded in demonstrating that a first seizure provoked by a vaccine-induced fever could later lower a child's threshold for seizures, causing a snowball effect and leading to a greater seizure condition with attendant brain damage. *See, e.g.*, *Weaver v. Sec'y of Health & Hum. Servs.*, No. 16-1494V, 2022 WL 12542485 (Fed. Cl. Spec. Mstr. Sept. 23, 2022), *reversed in part, vacated in part*, 164 Fed. Cl. 608 (2023), *remanded*, 2023 WL 3836239, at *2 (Fed. Cl. May 8, 2023 (determining on remand that vaccines induced a febrile seizure that caused and significantly aggravated the child's seizure disorder; no evidence of an underlying genetic cause for the disorder had been supplied); *Ginn v. Sec'y of Health & Hum. Servs.*, No. 16-1466, 2021 WL 1558342, at *6 (Fed. Cl. Spec. Mstr. Mar. 26, 2021) (finding that "a brief febrile seizure triggered by vaccinations can be the starting point for the development of epilepsy."). More prosaically, syncope after vaccination is a recognized Table injury—and in such instances, damages are almost always dependent on proof that a person's initial fainting from receipt of a vaccine caused them to experience some form of serious consequential harm (such as injury to the cranium or face).

progressive or relapsing forms of comparable illnesses. But nothing has been offered *in this case*, direct or indirect, to suggest that MS is understood by medical science to proceed in this manner—*that its occurrence is likely dependent on some initial autoimmune event triggered by an environmental factor*.[22] None of the articles in this case specific to MS propose that individuals who experience TM are subsequently more likely to develop MS *due* to the first illness. (At most, articles like Langer-Gould support the conclusion that individuals *already* suffering from MS sub-clinically could have their symptoms manifestations hastened due to vaccination—but that is not consistent with a causation determination).

At bottom, it has not at all been preponderantly shown that TM likely "causes" MS. It is accordingly erroneous to view in isolation the first clinical manifestation of MS as a separate instance of vaccine-caused TM, when in fact that first symptom was (in retrospect) the precursor of something worse (and that likely was already underway). TM itself does not unerringly lead to MS in any event. And the kinds of environmental factors that can cause limited forms of CNS demyelination or nerve injury are not equally causative of MS.

2. Petitioner Mistakenly Relies on a Plausibility Standard

An overarching legal mistake evident in Petitioner's briefing is the extent to which she appears to rely on a standard of plausibility, rather than preponderance, in proving her causation theory. *See, e.g.*, Br. at 14, 24. In *Cerrone,* however, the Federal Circuit made clear that the lesser standard of scientific/medical plausibility does not govern the first *Althen* prong. *Cerrone,* 146 F.4th at 1122. Rather, a claimant must demonstrate it is "more likely than not" that the vaccine in question can cause the relevant injury. This is the standard I am applying in my analysis—and overall, I have not found it met.

3. Dr. Tornatore Has Not Established that MS Likely Begins Due to Cross-Reactivity with Nerve Myelin Components

Petitioner correctly notes that she is not *required* by Program case law to offer a mechanism for how the flu vaccine could cause MS (Br. at 11)—but then Dr. Tornatore goes ahead and attempts to do so in any event. Obviously, when an expert offers a mechanistic explanation in the context of their causation opinion, a special master may reasonably evaluate the probative strength of that showing. *Bender v. Sec'y of Health & Hum. Servs.*, No. 11-693V, 2018 WL 3679637, at *28 (Fed. Cl. Spec. Mstr. July 2, 2018) (explaining that petitioner had not offered any direct

[22] One can envision many forms of evidence that might help prove this point. For example, studies that showed how effective treatment of TM could *prevent* the subsequent onset of MS could credibly aid the theory that what causes TM can secondarily cause MS. But I am not aware of any such studies—whether filed in this case or even in existence—suggesting that a dysregulated immune response thought associated with TM could, if not arrested, result in a chronic disease.

scientific or medical evidence associating the relevant vaccines and TM, but instead attempted to bulwark her case via a discussion of mechanisms by which any vaccine might cause the alleged injury at hearing, and thus, it was therefore reasonable to evaluate her success on that front).

Dr. Tornatore's opinion reflects what many experts attempt to do when similarly faced with proving how a vaccine could initiate an autoimmune process. He endeavors to identify an amino acid sequence from a component of the flu vaccine, and a mimicking sequence from a target antigen from nerve tissues (here, MBP); he offers some evidence that the target has been shown to be cross-reactive from reputable studies involving neuropathic harms; and he seeks to root all of the above in the context of MS.

