In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 22-1408V
Filed: July 23, 2026

ANNAMMA VARUGHESE,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Kirk Tripp Otto, Siri & Glimstad, LLP, Richmond, VA, for petitioner.
Mallori Browne Openchowski, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On September 30, 2022, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that she suffered transverse myelitis ("TM") caused by the influenza ("flu") vaccination that she received on September 21, 2021. (ECF No. 1.) For the reasons set forth below, I conclude that petitioner is *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

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

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

[2] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

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

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

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

A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally

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

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

II. Procedural History

Petitioner filed medical records, a Vaccine Adverse Event Reporting System ("VAERS") report, declarations by petitioner and her spouse, and a Statement of Completion in October of 2022. (ECF Nos. 7-8; Exs. 1-6.) The case was initially assigned to another special master. (ECF Nos. 10-11.) Petitioner filed additional medical records in August of 2023 (ECF No. 18; Ex. 7), and respondent then filed his Rule 4(c) Report in September of 2023 (ECF No. 21). Respondent recommended against compensation, arguing that petitioner's medical records were inadequate to meet the *Althen* test and stressing that symptom onset occurred within 24 hours of vaccination. (*Id.* at 8-9.)

The parties then exchanged multiple rounds of expert reports, with neuroimmunologist Lawrence Steinman, M.D., opining on petitioner's behalf (Exs. 8, 51,[3] 52), and neurologist Alexander Merkler, M.D., and immunologist You-Wen He, M.D., Ph.D., opining on respondent's behalf (Exs. A, C, E-F). Thereafter, the previously presiding special master directed respondent to file a motion for a ruling on the written

[3] Petitioner filed Dr. Steinman's first supplemental report at ECF No. 29-1 without proper bates stamping. However, for the purposes of this decision, Dr. Steinman's first supplemental report will be referred to as Exhibit 51, the exhibit designation reflected on the notice of filing (ECF No. 29).

record after the parties confirmed they would not file further expert reports. (ECF No. 37; Non-PDF Scheduling Order, filed Feb. 10, 2025.)

The case was subsequently reassigned to the undersigned in February of 2025 (ECF No. 38), and respondent filed his motion for a ruling on the written record on May 1, 2025 (ECF No. 45). That motion is fully briefed as of August 6, 2025. (ECF No. 47 (petitioner's response); ECF No. 50 (respondent's reply).) Based on my review of the docket, the record, and the parties' briefs, I agree that the parties have had a full and fair opportunity to develop the record and that it is appropriate to resolve this case on the existing record.[4] *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Sec'y of Health & Human Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020).

III. Factual Summary

The facts of this case are largely not disputed and need not be discussed at length. Petitioner received the flu vaccination at issue on September 21, 2021, at 66 years of age. (Ex. 3, p. 7.) Although she was not in perfect health, her prior medical history is not at issue. Three days after her vaccination, on September 24, 2021, petitioner presented to the emergency department with complaints of chest pain radiating to her back with bilateral leg weakness and numbness as well as nausea. (Ex. 4, p. 683.) At that time, she reported a gradual onset of symptoms over the course of two days, placing onset the day after vaccination. (*Id*.) Petitioner's initial neurological exam was unremarkable; however, she was admitted to the hospital for observation and a workup for possible Guillain Barré Syndrome ("GBS"). (*Id.* at 686-87.) On September 26, 2021, petitioner was discharged with a differential diagnosis of GBS versus a spinal cord lesion versus polyradiculopathy. (*Id*. at 679-80.) However, when petitioner later followed up with her primary care provider for ongoing symptoms (*Id.* at 632-41), the primary care provider felt that transverse myelitis ("TM") was likely, and petitioner was advised to return to the emergency department after a repeat MRI suggested "progression of demyelinating or inflammatory process" (*Id.* at 381-85, 620, 624-45, 641). Petitioner was admitted to neurology and completed a five-day course of IV Solu-Medrol. (*Id*. at 320, 376-85).

On October 10, 2021, the pharmacy where petitioner received her vaccination submitted a VAERS report indicating that petitioner experienced symptoms, which were later diagnosed as transverse myelitis, beginning on September 22, 2021, the day after vaccination. (Ex. 5, pp. 1-2.) Petitioner continued to treat and evaluate for possible transverse myelitis. (*See generally* Ex. 4.) Ultimately, she sought a second opinion from neurologist Buse Sengul, M.D., on February 28, 2022. (*Id.* at 184-89.) Dr. Sengul's impression was, "66-year-old woman with longitudinally expansile transverse myelitis, negative AQ P4 and MOG antibodies, paraneoplastic panel negative as well as cancer screening. This is truly an idiopathic transverse myelitis, likely provoked by

[4] In connection with her motion response, petitioner filed additional medical literature. (ECF No. 48; Exs. 54-59.) In reply, respondent argued the additional literature is inapposite but did not object to its filing. (ECF No. 50, pp. 2-3.)

vaccine, which can be seen with MOGAD although she remains to be seronegative." (*Id*. at 188.) Notably, however, the history recorded by Dr. Sengul does not document the preceding vaccination. (*Id*. at 184.) Accordingly, it is unclear what Dr. Sengul's understanding was with regard to the latency between petitioner's flu vaccination and the onset of her TM, though petitioner did consistently report a 24-hour onset both before and after this evaluation. (*Id.* at 159, 356, 683; Ex. 5, pp. 1-2.)

Declarations by petitioner and her spouse are consistent with the above. (*See* Exs. 1-2.) Neither petitioner nor respondent has identified any treating physician opinion as informative of the cause of petitioner's condition apart from the above-referenced notation by Dr. Sengul. (*See* ECF Nos. 1, 21, 45, 47.)

IV. Expert Summary

a. Petitioner's initial report by Dr. Steinman[5]

Dr. Steinman opined that the condition at issue is TM. (Ex. 8, p. 8.) However, although petitioner suffered TM, she tested negative for the two known antigens associated with TM, MOG and AQP-4, suggesting she did not mount an immune response to either antigen. (*Id*. at 8-9.) Instead, Dr. Steinman suggested that petitioner's TM is explained by molecular mimicry resulting from cross-reaction between components of the flu vaccine and neurofascin, a molecule found at the Node of Ranvier in the central and peripheral nervous systems that has been implicated in TM as well as neuromyelitis optica spectrum disorder ("NMOSD"). (*Id*. at 9, 12 (citing Matthew N. Rasband & Elior Peles, *The Nodes of Ranvier: Molecular Assembly and Maintenance*, 8 COLD SPRING HARBOR PERSPECTIVES BIOLOGY, 2016, at 1 (Ex. 17); Fang Guo & Yong-Bo Zhang, *Clinical Features and Prognosis of Patients with Guillain-Barré and Acute Transverse Myelitis Overlap Syndrome*, 181 CLINICAL NEUROLOGY & NEUROSURGERY 127 (2019) (Ex. 18); Jun-ichi Kira et al., *Anti-Neurofascin Autoantibody and Demyelination*, 130 NEUROCHEMISTRY INT'L, 2018, at 1 (Ex. 19); Kun Jia et al., *Anti-Neurofascin-155 Antibody-Positive Neuromyelitis Optica Spectrum Disorders*, 398 J. NEUROLOGICAL SCIS. 16 (2019) (Ex. 20)).) Specifically, Dr. Steinman indicated that a BLAST[6] search confirmed that influenza hemagglutinin contained in the Fluad vaccine

[5] Dr. Steinman received his medical degree from Harvard University in 1973. (Ex. 9, p. 1.) He completed a surgical internship and two residencies, one in neurology and one in pediatrics, at Stanford University Hospital. (*Id.*) Thereafter, Dr. Steinman completed a fellowship in chemical immunology at Weizmann Institute of Science and also served as a visiting fellow at the National Institutes of Health. (*Id.*) He maintains his license to practice medicine in California and is board-certified in neurology. (*Id.* at 2; Ex. 8, p. 1.) Currently, Dr. Steinman serves as a Professor at Stanford University, Departments of Neurology and Neurological Sciences, Pediatrics, and Genetics. (Ex. 9, p. 1.) In his clinical practice, he cares for patients with neuroinflammatory disorders, including transverse myelitis. (Ex. 8, p. 1.) Dr. Steinman has co-authored over 600 peer-reviewed publications, with a focus on neuroinflammation in the nervous system. (*Id*.; Ex. 9, pp. 5-51.)

[6] Basic Local Alignment Search Tool ("BLAST") "finds regions of similarity between biological sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance of the matches. BLAST can be used to infer functional and evolutionary relationships between sequences as well as help identify members of gene families." *Blast Local*

shares a six of ten amino acid sequence with neuorfascin, **AG**SG**II**IS**DT** (hemagglutinin) versus **AG**DT**II**FR**DT** (neurofascin). (*Id*. at 23-24.)

Dr. Steinman opined that this degree of homology is relevant based on his citation to a number of papers, which he described as demonstrating that sequences of five out of 12 identical amino acids have been shown to be sufficient to trigger neuroinflammation. (Ex. 8, pp. 15, 24 (citing Anand M. Gautam et al., *A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis*, 176 J. EXPERIMENTAL MED. 605 (1992) (Ex. 27); Anand M. Gautam et al., *Minimum Structural Requirements for Peptide Presentation by Major Histocompatibility Complex Class II Molecules: Implications in Induction of Autoimmunity*, 91 PNAS 767 (1994) (Ex. 28); Anand M. Gautam et al., *A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis*, 161 J. IMMUNOLOGY 60 (1998) (Ex. 29)).) He also asserted that the significance of this finding is further supported by the Immune Epitope Database ("IEDB"),[7] which shows that "APAAGDTIISL" has been studied in humans. (*Id*. at 25.) However, although Dr. Steinman included a screenshot documenting the IEDB search results, the screenshot does not indicate what has been observed about this antigen. (*See id*.)

