In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 21-1231V

ADWOA AMPOFO-ADDO,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: July 31, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Maximillian J. Muller, Muller Brazil, LLP, Dresher, PA, for Petitioner.

Rachelle Bishop, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On April 16, 2021, Adwoa Ampofo-Addo filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that her receipt of a tetanus, diphtheria, and acellular pertussis ("Tdap") vaccine on April 17, 2018, as well as measles-mumps-rubella ("MMR") and varicella vaccines administered on April 24, 2018, caused her to suffer myelin oligodendrocyte glycoprotein antibody-associated disease ("MOGAD"), and/or MOG-positive acute disseminated encephalomyelitis ("ADEM"). *See* Amended Petition, dated July 31, 2024 (ECF No. 41).

A two-day Entitlement Hearing was held in Washington, D.C., on November 12–13, 2024. Having reviewed the record, all expert reports, the medical records, and associated literature, I hereby find that Petitioner is not entitled to an award of compensation.

[1] Under Vaccine Rule 18(b), each party has fourteen (14) 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) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

I. Factual Background

Pre-Vaccination History

Petitioner (37 years old when she received the vaccines in question) has a medical history significant for an ectopic pregnancy in 2017, suspected allergic rhinitis, and migraines. Ex. 5 at 126, 204; Ex. 6 at 9; Ex. 11 at 24–24; Ex. 22 at 52; Ex. 53 at 145. More significantly, the record establishes that Petitioner was ill in the days before receiving the first vaccine at issue in this case. Thus, on April 14, 2018, Petitioner went to University Hospitals Twinsburg Urgent Care in Twinsburg, Ohio ("Twinsburg") complaining of a cough and congestion that was "not getting better" after a week (consistent with a similar complaint from several weeks before). Ex. 23 at 117, 118–19. She was instructed to follow up with her primary care provider for treatment of a "recurrent upper respiratory infection ["URI"] and cough." *Id.* at 119.

Vaccination and ER/Urgent Care Visits

Three days after treatment for her URI (now April 17, 2018), Petitioner visited an urgent care facility for a medical examination, and to obtain lab work needed to fulfill her U.S. immigration requirements. *See generally* Ex. 16. She received a Tdap vaccine—and the following week (April 24, 2018) returned to the same urgent care provider for receipt of MMR and varicella vaccines. Ex. 1 at 2; Ex. 16 at 16. There are no medical records from the intervening seven days between the two vaccination events, and no close-in-time evidence of complaints specific to the vaccines. But in the days and weeks not long after the second vaccine administration event, Petitioner sought urgent care several times.

For example, on April 27, 2018 (three days after the second vaccination event), Petitioner went to the University Hospitals Ahuja Medical Center Emergency Department ("Ahuja ED") in Beachwood, Ohio, for treatment of sinus congestion, cough, fever, headache, and eye pain. Ex. 23 at 15–30, 50. The records memorializing this ED visit contain no history summary setting forth how long these symptoms had existed, or when they first manifested. Petitioner was diagnosed with a sinus headache and prescribed amoxicillin, Sudafed, Mucinex, and Flonase. *Id.* at 43.

Three days later (April 30, 2018), Petitioner returned to Twinsburg, complaining of continued fevers, urinary frequency, and delayed menstruation, with her overall ill feeling unchanged since she had been last seen on April 27th. Ex. 23 at 187. Petitioner's urine culture revealed positive human chorionic gonadotropin (evidence of a possible pregnancy[3]), plus abnormal protein levels, heightened white blood cells ("WBC"), and bacteria. *Id.* at 178–79. She

[3] "Chorionic gonadotropin can be detected by immunoassay in the maternal urine within days after fertilization and thus provides the basis for the most commonly used pregnancy tests." *Chorionic Gonadotropin*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=79195 (last visited July 2, 2025).

was diagnosed with acute sinusitis and again discharged. *Id.* at 191–92.

That same day, however, Petitioner went back to the Ahuja ED reporting more urinary frequency and urgency, as well as an ongoing headache and congestion. Ex. 23 at 136. A transabdominal transvaginal ultrasound performed at this time did not confirm a gestational sac in the uterus, and imaging showed a mass-like area of tissue between the left ovary and the uterus. Ex. 23 at 34, 144, 146–47. Petitioner was diagnosed with a urinary tract infection and advised to follow up with obstetrics for repeat evaluation. *Id.* at 145, 147.

Petitioner returned to the Ahuja ED on May 3, 2018 (now nine days after the second vaccination event), reporting abdominal distention, lack of bowel movements, urinary frequency, and a two-day history of nausea and vomiting. Ex. 23 at 236. It was noted that Petitioner had been diagnosed as pregnant a few days before. *Id.* A catheter was inserted to "relieve [] [Petitioner's] abdominal distention," but her kidney functioning was deemed significantly worse compared to her last visit. *Id.* at 238. Petitioner was admitted with a diagnosis of acute renal insufficiency and an adnexal mass, and was later transferred to University Hospitals Cleveland Medical Center for further evaluation. *Id.* at 238–40, 251; Ex. 21 at 311, 1351.

Hospitalization and Relevant Treatment

At the start of her hospital admission, Ms. Ampofo-Addo reiterated the medical history she had previously provided other treaters, but also now noted that on April 30, 2018 (six days after receiving the second set of vaccines at issue), she had found it difficult to empty her bladder, and also experienced constipation, worsening lower extremity weakness, a noticeable change in her gait, bilateral feet tingling and pain with light touch, and development of a whole-body tremor. Ex. 13 at 235, 274, 329. She also noted that she recalled becoming sick the week of April 9, 2018—prior to her vaccinations—and that the illness arose after a visit from a family member from Ghana. *Id.* at 232, 267. Over the first night of her admission Petitioner developed a fever of 102.2 degrees shortly after administration of Tylenol. *Id.* at 296.

Petitioner subsequently underwent a brain MRI, which yielded evidence of "patchy areas of nonspecific abnormal increased signal on the FLAIR and T2 weighted images involving subcortical white matter and to a lesser degree the cortex within the cerebral hemispheres bilaterally" with "[a]dditional areas of ill-defined increased signal identified within the thalami and brainstem." Ex. 13 at 91. The etiology for these findings, however, was deemed uncertain. *Id.* A cervical MRI was also performed at this time, and it revealed "nonspecific vague increased intramedullary signal on the T2 images overlying the cervical cord." *Id.* at 94.

A few days into her hospitalization, Petitioner had a consult in early May 2018 with Hesham Abboud, M.D., Ph.D., a neuroimmunologist. Ex. 13 at 274–75. Dr. Abboud's exam

revealed "slow saccades, pathological hyperreflexia, LUE>RUE rest/postural/action coarse tremor, reduced vibration sense distally and a positive Romberg's sign."[4] *Id.* at 274. Additional testing provided evidence of pleocytosis with a WBC count of 205, a red blood cell count of 2, and 35% neutrophils, 35% lymphocytes, and 30% monocytes, as well as signs of high protein at 79 mg/dL and elevated myelin basic protein of 13.7 mcg/L. *Id.* at 100–35, 330, 333–34, 365, 502. Petitioner also consulted with allergy and immunology fellow Nancy Joseph, D.O. *Id.* at 325. Although Dr. Joseph maintained that an exact etiology for Petitioner's condition remained unknown at the time, the normal immunoglobulin values revealed by testing suggested to her that an immunodeficiency disease was "less likely," although she allowed for the possibility of an adverse rection to the rubella component of the MMR vaccine (as had been reported in a prior case report of which Dr. Joseph was aware). *See* S. Holt et al., *Diffuse Myelitis Associated with Rubella Vaccination*, 2 Br. Med. J. 1037 (1976) ("Holt") (describing two teenage girls who received rubella vaccines and developed sensory and motor impairment in their legs, one in four days after vaccination, and the other two weeks after vaccination).[5]

Petitioner subsequently began to receive antiviral and antibiotic medications after consult with the hospital's infectious disease team. Ex. 13 at 333, 342. Mohamad Yasmin, M.D., noted that Petitioner's mental status and lumbar puncture results were inconsistent with bacterial meningitis, but that "[p]ossible pathology may involve immune response to a preceding infection or vaccination that cross-reacts with nerve components (molecular mimicry)." *Id.* Antibiotic and antiviral coverage were discontinued on May 7th and 8th, respectively, following negative bacterial cultures, inconsistent findings of bacterial meningitis, and a remaining unidentified etiology. *Id.* at 399. However, Petitioner's WBC remained relatively high. *Id.* at 405.

Petitioner's condition had noticeably worsened by May 7, 2018. Ex. 13 at 403. She was tested and later found positive for MOG-IgG antibodies, with titer levels of 1:20 (which Petitioner's neurologic expert later deemed a low positive result). Ex. 6 (ECF No. 1-9) at 29; Tr. at 60. But despite the lack of a clear diagnosis of an immune-mediated illness, her treating physicians ordered empiric plasmapheresis given the likelihood of an autoimmune process." Ex. 13 at 369. On May 11, 2018, Petitioner had elevated WBCs of 11.9 with 1.9% immature granulocytes and increased neutrophils and monocytes, and on May 12, 2018, she had elevated WBCs of 12. *Id.* at 499–500. An echovirus lab test also revealed highly abnormal results on May

[4] "Romberg Sign" is defined as the "swaying of the body or falling when standing with the feet close together and the eyes closed; the result of loss of joint position sense, seen in … diseases affecting the posterior columns." *Romberg Sign*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=106448&searchterm=Romberg+sign (last visited July 29, 2025).