But this showing fails, for the reasons I have often stated in prior cases. *McKown v. Sec'y of Health & Hum. Servs*., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (explaining that "merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or vaccine in question" (emphasis omitted)). In isolation, molecular mimicry is a reliable scientific concept that *can* explain how autoimmune diseases occur—but it is not a blanket explanation for every single possible autoimmune disease, and it has not in this case been shown to be a likely explanation for MS's pathogenesis, despite its relevance to other demyelinating disease processes. Thus,

> (a) homology demonstrations alone, based on amino acid sequence similarity, do not establish vaccination is likely to cause an antibody-driven cross-reaction,
>
> (b) MS has not been shown to be mediated *at the outset* (as opposed to *during* disease course, in the midst of damaging processes already underway) by an autoimmune attack on MBP, or MOG for that matter, and
>
> (c) many of the studies offered to suggest that MS *could* be so driven involve either direct transfer of peptide sequences based on the goal of a cross-reaction, occur in the context of an animal study intended to provoke an experimentally-observable response (like EAE), or involve existing MS patients (rather than disease initiation)—and hence are not comparable to the immune stimulation provided by vaccination.

In the end, this was yet another case in which a Petitioner attempts to offer the otherwise-reliable medical theory of molecular mimicry—which "works" in conjunction with some kinds of demyelinating autoimmune diseases—as an explanation for how a vaccine could spark an entirely

different autoimmune disease, but without linking up all elements of the theory with sufficient reliable scientific or medical evidence.

4. The "Marmoset Paper" Does not Constitute Novel Scientific Thinking Supportive of Petitioner's Causation Theory

Despite Petitioner's embrace of the Marmoset Paper as reflecting critical and recent thinking about MS's pathogenesis bearing on this case, a careful reading of the article reveals less support for causation than Petitioner supposes. The main thrust of this article is a defense of a particular form of animal EAE study, and while it reviews various other published marmoset-subject studies that bear on the case, Marmoset Paper was not directly intended to comment on the kinds of issues at stake in this matter. Moreover, its author not only outlines an "inside out" pathogenic theory for MS *inconsistent* with a peripherally-administered vaccine causing systemic inflammation that subsequently goes to the CNS, but seems to *favor* that explanation (while admitting that medical science still knows too little about MS's pathogenesis to fully rule out *any* explanation for it). Marmoset Paper at 22.

In addition, even if the Marmoset Paper's primary focus is ignored and weight is given to the results of the studies it references, the article does not appreciably advance the concept that a peripherally-administered flu vaccine could spark an aberrant immune process leading to MS. Its discussion of MOG, for example, did not involve the generation of anti-MOG antibodies against this myelin-found protein due to vaccination, but instead direct transfer of peptides already known to be homologous. In addition, the discussed study was not itself ever filed in this case—and MOG is not likely particularly relevant to MS in any event.[23]

Given the foregoing, how does the Marmoset Paper actually assist Petitioner's case? It is facially equivocal in concluding whether *any* environmental factor like vaccination could even trigger the disease process (although it clearly does not rule this possibility out). It offers a reasoned defense of EAE marmoset models in investigating MS, but the merits of different experimental methodologies or approaches are not all that relevant to whether the flu vaccine can cause MS. And the specific studies it mentions that *do* go to the questions of causation disputed in this case only offer limited support for an external trigger as initiating MS, while supporting alternative

[23] Current medical science favors MOG-associated demyelinating diseases (MOGAD) as *distinguishable* from MS—not that it is believed that MOG antibodies likely drive MS in any manner. *See, e.g.*, *Garris*, 2025 WL 2401999, at *10 ("MS patients are not commonly found to possess anti-MOG antibodies in the first place"). In addition, the Marmoset Paper was published six years ago, and relied on studies from prior to that time. *See generally* Marmoset Paper at 49-58 (bibliography). Thus, while it is hardly outdated, it does not reflect the most up-to-date thinking on the relevance of MOG antibodies to MS. And again, the goal of the Marmoset Paper (to defend this kind of model for MS research) should be kept in mind as well, since its author is unapologetically seeking to demonstrate the importance of the model—a different aim from defending the accuracy of MOG-oriented studies when applied to MS.

etiologies that would *rule out* vaccine causation if found to be scientifically correct. I thus do not find the Marmoset Paper warrants the significance Petitioner or her expert has proposed it merits.