Additionally, Dr. Steinman indicated that "AGDTIFRDT," the sequence identified in neurofascin, is in a region of the immunoglobulin domain between Ig4 and Ig5, which means it is "capable of interactions with the extracellular milieu where antibody and T cell receptor might interact." (Ex. 8, p. 26 (citing Lucas M. Chataigner et al., *Structural Insights into the Contactin-1 Neurofascin 155 Adhesion Complex*, 13 NATURE COMMC'NS, 2022, at 1 (Ex. 39)).) He opined that "[a]n antibody targeting this region would more likely than not disrupt paranodal architecture." (*Id*.) Dr. Steinman indicated that neurofascin also has "remarkable homology" with GlialCAM, which is also highly related to Contactin-1. (*Id*. at 22, 26 (citing Tobias V. Lanz et al., *Roadmap for Understanding Mechanisms on How Epstein-Barr Virus Triggers Multiple Sclerosis and for Translating These Discoveries in Clinical Trials*, 12 CLINICAL & TRANSLATIONAL IMMUNOLOGY, 2023, at 1 (Ex. 35)).) The above referenced sequence "AGDTIIFRDT" is adjacent to the homology between GlialCAM and neurofascin, which occurs at "TIIFRDT." (*Id*. at 27.) Dr. Steinman did not explicitly indicate why the similarity between neurofascin and GlialCAM is relevant; however, in substantiating that a five of 12 amino acid sequence can support molecular mimicry, he cited a paper by his own research group demonstrating that the Epstein-Barr virus can cause multiple sclerosis, another central nervous system demyelinating condition, via molecular mimicry involving GlialCAM. (*Id*. at 18-19 (citing Tobias V. Lanz et al., *Clonally Expanded B*

Alignment Search Tool, NAT'L LIBR. MED., https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited July 15, 2026).

[7] The Immune Epitope Database ("IEDB") "catalogs experimental data on antibody and T cell epitopes studied in humans and other animal species in the context of infectious diseases, allergy, autoimmunity and transplantation. The IEDB also hosts epitope prediction and analysis tools." IMMUNE EPITOPE DATABASE & TOOLS, https://www.iedb.org/ (last visited July 15, 2026).

Cells in Multiple Sclerosis Bind EBV EBNA1 and GlialCAM, 603 NATURE 321 (2022) (Ex. 30)).)

Because onset of petitioner's TM occurred about 24 hours post-vaccination, Dr. Steinman indicated that "there MUST be a recall response" to explain the timing in this case. (Ex. 8, p. 27.) Thus, he confirmed that petitioner received flu vaccinations in 2019-2021. (*Id*. at 27-28 (citing Ex. 3, pp. 5-7).) He opined that all three vaccines "had enormous degrees of homology." (*Id*. at 28.) That is, "[a]ll three vaccines including the 2021-2022 vaccine had at least 5 identical amino acids in the stretch of hemagglutinin with the sequence in the 2022 vaccine – AGSGIIISDTP – shared with the neurofascin mimic." (*Id*. at 30.) Thus, he opined there is "a high likelihood" that there would be a recall response from the two prior vaccines. (*Id*.) Dr. Steinman indicated that a recall response can result in neurologic symptoms within a day or two based on two epidemiologic papers by Schonberger et al. and Park et al. respectively, and an experimental study by Lai et al. (*Id*. at 31-33 (citing Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979) (Ex. 40); Yong-Shik Park et al., *Clinical Features of Post-Vaccination Guillain-Barré Syndrome (GBS) in Korea*, 32 J. KOREAN MED. SCI. 1154 (2017) (Ex. 41); Wendy Lai et al., *Transcriptional Control of Rapid Recall by Memory CD4 T Cells*, 187 J. IMMUNOLOGY 133 (2011) (Ex. 47)).) Dr. Steinman asserted that the Lai et al. study shows that a recall response can occur as early as 6 hours after antigenic exposure and the Schonberger et al. study described an increased risk of GBS 0-1 days after vaccination. (*Id*. at 32-33 (citing Lai et al., *supra*, at Ex. 47; Schonberger et al., *supra*, at Ex. 40, p. 8 fig. 5).)[8] He also indicated that tuberculin tests, which can be read as positive within 24 hours, further demonstrate that antigen-specific recall responses can occur within that timeframe. (*Id*. at 32 (citing Tiroumourougane V. Serane & Bhuvaneswari Kothendaraman, *Brief Report: Tuberculin Test Can Be Read After 24 Hours in Children*, 60 J. TROPICAL PEDIATRICS 157 (2014) (Ex. 43); Lin Fan et al., *Variation of* Mycobacterium tuberculosis *Antigen-Specific IFN-γ and IL-17 Responses in Healthy Tuberculin Skin Test (TST)-Positive Human Subjects*, 7 PLOS ONE, 2012, at 1 (Ex. 44); T. Kardjito & J.M. Grange, *Immunological and Clinical Features of Smear-Positive Pulmonary Tuberculosis in East Java*, 61 TUBERCLE 231 (1980) (Ex. 45)).)

Given that Dr. Steinman has opined that a medical theory exists to explain how petitioner's vaccine can cause TM, and given that he opined that a recall response can explain how that can occur within one day of vaccination, Dr. Steinman further opined that there is a logical sequence of cause and effect that demonstrates that petitioner's vaccination did cause her injury. (Ex. 8, pp. 38-39.) In addition to the sequence of events he proposed, he noted that petitioner had no infectious illness at the time of her

[8] Dr. Steinman further highlighted that the bar graph in figure 5 shows that there were more cases of GBS observed on days 0-1 than on days 35 and 41. (Ex. 8, p. 33 (discussing Schonberger et al., *supra*, at Ex. 40, p. 8 fig. 5).) Based on my review of his report and my experience in prior cases, Dr. Steinman is presumably asserting that this finding is significant because, while it remains controverted that the day 0-1 cases reflect a causal relationship, it is generally not disputed that days 35 and 41 fall within the period of elevated risk. (*Id*.)

vaccination and that none of her preexisting conditions are known to provoke TM. (*Id*. at 39.)

b. Respondent's initial report by Dr. Merkler[9]

Dr. Merkler agreed that petitioner suffered TM and that the onset of her condition occurred about 24 hours following her flu vaccination. (Ex. A, pp. 5-6.) He also agreed that petitioner's TM was not found to be related to any other condition, meaning that it was idiopathic. (*Id*. at 6.) However, citing the lack of any clear epidemiologic signal, Dr. Merkler did not agree that the flu vaccine can cause TM. (*Id*. at 6-7 (citing ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al. eds., 2012) [hereinafter 2012 IOM Report] (Ex. 50, pp. 338-39); AGENCY FOR HEALTHCARE RSCH. & QUALITY, NO. 14-E002-EF, SAFETY OF VACCINES USED FOR ROUTINE IMMUNIZATION IN THE UNITED STATES (2014) (Ex. A, Tab 5);[10] Roger Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis*, 63 CLINICAL INFECTIOUS DISEASES 1456 (2016) (Ex. A, Tab 2) (*see also* Ex. C, Tab 3)).) Regarding Dr. Steinman's molecular mimicry theory, Dr. Merkler acknowledged that neurofascin autoimmunity has been suggested vis-à-vis GBS but disagreed that neurofascin has been implicated in the pathology of TM. (*Id*. at 7 (citing Harald Prüss et al., *Neurofascin as Target of Autoantibodies in Guillain-Barré Syndrome*, 124 BRAIN, 2011, at 1 (Ex. A, Tab 8); Emily K. Mathey et al., *Neurofascin as a Novel Target for Autoantibody-Mediated Axonal Injury*, 204 J. EXPERIMENTAL MED. 2363 (2007) (Ex. A, Tab 7); O. Stich et al., *Prevalence of Neurofascin-155 Antibodies in Patients with Multiple Sclerosis*, 364 J. NEUROLOGICAL SCIS. 29 (2016) (Ex. A, Tab 10)).) He also disagreed that Dr. Steinman's findings supporting molecular mimicry between the Epstein-Barr virus and GlialCAM can be generalized to other molecular exposures. (*Id*.) And, in any event, even if crediting Dr. Steinman's theory, Dr. Merkler did not agree that exposure to vaccination can result in neuroimmunological complication within 24 hours, opining instead that such an injury would take anywhere from five days to weeks to develop. (*Id*. at 6-8 (citing

[9] Dr. Merkler received his medical degree from New York University School of Medicine in 2010. (Ex. B, p. 1.) He completed an internship in internal medicine and a residency in neurology at New York Presbyterian Hospital -Weill Cornell Medical College in 2011 and 2014 respectively, serving as chief resident in his final year. (*Id.*) Thereafter, Dr. Merkler completed a fellowship in neurocritical care at Cornell/Columbia University and earned a Master's Degree in Clinical and Translational Investigation from Cornell University. (*Id.* at 2; Ex. A, p. 1.) He maintains his medical license in New York and is board-certified in neurology with subspeciality certification in neurocritical care. (Ex. B, p. 2; Ex. A, p. 1.) Currently, Dr. Merkler serves as an Assistant Professor of Neurology and Neuroscience at Weill Cornell Medical College and the Brain and Mind Research Institute. (Ex. A, p. 1; Ex. B, p. 2.) Additionally, he serves as an attending neurologist at New York Presbyterian Hospital, where he routinely cares for patients with neurological disorders, including transverse myelitis. (Ex. B, p. 2; Ex. A, p. 1.) Dr. Merkler has co-authored over 100 peer-reviewed publications on neurological disorders. (Ex. B, pp. 14-22; Ex. A, p. 1.)

[10] Some of the articles of medical literature referenced in Dr. Merkler's initial report at Exhibit A were filed by respondent with erroneous bates stamping. For example, the notice of filing designates the report authored by the Agency of Healthcare Research and Quality as Exhibit A, Tab 5 (*see* ECF No. 44-1); however, the bates stamping marks the report as Exhibit C, Tab 5. For the purposes of clarity, this decision refers to the medical literature filed at ECF No. 44 by the exhibit designations reflected on the notice of filing.