[5] Holt was not filed as an exhibit in this case, but it is referenced by Petitioner's Expert, Dr. Akbari, in his expert report, as well as in a report prepared by Dr. Forsthuber, and mentioned and cited by Respondent in his Rule 4(c) Report as well as articles and studies offered by the Respondent. *See, e.g.*, Ex. H at 155; Ex. I at 6; Ex. F Tab 5 at 23.

13, 2018—1:80 (normal range <1:10) for echovirus 7 and 30—although subsequent histories or summaries from the remainder of Petitioner's treatment course do not document further consideration of these findings and their significance. *Id.* at 125. Petitioner underwent a total of five cycles of plasmapheresis and two days of IVIG treatment between May 8 to May 18, 2018—both of which were deemed to have helped ameliorate Petitioner's condition. *Id.* at 539, 569.

Diagnosis and Subsequent Treatment

On May 18, 2018, and after slightly more than two weeks as a hospital in-patient, Petitioner was discharged to an acute in-patient rehabilitation center with the final diagnoses of ADEM post-vaccination, acute urinary retention, and unspecified abnormal involuntary movements. Ex. 13 at 557, 567. At discharge, she exhibited a mild full-body and resting bilateral hand tremor, full strength, intact sensation, subjective numbness in her left foot without loss of sensation on exam, intermittent chest tightness complaints, and the ability to walk with assistance. *Id.* at 568–69. Some lab work results that might have shed further light on the etiology of her condition remained outstanding as of discharge.[6] Petitioner was advised to follow up with an obstetrician, urology, and Dr. Abboud. *Id.* at 570. For approximately a week in mid-May 2018, Petitioner received in-patient physical therapy ("PT") and occupational therapy ("OT") at Beachwood Rehabilitation Hospital. *See generally* Ex. 17.

A few days later (May 22, 2018), Petitioner saw Dr. Harold Mars, a neurologist, complaining of "a tight band-like sensation about the waist," which corresponded to a T6–T8 sensory level. Ex. 13 at 31. Upon examination, Dr. Mars noted Petitioner possessed diffusely hyperactive deep tendon reflexes and a sensory level at T6–T8, concluding that her condition was "predominantly transverse myelitis ["TM"]." *Id.* at 32. Although Dr. Mars was unable to exclude other demyelinating disorders at that time, he did note that TM with brain lesions could reflect an "autoimmune response to vaccine received a few weeks previously." *Id.* He recommended that Petitioner undergo continued brain and spine MRIs in order to monitor for recurrent disease (consistent with a protocol to watch for multiple sclerosis). *Id.* Petitioner was at this time noted, however, to have significantly improved since her in-patient rehab stay, and could not transfer independently, walk independently for more than 150 feet, and climb stairs. *Id.* at 328–30.

Petitioner had a follow-up appointment with Dr. Abboud on June 4, 2018. Ex. 20 at 25. She exhibited decreased sensation in her left lower extremity, but her exam was otherwise unremarkable. *Id.* at 28. After reviewing Petitioner's extensive testing results, Dr. Abboud recommended additional MOG antibody testing, another brain and cervical MRI, and PT and OT. *Id.* at 42. Dr. Abboud proposed that if Petitioner's antibodies remained positive, or the MRI

[6] These lab testing categories include Listeria IgG, Mayo CDS1 panel for demyelinating diseases, JCV with IgG antibody, echovirus antibody, Strep pneumococcal IgG antibody, and coxsackie, rubella, and echovirus. Ex. 13 at 569-70.

imaging revealed new lesions, then long-term immunosuppressive therapy would need to be considered for treatment of "presumed NMO."[7] *Id.* Petitioner was now diagnosed with "anti-MOG positive post-vaccination encephalomyelitis vs NMOSD." *Id.*

Later that same June, Petitioner began outpatient PT and OT. She reported at this time that she had resumed the majority of her activities of daily living. Ex. 4 at 16. Her therapist documented some positive upper body motor neurological findings, but overall Petitioner's "movement quality, balance, and strength are virtually normal." *Id.* at 8. Petitioner attended three visits, after which she was discharged with "excellent gains in strength, balance, and vestibular-ocular function." *Id.* at 16. She further noted that she was "90–95 percent recovered" and that she had regained "95 percent of her strength." *Id.* at 16–19.

Petitioner returned to Dr. Abboud on August 9, 2018. Ex. 20 at 15–24. Repeat MRIs, completed on June 20, 2018, revealed "near complete resolution of the abnormal signal in the brain and cord." *Id.* at 18–22, 23. Dr. Abboud deemed Petitioner to be "doing great," with a residual mild and tolerable "cold feeling in her right leg and some vibration feeling in the left hip." *Id.* at 15. His diagnosis remained "anti MOG positive post-vaccination encephalomyelitis vs. NMOSD." *Id.*

Since the summer of 2018, Petitioner has experienced no significant regression or relapse in her health—and support for the MOGAD diagnosis considered in this case is variable. For example, in November 2018, Petitioner underwent Aquaporin 4 and MOG antibody testing, but the results were now negative. Ex. 6 at 37–38; Ex. 20 at 49. At a follow-up visit that December with Dr. Abboud, it was proposed that these results (coupled with her improved physical condition) supported the conclusion that her April–May 2018 illness was "a one-time event related to vaccination" and "more consistent with monophasic post-vaccinal demyelination." Ex. 20 at 14. Dr. Abboud also stated that no additional follow up would be needed should her repeat brain MRI in June 2019 was negative. *Id.* In the following two to three years, Petitioner has intermittently sought treatment for neurologic concerns, but physical exams and testing have not revealed any concerning evidence of an ongoing disease process.

II. Witness Testimony

A. *Fact Witness – Ms. Ampofo-Addo*

Ms. Ampofo-Addo was the only fact witness to testify at the hearing. *See generally* Tr. at

[7] Neuromyelitis Optica is defined as a "demyelination of the optic nerve and the spinal cord . . . marked by diminution of vision and possibly blindness, flaccid paralysis of the extremities, and sensory and genitourinary disturbances." *Neuromyelitis Optica*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92610&searchterm=neuromyelitis+optica (last visited July 2, 2025).

5–54. She began her testimony describing her pre-vaccination medical history. *Id.* at 11. The only major issue she had experienced was an ectopic pregnancy that occurred in 2017. Tr. at 11. She also had ongoing sinus problems, including nasal congestion and an itchy throat, and she was diagnosed with a dust allergy. *Id*. Three days before receiving the Tdap vaccine, Petitioner went to urgent care to seek treatment for a cold and cough that had "lingered" longer than a usual illness. *Id.* at 13.

Petitioner received the Tdap vaccine on April 17, 2018, followed by the MMR and varicella vaccines on April 24th. Tr. at 14. She received these vaccines as part of her application for permanent residency in the United States. *Id.* She remembers thinking it was "unusual" to receive two vaccines at once, but does mention any pain or other symptoms after receiving these vaccines. *Id.* at 15. Three days after she received the MMR and varicella vaccines, Petitioner experienced sinus congestion, cough, fever, headache, and eye pain. *Id*. Six days after she received the MMR and varicella vaccines, Petitioner recalled that she still had a headache, in addition to other symptoms such as difficulty urinating and fever. *Id.* at 16. At that time, it was determined that Petitioner was pregnant. *Id.* at 17.

On May 3, 2018, Petitioner went to the Ahuja ED. Tr. at 18–19. She testified that she was suffering from abdominal distention, lack of bowel movements, difficulty urinating, nausea, and vomiting. *Id*. at 19. At that time, a foley catheter was inserted to relieve Petitioner's abdominal distention. *Id*. Petitioner explained that her doctor was concerned about the functioning of her kidneys, so she was transferred to the Cleveland Clinic. *Id*. at 20. She recalled that on the day of the transfer, she began to experience a full body tremor, lower extremity weakness, bilateral tingling and numbness in the feet, pain with light touch, and difficulty walking. *Id*. at 21–22. Petitioner testified that she was "in so much pain [that she] couldn't explain or know where the pain was coming from," and that the "upper part" of her body was shaking. *Id*. at 22–23.

Once admitted to the Cleveland Clinic, Petitioner's symptoms worsened. Petitioner testified that after four days in the hospital, she could no longer walk due to the pain and numbness in her feet. Tr. at 26. She began to experience tremors in her hands that prevented her from dressing herself or doing "anything that a normal person could… do for themselves." *Id*. Petitioner recalled that while she was in the hospital, Dr. Joseph posited that her symptoms may be a result of an adverse reaction to the vaccines. *Id*. at 28–29. Petitioner testified that the treatments of plasmapheresis and IVIG began to work, and she regained feeling and mobility. *Id*. at 29. At some point during her stay at the clinic, Petitioner experienced a miscarriage. *Id*. at 30. Petitioner was discharged from the Cleveland Clinic to Beachwood Rehab Hospital, where she spent a week doing physical and occupational therapy. *Id*. at 31. Petitioner recalled that her mobility had improved between her stay at the Cleveland Clinic and her time at Beachwood, though she had not regained full mobility and continued outpatient physical therapy through the summer. *Id*. at 32–34.