5. Other Literature Filed in this Case is Inapposite or Fails to Preponderantly Support Flu Vaccine-MS Causation

Many items of literature offered by Dr. Tornatore involve distinguishable injuries (*see, e.g.*, Schonberger (Guillain-Barré syndrome), Rowhani-Rahbar (ADEM), and Nakamura (TM and ADEM)), or vaccines (Langer-Gould (focus on Hepatitis B and HPV)). I reasonably give such literature a bit less weight in comparison to articles more specific to MS.

Even those more MS-oriented items of literature were less helpful to the Petitioner's case than billed. Markovic-Plese, for example, demonstrated cross-reactivity in the context of an existing MS patient, and thus does not stand as robust proof that the flu vaccine's antigens can *prompt* MS *ab initio,* via autoantibody cross-reactivity. Bielekova relies on uncorroborated assumptions about MS's potential propagation from an external/environmental insult, involved a very small sample of subjects, and (in a study that actually aimed to seek ways to better treat MS) reached inadvertent conclusions about cross-reactivity with MBP that does not suggest MS likely initiates in this manner.

Langer-Gould (which has in other cases been relied upon as evidence that vaccines might hasten MS's clinical manifestations) actually stands for the greater conclusion that commonly administered vaccines (like the flu vaccine) are *not* reliably associated with MS. To the extent it suggests otherwise, its findings are simply consistent with the understanding that an immune stimulus can produce MS clinical symptoms (flares in existing patients; first manifestations in individuals with sub-clinical courses)—not that the vaccine's immune stimulation *causes* MS. And there are at the same time—and in addition to Langer-Gould—other epidemiologic studies standing for the conclusion that the flu vaccine does not likely cause MS. *See, e.g.*, Hapfelmeier.

6. Not Enough is Yet Known About MS's Etiology To Credibly Implicate Vaccines in its Pathogenesis

In many vaccine injury cases, a claimant can point to the fact that a vaccine's wild viral or bacterial analog is itself associated with the alleged injury. Here, by contrast, there is no comparable known infectious basis for MS relevant to the vaccines covered in the Program. A. Hernandez et al., *Multiple Sclerosis*, in THE AUTOIMMUNE DISEASES 735, at 745 (N. Rose and I. Mackay, eds., 2014 (Fifth Ed.)). Dr. Sriram endorsed this view as a neurologist conversant with the study and treatment of MS—along with the concurrent fact that MS's cause remains unknown, and even that the theory that it proceeds via an autoimmune process has many holes left to be filled. Sriram Rep. at 4–5.

Dr. Tornatore of course attempted to maintain the opposite. But although he clearly possesses the expertise needed to opine on the subject, he did not link his theories (in a case in which he was acting as an advocate, well-aware of the legal requirements for a successful Vaccine Act claim) to evidence that would establish that his opinion possesses wide-spread support in the medical community. Ultimately, it was not shown in this case that enough is known about MS to conclude that a foreign/environmental agent could *likely* instigate it (and in fact, items of literature like the Marmoset Paper seem to deem this unlikely). While at some later point a study might better elucidate how some environmental factor analogous to vaccination could spark MS, that kind of reliable research remains to be performed. The studies and articles filed in this case may establish that within MS's ongoing processes cross-reactivity leading to demyelination occurs—but this is not the same as evidence suggesting vaccination instigates it.

7. Dr. Tornatore's Personal Expertise Was Not Enough to Render his Overall Opinion Sufficient to Establish His Proposed Theory by a Preponderance

Petitioner undoubtedly found in Dr. Tornatore an expert with significant personal expertise in the study and treatment of MS, and he was well-qualified to offer an expert opinion in this case (even if his knowledge of immunology as a separate discipline is somewhat comparatively less than his grasp of neurology). But as I recently observed in a different matter also involving Dr. Tornatore's work as an expert, "I discount the opinion offered on Petitioner's behalf more than I find fault in the personal qualifications of the expert espousing it." *Kazcerowski v. Sec'y of Health & Hum. Servs.,* No. 21-758V, 2025 WL 2798865, at *38 (Fed. Cl. Spec. Mstr. Aug. 28, 2025).

I have the same reaction here to Dr. Tornatore's opinion. While logically presented, it over-relied on the assumption that what can cause TM can also cause MS, put too much emphasis on a showing of homology of the sort repeatedly rejected as evidentiarily sufficient, and assumed things about MS's course or etiology that are not fully accepted in the medical community (even if the articles selected to prove these contentions contained reliable conclusions about the studies they considered).