Schonberger et al., *supra*, at Ex. 40; Divya Mamootil & Anmol Grewal, *Viral Versus Vaccine-Associated Acute Transverse Myelitis with Neuromyelitis Optica Immunoglobulin G Antibody and Myelin Basic Protein: A Case Report*, 14 CUREUS, 2022, at 1 (Ex. A, Tab 6); Chumpol Anamnart et al., *Newly Diagnosed Neuromyelitis Optica Spectrum Disorders Following Vaccination: Case Report and Systematic Review*, 58 MULTIPLE SCLEROSIS & RELATED DISORDERS, 2022, at 1 (Ex. A, Tab 1); Chris Wu et al., *Case Report: Hemorrhagic Longitudinally Extensive Transverse Myelitis*, CASE REPS. NEUROLOGICAL MED., 2016, at 1 (Ex. A, Tab 12); Marcelo Adriano da Cunha e Silva Vieira et al., *Transverse Myelitis with Brown-Sèquard Syndrome After H1N1 Immunization*, 70 ARQUIVOS DE NEURO-PSIQUIATRIA 555 (2012) (Ex. A, Tab 11); Isabelle Korn-Lubetzki et al., *H1N1 Vaccine-Related Acute Transverse Myelitis*, 13 ISRAEL MED. ASS'N J. 249 (2011) (Ex. A, Tab 4); Li Gui et al., *Acute Transverse Myelitis Following Vaccination Against H1N1 Influenza: A Case Report*, 4 INT'L J. CLINICAL & EXPERIMENTAL PATHOLOGY 312 (2011) (Ex. A, Tab 3)).) Accordingly, he opined that it is more likely than not that petitioner's vaccination was not a causal factor in her development of TM. (*Id*. at 8.)

c. Respondent's initial report by Dr. He[11]

Like Dr. Merkler, Dr. He opined that, although TM can be parainfectious and post-vaccinal TM has been reported, epidemiologic evidence has not indicated that vaccines cause TM. (Ex. C, pp. 5-6 (citing INST. OF MED., *Influenza Vaccine: Transverse Myelitis*, *in* ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY 309-10 (Kathleen Stratton eds., 2012) (Ex. C, Tab 2)[12]; Baxter et al., *supra*, at Ex. C, Tab 3; Matthew Z. Dudley et al., *The State of Vaccine Safety Science: Systematic Reviews of the Evidence*, 20 LANCET INFECTIOUS DISEASES e80 (2020) (Ex. C, Tab 4); Agency for Healthcare Rsch. & Quality, Safety of Vaccines Used for Routine Immunization in the United States, at 1 (unpublished manuscript) (Ex. C, Tab 5)).)[13] He additionally stressed that between 15-30% of cases of TM remain classified as idiopathic after thorough evaluation. (*Id*. at 5 (citing Benjamin Greenberg, *Transverse Myelitis:*

[11] Dr. He received his medical degree in 1986 from The Fourth Military Medical University in Xian, China. (Ex. D, p. 1.) In 1996, he earned his Ph.D. in immunology from the University of Miami School of Medicine. (*Id.*) Thereafter, he served as a senior fellow at the Howard Hughes Medical Institute and the University of Washington, Department of Immunology. (*Id.*) Since completing his fellowship training, Dr. He has held various academic appointments at Duke University School of Medicine. (*Id.*) Currently, he serves as a Professor Integrative Immunobiology in the Department of Integrative Immunobiology and as Director for an advanced immunology course. (*Id.*; Ex. C, p. 1.) Dr. He has been conducting research in immunology since 1986 with a focus on innate and adaptive immunity against viral infections, as well as tumors. (Ex. C, p. 1.) He has co-authored over 100 peer-reviewed publications on immunology. (*Id.* at 2; Ex. D, pp. 7-18.)

[12] Both Dr. Steinman and Dr. He provided as a reference the 2012 IOM report entitled: ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY. (Ex. 50; Ex. C, Tab 2.) Dr. Steinman filed a copy of the entire report whereas Dr. He only filed a section of the report that explicitly focuses on TM and the flu vaccine. (*Compare* Ex. 50, *with* Ex. C, Tab 2.) Therefore, both of these exhibits refer to the same 2012 report.

[13] Some of the docket text accompanying respondent's filing incorrectly identifies Dr. He's initial report as Exhibit A, duplicative of the designation for Dr. Merkler's initial report. However, the exhibit itself is correctly bates stamped as Exhibit C.

Etiology, Clinical Features, and Diagnosis, UPTODATE (last updated Jan. 23, 2024) (Ex. C, Tab 1)).)

Regarding Dr. Steinman's molecular mimicry theory, Dr. He opined that three scientific findings have "strongly challenged" the theory of molecular mimicry: (1) massive sequence sharing between microbial pathogens and human proteome; (2) frequent detection of cross-reactive antibodies and T lymphocytes in humans; and (3) advances in the understanding of host tolerance mechanisms. (Ex. C, p. 7.) Thus, he opined:

> Given these many layers of immune regulatory/tolerant mechanisms, the critical determinant of autoimmune disease development upon antigen stimulation is the strength/ extent of the immune activation induced by the overall immunological encounters (either infections or immunizations), not the molecular mimicry (aka linear sequence or structural homologies) between foreign antigens and self-proteins. On rare occasions, strong immune activation conditions such as certain types of infections and potent immune stimulations can break the many layers of immune regulatory mechanisms to cause autoimmune diseases. However, there is no evidence to support that seasonal influenza vaccine can induce strong immune activation to break immune tolerance to cause TM.

(*Id*. at 11.) Moreover, Dr. He opined that the specific sequence homologies discussed by Dr. Steinman "are entirely expected." (*Id.* at 16.) For example, Kanduc et al. showed that a stretch of six consecutive amino acids in H5N1 influenza virus shares sequence similarity with 2,332 different human proteins. (*Id*. (citing Darja Kanduc et al., *Massive Peptide Sharing Between Viral and Human Proteomes*, 29 PEPTIDES 1755 (2008) (Ex. C, Tab 7, tbl. 6)[14]).) Dr. He criticized the studies by Gautam et al. because they used complete Freund's adjuvant, "a powerful immune adjuvant," which means the molecular mimics "are not working by themselves." (*Id*. at 13 (discussing Gautam et al., *supra*, at Ex. 27; Gautam et al., *supra*, at Ex. 28; Gautam et al., *supra*, at Ex. 29.) When juxtaposed with other studies, such as Lanz et al., Dr. He opined that the evidence shows that "the critical determinant of autoimmune disease development upon antigen stimulation is the strength/extent of the immune activation induced by the overall immunological encounters (either infections or immunizations), not the molecular mimicry (aka linear sequence or structural homologies) between foreign antigens and self-proteins." (*Id*.)

Dr. He also disagreed that 24-hours would be enough time for the immunopathogenesis at issue. (Ex. C, pp. 16-17.) Instead, he opined that it takes at least three days for the innate immune response to develop and seven to ten days for activation of the adaptive immune response following antigen exposure. (*Id*. at 16

[14] Respondent filed the Kanduc et al. article referenced by Dr. He in his report as Exhibit C, Tab 7. However, the filing appears to be incomplete. While the filing suggests that the article includes twelve pages total, only the first four pages of the article are viewable. Pages five through twelve are blank with the exception of the bates stamping.

(citing Matthew A. Williams & Michael J. Bevan, *Effector and Memory CTL Differentiation*, 25 ANN. REV. IMMUNOLOGY 171 (2007) (Ex. C, Tab 29, p. 2 fig. 1); Lina Sun et al., *T Cells in Health and Disease*, 8 SIGNAL TRANSDUCTION & TARGETED THERAPY, 2023, at 1 (Ex. C, Tab 30, p. 5 fig. 3)).) He opined that the Lai et al. study is not informative because it did not examine an in vivo immune response, explaining that

> [t]he fast response to antigen-stimulation by memory T cells is well-established in the immunology field. However, the CD4+ T cell activation in vitro was not subjected to the multiple layers of host regulatory mechanisms as in vivo, therefore, using the in vitro kinetics of memory CD4+ T cell response to make conclusion that memory CD4+ T cells will rapidly attack self-tissue in vivo is scientifically unreliable.

(*Id*. at 17 (discussing Lai et al., *supra*, at Ex. 47).) Dr. He also disagreed with Dr. Steinman's interpretation of the data within the Schonberger et al. study. (*Id*. at 17-18 (discussing Schonberger et al., *supra*, at Ex. 40).)

d. Dr. Steinman's first supplemental report

In response to respondent's experts, Dr. Steinman questioned the value of the Baxter et al. study relative to this case given that the seasonal flu vaccine changes each year. (Ex. 51, pp. 1-2, 4 (discussing Baxter et al., *supra*, at Ex. A, Tab 2).) Although respondent's experts cite Baxter et al. as an example of a study that found no association between the flu vaccine and TM, Dr. Steinman pointed out that the study was published in 2016 and examined the season flu vaccines for 2007 through 2012. (*Id.* at 1-2 (citing Baxter et al., *supra*, at Ex. A, Tab 2, p. 2 tbl. 1).) By contrast, Dr. Steinman noted that his theory is specific to components of the 2019 through 2021 vaccines, which were not contained in the prior years examined by Baxter et al. (*Id*. at 2.) Dr. Steinman suggested the 2012 IOM report likewise could not have addressed a vaccine formulation that did not yet exist at that time. (*Id*. at 2-3 (discussing 2012 IOM Report, *supra*, at Ex. 50).) Dr. Steinman also further defended his interpretation of the Schonberger et al. study as supporting a one-day post-vaccination onset. (*Id*. at 7-9 (discussing Schonberger et al., *supra*, at Ex. 40).) Furthermore, he seemed to characterize the treating physicians' diagnosis of idiopathic transverse myelitis as a failure to consider the vaccine as a potential cause. (*Id*. at 1.)