Petitioner experienced a "band-like" sensation of tightness around her upper trunk and tinging and pain in her lower extremities that continued after being released from the hospital. Tr. at 33. She recalled that Dr. Abboud told her that she had tested positive for the anti-MOG antibody and prescribed Gabapentin, which Petitioner took until her nerve pain ceased. *Id* at 33–34. Two months after receiving her vaccines, Petitioner reported migraines and blurry vision that is ongoing. *Id*. at 35.

Petitioner recalled seeing Dr. Abboud again four months after her vaccinations, and he requested that she be tested again for the anti-MOG antibody. Tr. at 36. On November 16, 2018, Petitioner tested negative for the anti-MOG antibody. *Id*. at 38–39. Petitioner remembered Dr. Abboud telling her that because of her negative test results, her symptoms from earlier that year were likely a "one-time event related to vaccination." *Id*. at 39. Petitioner explained that Dr. Abboud told her to avoid live vaccines if possible so that she did not have an "adverse reaction." *Id*. at 38.

Petitioner became pregnant and gave birth to her first child in August 2019. Tr. at 38. In June 2020, Petitioner saw a new neurologist reporting coldness and tinging in her lower left extremity that had been continuous since her hospitalization in 2018. *Id*. at 41. Petitioner returned to the neurologist in July 2022 and reiterated her concerns about sensory symptoms in her legs and migraines. Petitioner stated that she is seeing a therapist to help process her trauma surrounding the hospitalization and continued symptoms. *Id*. at 43–44. Petitioner testified that her symptoms impacted her daily life, including her ability to work, engage in recreation, volunteer, and interact with her family. *Id*. at 45–46.

On cross-examination, Petitioner acknowledged that a family member had visited her from Ghana around the time she first became sick. Tr. at 50-51. She denied records indicating that this individual had been sick, but acknowledged the individual may at least have had a cold. *Id.* at 51.

B. *Petitioner's Experts*

1. Dr. Syed Rizvi – Dr. Rizvi, a neuroimmunologist, prepared two written reports for the Petitioner and testified at trial. Report, dated April 11, 2023, filed as Ex. 30-1 (ECF No. 29-2) ("First Rizvi Rep."); Report, dated October 4, 2023, filed as Ex. 46-1 (ECF No. 31-2) ("Second Rizvi Rep."). He proposed that the Tdap, MMR, and varicella vaccines that Petitioner received likely played a significant role in the development of Petitioner's MOGAD. Tr. at 59.

Dr. Rizvi is a neuroimmunologist in academic practice at Brown Neurology, and Professor of Clinical Neurology at the Alpert Medical School of Brown University. First Rizvi Rep. at 1. He routinely cares for patients with a wide variety of autoimmune conditions, such as ADEM/MOGAD and multiple sclerosis. *Id.* Dr. Rizvi received his medical degree from Dow Medical School in Karachi, Pakistan. Curriculum Vitae, dated April 25, 2023, filed as Ex. 31-1 (ECF No. 29-3) ("Rizvi CV") at 1. He is board certified by the American Board of Psychiatry and

Neurology to practice neurology in the state of Rhode Island. *Id.* Dr. Rizvi also received fellowship training in multiple sclerosis and clinical neuroimmunology in 2000. First Rizvi Rep. at 1. He previously taught neurology at Stony Brook University Hospital before accepting a position at the Alpert Medical School. *Id.* at 2. He has acted a principal investigator in over twenty-five clinical trials, authored over twenty journal articles in his field, and continues his clinical practice today. *Id.* at 1–8.

Dr. Rizvi began his testimony by describing MOGAD as a newly-identified classification for an autoimmune disease in which the body's immune system attacks MOG proteins found in the myelin sheath covering central nervous system ("CNS") nerves, causing demyelination and inflammation. Tr. at 60. Although the exact etiology of MOGAD is unknown, it is generally thought to occur after an infection or, more rarely, a vaccination. *Id*. at 60–61. Dr. Rizvi discussed the diagnostic criteria for MOGAD, opining that Petitioner's "core clinical demyelinating event" was myelitis or ADEM. *Id*. at 66.

Dr. Rizvi acknowledged that Petitioner's antibody titer levels (as first tested on May 7, 2018) were not by themselves high enough to support a diagnosis of MOGAD. Tr. at 63–64; Ex. 6 (ECF No. 1-9) at 29. But he opined that, when combined with her negative test for aquaporin antibodies and her supporting MRI features of longitudinally extensive myelitis and multifocal brain lesions, the results pointed toward MOGAD. *Id*. at 64–65; Rizvi First Rep. at 6 (citing B. Banwell et al., *Diagnosis of Myelin Oliogdendrocyte Glycoprotein Antibody-Associated Disease: International MOGAD Panel Proposed Criteria*, 22 Lancet Neurol. 268, 276 Fig. 3 (2023), filed as Ex. 49 (ECF No. 52-2) ("Banwell")). In addition, Petitioner's ongoing symptoms were consistent with her diagnosis (other than her tremors). Tr. at 99, 115. And he maintained that the timing of the onset of Petitioner's symptoms after her vaccinations was consistent with studies of other patients that developed similar disorders. *Id*. at 101.

Dr. Rizvi went on explain his causation theory connecting Petitioner's vaccinations to her MOGAD. Tr. at 78. He opined that Petitioner's vaccinations triggered the production of MOG antibodies through a process known as molecular mimicry, in which "there is a sharing of antigen between… the host and the pathogen, epitope sharing, so that what happens is when your immune system gets activated, there is… a mechanism where it's attacking both certain parts of the host and the pathogen." *Id*. at 79. In Petitioner's case, Dr. Rizvi believed that via molecular mimicry, "certain components of her central nervous system were attacked as well." *Id*. Petitioner's exposure to vaccines, combined with her pregnancy and possible genetic predisposition to autoimmune disorders, produced her "abnormal immune response resulting in autoimmunity and MOGAD." *Id*. at 81. Dr. Rizvi later acknowledged, however, that there was nothing in Petitioner's medical records to suggest a predisposition or genetic susceptibility to autoimmunity. *Id*. at 130. He also did not specify anything about the nature of the cross-reactivity between any of the vaccines that Petitioner received and the MOG protein. *Id*. at 131.

Dr. Rizvi further sought to rebut the contention of Respondent's experts that Petitioner's MOGAD was caused by a possible preceding viral infection (reflecting a para-infectious myelitis, as Dr. Roos opined). Tr. at 82, 472–73. He concluded instead, based on Petitioner's medical records, that (as seemed to be the view of Petitioner's treating physician) her pre-vaccination symptoms were likely bacterial in origin. *Id*. at 84. In Dr. Rizvi's view, Petitioner did not demonstrate symptoms of a viral infection during her various pre-vaccination presentations—he would have expected someone with a "significant viral illness" to show "more flu-like symptoms" and have "a lot more congestion." *Id*. at 85. Dr. Rizvi also confirmed that Petitioner's neurological symptoms began around April 30th, six days after her second vaccination event. *Id*. at 87. He found it significant that four of Petitioner's treating physicians, including an immunologist, two infectious disease specialists, and a neurologist, all allowed for the possibility that Petitioner's symptoms could be connected to her recent vaccinations. *Id*. at 90–93. However, Dr. Rizvi did later acknowledge that most cases of MOGAD are post-infectious. *Id*. at 470. And he could identify no large-scale studies connecting MOGAD to vaccination. *Id*. at 132.

Dr. Rizvi similarly sought to undermine the argument that an echovirus infection (as evidenced by Petitioner's moderately elevated antibody titers for echovirus-7 and echovirus-30 after her hospitalization) explained her MOGAD. Tr. at 93. Due to a lack of repeat testing to show the change in antibodies over time, he could not determine the time frame in which Petitioner had likely experienced an active echovirus infection. *Id*. at 94. Petitioner also did not show specific clinical symptoms of echovirus, and thus "in the absence of real typical clinical presentation," physicians would tend to discount such test results as not meaningful. *Id*. at 96. He later admitted, however, that Petitioner's treating physicians never ruled out echovirus as a cause of her symptoms, and that he could not himself rule out echovirus given Petitioner's positive titers. *Id*. at 126, 480.

Dr. Rizvi addressed an error in his first expert report, where he stated that Petitioner was treated with steroids while in the hospital. Rizvi First Rep. at 4. According to the case report specifically written about Petitioner, she was never treated with steroids. N. Kumar et al., *Case Report: Postvaccination Anti–Myelin Oligodendrocyte Glycoprotein Neuromyelitis Optica Spectrum Disorder*, 22 Int. J. MS Care 85, 87 (2020), filed as Ex. 43 (ECF No. 29-15) ("Kumar"). Dr. Rizvi acknowledged the error, and stated that while most MOGAD patients are treated with steroids, Petitioner's treating physicians likely did not likely do so due to her pregnancy. *Id*. at 130.