I was not required to accept Dr. Tornatore's *ipse dixit*. Rather, I am ultimately to base my decision on the evidence offered *in this case*. And I do not find that the balance of proof favors causation, even applying the Program's preponderant standard of "fifty percent and a feather"—for that standard is not easy to meet even if it does not require certainty. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed. Cir. 1993) (in causation-in-fact cases, "the heavy lifting must be done by the petitioner, and it is heavy indeed"). Dr. Tornatore's opinion was based on partial evidence that did not effectively link up into a persuasive causation theory. He relied too much on somewhat-stale studies, blanket applications of molecular mimicry to a context in which

it may not work, and assumptions about MS's etiology—including in particular that it likely begins *due* to an external/environmental trigger.

B. *Prong Two*

Petitioner has also not preponderantly established that the flu vaccine she received "did cause" her MS. Treaters certainly did not link the two. And to the extent they discussed both, it was only to acknowledge the fact that Petitioner's MS onset *literally* post-dated vaccination (hence a mere temporal relationship). Ex. 6 at 48. At most, at the time it was possible that Petitioner had only experienced a one-time, monophasic injury, TM, treaters allowed for an association. But later treaters who diagnosed Petitioner's MS, like Dr. Shoemaker, expressly discounted the flu vaccine as a causal factor. Ex. 6 at 84. Thus, the actual injury Petitioner experienced was not deemed likely associated with the flu vaccine.

Admittedly, the record does establish Petitioner received the flu vaccine while apparently suffering from the flu—an occurrence Dr. Tornatore deemed significant. First Tornatore Rep. at 3. But there is no evidence Petitioner experienced any kind of unusual, post-vaccination reaction that would suggest an aberrant immune response (perhaps characterized by excessive inflammation). Rather, within three days of vaccination, she began to experience neurologic-like symptoms that later reflected her MS onset. Petitioner has not otherwise established that receipt of a flu vaccine while experiencing an intercurrent infection is contraindicated or suggests a risk.

C. *Prong Three*

The timeframe in which Petitioner's neurologic symptoms presented was too short, measured from the date of vaccination, to be deemed medically acceptable (even if I were to assume the flu vaccine "can cause" MS).

First, the record best supports the conclusion that Petitioner's early, MS-associated symptoms—numbness and tingling—manifested March 29, 2019, no later than *three days post-vaccination*, when she (by her own admission) began experiencing numbness and tingling that lead her to seek chiropractic help, followed by ED visits on April 2 and 3, 2019, and then to her hospitalization, when TM was first proposed as explanatory. Peterson Aff. at 2; Ex. 7 at 67.

This timeframe is far too short for a disease involving not only actual clinical manifestations, but manifestations attributable to or associated with lesions identified on MRI only a few days after clinical symptoms occurred. Dr. Sriram persuasively established that an antibody-mediated disease process (which is how Petitioner's injury would unfold if her theory were accepted) would not likely occur in so short a timeframe—and Dr. Tornatore did not convincingly rebut these contentions, over-relying on literature specific to other kinds of diseases like Guillain-

Barré syndrome or ADEM. (The experts' disagreement about the relationship between the oligoclonal bands and lesion imaging findings was ultimately without moment, for there is no dispute that by the time they were observed, Petitioner's MS had begun—three days post-vaccination. And the oligoclonal bands only corroborate that Petitioner's initial symptoms did not constitute simply a one-time occurrence of a form of myelitis).

Second, the evidence of enhancing lesions (meaning more recent) as revealed by Petitioner's MRIs (first performed April 3, 2019) raises the strong possibility that Petitioner's MS *already existed* at the time of vaccination a week before. I have noted in other cases that lesions often predate symptoms in MS. *Porch*, 2023 WL 21875, at *21 (noting that MS is a chronic disease process impacting the CNS and that it "can be subclinical and symptomatically silent for a longer period of time, with MS-characteristic brain lesions often discovered in the absence of symptoms, or non-recent lesions discovered only after clinical manifestations"). Even Dr. Tornatore allowed that the lesions could be as "old" as two to three weeks by the time they were observed—a timeframe which could support the determination that the lesions began developing up to two weeks prior to vaccination. It cannot be concluded on this record that it is *likely* the lesions sprung into existence within a week of vaccination *and* also concurrent with appearance of actual symptoms.