Noting in particular the positive reception for the 2022 study by Lanz et al., Dr. Steinman does not accept Dr. He's opinion that molecular mimicry is an outdated theory. (Ex. 51, pp. 4-5 (discussing Lanz et al., *supra*, at Ex. 30).) And, whereas Dr. Merkler opined that the findings from Lanz et al. cannot be generalized to other molecular mimics, Dr. Steinman reiterated the view that the Lanz et al. study's findings, especially when coupled with the prior Gautam et al. studies, does support the proposition that five identical amino acids out of a sequence of 12 can trigger clinically relevant neuroinflammation. (*Id*. at 3 (discussing Lanz et al., *supra*, at Ex. 30; Gautam et al., *supra*, at Ex. 27; Gautam et al., *supra*, at Ex. 28; Gautam et al., *supra*, at Ex. 29).) Moreover, Dr. Steinman disagreed with Dr. He's criticism of the Gautam et al. studies'

use of complete Freund's adjuvant, explaining that within the studies some peptides, such as Ac3.5.6 did not induce paralysis despite the use of the adjuvant. (*Id.* at 5-7 (citing Gautam et al., *supra*, at Ex. 27).) Therefore, the use of the adjuvant did not invalidate the findings. (*Id*. at 6-7.)

e. Dr. Merkler's supplemental report

In his second report, Dr. Merkler stressed the validity of a diagnosis of idiopathic TM as "well-established in medical practice and is applied when a thorough investigation fails to identify a known cause." (Ex. E, p. 1 (citing Transverse Myelitis Consortium Working Group, *Proposed Diagnostic Criteria and Nosology of Acute Transverse Myelitis*, 59 NEUROLOGY 499 (2002) [hereinafter TM Consortium] (Ex. E, Tab 3)).) Idiopathic TM accounts for as many as 19% of patients diagnosed with TM. (*Id*. (citing Olwen C. Murphy et al., *Identification of Specific Causes of Myelopathy in a Large Cohort of Patients Initially Diagnosed with Transverse Myelitis*, 442 J. NEUROLOGICAL SCIS., 2022, at 1 (Ex. E, Tab 1)).) The diagnosis of idiopathic TM is correct in this case because "all other possible etiologies for which there are diagnostic tests approved for human use by the FDA, including infections, and autoimmune diseases were ruled out based on [the] available clinical data." (*Id*.) Although Dr. Merkler does not dispute Dr. Steinman's observation that the Baxter et al. study and 2012 IOM report pre-dated the vaccination at issue in this case, he opined that these publications remain informative of the broader understanding of the potential relationship between vaccines and TM. (*Id*. (discussing Baxter et al., *supra*, at Ex. A, Tab 2; 2012 IOM Report, *supra*, at Ex. 50).) Dr. Merkler stressed that he does not disparage the molecular mimicry hypothesis, but he maintained that it "is an interesting hypothetical mechanism and does not establish causality in this case without robust supporting epidemiological evidence to confirm it." (*Id*. at 2.) And, while Dr. Merkler acknowledged that some autoimmune phenomena can develop rapidly in rare cases, "the latency period for the onset of transverse myelitis after an immunological trigger is longer than 24 hours." (*Id*.) He opined that isolated case reports of TM occurring in such a timeframe do not imply causation. (*Id*.)

f. Dr. He's supplemental report

In response to Dr. Steinman's assertion that the Baxter et al. study and 2012 IOM report are not informative of the specific molecular mimics he identified, Dr. He responded that "no matter how many homologies (aka, molecular mimics) can be found between the 2021 influenza vaccine and host proteins, mere sequence homologies will not cause autoimmune diseases as the IOM clearly stated."[15] (Ex. F, p. 2.) Dr. He indicated that

> [T]he IOM committee has unequivocally stated that "Both naturally occurring and postinfectious cross-reactive antibodies and T cells are

[15] Dr. He also noted that the Dudley et al. study cited in his initial report was from 2020. (Ex. F, p. 2 (citing Dudley et al., *supra*, at Ex. C, Tab 4).)

> relatively common and most frequently not pathogenic. Cross-reacting antibodies can also be secondary to nonspecific tissue injury rather than involved in primary injury to the tissue. Moreover, in some circumstances, infection with viruses that express antigens having immunologic cross-reactivity with self-proteins can actually protect against autoimmune disease in certain animal models."

(*Id*. at 2-3 (quoting 2012 IOM Report, *supra*, at Ex. 50, pp. 99-100).)

Thus, Dr. He reiterated his view that autoimmune disease development is explained by the strength and extent of the immune response at issue rather than the presence of molecular mimicry. (Ex. F, p. 3.) He opined that Dr. Steinman's opinion lacks "a balanced view on the ying and yang force of the host immune system." (*Id.*) Dr. He clarified that there is no argument as to the validity of the Gautam et al. studies, but reiterated that these studies underscore the need for a strong immune activation to break immune tolerance. (*Id*. at 4 (discussing Gautam et al., *supra*, at Ex. 27; Gautam et al., *supra*, at Ex. 28; Gautam et al., *supra*, at Ex. 29).) Moreover, these studies are mostly focused on the innate immune system. (*Id*.)

Dr. He also maintained that, even accounting for the presence of a recall response, the timeframe between petitioner's vaccination and onset of her TM is too short for a vaccine-induced activation of innate and adaptive immune responses leading to attack of neural tissue. (Ex. F, p. 5.) While the innate immune response is fully functional within three days, it takes the adaptive immune response at least seven days to be fully activated. (*Id*. (citing Richard Warrington et al., *An Introduction to Immunology and Immunopathology*, 7 ALLERGY ASTHMA & CLINICAL IMMUNOLOGY, 2011, at 1 (Ex. F, Tab 1);[16] Williams & Bevan, *supra*, at Ex. C, Tab 29; Bali Pulendran & Rafi Ahmed, *Immunological Mechanisms of Vaccination*, 12 NATURE IMMUNOLOGY 509 (2011) (Ex. C, Tab 24); Michael J. Carter et al., *The Antibody-Secreting Cell Response to Infection: Kinetics and Clinical Applications*, 8 FRONTIERS IMMUNOLOGY, 2017, at 1 (Ex. F, Tab 2)).) Moreover, antigen-specific B cells do not become antibody secreting cells until at least 8 days after a primary vaccination and at least five days after a secondary vaccination. (*Id*. at 5-6 (citing Carter et al., *supra*, at Ex. F, Tab 2, p. 9 fig. 2).)[17] A recall response does not develop until day four post-vaccination and both B cells and antigen-specific T cells take at least five days to activate and expand. (*Id.* at 5 (citing Carter et al., *supra*, at Ex. F, Tab 2; Williams & Bevan, *supra*, at Ex. C, Tab 29).) Thus, according to Dr. He, it is "impossible" for T and B cells to be activated within two days of a flu vaccination even in the context of a recall response. (*Id.*) Dr. He disagreed again

[16] In his report, Dr. He's bibliography indicates that reference 4 refers to: J.S. Marshall et al., *An Introduction to Immunology and Immunopathology*, 14 ALLERGY ASTHMA & CLINICAL IMMUNOLOGY 49 (2018). (*See* Ex. F, p. 8.) However, this decision cites the article respondent actually filed. (*See* Ex. F, Tab 1.)

[17] Figure two is a line graph comparing the relative frequency of antigen-specific antibody secreting cells by day as among bacterial and viral infections, primary vaccination, and secondary vaccination. (Carter et al., *supra*, at Ex. F, Tab 2, p. 9 fig. 2.) The figure reflects that only the line for bacterial and viral infection rises above the x-axis before day four. (*See id.*)

with Dr. Steinman's interpretation of the Schonberger et al. data and stressed that Schonberger et al. examined GBS rather than TM. (*Id.* (discussing Schonberger et al., *supra*, at Ex. 40).)

g. Dr. Steinman's second supplemental report

In his final report, Dr. Steinman stressed that respondent's suggestion that petitioner's TM is idiopathic – which he characterized as a euphemism for "I don't know" – must be balanced against his presentation of a causal explanation that should be considered sound and reliable. (Ex. 52, p. 1.) And, regarding Dr. Merkler's emphasis on epidemiology, he indicated that it would not be realistic to expect any epidemiology to be available given the recency of the specific flu vaccine formulations at issue. (*Id*.) He charged that Dr. Merkler seeks scientific certainty. (*Id*. at 1-2.) In that regard, he cited an additional study as validation of the findings of Lanz et al. (*Id*. at 2 (citing Neda Sattarnezhad et al., Antibody Reactivity Against EBNA1 and GlialCAM Differentiates Multiple Sclerosis Patients from Healthy Controls (Jan. 5, 2025) (unpublished manuscript) (Ex. 53))[18].)

Dr. Steinman agreed with Dr. He's statement that the immune system involves "ying and yang" forces. (Ex. 52, p. 4.) However, in response to Dr. He's statement that "mere sequence homologies will not cause autoimmune disease," Dr. Steinman indicated

> Petitioner's theory goes far beyond showing "mere sequence homologies". The hierarchical search describes regions in the vaccine that have met the criteria of four peer reviewed papers, the three Gautam papers and the Lanz paper, and now a fifth paper cited here as Supplementary Reference 1. In addition, the region in the vaccine has been studied by others and is described extensively in Petitioner's reports.

(*Id*. at 3.)