2. Omid Akbari, Ph.D. – Dr. Akbari is an immunologist, and he both testified on Petitioner's behalf and prepared two written reports. Report, dated Oct. 3, 2023, filed as Ex. 50-1 (ECF No. 31-6) ("First Akbari Rep."); Report, dated May 16, 2024, filed as Ex. 92-1 (ECF No. 38-2) ("Second Akbari Rep."). He posited that Petitioner's immune response to the MMR vaccine triggered her ADEM and MOGAD via multiple mechanisms, including inflammasome

activation[8] and then adaptive immune response-driven molecular mimicry. First Akbari Rep. at 24.

Dr. Akbari is both a director and professor of Immunology at the University of Southern California Keck School of Medicine. First Akbari Rep. at 2; Curriculum Vitae, dated Oct. 13, 2023, filed as Ex. 51-1 (ECF No. 31-7) ("Akbari CV") at 2. He received both his bachelor's and master's degrees from University College London before receiving a Ph.D. in cellular and molecular immunology from the Nation Institute for Medical Research in London, England. Akbari CV at 1. Afterwards, he completed a postdoctoral fellowship at Stanford University. *Id.* Dr. Akbari worked as an assistant professor at the Children's Hospital of Boston before joining the faculty of the Keck School of Medicine in 2008. *Id.* He has and continues to serve on the editorial board of several journals, and he has numerous publications in the field of immunology research. *Id.* at 5–7. His research largely focuses on analyzing "cytokines and inflammation with the focus specifically on dysregulated immune responses." First Akbari Ret. at 2. Dr. Akbari is not a medical doctor, however, and does not diagnose or treat patients with neurological diseases in a clinical setting.

Dr. Akbari began his testimony by describing how vaccines can trigger the body's immune system, particularly the mechanism through which vaccines activate inflammasomes that trigger inflammatory responses and enhance immune response. Tr. at 154–56. He testified that molecular mimicry, which he described as the process through which foreign antigens closely resembling the body's own proteins "confuse the immune system," "causing it to mistakenly attack the body's own tissue," was the likely mechanism through which Petitioner's vaccinations caused her MOGAD. *Id*. at 156–61. While Dr. Akbari acknowledged that he did not cite any peer-reviewed literature about molecular mimicry in his expert report, he affirmed the existence of such literature. *Id*. at 200.

He also discussed the activation of the inflammasome. While the scientific community is still working towards a complete understanding of the innate immune response to vaccinations, it is well established that all effective vaccines must induce an inflammasome response. Tr. at 155–56. Dr. Akbari proposed that antigenic elements in the MMR and Tdap vaccines could infect host immune cells and lead to inflammasome activation—which in turn would lead to the production of cytokines and encourage subsequent inflammation. First Akbari Rep. at 15; *see also* Second Akbari Rep. at 2 (citing B. Pulendran & R. Ahmed, *Immunological Mechanisms of Vaccination*, 12 Nat. Immunol. 509 (2011), filed as Ex. 93 (ECF No. 38-3) ("Pulendran & Ahmed") (discussing

[8] The inflammasome is "a complex of cryopyrin, caspase-1, and other proteins, found in phagocytic cells and related to the body's system of innate immunity. Assembly of the inflammasome leads to activation of caspase-1 and resultant cleavage and activation of interleukins IL-1ß and IL 18 in the inflammatory response." Inflammasome, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=25203&searchterm=inflammasome (last visited July 22, 2025).

the mechanisms of vaccine induced immunity); E. Latz et al., *Activation and Regulation of the Inflammasomes*, 13 Nat. Rev. Immunol. 397 (2013), filed as Ex. 94 (ECF No. 38-4) (reviewing the biologic mechanisms that facilitate inflammasome-mediated immune response)).

Dr. Akbari offered three ways the MMR vaccine could activate the inflammasome. First, the viral components of the MMR vaccine are recognized by Pattern Recognition Receptors on immune cells and activate the inflammasome. Second Akbari Rep at 2. Second, the MMR vaccine can induce the production of reactive oxygen species that leads to the release of mitochondrial DNA from damaged cells, which then activates the inflammasome. *Id.* Third, that intracellular sensors of viral RNA (contained in the vaccine) can trigger downstream signaling pathways leading to inflammasome assembly. *Id.* at 2–3.

After the inflammasome response, an adaptive immune response—in which the immune system learns to recognize and react to foreign antigens—is activated. First Akbari Rep. at 7. It is at this point that pieces of foreign antigens would need to migrate to the lymph nodes in order to interact with B and T cells. *Id.* Dr. Akbari emphasized the subsequent role of regulatory and effector T cells in triggering Petitioner's ADEM. Tr. at 208. In his opinion, an imbalance in the types of T cells in the body can cause different ailments. In particular, Dr. Akbari proposed that "if the T effector [cells start to increase beyond] a usual number" and start to "dominate" the Tregs, "then the T effectors can cause autoimmunity" such as ADEM. *Id*. Dr. Akbari testified that there is a direct causal link between vaccination, T cell imbalance, and ADEM based on the Petitioner's likely genetic predisposition to certain autoimmune diseases. *Id*. at 210–211. While he acknowledged a lack of peer-reviewed research that demonstrates a causation beyond correlation connecting these conditions, Dr. Akbari maintained that his theory is "very viable." *Id*. at 210.

Dr. Akbari mentioned that some of the circumstances surrounding Petitioner's vaccination, including Petitioner's pregnancy and the fact that she received three vaccines in a short time frame, may have contributed to the development of her ADEM and/or MOGAD. Tr. at 165, 189. However, he could not commit to the opinion that all *three* vaccines were necessary to trigger her ADEM. *Id*. at 225–26. Additionally, Dr. Akbari briefly touched on Petitioner's elevated echovirus titers, agreeing with Dr. Rizvi that he would not consider test results revealing a single elevated titer to be clinically significant ("one titer doesn't mean anything"). *Id*. at 186.

Notably, Dr. Akbari did not identify anything specific about the nature of the proposed cross-reactivity in this case. Thus, although the MOG antibody would presumably be driving the disease process at issue, he provided no direct testimony as to how the vaccines at issue would cause the production of this autoantibody, whether due to mimicking similarity between vaccine components and the MOG protein in self-tissues or via some other mechanism. At most, he referenced a case study where the authors found amino acid sequence similarities between two T-cell epitopes of tetanus toxoid and MOG-specific T cells in MS patients. A. Virupakshaiah et. al.,

Life-Threatening MOG Antibody-Associated Hemorrhagic ADEM With Elevated CSF IL-6¸ 11 Neurol. Neuroimmunol. Neuroinflamm. 1, (2024), filed as Ex. 120 (ECF No. 55-3) ("Virupakshaiah"). But he did not address the low expect values for each of these similarities, nor challenge the veracity of the results of homologic research performed by Respondent's immunologic expert (which did not find sufficient homology between the relevant vaccine components and self-structures to find cross-reactivity likely). *See* Analysis of Dr. Forsthuber's BLAST Search Results, dated Nov. 21, 2024, filed as Ex. K. (ECF No. 54-1) ("Forsthuber BLAST") at 3–4.[9]

C. *Respondent's Experts*

1. Dr. Karen L. Roos – Dr. Roos, a neurologist, filed two reports for Respondent and testified at the hearing. Report, dated July 27, 2023, filed as Ex. A (ECF No. 30-1) ("First Roos Rep."); Report, dated January 22, 2024, filed as Ex. E (ECF No. 35-1) ("Second Roos Rep."). Dr. Roos opined that the Petitioner was misdiagnosed, and likely suffered from a postinfectious/parainfectious myelitis with a mild encephalitis component that was fully resolved in three months, rather than MOGAD. First Roos Rep. at 10.

Dr. Roos is a board-certified neurologist specializing in infectious diseases and serves as the John and Nancy Nelson Professor Emerita of Neurology at the Indiana University School of Medicine, where she has taught neurology since 1985. Curriculum Vitae, filed as Ex. B (ECF No. 30-11) ("Roos CV"). She obtained her undergraduate degree from the University of Pittsburgh, and her medical degree from the Drexel University School of Medicine. *Id* at 1. She then completed her residency in neurology and training in electroencephalography at the University of Virginia Medical Center. *Id*. She has over 40 years of experience in treating patients with neurological disorders with a specialization in neurological infectious diseases. First Roos Rep. at 1. She has authored or edited six textbooks and has published 250 manuscripts relating to neurology. *Id*. While Dr. Roos conceded that she had never diagnosed anyone with MOGAD, she has treated over 160 para- and postinfectious myelitis patients. Tr. at 333, 360.

Dr. Roos contended at the outset that, in her view, ADEM was not a reasonable diagnosis for Petitioner's symptoms. Tr. at 258. Rather, the medical record established that Petitioner had experienced a systemic infectious disease prior to her vaccinations which likely caused a myelitis (inflammation of the spinal cord), resulting in her neurological symptoms. *Id*. Dr. Roos noted that Petitioner's CSF cell count was greater than 50, when one would expect a lower count for an

[9] According to its own website, the "Basic Local Alignment Search Tool" (BLAST) "finds regions of local similarity between sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance of matches. BLAST can be used to infer functional and evolutionary relationships between sequences as well as help identify members of gene families." *See* https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited July 29, 2025). It is common in the Program for immunology experts to utilize BLAST searches when arguing about whether a vaccine's protein components mimic self-structures.