Indeed, if Petitioner's lesions existed for more than seven days from when they were discovered, it is likely her MS *preceded* vaccination—meaning the vaccine *could not have caused it*. *McDaniel v. Sec'y of Health & Hum. Servs*., No. 17-1322V, 2023 WL 4678688, at *33 (Fed. Cl. Spec. Mstr. June 26, 2023) (finding that Petitioner could not satisfy Althen prong two where onset of immune-mediated myopathy occurred before vaccination). The fact that Petitioner was only *diagnosed* with MS later, after treaters were able to factor in a combination of clinical evidence with test results, does not mean it likely began post-vaccination as well. *Flowers v. Sec'y of Health & Hum. Servs*., No. 20-285V, 2024 WL 2828211, at *12 (Fed. Cl. Spec. Mstr. May 8, 2024) ("[i]t is a foundational matter of Vaccine Program law that onset occurs at first manifestation of a symptom, *regardless* of whether the disease it foretells could be diagnosed at that time—and thus whether the onset symptoms would be clearly understood to reflect the start of the illness"), *mot. for review den'd,* 173 Fed. Cl. 613 (2024).

Of course, articles like Langer-Gould support the conclusion that the flu vaccine could (in a younger population) hasten the clinical "arrival" of MS—seeming to open the door to the possibility that Petitioner's vaccination still played some role in "unmasking" her MS. But this is not the same as a finding that the flu vaccine directly caused or triggered it (nor could it have been responsible for a process that was already underway at the time of vaccination). And as Langer-

Gould determined, the *ultimate* risk from vaccination was low no matter *when* the MS symptoms appeared.[24]

IV. This Matter was Appropriately Dismissed Without Trial

I am opting to dismiss this case on the existing record, and without holding a hearing. Determining how best to resolve a case is a matter that lies generally within my discretion, but I shall explain my reasoning.

Prior decisions have recognized that a special master's discretion in deciding whether to conduct an evidentiary hearing "is tempered by Vaccine Rule 3(b)," or the duty to "afford[] each party a full and fair opportunity to present its case." *Hovey*, 38 Fed. Cl. at 400–01 (citing Rule 3(b)). But that rule also includes the obligation of creation of a record "sufficient to allow review of the special master's decision." *Id*. Thus, the fact that a claim is legitimately disputed, such that the special master must exercise his intellectual faculties in order to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes without a hearing—although they should only so act if a party has been given the proper "full and fair" chance to prove their claim.

In addition, there is another overarching consideration at play. Special masters are intended to develop "on the job" expertise from deciding entitlement in the numerous Vaccine Act claims that exist at the Court of Federal Claims. *Hodges*, 9 F.3d at 961 ("Congress assigned to a group of specialists, the Special Masters within the Court of Federal Claims, the unenviable job of sorting through these painful cases and, *based upon their accumulated expertise in the field*, judging the merits of the individual claims") (emphasis added). Over time, the special masters become conversant in the kinds of theories presented in certain cases, and whether those theories have sufficient scientific and/or factual validity. And those theories often overlap from one case to the next. So where a theory of causation is reasonably doubted—and that doubt stems from the special master's prior exposure to the theory in many prior cases—that theory should not be entertained anew (except where the claimant can point to novel scientific or medical understanding sufficient to breathe life into a theory previously deemed wanting). Only by doing so can special masters ensure that the Program focuses on fairly compensable claims, and husband their judicial resources in an efficient manner.

My review of the record here plus Petitioner's arguments have convinced me that she did not preponderantly establish that the flu vaccine can cause MS. I reach that determination on the basis of Dr. Tornatore's reports, which were clear in their arguments. I did not need to litigate the

[24] Notably, Petitioner has not alleged a claim of significant aggravation in this case—and I do not find it has been shown the flu vaccine can worsen an existing case of MS. In fact, I have found precisely the opposite in several prior matters. *See, e.g.*, *L.Z.*, 2018 WL 5784525, at *22 (rejecting petitioner's claim that the flu vaccine significantly aggravated his pre-vaccination MS).

matter via a live proceeding to reach the determination I have. The inquisitorial function of special masters in the Vaccine Program compels them to steer cases in the most sensible direction, based on the facts presented as well as the special master's experience with comparable claims.

Because my preliminary review of the filings did not suggest (based on my experience with comparable cases) that this matter was likely to succeed, I asked Petitioner to establish whether, and how, I might be wrong. Despite due opportunity, Petitioner has not succeeded in doing so.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Petitioner cannot make such as showing. She therefore is not entitled to compensation.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[25]

IT IS SO ORDERED.

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

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