Dr. Steinman also disagreed with Dr. He's citation to Carter et al. (Ex. 52, p. 4.) According to Dr. Steinman, the chart depicted in Dr. He's report, though it comes from the Carter et al. paper, does not actually reflect the data generated by the study. (*Id*. (discussing Carter et al., *supra*, at Ex. F, Tab 2, p. 9 fig. 2).) In fact, citing Table 1 within the study, he observed that in most instances measurements were not even taken until day 7. (*Id*. at 4-5 (citing Carter et al., *supra*, at Ex. F, Tab 2, pp. 4-7 tbl. 1).) Dr. Steinman reiterated his reliance on the Schonberger et al. and Park et al. papers. (*Id*. at 5 (citing Schonberger et al., *supra*, at Ex. 40; Park et al., *supra*, at Ex. 41).)

[18] The manuscript was subsequently published in March of 2025. *See* Neda Sattarnezhad et al., *Antibody Reactivity Against EBNA1 and GlialCAM Differentiates Multiple Sclerosis Patients from Healthy Controls*, 10 PNAS, 2025, at 1. However, petitioner did not file the publication as an exhibit in this case.

V. Analysis

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," showing that the subject vaccine can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006)). Such a theory need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *See Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [the proposed causal] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (citation omitted) (first quoting *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010); then quoting *Knudsen*, 35 F.3d at 548-49). That is, although petitioners are not required to prove their theories in any one way, they must in all events support their proffered theory with sound and reliable scientific explanation, *Boatmon*, 941 F.3d at 1359, and provide evidence that establishes on balance that the vaccine at issue can more likely than not cause the injury at issue, *Cerrone v. Sec'y of Health & Human Servs.*, 146 F.4th 1113, 1120-23 (Fed. Cir. 2025).[19]

In his motion, respondent argues that relying on the concept of molecular mimicry broadly would be too generic to support a theory of causation.[20] (ECF No. 45,

[19] In the motion papers, the parties debated whether a plausible theory of causation is sufficient to meet petitioner's burden of proof under *Althen* prong one (ECF No. 45, p. 11; ECF No. 47, pp. 7-11), with respondent ultimately arguing in his reply brief that the Federal Circuit resolved the question in *Cerrone*, which was issued subsequent to the filing of petitioner's brief (ECF No. 50, pp. 1-2 (citing *Cerrone*, 146 F.4th at 1121 n.3)). I agree with respondent, but I also stress that *Cerrone* did not announce a new standard. As respondent indicated in his initial brief (ECF No. 45, p. 11), the Federal Circuit has long held that a plausible theory of causation is insufficient in this program. *Cerrone*, 146 F.4th at 1121 (emphasizing that "we have repeatedly stated that 'simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof.'" (quoting *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014))).

[20] Consistent with what respondent argues here, prior cases have explained that molecular mimicry is "a generally accepted scientific principle," but "mere invocation of the scientific term does not carry a petitioner's burden in a Program case." *Deshler v. Sec'y of Health & Human Servs.*, No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec'y of Health & Human Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019)). In particular, identifying sequence homology is not enough standing alone because, as Dr. He stressed, being able to identify a homology "does not necessarily mean the similarity has significance to the immune system." *Tullio v. Sec'y of Health & Human Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *aff'd*, 149 Fed. Cl. 448 (2020); *see also Caredio ex rel. D.C. v. Sec'y of Health & Human Servs.*,

pp. 12-13.) Instead, petitioner must show that it applies to the specific vaccination and condition at issue. (*Id*. (citing *W.C. v. Sec' of Health & Human Servs*., 704 F.3d 1352, 1360 (Fed. Cir. 2013)).) In this case, however, no scientific studies are available that associate the flu vaccine with TM. (*Id*. at 13-15.) Moreover, respondent argues that Dr. Steinman's theory is speculative. (*Id*. at 15-17.) In light of Dr. He's opinion, respondent argues that an immune response to vaccination cannot be equated to an infection-induced immune response and, therefore, Dr. Steinman's reliance on papers such as Lanz et al. and Bjornevik et al. that study EBV infection as a cause of MS is misplaced. (*Id*. at 15-16 (discussing Lanz et al., *supra*, at Ex. 30; Kjetil Bjornevik et al., *Longitudinal Analysis Reveals High Prevalence of Epstein-Barr Virus Associated with Multiple Sclerosis*, 375 SCIENCE 296 (2022) (Ex. 31)).) Dr. Steinman's use of BLAST to compare neurofascin and influenza hemagglutinin is otherwise insufficient without more. (*Id*. at 16-17.)

In response, petitioner stresses that, though medical literature is valuable, petitioner is permitted to rely on circumstantial evidence and is not obligated to demonstrate scientific certainty, especially given the rarity of the condition at issue. (ECF No. 47, pp. 11-12.) Petitioner contends that in the search for medical proof, respondent's experts impermissibly "downplay" Dr. Steinman's opinion. (*Id.* at 11.) Petitioner notes that prior petitioners have been found entitled to compensation for TM resulting from various vaccinations.[21] (*Id*. at 12.) TM is known to be an autoimmune inflammatory condition affecting the spinal cord that can be post-infectious and, moreover, the National Institute of Neurological Disorders and Stroke ("NINDS") recognizes that TM may also present as a post-vaccination phenomenon. (*Id*. at 13 (citing *Transverse Myelitis*, NAT'L INST. NEUROLOGICAL DISORDERS & STROKE, https://www.ninds.nih.gov/health-information/disorders/transverse-myelitis (last reviewed Nov. 28, 2023) (Ex. 14)).) Petitioner challenges the notion that the available epidemiology is informative. (*Id*. at 13-17.) Describing the steps included in Dr. Steinman's reports, petitioner endorses Dr. Steinman's reliance on molecular mimicry to explain how the flu vaccine can cause TM (*Id*. at 17-23), and she cites prior cases in

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No. 17-0079V, 2021 WL 4100294, at *31 (Fed. Cl. Spec. Mstr. July 30, 2021) ("[D]emonstration of homology alone is not enough to establish a preponderant causation theory." (emphasis omitted) (citing *Schultz v. Sec'y of Health & Human Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020))), *mot. for rev. denied*, No. 17-79V, 2021 WL 6058835 (Fed. Cl. Dec. 3, 2021). Thus, for example, in *Brayboy*, an omnibus proceeding addressing autoimmune premature ovarian insufficiency, the special master found it sufficient that the petitioners had, more robustly, "identified cross-reaction between components of the vaccine and proteins in the body that are directly responsible for the health and productivity of the organ at issue," while also noting that further requiring additional steps, or insisting on direct, testable evidence would impermissibly heighten petitioner's burden of proof. *Brayboy v. Sec'y of Health & Human Servs.*, No. 15-183V, 2021 WL 4453146, at *19 (Fed. Cl. Spec. Mstr. Aug. 30, 2021). Ultimately, when assessing theories based on molecular mimicry in light of petitioner's preponderant burden of proof, "[t]he line must be drawn somewhere between speculation and certainty." *Id*.

[21] Specifically, petitioner cites *White v. Secretary of Health & Human Services*, No. 15-1521V, 2019 WL 7563239 (Fed. Cl. Spec. Mstr. Dec. 19, 2019) (finding the HPV vaccine caused TM) and *Le v. Secretary of Health & Human Services*, No. 16-1078V, 2023 WL 3049203 (Fed. Cl. Spec. Mstr. Mar. 30, 2023) (finding the Tdap vaccine caused TM). (ECF No. 47, p. 12.)

which Dr. Steinman's use of BLAST and citation to the Gautam et al. papers have been accepted (*Id.* at 23).[22] She is critical of respondent's experts' competing opinions. (*Id*. at 24-33.)

It is not necessary to resolve the parties' competing positions on molecular mimicry more broadly. Even crediting the applicability of molecular mimicry to TM and accepting arguendo Dr. Steinman's use of BLAST and his reliance on the Gautam et al., Lanz et al., and Bjornevik et al. papers, I am not persuaded as a threshold matter that petitioner has preponderantly demonstrated that TM can be explained by an autoimmune attack on neurofascin, as Dr. Steinman's proffered theory requires.[23] Absent Dr. Steinman's specific molecular mimicry theory, the evidence on this record, including the additional literature petitioner filed in connection with her motion response, is not otherwise sufficient to meet petitioner's preponderant burden of proof under *Althen* prong one. Ultimately, Dr. Steinman cited four publications to support neurofascin as found in the Node of Ranvier as having been implicated in TM. (Ex. 8, pp. 8-9, 22 (citing Rasband & Peles, *supra*, at Ex. 17; Guo & Zhang, *supra*, at Ex. 18; Kira et al., *supra*, at Ex. 19; Jia et al., *supra*, at Ex. 20).) However, although these papers do support the general idea that neurofascin may be affected by demyelination in the central nervous system, there are several reasons why they do not readily support Dr. Steinman's theory in this case.

First, contrary to what Dr. Steinman suggests, none of these papers invoke an attack against neurofascin as an explanation specifically for the pathogenesis of acute monophasic TM as is at issue in this case. One case series does discuss an autoimmune attack against the Nodes of Ranvier as a potential explanation for "GBS/ATM overlap syndrome;" however, that case report does not seek to implicate neurofascin antibodies. (Guo & Zhang, *supra*, at Ex. 18, pp. 4-5.) Instead, they note only that three of the subjects tested positive for anti-ganglioside antibodies. (*Id*. at 2.) Rather, the literature cited by Dr. Steinman as a whole generally discusses neursofasin antibodies in the context of chronic conditions including MS, NMOSD, and CIDP.

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[22] Specifically, petitioner cites *Pasco v. Secretary of Health & Human Services*, No. 16-500V, 2022 WL 6616736 (Fed. Cl. Spec. Mstr. Sept. 23, 2022); *Mullins v. Secretary of Health & Human Services*, No. 19-320V, 2024 WL 4045424 (Fed. Cl. Spec. Mstr. Aug. 8, 2024); *Gardner v. Secretary of Health & Human Services*, No. 17-1851V, 2023 WL 9288070 (Fed. Cl. Spec. Mstr. Dec. 21, 2023); *White*, 2019 WL 7563239. (ECF No. 47, p. 23.)