ADEM diagnosis. *Id*. at 262. Petitioner also had not reported experiencing encephalopathy, nor did she demonstrate any decreased level of consciousness, which is "really important" for a diagnosis of ADEM. *Id*. at 263. As such, Dr. Roos would "find it difficult" to diagnose Petitioner with ADEM. *Id*. (in addition, even if ADEM were a fair diagnosis given the record, Dr. Roos opined, it was more likely attributable to an infectious process. Dr. Roos reiterated Dr. Rizvi's concession that 95% of ADEM cases follow an antecedent infection, adding that every case of ADEM that she had ever seen had been triggered by an antecedent infection. *Id*. at 265, 268).

Dr. Roos pointed out other aspects of the record that suggested Petitioner's neurologic symptoms, however properly diagnosed, had an infectious origin. She noted, for example, that Petitioner's choice to go to urgent care on April 14, 2018 (hence prior to her first vaccination) for her cough and congestion established that such symptoms were more severe than what would be associated with her allergic rhinitis. Tr. at 269–70. In fact, Dr. Roos characterized Petitioner's complaints of cough, cold, congestion, fever, and headaches as most likely evidence of a viral infection. *Id*. at 271–72. And later on, Petitioner's "moderately elevated" echovirus-7 and echovirus-30 titers (1:80), in combination with her symptoms of infection, suggested a specific explanation for her condition: an echovirus infection. *Id*. at 266–67. Echovirus-30 is neurovirulent, meaning that it infects the CNS, and is much more common in adults than ADEM. *Id*. at 276–77. It also is not a "latent" virus, so Petitioner's positive serology result was consistent with a current or recent infection. *Id*. at 278–79. And as Petitioner's treating physicians were seemingly unaware of her echovirus titer results, their presence was never factored into her diagnosis. *Id*. at 326.

Dr. Roos also took issue with Dr. Rizvi's opinion that Petitioner either had MOGAD or MOG-positive ADEM. Tr. at 280. In particular, Petitioner did not meet the diagnostic criteria necessary for MOGAD. *Id*. at 289. Petitioner's "low-positive" MOG antibody titer of 1:20 (as identified on testing performed on May 7, 2018) is "seen in many para and postinfectious neurological disorders," and was lower than what one would expect for a MOGAD patient. *Id*. at 282–83; Ex. 6 at 29. And Petitioner's treating neurologist did not identify any of the "classic and expected neuroimaging abnormalities of MOG" from Petitioner's brain MRI. *Id*. at 285. Thus, Petitioner's clinical presentation was inconsistent with MOGAD—especially in light of the low antibody titers. *Id*. at 288.

Most damming to Dr. Rizvi's theory, according to Dr. Roos, was his failure to exclude alternative diagnoses before settling on MOGAD—a required element of the relevant diagnostic criteria. Tr. at 292; Banwell at 9 fig. 3. And the case report that was written about Petitioner's treatment, Kumar, was also not proof that the diagnosis was correct, especially since the report's conclusions were unreliable (and also given the low weight case reports should receive generally as evidence of causation). Tr. at 294–95. In Dr. Roos's opinion, the tests chosen and written about by the physicians "tilted [the conclusion] toward what they wanted it to be." *Id*. at 297. In general, Dr. Roos felt that causation could "not depend on case reports and case series," as case reports can be "swayed" to fit a physician's theory. *Id*. at 299.

Dr. Roos concluded her testimony by discussing multiple studies regarding the connection between vaccines and ADEM. Tr. at 300. In particular, she cited a long-term prospective study which found no increase in the risk of ADEM after many common vaccinations, including those received by Petitioner. *Id*. at 310; Y. Chen et al., *Vaccines and the Risk of Acute Disseminated Encephalomyelitis*, 36 Vaccine 3733, 3733–39 (2018), filed as Ex. I (ECF No. 44-2) ("Chen") (no increase in the risk of ADEM after administration of the Hepatitis B, influenza, polio (live), diphtheria, pertussis (acellular), tetanus, measles, mumps, rubella, Japanese Encephalitis, meningitis, hepatitis a, varicella, and rabies vaccines).

2. Dr. Thomas Forsthuber – Dr. Forsthuber is an immunologist (although also a medical doctor, unlike Dr. Akbari), and he prepared three written reports in this case and testified for Respondent. Report, dated July 27, 2023, filed as Ex. C (ECF No. 30-12) ("First Forsthuber Rep."); Report, dated February 1, 2024, filed as Ex. F (ECF No. 36-1) ("Second Forsthuber Rep."); Report, dated July 30, 2024, filed as Ex. G (ECF No. 42-1) ("Third Forsthuber Rep.").

Dr. Forsthuber is a Professor of Immunology at the University of Texas at San Antonio and an Adjunct Professor of Microbiology & Immunology at the University of Texas Health Sciences Center in San Antonio. First Forsthuber Rep. at 1. He received his M.D. from the University of Tübingen in Germany and completed his residency in pathology at University Hospitals Cleveland. Curriculum Vitae, dated July 28, 2023, filed as Ex. D (ECF No. 30-34) ("Forsthuber CV") at 2. He is board certified in anatomical and clinical pathology by the American Board of Pathology. *Id*. Dr. Forsthuber has over twenty-five years of experience in immunology and has published over one hundred scholarly publications, reviews, and book chapters in his field. First Forsthuber Rep. at 1; Forsthuber CV at 21–38. His research focuses on immunology, multiple sclerosis, and autoimmune heart disease. First Forsthuber Rep. at 1. Dr. Forsthuber does not currently treat patients, however, and has never treated a patient with encephalomyelitis. *Id.* at 2.

Dr. Forsthuber began his testimony by criticizing the first component of Dr. Rizvi's mechanistic theory: vaccine activation of the inflammasome. Tr. at 383. While a vaccination might instigate some level of inflammasome activation at the site of the injection, Dr. Forsthuber saw "no reason that a vaccination in the arm should lead to an inflammasome activation in the brain." *Id*. at 384–85. Additionally, while the activation of inflammasomes is the "first step in a cascade" of immune system activation that could "eventually lead to molecular mimicry," there is "no direct link" between inflammasomes and an adaptive process of autoimmune cross-reactivity due to molecular mimicry. *Id*. at 387.

Dr. Forsthuber next discussed the link that Dr. Akbari emphasized between the inflammasomes and the production of cytokines, particularly interleukin-1 (IL-1), and the role those cytokines would putatively play in impacting T cell regulation of the immune response. Tr. at 395. Although he acknowledged that cytokine levels could be impacted by vaccines and infections alike, "the levels of cytokines that you see after infection are typically much, much

higher than what you see after vaccination." *Id*. at 397. As a result, an infection would "absolutely" induce more inflammatory cytokines than even the receipt of multiple vaccinations, as the small number of cytokines produced by a vaccination are "short-lived, 24 to 48 hours," whereas those produced by an infection are generated for days or weeks. *Id*. at 399.

Dr. Forsthuber went on to consider the next element of Dr. Akbari's theory, which proposed that interference with regulatory T cells ("Treg") could encourage ADEM. Tr. at 388. While Tregs do "play a role in in preventing excessive immune activation," there is "no direct role for regulatory T cells" in the pathogenesis of ADEM. *Id*. at 391. Because even a small number of T cells can prevent autoimmunity, the kind of massive disruption of T cells required to render them ineffective (and hence resulting in the "breaking" of immune tolerance) would involve a "cataclysmic event." *Id*. at 393–94. Dr. Forsthuber also pointed out that Dr. Akbari had failed to explain how a dysregulation in T cells would trigger ADEM specifically, rather than produce other autoimmune disorders. *Id*. at 393. And existing medical literature "debunks the claim that vaccination … somehow alters the T regulatory cell balance." *Id*. at 400; A. de Wolf et al., *Regulatory T Cell Frequencies and Phenotypes Following Anti-Viral Vaccination*, 12 PLoS ONE 6 (2017), filed as Ex. G Tab 1 (ECF No. 42-2) ("de Wolf") (finding that vaccines with a "safe profile" had minimal and insignificant impact on the homeostasis of Treg cells).

Dr. Forsthuber then criticized the theory of molecular mimicry and its application to this case. Tr. at 401. In his opinion, there are "very few examples where molecular mimicry [as casual of an autoimmune injury] has been robustly supported by the evidence." *Id*. at 402. Further, none of the evidentiary elements required to corroborate molecular mimicry as causal of a disease (epidemiological evidence, sequence or structural similarity between pathogens and self-antigens, and cross-reactivity) had been demonstrated to support a link between ADEM and vaccination. *Id*. at 403. In his opinion, "the literature on MMR vaccine, varicella vaccine and Tdap vaccine is pretty clear that there is no… robust support that [they cause] ADEM or MOGAD," or that the receipt of multiple vaccinations in a short period of time would make someone more likely to develop ADEM. *Id*. at 404, 409.

In addition, Dr. Forsthuber found no significant similarity between the amino acid sequences of tetanus and MOG, as allegedly supported by Virupakshaiah. Forsthuber Blast at 3–4. Instead, what Dr. Forsthuber found from his own search was that Virupakshaiah's authors had erred in their analysis because the E values of the two regions did not match up to the E values of his BLAST results. *Id.* In addition, the authors of the study expanded the BLAST search parameters beyond the standard settings, which allowed the inclusion of insignificant sequence alignments. *Id.* Dr. Forsthuber's own analysis of the proposed sequences resulted in no matches or significant sequence alignments between the amino acid sequences of tetanus and MOG. *Id.*

While Dr. Forsthuber did not commit to echovirus as a specific cause of the Patient's ADEM, he did agree with Dr. Roos that an upper respiratory infection of an unspecified nature was the most likely trigger of Petitioner's illness. Tr. at 432. In fact, he was conflicted as to whether

any vaccine could cause a neurological disease. *Id*. at 447. He stressed that Petitioner's treating physicians did not do a thorough infectious disease workup to rule out alternative causations. Finaly, he opined that Petitioner's existing conditions of pregnancy and an upper respiratory infection would not increase her susceptibility to an adverse reaction to vaccination. *Id*. at 455.

III. Procedural History

Respondent filed his Rule 4(c) Report contesting Petitioner's right to compensation on November 30, 2022. *See* Report, dated Nov. 30, 2022 (ECF No. 28). Thereafter, the process of obtaining expert reports began, with the final report from Dr. Forsthuber filed in July 2024. The parties submitted pre-hearing briefs and a two-day Entitlement Hearing took place in November 2024. After the Entitlement Hearing, the Respondent submitted medical literature and findings mentioned at the hearing.[10] In addition, both parties indicated their desire to, and later submitted, post hearing briefs. Petitioner's Post Hearing Brief, dated Feb. 6, 2025 (ECF No. 63) ("Br."); Respondent's Post Hearing Brief, dated Feb. 6, 2025 (ECF No. 62) ("Opp.").

IV. Applicable Legal Standards

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 his 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 v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[11] There is no Table claim for vaccine-caused MOGAD or ADEM.

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

---

[10] *See generally* Forsthuber BLAST; M. Varrin-Doyer et. al., *MOG Transmembrane and Cytoplasmic Domains Contain High Stimulatory T-cell Epitopes in MS*, 2 NEUROL. NEUROIMMUNOL. NEUROINFLAMM. 1 (2014), filed as Ex. L (ECF No. 54-2); R. Weissert, et. al. *High Immunogenicity of Intracellular Myelin Oligodendrocyte Glycoprotein Epitopes,* 169 J. IMMUNOL. 548 (2022), filed as Ex. M (ECF No. 54-3); Supp. App. to B. Banwell, et. al., *Diagnosis of Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: International MOGAD Panel Proposed Criteria,* 22 LANCET NEUROL. 268 (2023), filed as Ex. N (ECF No. 54-4).

[11] 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).

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). 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 v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(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 even a generally accepted medical theory. *Andreu ex rel. Andreu v. Sec'y of Dep't 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 Dep't of Health & Hum. Servs*., 121 Fed. Cl. 230, 245 (Fed. Cl. May 6, 2015).

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 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. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (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 den'd*, 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. den'd 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. den'd* (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) (determining that 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).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *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*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to 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. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often 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 per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (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.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) 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. *La Londe 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. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

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

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

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See*, *e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the

credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor 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.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 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. App'x 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. MOGAD and Program Treatment of it as a Vaccine Injury

MOGAD is a rare, antibody-mediated inflammatory demyelinating disorder of the CNS that presents in a variety of ways—including optic neuritis, ADEM, and TM. E. Sechi et al., *Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD): A Review of Clinical and MRI Features, Diagnosis, and Management*, 13 Front Neurol. June 17, 2022, at 2–3, filed as Ex. C Tab 14 (ECF No. 30-25) ("Sechi"); Tr. at 59–60. It occurs when MOG autoantibodies mistakenly attack MOG proteins on oligodendrocytes (the cells responsible for the creation of myelin in the

CNS), leading to nerve demyelination, and then to the clinical symptoms of the disorders associated with MOGAD (like ADEM). Sechi at 3. Significantly, and as acknowledged in this case, the MOG antibody itself has not been *proven* to be pathogenic—hence the name "MOG-antibody-*associated* disease (MOGAD)." Tr. at 60, 172 (emphasis added). MOGAD's exact etiology is unknown, although it is generally thought to occur after an infection or, more rarely, after a vaccination. Tr. at 60–61.

Medical science has only recently identified the MOG autoantibodies and observed their potential relationship to the kinds of CSN-oriented demyelinating injuries that characterize MOGAD. Accordingly, there are few Program cases addressing MOGAD as a vaccine injury. Petitioners have been successful, however, in demonstrating the anti-MOG antibody was vaccine-induced, causing an individual to experience a CNS demyelinating injury. *See, e.g.*, *White v. Sec'y of Health & Hum. Servs.*, No. 15-1521V, 2019 WL 7563239 (Fed. Cl. Spec. Mstr. Dec. 19, 2019) (HPV vaccine caused anti-MOG antibody-mediated TM). I have also so found. *Hock v. Sec'y of Health & Hum. Servs.*, No. 21-945V, 2024 WL 3826125 (Fed. Cl. July 12, 2024) (holding Petitioner satisfied his burden of proof for his claim that the influenza vaccine caused his MOGAD); *L.C. v. Sec'y of Health & Hum. Servs.*, No. 17-722V, 2021 WL 3630315 (Fed. Cl. July 2, 2021) (finding Tdap vaccine caused MOGAD in a minor).

I note, however (and as reflected in my own decisions involving MOGAD), that there is a paucity of scientific understanding of this new diagnostic classification—and hence that the showing deemed preponderant in cases involving MOGAD was still somewhat limited. *See, e.g.*, *L.C.*, 2021 WL 3630315 at *17–19; *Hock*, 2024 WL 3826125 at *19–20. For example, in *Hock*, I found the Petitioner satisfied causation because despite the somewhat-underwhelming evidence applicable to causation, Respondent did not sufficiently rebut the factors suggesting Petitioner's showing was preponderantly established. 2024 WL 3826125, at 20–22.

Thus, while there is nascent Program recognition that vaccination *might* play a role in the propagation of these MOG antibodies, the question is far from resolved. It cannot be said based on the paltry number of decisions that the Program *favors* a vaccine-MOGAD connection. And, as I have indicated before, I am not otherwise prepared to find in favor for *all* petitioners that bring forward MOGAD claims post-vaccination, just because the injury involves demyelination and/or is believed in part to be associated with certain autoimmune neuroinflammatory conditions. *See, Hock*, 2024 WL 3826125, at *19. This is especially so given that MOGAD-specific literature remains limited[12] (likely related to the fact that widespread commercial testing for the autoantibody only became available in the past decade). The understanding of MOGAD will evolve as medical science continues to study this disease—and this in turn ensures variability in

[12] Dr. Rizvi conceded that there are no large epidemiological studies focusing on MOGAD. First Rizvi Rep. at 5.

how Vaccine Act cases involving this injury are resolved, with the individual evidentiary showings made from one case to the other most dispositive.

II. Petitioner Has Not Carried Her *Althen* Burden of Proof[13]

A. *Prong One*

Petitioner contends that that the Tdap, MMR, and/or Varicella vaccine can cause MOG-positive ADEM or MOGAD. In support, Dr. Akbari (Petitioner's primary expert on the immunologic issues relevant to causation) offered a lengthy, multi-step theory that requires the vaccines in question to impact every step of the immune process in order to reach the conclusion that the vaccines caused the Petitioner's injury.

I am familiar with the contours of the theory Dr. Akbari has espoused (which implicates the inflammasome and its impact on the innate response, and then goes on to propose a role for the putatively-causal vaccine to affect Treg cells and the adaptive immune response in producing cross-reactive autoantibodies). I have actually heard him personally propose it before—but I deemed the theory unreliable and insufficiently corroborated by independent scientific or medical evidence. *Efron v. Sec'y of Health & Hum. Servs.*, No. 20-1405V, 2025 WL 408219, at *21 (Fed. Cl. Spec. Mstr. Jan. 2, 2025) (denying entitlement to petitioner who could not show by a preponderance of the evidence that his anhidrosis could be vaccine-caused; noting that Dr. Akbari's causal theory lacked sufficient evidence to support it).

The theory was no better substantiated in this matter. First, Petitioner has failed to show a causal connection between inflammasome activation and ADEM or MOGAD. The literature offered does not support the conclusion that vaccines pathologically impact inflammasome stimulation. For example, Pulendran & Ahmed does not state that the MMR vaccine triggers inflammasome activation. At best (and only with respect to the Tdap vaccine), it references *other* studies that have demonstrated that alum (an adjuvant contained in some vaccines) impacts the function of the inflammasome, but the article concedes that the process is poorly understood. Pulendran & Ahmed at 4. Dr. Forsthuber offered a study that confirms how little is known about the impact of alum adjuvant-containing vaccines on the inflammasome. H. Hogenesch, *Mechanism of Immunopotentiation and Safety of Aluminum Adjuvants*, 3 Front Immunol. 406 (2013), filed as Ex. G Tab 4 (ECF No. 42-5). And Dr. Akbari otherwise did not corroborate the larger connection between vaccine-induced inflammasome activation and ADEM or MOGAD—and therefore his arguments about its initial, critical role in what follows amount to trying to "spin" an acknowledged feature of the innate immune system response into something pathologic.