[23] Of course, petitioner is not obligated to come forward with a specific biologic mechanism to meet her burden of proof. *Kottenstette v. Sec'y of Health & Human Servs*., 861 F. App'x 433, 440-41 (Fed. Cir. June 15, 2021) (citing *Knudsen*, 35 F.3d at 549 and *Simanski v. Sec'y of Health & Human Servs.*, 671 F.3d 1368, 1384 (Fed. Cir. 2012)). However, because she does seek to meet her burden of proof under *Althen* prong one at least in part via presentation of such a mechanism – in this case, molecular mimicry – the soundness of that assertion must be assessed. *E.g*., *Howard v. Sec'y of Health & Human Servs*., No. 16-1592V, 2022 WL 4869354, at *24 (Fed Cl. Spec. Mstr. Aug. 31, 2022) ("[P]etitioners are never required to establish [a] mechanism—but they often attempt to do so, and therefore it is reasonable to evaluate their success in the effort."), *mot. for rev. denied sub nom.*, *Howard v. United States*, No. 16-1592V, 2023 WL 4117370 (Fed. Cl. May 18, 2023), *aff'd per curiam sub nom.*, *Howard v. Sec'y of Health & Human Servs.*, No. 2023-1816, 2024 WL 2873301 (Fed. Cir. June 7, 2024).

Second, even for those conditions that are more explicitly discussed, this literature characterizes a role for neurofascin antibodies as merely an "emerging" area of research (Kira et al., *supra*, at Ex. 19, p. 6; *see also* Jia et al., *supra*, at Ex. 20, p. 3 (noting that "further studies are needed to elucidate the pathogenesis of NMOSD in relation to the presence of antibodies against . . . [neurofascin]155")), undercutting the suggestion that an autoimmune attack against neurofascin is an accepted cause of any condition, let alone TM by extension. For example, Dr. Merkler noted on respondent's behalf that a later study by Stich et al. was unable to find a difference in the prevalence of neurofascin 155 antibodies as between MS patients and controls. (Ex. A, p. 7 (discussing Stich et al., *supra*, at Ex. A, Tab 10, p. 3).) Indeed, Dr. Steinman initially raised an attack against neurofascin because he had noted that petitioner tested negative for antibodies against two targets more commonly associated with TM, MOG and AQP-4. (Ex. 8, pp. 8-9.)

Third, the literature suggests that anti-neurofascin antibodies, as antibodies against paranodal proteins, "are associated with unique features and these conditions are collectively termed nodopathy or paranodopathy." (Kira et al., *supra*, at Ex. 19, p. 1.) Indeed, these papers note that injury to neurofascin is viewed as a potential explanation for conditions that combine both peripheral and central nervous system damage. (Jia et al., *supra,* at Ex. 20, p. 1; Kira et al., *supra*, at Ex. 19, pp. 3, 6.) Thus, for example, Dr. Merkler opined that neurofascin is more likely to be important to GBS or CIDP than to TM. (Ex. A, p. 7.) Even Dr. Steinman's own report suggests that neurofascin is more readily implicated in the pathogenesis of GBS. (Ex. 8, p. 22 (stating that "[i]mmunity to neurofascin is critical to the triggering of inflammatory neuropathy (GBS), and in some cases transverse myelitis . . .").)

Prior petitioners have been compensated for post-flu vaccine TM. *E.g.*, *Jane Doe 93 v. Sec'y of Health & Human Servs.*, No. [redacted], 2011 WL 2326966 (Fed. Cl. Spec. Mstr. May 9, 2011); *J.S. v. Sec'y of Health & Human Servs*., No. 14-851V, 2018 WL 11731139 (Fed. Cl. Spec. Mstr. Apr. 9, 2018); *Songero v. Sec'y of Health & Human Servs*., No. 18-300V, 2025 WL 3013090 (Fed. Cl. Spec. Mstr. Oct. 3, 2025). However, given the cause-in-fact context, petitioners do not always succeed in demonstrating that the flu vaccine can cause TM. *E.g.*, *Caves v. Sec'y of Health & Human Servs*., No. 07-443V, 2010 WL 5557542 (Fed. Cl. Spec. Mstr. Nov. 29, 2010), *mot. for rev. denied*, 100 Fed. Cl. 119 (2011), *aff'd per curiam*, 463 F. App'x 932 (Fed. Cir. 2012); *Bowling v. Sec'y of Health & Human Servs*., No, 18-109V, 2023 WL 6846491 (Fed. Cl. Spec. Mstr. Sept. 20, 2023); *Johnson v. Sec'y of Health & Human Servs.*, No. 18-410V, 2025 WL 1942989 (Fed. Cl. Spec. Mstr. June 17, 2025). Moreover, the undersigned is not aware of any prior TM case in which a petitioner has presented the specific neurofascin-based molecular mimicry theory advanced in this case.

A limited number of prior cases discuss neurofascin, but they all involved conditions other than TM and, moreover, often rejected or did not reach the question of whether molecular mimicry against neuorfascin was preponderantly supported. In fact, consistent with the above, these cases mostly involved peripheral nerve disorders. *Davis v. Sec'y of Health & Human Servs.*, No. 14-978V, 2022 WL 1654743, at *33 (Fed.

Cl. Spec. Mstr. Apr. 27, 2022) (alleging the flu vaccine caused CIDP in which one of the theories presented by Dr. Steinman involved molecular mimicry between a component of the flu vaccine and contactin-1 and neurofascin; however, the special master did not reach any conclusions about this theory as there was no record evidence of the presence of either contactin-1 or neurofascin in petitioner); *H.C. v. Sec'y of Health & Human Servs.*, No. 16-4V, 2022 WL 2825395, at *28 (Fed. Cl. Spec. Mstr. May 9, 2022) (alleging the flu vaccine caused Ramsay Hunt syndrome in which Dr. Zamvil proposed homology between flu A hemagglutinin and contactin-associated protein and/or neurofascin, which was ultimately rejected); *Gatto v. Sec'y of Health & Human Servs.*, No. 21-924V, 2025 WL 972817, at *6-7, 20-21 (Fed. Cl. Spec. Mstr. Feb. 28, 2025) (alleging that the meningococcal vaccine caused GBS in which Dr. Steinman identified homology between the diphtheria toxin and contactin and neurofascin; however, the Chief Special Master rejected Dr. Steinman's theory); *Borgelt v. Sec'y of Health & Human Servs*., No. 23-1051V, 2026 WL 267899, at *20-21 (Fed. Cl. Spec. Mstr. Jan. 5, 2026) (finding that the record did not preponderantly support petitioner's theory that the flu vaccine can worsen anti-neurofascin CIDP). In one prior case, Dr. Steinman presented multiple proposed homologies, including against neurofascin, to explain how a vaccine can cause ADEM, a central nervous system demyelinating disorder. *Mullins v. Sec'y of Health & Human Servs.*, No. 19-320V, 2024 WL 4045424 (Fed. Cl. Spec. Mstr. Aug. 8, 2024). However, although the petitioner in that case was found entitled to compensation, molecular mimicry against neurofascin was not isolated as being dispositive of the special master's *Althen* prong one analysis and, in fact, the special master explicitly remarked that she would have reached the conclusion that vaccines can cause ADEM even if petitioner has not presented any of the multiple examples of potential homology. *Id*. at *45; *see also Byrd v. Sec'y of Health & Human Servs*., No. 20-1476V, 2024 WL 4003061 (Fed. Cl. Spec. Mstr. July 8, 2024) (similarly finding petitioner entitled to compensation for GBS caused by the pneumococcal vaccine where multiple molecular mimics were proposed, including one against neurofascin).

In light of all of the above, petitioner has not presented preponderant evidence on this record of a sound and reliable medical theory that demonstrates that the flu vaccine can cause TM.

a. *Althen* prong three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* at 1281. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id*.; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *mot. for recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd per curiam*, 503 F. App'x 952 (Fed. Cir. 2013);

Koehn v. Sec'y of Health & Human Servs., No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied sub nom.*, *C.K. v. Sec'y of Health & Human Servs.*, 113 Fed. Cl. 757 (2013), *aff'd sub nom.*, *Koehn v. Sec'y of Health & Human Servs.*, 773 F.3d 1239 (Fed. Cir. 2014).

Here, there is no dispute that petitioner's TM developed as little as 24 hours after the vaccination at issue.[24] (ECF No. 45, p. 20; ECF No. 47, p. 38.) Moreover, based on the medical history discussed above, I conclude that the evidence preponderates in favor of a finding that onset of petitioner's TM occurred approximately 24-hours post-vaccination. (Ex. 4, pp. 159, 683; Ex. 5, pp. 1-2.) What remains controversial is whether molecular mimicry can manifest a neurologic injury such as TM within 24 hours.

Respondent argues the timing is too short. (ECF No. 45, pp. 20-21.) He contends that Dr. He persuasively explained that a 24-hour latency is inconsistent with the multi-step innate and adaptive immune responses necessary to explain how molecular mimicry would result in TM. (*Id.* at 20.) He additionally argues that Dr. Steinman's reliance on the Schonberger et al. study is unpersuasive. (*Id*.) Petitioner argues that the short latency can be explained by a recall response. (ECF No. 47, pp. 37-40.) In particular, petitioner notes Dr. Steinman's reliance on the Lai et al. study to demonstrate that CD4 T cells respond to antigen within 6 hours. (*Id.* at 39.) Additionally, petitioner observes that Dr. Steinman cited the Schonberger et al. study to demonstrate that a one-day onset has been seen in humans and not merely in animal model studies. (*Id*. at 39-40.)