[13] I address herein only the *Althen* prongs relevant to my analysis, since all three prongs must be met to obtain entitlement. *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 566 F. App'x 976, 979 (Fed. Cir. 2014).

Second, Dr. Akbari failed to explain how innate immune cell activation at the injection site would cause any kind of immune cells to travel from the periphery of the body to the CNS. In reaction, Dr. Forsthuber offered more compelling evidence that vaccine-induced inflammasome activation is localized, and any elevation in the presence of pro-inflammatory cytokines is still far below the number of cytokines expected to influence Tregs, or trigger ADEM or MOGAD. Tr. at 399–400 (discussing de Wolf).

Dr. Akbari's contentions about the negative impact of Treg cells in promoting autoimmune diseases were also not persuasively tied to vaccination. This component of his theory posited that the dysregulation of these cells prevents them from successfully suppressing autoimmune reactions resulting in disease. But he has not specified *how* those cells become dysregulated by vaccination—resorting instead to the conclusory reasoning that vaccines can cause dysregulation in those with a genetic predisposition (without also establishing *what* Petitioner's genetic propensity was, let alone offering sufficient evidence that Treg cells are critical to the pathogenesis of ADEM and other autoimmune diseases—even if immune tolerance breakdown plays some role in such conditions). Tr. at 209; First Akbari Rep. at 12. And Dr. Forsthuber (again) effectively rebutted such points, offering evidence that vaccines and infections could actually *increase* the number of Tregs (and thus prove to be encouraging of immune resistance to autoimmune reactions). Second Forsthuber Supp. Rep. at 9 (citing K. Mills, *Designer Adjuvants for Enhancing the Efficacy of Infectious Disease and Cancer Vaccines Based on Suppression of Regulatory T Cell Induction*, 122 Immunol. Lett. 108 (2009), filed as Ex. G Tab 9 (ECF No. 42-10), at 110.

Otherwise, little in the way of more specific evidence that the vaccines at issue have been shown to spur the immune system to create MOG-antibodies via molecular mimicry has been offered. And Petitioner's experts did not successfully identify possible sequences or similar homologic structures between components of the vaccines and the MOG protein. Dr. Rizvi offers support for molecular mimicry between vaccines and CNS diseases unrelated to what the Petitioner experienced, while failing to identify a vaccine or vaccine component that could cause ADEM or MOGAD. Tr. 131:2–16. Dr. Akbari also failed to identify any homologies or structural sequences between the MOG-antibody and the varicella or MMR vaccines, and dismissed the need for a structural or sequential homology. Second Akbari Rep. at 9–10. While this kind of showing is not required of claimants, their experts often spend a great deal of time hoping to substantiate it anyway. *See, e.g., A.T. v. Sec'y of Health & Hum. Servs.*, No. 16-393V, 2021 WL 6495241, at *23–25 (Fed. Cl. Dec. 17, 2021); *L.R. v. Sec'y of Health & Hum. Servs.*, No. 16-922V, 2024 WL 1912575, at *18–21 (Fed. Cl. Mar. 28, 2024).

At most, Dr. Akbari referenced the Virupakshaiah case study as establishing sequence homology between tetanus toxoid protein components and the MOG-protein. But its authors did not conclude that Tdap vaccine had a causal role in the development of MOGAD, and merely

identified two T-cell epitopes of tetanus toxoid that share an amino acid sequence with MOG-specific T cells in *MS* patients. Virupakshaiah at 5. This is not enough of a basis upon which to conclude that a tetanus-containing vaccine will likely lead to a cross-reactive process mediated by autoantibodies *and* resulting in MOGAD. By contrast, Dr. Forsthuber found no significant similarity between the amino acid sequence of TT and MOG based upon his own searches. Forsthuber Blast at 3–4. While I do not give great weight to BLAST searches as a general matter (since a showing of homology alone is never enough to prove causation),[14] it is telling in this case that Petitioner's immunology expert made little effort to establish homology—while Respondent's expert offered reliable evidence, based on his own review (and one which he was qualified to conduct), to refute its likelihood.

Molecular mimicry is in this case (as in many others) invoked as the mechanistic "skeleton key" that unlocks how an autoimmune disease process may have occurred. But it has not been fleshed out with sufficient corroborative evidence specific to the vaccines at issue or to MOGAD, and thus alone does not carry the day for Petitioner on the issue of causation. *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)); *Forrest v. Sec'y of Health & Hum. Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019), ("[A] simple invocation of the term 'molecular mimicry' does not carry a petitioner's burden of proof. As explained by the Court of Federal Claims, 'Without any empirical evidence that the theory actually applies to the [vaccine and the disease in question], the first prong of *Althen* would be rendered meaningless.'" (*quoting Caves v. HHS*, 100 Fed. Cl. 119, 135 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012) (per curiam)).

At bottom, Dr. Akbari's theory seeks to accomplish what many other unsuccessful claimants attempt: describe expected ways in which vaccines impact the immune system, and then convert that into a narrative of an aberrant process resulting in illness. But a theory that requires the vaccine to impact the immune system at so many points (stimulating the production of cytokines, defeating the immune system's self-regulating safeguards, and then encouraging an aberrant adaptive immune response due to the creation of cross-reacting MOG autoantibodies) cannot be found to have met the "more likely than not" standard unless its various subcomponents

[14] Indeed, I have found for Petitioners in MOGAD cases even where they *did not* show structural or sequential homologies between a vaccine and the MOG-protein. *See, e.g.*, *Hock*, 2024 WL 3826125; *L.C.*, 2021 WL 3630315. But I did so because of MOGAD's novelty as a diagnosis, the fact that the vaccines at issue had a history of being associated with comparable CNS demyelinating diseases, and because both parties had agreed that the "relevant cross-reactive process herein is 'more complicated than simple amino acid sequence homology.'" *Hock*, 2024 WL 3826125; *L.C.*, 2021 WL 3630315, at *19.

have preponderant support. Here, they do not.[15] And Dr. Akbari's opinion was soundly rebutted by Dr. Forsthuber, a very qualified immunologist who provided cogent testimony. I ultimately found Dr. Forsthuber far more persuasive on the causation question than Dr. Akbari. It is well-recognized in the Program that special masters are not compelled to accept the *ipse dixit* of experts at face value. *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co.,* 522 U.S. at 146). And in this case, the overall balance of evidence favored Respondent, even if Dr. Akbari possessed sufficient personal expertise on immunologic matters to propose a causation opinion.

I thus do not find that the "can cause" prong was established, given the mix of evidence and testimony presented *in this case*. This is not to say that "the door is closed" (from the Program's standpoint) on the question of whether MOGAD could be vaccine-associated. The subject is very much under study at this time—and as more medical and scientific evidence is generated on the topic, it will likely become easier to ascertain in Vaccine Act cases the strength of the putative vaccine association. But it certainly cannot be *presumed* that MOGAD is likely vaccine caused, in some or all circumstances. Rather, the discovery of the MOG antibody raises questions about its role in propagating disease, as well as its origins—and as science evaluates the matter, it is likely that vaccine injury cases involving MOGAD will continue to turn on the strength of evidence offered in the case at hand.

B. *Prong Two*

As revealed from the discussion of the trial testimony, Respondent challenges Petitioner's MOGAD diagnosis. Opp. at 15–16. He contends that Petitioner has not provided sufficient evidence, in the form of supporting clinical or MRI features, to establish MOGAD, especially given the low-positive MOG antibody titer levels revealed in testing. *Id.* at 16–17.

Respondent's arguments have independent support. For example, authority establishes that a low positive MOG-IgG test must usually be supplemented with evidence of both a negative AQP4-IgG test and at least one supporting clinical or MRI feature to satisfy the MOGAD diagnosis requirement. Banwell at 9 Fig. 3. Here, however, although Petitioner did obtain a negative AQP4-IgG test, the MRI results were not sufficient to confirm MOGAD. Ex. 13 at 290–91. And Dr. Roos

[15] I give relatively little weight to Kumar—the case study specific to the Petitioner herself. It is factually accurate, but it observes consistently the limitations of its findings, which are largely based on the temporal relationship to vaccination (and it notes other associations between vaccination and diseases featuring demyelination, like MOGAD, are also temporal only). Kumar at 87. It relies largely on other case reports or series to reach its conclusions. *Id.* at 90. It seems to assume that vaccination's aberrant impact was heightened by Petitioner's concurrent pregnancy and illness. *Id.* at 89. And most significantly, it is foundationally difficult to find probative a case report *written about the relevant petitioner*—since the speculation it raises about causation is the very subject of this matter, and numerous items of reliable evidence pro and con were offered. To find a petitioner-specific case report to be worthy of evidentiary weight (e.g., to rule that "a case report about a petitioner's adverse vaccine reaction supports her contention that she had an adverse vaccine reaction"), I would require additional corroborative evidence that the case report's speculation was later shown to be more than that.

noted other aspects of Petitioner's presentation that were not clinically consistent with ADEM (a form of how MOGAD might present). Tr. at 258–63.[16]

But even assuming that Petitioner's MOGAD diagnosis was correct, she has failed to carry her burden of preponderantly establishing that the vaccines she received "did cause" that injury.

To begin, the record lacks evidence that Petitioner experienced *any* kind of reaction from the vaccines. Tr. at 15. Petitioner was clearly experiencing infectious symptoms during the timeframe in which she was vaccinated, but these cannot be linked to the vaccines (and they do not reflect the kinds of symptoms often viewed as vaccination-associated "malaise"). And there is no evidence that would establish the presence of vaccine-associated inflammation (which Dr. Akbari's theory of inflammasome stimulation would require for corroboration).

This leads to the second, and more compelling, evidentiary factor militating against a finding of causation: the Petitioner's obvious URI symptoms. The etiology of those symptoms cannot be ascertained on this record. But their *existence* and *severity* is plain. Thus, the record clearly shows that (a) Petitioner was very ill, with little improvement, for a week or more prior to her receipt of the Tdap vaccine, and (b) she experienced comparable, recurrent symptoms a few days after the second vaccination event on April 27, 2018. Ex. 23 at 117–19, Ex. 23 at 15–30, 50; *see also* Tr. at 14. Only by April 30th did she begin to report symptoms that could be reflective of a neurologic condition (urinary frequency and urgency)—and while this occurred post-vaccination, it was also in the wake of her confirmed URI treatment on two occasions.[17] And she admitted in her testimony (as corroborated by record proof) that her illnesses predated vaccination—and there is other proof suggesting Petitioner told contemporaneous treaters that she might have become ill after being exposed to a relative who had traveled from abroad (although she disputes how ill this individual actually was).

All of the foregoing supports the conclusion that both before and then during the time Petitioner was vaccinated, she was also ill with some kind of infectious process. And an infection

[16] The evidence far more strongly preponderates against the conclusion that Petitioner experienced ADEM, since (as Dr. Roos opined) the Petitioner's symptoms did not align with recognized diagnostic criteria. First Roos Rep. at 8. The Petitioner experienced several weeks of URI symptoms that developed after visiting her sick relative, test results showed "abnormally high echovirus" titers, Petitioner's physicians did not have an explanation for her coagulopathy—which is reported in echovirus 7 infections, negative subsequent anti-MOG antibody test results during the "acute period" of the affliction, and testing and post-hospitalization neurological testing and provider visits shows that her symptoms abated approximately three months from her onset—meaning she did not experience a neurological deficit lasting at least six months. *Id*. at 8–9.

[17] The record also reveals that some of Petitioner's symptoms as of April 30, 2018, could be attributable to an ectopic pregnancy. *See* Ex. 21 at 288; Ex. 23 at 191–92. However, this does not appear to have played a role in her subsequent illness, and I deem arguments by Drs. Rizvi or Akbari that Petitioner's pregnant condition was an aspect of her course later to be speculative.

could be causal of MOGAD, as literature filed in this case allows. *See* Sechi at 13; Banwell at 5 (stating that infectious episodes—mostly respiratory—often preceded ADEM, which is a presentation of MOGAD). If so, this undermines Petitioner's contention that the vaccines she received were also a substantial factor in causing her MOGAD.[18] And her experts did not adequately diminish the significance of this evidence. Of course, claimants need not affirmatively *exclude* all alternative factors that could explain an alleged vaccine injury. *Efron*, 2025 WL 408219, at *22 (citing *M.R. v. Sec'y of Health & Hum. Servs.*, No. 16-1024V, 2023 WL 4936727, at *30 (Fed. Cl. Spec. Mstr. June 30, 2023)). But they have *some obligation* to grapple with the record when it establishes the existence of such countervailing factors—and Petitioner failed here to do so.

There is also the proof of an echovirus infection as the possible causal infection. Respondent has made some reasonable points about echovirus—how it could be consistent with Petitioner's symptoms, the fact that she did test positive for it, that it can be neurovirulent, and the fact that the timing of the testing result did not preclude the possibility that such an infection existed at the time of Petitioner's onset of MOGAD-like symptoms. In response, Petitioner's experts unpersuasively denied the significance of testing confirming the presence of echovirus (while simultaneously calling upon me to find the very low titer levels of MOG antibodies revealed by testing to be strong evidence of vaccine causation). First Rizvi Rep. at 10; Second Akbari Rep. at 23. Dr. Rizvi in fact acknowledged the possibility of echovirus as the cause—and although he argued that echovirus is more common in children, and that he was unaware of evidence supporting a connection with MOGAD, the evidence associating *vaccines* with MOGAD is not all that much more robust. First Rizvi Rep. at 10; Second Rizvi Rep. at 3.

The fact that Petitioner herein was unquestionably ill at the time of vaccination distinguishes this case from the other MOGAD cases I have decided where causation was found. In *L.C.*, for example, a child's neurologic symptoms occurred a few weeks after vaccination, but in a context of general good health, with far less direct evidence that the child had been previously sick. *L.C.,* 2021 WL 3630315, at *1. The evidence that the *Hock* petitioner was experiencing some kind of intercurrent infection in the weeks prior to vaccination was a bit stronger, but that Petitioner had largely recovered by the time he received the flu vaccine (which this Petitioner was not administered). *Hock,* 2024 WL 3826125, at *21. I deemed this kind of evidence too inconclusive to give significant weight in those cases. But in this matter, the evidence of a preexisting, concurrent illness is substantially greater.

Petitioner can point to instances in the record where treaters allowed for the possibility of

[18] Petitioner has argued that she can still prevail even if other factors (the echovirus infection, her intercurrent illness, etc.) were partially causal, because claimants only need show that a vaccine was a "substantial factor" and "but for" cause of their injury. Petitioner's Pre-Hearing Brief, dated Aug. 12, 2024 (ECF No. 43) at 30 (citing *Shyface*, 165 F.3d at 1352); Br. at 25. But that only holds if I *first* had found that the vaccines at issue could cause MOGAD—and I do not so find in this case (nor have Respondent's experts conceded vaccine causation).

vaccine causation. While such views are entitled to some evidentiary weight, they are not *per se* controlling of the outcome. *Al-Uffi v. Sec'y of Health & Hum. Servs*., No. 13-956V, 2017 WL 1713113, at *13 (Fed. Cl. Spec. Mstr. Feb. 22, 2017). In addition, some of these opinions appear to have reflected speculation or reliance on unreliable case studies. For example, Dr. Abboud noted that Petitioner's symptoms could be related to a demyelinating process, including ADEM post-vaccinal demyelination, or an atypical CNS infection. Ex. 21 at 327–28. But this observation seemed to reflect reasoned speculation based on timing between onset and receipt of vaccines, rather than an informed interpretation of the overall record (including Petitioner's illness throughout the time of her vaccination). Dr. Joseph also proposed that Petitioner's symptoms could reflect an adverse reaction to the rubella compound of the MMR vaccine. Ex. 21 at 368. But she relied expressly on Holt - an outdated case report that does not involve MOGAD at all. Tr. at 62.

Dr. Yasmin provided a bit more substantiation for his view of a possible vaccination association. *See* Ex. 13 at 399. But he hypothesized that either *an infection or a vaccination* cross-reacted with a nerve component via molecular mimicry to cause Petitioner's condition. *Id.* That view is consistent with the opinions offered by Petitioner's experts—and does not exclude the possibility of a solely-viral cause (something Petitioner did test positive for—findings reached *after* Dr. Yasmin expressed this view).[19] It otherwise is not strong support for the conclusion that vaccine and infection acted synergistically to cause Petitioner's MOGAD—especially since I am finding it not preponderantly demonstrated that any of the vaccines Petitioner received can cause it.

What remains is the fact that Petitioner's most likely neurologic symptoms arose within two weeks of her first vaccination event. But as the Circuit has stated, "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 1274 at 1278 (citing *Grant*, 956 F.2d at 1149). Petitioner has not met that burden. Despite the fairly robust evidence that Petitioner was ill before and around the time of her vaccinations, I cannot on this record identify *what* explanation for Petitioner's general complaints has the most preponderant support (and I do not *ever* purport to diagnose the cause of any Program claimant's injury in performing my duties as a special master). But the overall murkiness of this evidentiary balance prevents a finding that one, or a combination, of Petitioner's vaccines were likely a substantial factor in causing MOGAD.

[19] Dr. Yasmin expressed his view on May 7, 2018, but Petitioner's test results showing abnormally high echovirus antibody levels were reported later, on May 13, 2018. ECF 14-2 at 135, 399.

CONCLUSION

Despite the uncertainty still surrounding MOGAD, I can determine *in this case*—and based on the evidence before me—that Petitioner did not carry her preponderant burden of showing causation. I am therefore compelled to deny 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.[20]

IT IS SO ORDERED.

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

[20] 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.