For purposes of this discussion under *Althen* prong three, despite having concluded that petitioner has not met her burden under *Althen* prong one, I assume arguendo that a recall response to vaccination could result in the relevant molecular mimicry.[25] Granting petitioner that premise, petitioner has not preponderantly

[24] In their briefs, the parties discuss the onset as being "within 24 to 48 hours." (ECF No. 45, p. 20; ECF No. 47, p. 38.) This characterization is based on Dr. Steinman's couching of the timing issue. However, based on the positions staked out by the experts, the distinction between a 24-hour onset and a 48-hour onset is immaterial. Dr. Steinman has opined that a 24-hour onset is biologically plausible without distinguishing a 48-hour onset as being any more plausible and respondent's experts have, conversely, opined that anything less than three or four days is implausible.

[25] In his motion, respondent also raises within his *Althen* prong three argument a comment by Dr. He that, if Dr. Steinman's hypothesis were correct, then the Baxter et al. study would have detected many more cases of post-vaccination TM due to immune recall response. (ECF No. 45, p. 20 (discussing Ex. C, p. 19).) Petitioner then devoted significant attention to this issue, arguing that the Baxter et al. data can be viewed in such a way as to buttress Dr. Steinman's view. (ECF No. 47, pp. 41-43 (discussing Baxter et al., *supra*, at Ex. C, Tab 3; Baxter et al., Supplemental Tables 1 & 2: Relative Risk of Transverse Myelitis in the 2-42 Day Risk Interval Following Vaccines, Compared to Remainder of the 9 Months Post Vaccination (Ex. 58) [supplement to Ex. C, Tab 3]).) However, Dr. He's assertion speaks to his overall view that an immune response to vaccination would not result in autoimmunity. (Ex. C, pp. 17-18.) Although Dr. He's comment was included under the heading "Timing," I take Dr. He to be stating that the Baxter et al. study provides evidence that a recall response is no more likely than a primary immune response to result in autoimmunity, which is not a question that bears directly on whether a recall response would accelerate the timing of onset relative to a primary response. I do not find it necessary to resolve this question. Nor do I find petitioner's reinterpretation of the Baxter et al. data to be persuasive.

demonstrated that molecular mimicry can cause a neurologic injury within 24-hours, even in the context of a recall response. Thus, petitioner cannot meet her burden of proof under *Althen* prong three for reasons entirely independent of her failure to meet her burden of proof under *Althen* prong one. Indeed, the timing of onset is the biggest issue in this case and would be fatal to petitioner's case even if she met her burden of proof under *Althen* prong one.

Dr. He is persuasive in explaining that even in the context of a recall response, the overall time needed for the complete immune response to develop is days, not hours. (Ex. F, pp. 5-6.) Although Dr. Steinman questioned whether a particular graph cited by Dr. He was supported by underlying study data (Ex. 52, pp. 4-5 (discussing Carter et al., *supra*, at Ex. F, Tab 2, p. 9 fig. 2)), he did not actually refute Dr. He's assertion as a matter of basic immunologic understanding. In fact, Dr. Steinman described the graph at issue as "idealized," not incorrect. (*Id*. at 5.) But in any event, Dr. He cited several other publications for the proposition that even a recall response takes several days to fully develop. (*E.g.*, Warrington et al., *supra*, at Ex. F, Tab 1; Williams & Bevan, *supra*, at Ex. C, Tab 29; Pulendran & Ahmed, *supra*, at Ex. C, Tab 24.) Moreover, Dr. Steinman himself presented the complete 2012 IOM report on adverse effects of vaccines, which indicates that a recall response reduces the time for the initial lag phase of an immune response from 4-7 days for a primary response down to 1-3 days, meaning that the recall response does not begin to build until after that initial period of at least 24 hours. (2012 IOM Report, *supra*, at Ex. 50, p. 87.) Dr. Steinman's counter presentation seeking to show that a recall response can nonetheless result in disease more rapidly is based primarily on three sources of evidence, none of which is persuasively presented.[26]

First, Dr. Steinman relies on papers by Lai et al. and Bartholomäus et al. to demonstrate that CD4 T cells can respond to antigen within six hours and enter the central nervous system within one to two days. (Ex. 8, pp. 32, 34-35 (citing Lai et al., *supra*, at Ex. 47; Ingo Bartholomäus et al., *Effector T Cell Interactions with Meningeal Vascular Structures in Nascent Autoimmune CNS Lesions*, 46 NATURE 94 (2009) (Ex. 49)).) The fact that the Lai et al. study shows an initial T cell response occurring within six hours is not disputed; however, Dr. He persuasively notes that this finding alone is not informative. (Ex. C, pp. 16-17.) The fact that T cells respond at six hours within an in vitro experiment does not necessarily speak to how long it would take these cells to respond and meaningfully develop in vivo, given the overall interplay of forces that make up the human immune system. (*Id*. at 17 (discussing Lai et al., *supra*, at Ex. 47).) Moreover, even taking the Lai et al. finding at face value, the time it takes T cells to initially respond to an antigen is only the first step in what Dr. He explains to be a multi-

[26] In reaching this conclusion, I have also considered the fact that petitioner's treating neurologist, Dr. Sengul, opined that petitioner's TM was provoked by her vaccination. (Ex. 4, p. 188.) Although Dr. Sengul's opinion is primarily discussed under *Althen* prong two, the same reasoning applies with respect to *Althen* prong three. Even assuming arguendo that Dr. Sengul was opining with specific respect to a 24-hour onset, the available evidence is limited to the mere fact that Dr. Sengul was willing to attribute petitioner's TM to vaccination. (*See id.* at 184-89.) Without more, it cannot be said that Dr. Sengul's opinion on this question would be any more reliable than Dr. Steinman's.

step process. (*Id.*) An initial T cell response occurring in as little as six hours does not imply that the full immune response develops within that timeframe, let alone that it would result in the manifestation of neurologic injury within that timeframe. Dr. Steinman cited a prior case in which another special master credited his reliance on the Lai et al. study. (Ex. 8, p. 35 (discussing *E.M. v. Sec'y of Health & Human Servs*., No. 14-753V, 2021 WL 3477837 (Fed. Cl. Spec. Mstr. July 9, 2021).) However, in a prior case I rejected a petitioner's reliance on the Lai et al. study as support for a rapid onset of small fiber neuropathy because it showed only that T cells can respond to antigen challenge within a matter of hours, which is not equivalent to showing that the immune response can cause neurologic injury in that timeframe. *McGill v. Sec'y of Health & Human Servs*., No. 15-1485V, 2023 WL 3813524, at *35-36 (Fed. Cl. Spec. Mstr. May 11, 2023).)

Dr. Steinman also discussed a study by Bartholomäus et al. as further demonstrating that experimental autoimmune encephalomyelitis studies have shown that the first T cells sensitized to myelin enter the central nervous system between one to two-and-a-half days after transfer. (Ex. 8, p. 35 (citing Bartholomäus et al., *supra*, at Ex. 49).) However, it is explicitly noted that this occurred "before onset" of the EAE. (Bartholomäus et al., *supra*, at Ex. 49, p. 1.) Dr. Steinman stressed that "an experimental animal would not be able to share with an investigator subtle neurologic symptoms in a manner that petitioner did." (Ex. 8, p. 35.) However, I rejected this reasoning in a prior decision, explaining that: "While Dr. Steinman's point may be logical, it is inherently speculative. The study's finding was that clinical symptoms manifested at 3 days. The fact that mice cannot report subjective complaints is a limitation of the study, not evidence that onset was earlier than actually observed." *Greenslade v. Sec'y of Health & Human Servs*., No. 14-1140V, 2024 WL 3527665, at *39 (Fed. Cl. Spec. Mstr. June 28, 2024).

Second, Dr. Steinman has also put forward the idea that tuberculin tests, which can be read as positive within 24 hours, further demonstrate that antigen-specific recall responses can occur within that timeframe. (Ex. 8, p. 32 (citing Serane & Kothendaraman, *supra*, at Ex. 43; Fan et al., *supra*, at Ex. 44; Kardjito & Grange, *supra*, at Ex. 45).) However, tuberculin tests detect a hypersensitivity response, which is not informative of the type of immune reaction Dr. Steinman otherwise places at issue. (Williams & Bevan, *supra*, at Ex. C, Tab 29; Warrington et al., *supra*, at Ex. F, Tab 1.) I have explained in prior cases that these tests are not informative of the type of immune response involved in a recall response resulting in molecular mimicry and autoimmunity. *Defenza v. Sec'y of Health & Human Servs*., No. 18-1601V, 2026 WL 473297, at *8 (Fed. Cl. Spec. Mstr. Jan. 7, 2026), *mot. for rev. denied*, 2026 WL 1742021 (Fed. Cl. May 29, 2026), *appeal docketed*, No. 2026-2053 (Fed. Cir. July 20, 2026); *see also Greenslade*, 2024 WL 3527665, at *39 (similarly noting that respondent's expert had refuted that tuberculin tests are informative of the timing of a recall response and that Dr. Steinman had not adequately explained the relevance of the cited papers).

Third, Dr. Steinman relies on two epidemiologic studies – Schonberger et al. and Park et al. – for the proposition that vaccinations have led to neurologic injury in humans

within one day. As a threshold matter, respondent's experts reasonably question Dr. Steinman's reliance on these papers because they pertain to GBS rather than TM. (*E.g.*, Ex. A, pp. 7-8; Ex. F, p. 5.) Although both conditions can be demyelinating and epidemiologic evidence pertaining to GBS has been considered in prior cases in assessing the time needed for autoimmunity to develop more broadly, TM is a central nervous system condition whereas GBS is a peripheral nerve disorder. This distinction should be more carefully considered when assessing edge cases with respect to the minimum time needed for disease to develop. Indeed, though not dispositive, it is notable that there is no direct evidence on this record that TM can develop in as little as 24 hours.

But in any event, even accepting these studies as some evidence pertaining to TM, Dr. Steinman is not persuasive in suggesting that these two studies meaningfully support a causal relationship between the flu vaccine and GBS occurring just a single day post-vaccination. As with the Lai et al. study, Dr. Steinman cited a prior case in which another special master credited his reliance on the Schonberger et al. paper with respect to a one-day post-vaccination onset. (Ex. 8, pp. 35-36 (discussing *Quackenbush-Baker v. Sec'y of Health & Human Servs*., 14-1000V, 2018 WL 1704523 (Fed. Cl. Spec. Mstr. Mar. 14, 2018).) However, in a prior case I addressed Dr. Steinman's reliance on both the Schonberger et al. and Park et al. papers in greater detail, finding that neither provided strong evidence that vaccinations can cause GBS within 24 hours. *Defenza*, 2026 WL 473297, at *14-15, *17. There is no doubt that the Schonberger et al. study is often credited with regard to its overall epidemiologic signal. However, although Dr. Steinman cites a specific bar graph that shows a number of cases of GBS occurring on day "0-1" (Ex. 8, p. 33 (discussing Schonberger et al., *supra*, at Ex. 40, p. 8 fig. 5)), nothing in the paper calculates that these cases represent an increased risk. At best, the study observes that there is an increased risk of GBS for the first full week post-vaccination. (Schonberger et al., *supra*, at Ex. 40, pp. 7-9.) The Park et al. study, by contrast, has significant shortcomings. It observes a highly selective group of people seeking compensation for perceived vaccine injury and, moreover, does not indicate what criteria the compensating authority uses to adjudge eligibility. (Park et al., *supra*, at Ex. 41.) In that regard, it is also suspicious that the distribution curve among the observed cases is very different from the type of distribution seen in other studies such as Schonberger et al. (*Compare id.* at 3 fig. 1, *with* Schonberger et al., *supra*, at Ex. 40, p. 8 fig. 5.) And, finally, because epidemiology speaks to correlation, epidemiologic findings do not necessarily override contrary scientific understanding just because epidemiology is also a valuable scientific tool. *Accord Defenza*, 2026 WL 1742021, at *10 (explaining that the fact that petitioner is not obligated to present epidemiologic evidence "does not mean [] that special masters must overlook the nature and limitations of such evidence").

Accordingly, petitioner has not presented preponderant evidence on this record that the timing of the onset of her TM occurred during a period that would support a causal inference based on the theory of causation she has presented.

b. *Althen* prong two

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 1326-27; *Grant*, 956 F.2d at 1147-48. Medical records are generally viewed as particularly trustworthy evidence. *Cucuras*, 993 F.2d at 1528. However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. *See* § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) ("[T]here is nothing ... that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted.") A petitioner may support a cause-in-fact claim through either medical records or expert medical opinion. § 300aa-13(a). The special master is required to consider all the relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines,* 940 F.2d at 1528).

Here, respondent argues that the medical records at best include "cursory statements" that are limited to explaining that petitioner's TM followed her vaccination. (ECF No. 45, p. 19 (citing Ex. 4, pp. 159, 188).) However, respondent stresses that such statements do not constitute causal opinions. (*Id*. (citing *Moberly*, 592 F.3d at 1323).) Otherwise, respondent argues that petitioner has not presented any evidence that would distinguish her own TM from the typical manifestations of idiopathic TM. (*Id*. at 18.) Moreover, respondent suggests that petitioner's success in meeting *Althen* prong two is predicated on her satisfaction of *Althen* prongs one and three, yet he contends that Dr. Steinman's theory and reliance on a recall response to explain the timing of onset are speculative. (*Id*. at 17-18.)

Petitioner disagrees with respondent's assessment of the treating physician's statements. (ECF No. 47, pp. 35-36.) She quotes her neurologist, Dr. Sengul, as documenting the opinion that "this is truly an idiopathic transverse myelitis, likely provoked by vaccine, which can be seen with MOGAD although she remains to be seronegative." (*Id.* at 35 (quoting Ex. 4, p. 188).) She also argues that Dr. Steinman's opinion further supports a logical sequence of cause and effect implicating petitioner's vaccination as a cause of her TM and stresses that respondent's experts have not identified any alternative cause. (*Id*. at 35-37.)

I cannot agree with respondent's interpretation of Dr. Sengul's record. The statement that petitioner's TM was "likely provoked by vaccine" cannot reasonably be reduced to mere acknowledgement of the temporal proximity of the vaccination. Dr. Sengul's opinion, as the treating neurologist, is clearly *some* evidence supporting petitioner's case. However, there are three significant reasons why Dr. Sengul's opinion does not meaningfully assist petitioner in reaching a preponderance of the evidence under *Althen* prong two.

First, as respondent suggests, Dr. Sengul's impression arose from a later neurology evaluation of February 28, 2022. (Ex. 4, pp. 184-89.) The history taken at that encounter does not confirm that Dr. Sengul was aware of the specific 24-hour post-vaccination latency at issue. (*Id.* at 184.) However, as discussed above under *Althen* prong three, that latency is not only controverted, but persuasively so. Given that respondent is persuasive in demonstrating that a 24-hour latency does not support a causal inference, and given that Dr. Sengul's record is silent as to the timing of petitioner's prior vaccination relative to the onset of her TM, it cannot merely be assumed that Dr. Sengul's opinion embraces what the record evidence otherwise demonstrates would be an implausible causal connection. Instead, considering Dr. Sengul's February 28, 2022 record holistically, the more reasonable inference is that Dr. Sengul was aware only of the fact of the prior vaccination and not the specific timing.

Second, and relatedly, although respondent is not persuasive in arguing that Dr. Sengul's notation included no causal opinion at all, that opinion does seem to be based in significant part on the presence of an assumed temporal relationship between vaccination and injury. After all, but for noting the vaccine likely "provoked" the TM, Dr. Sengul otherwise documented that petitioner's TM was "truly idiopathic." (Ex. 4, p. 188.) When a treating physician relies in part on temporality to conclude a condition is vaccine-caused, this is not disqualifying and, in fact, can be "quite probative," when the evidence under *Althen* prongs one and three also supports a causal relationship. *Capizzano*, 440 F.3d at 1326. Here, however, petitioner has not met her burden of proof under either *Althen* prongs one or three. Even if Dr. Sengul was aware of the 24-hour latency, a treating physician's opinion must still be assessed for its reliability.[27] Without more, Dr. Sengul's opinion cannot be said to be any more reliable or any better supported than Dr. Steinman's. That is, Dr. Sengul's opinion as documented does not present any reasoning or explanation that could account for the issues discussed under *Althen* prong three.

And, third, it is difficult to separate Dr. Sengul's causal opinion from her explanation that vaccine-provoked TM "can be seen with MOGAD although she remains to be seronegative." (Ex. 4, p. 188.) As discussed above relative to *Althen* prong one, Dr. Steinman specifically devised a theory of causation based on neurofascin rather than either MOG or AQP-4 because petitioner was seronegative for either MOG or AQP4. (Ex. 8, pp. 8-9.) Consistent with Dr. Steinman's implicit acknowledgement that a causal relationship based on MOG cannot be demonstrated, Dr. Sengul's notation on its face expresses that the lack of a positive MOG finding is a reason to at least doubt that petitioner's TM was vaccine-caused. Nonetheless, Dr. Sengul still couches the

[27] *E.g.*, *Veryzer v. Sec'y of Health & Human Servs.,* 100 Fed. Cl. 344, 355 (2011) (explaining that "[t]he special master properly assessed the probative value of the statements provided by petitioner's physicians . . ."), *aff'd per curiam sub nom., Veryzer v. United States*, 475 F. App'x 765 (Fed. Cir. 2012); *Hughes v. Sec'y of Health & Human Servs*., No. 16-930V, 2021 WL 839092, at *20 (Fed. Cl. Spec. Mstr. Jan. 4, 2021) (noting that "[t]he views of treating physicians should be weighed against other, contrary evidence also present in the record . . ."), *mot. for rev. denied*, 154 Fed. Cl. 640 (2021); *see also Caves v. Sec'y of Health & Human Servs*., 100 Fed. Cl. 119, 136-37 (2011) (noting special masters must consider, but are not bound by, treating physician opinions), *aff'd per curiam*, 463 F. App'x 932 (Fed. Cir. 2012).

possibility of vaccine-causation on the fact that it has been shown that vaccination can result in MOG antibody-mediated disease. Thus, Dr. Sengul's opinion does not readily support the application of Dr. Steinman's theory of causation because Dr. Sengul entertained vaccine-causation based on the premise that, despite negative testing, petitioner might have undergone an immune response to MOG, which is different than the immune response Dr. Steinman theorized.

Petitioner otherwise argues that a logical sequence of cause and effect is supported by Dr. Steinman's opinion. (ECF No. 47, pp. 35-37.) However, this argument fails in light of the fact that Dr. Steinman's opinion has not been credited under either *Althen* prong one or prong three. Petitioner also argues that it is significant that no other cause of her TM is in evidence. (*Id.* at 36.) However, although petitioners can rely on the absence of other causes to help carry their burden of proof, *Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1151 (Fed. Cir. 2007), "neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149). Respondent is persuasive in suggesting that there is nothing available on this record that distinguishes petitioner's own TM from idiopathic TM. (ECF No. 45, p. 18.) Moreover, as respondent's experts noted, idiopathic TM is well-accepted as a diagnosis. (*E.g.*, Ex. C, p. 5; Ex. E, p. 1.) Indeed, that was Dr. Sengul's actual diagnosis. (Ex. 4, p. 189.)

Accordingly, petitioner has not presented preponderant evidence on this record of a logical sequence of cause and effect that demonstrates that the flu vaccine did cause her own TM.

VI. Conclusion

Regardless of the cause of petitioner's condition, I am mindful of the pain and suffering she has endured. Moreover, I appreciate that, at first blush, the onset of TM occurring the day after a flu vaccine offers a striking coincidence. However, for all the reasons discussed above, the onset of petitioner's TM occurred too soon after vaccination to be causally related and petitioner has not met her preponderant burden of proof under any of the three parts of the applicable *Althen* test for demonstrating causation-in-fact. Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioner is not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

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