

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-945V

JUSTIN HOCK,	*	Chief Special Master Corcoran
	*	
Petitioner,	*	Dated: July 12, 2024
	*	
v.	*	
	*	
SECRETARY OF HEALTH AND	*	
HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

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

Eleanor Hanson, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT¹

On February 18, 2021, Justin Hock filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the “Vaccine Program”).² Petitioner alleges that an influenza (“flu”) vaccine he received on October 8, 2018, caused him to develop myelin oligodendrocyte glycoprotein antibody-associated disease (“MOGAD”). ECF No. 19. A two-day Entitlement Hearing was held on December 14-15, 2023. Now, having heard the witnesses at hearing and reviewed the record, I find Petitioner is entitled to 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 Ruling will be available to the public in its present form. *Id.*

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

I. Factual Background

Pre-Vaccination History

Mr. Hock was born on November 14, 1985. Ex. 2 at 6. Prior to receiving the vaccine at issue, he had no documented history of neurological disease (although the parties dispute whether his MOGAD might have predated vaccination). Petitioner was formerly an IV drug user, and sustained a gunshot wound to his left leg in 2016. Ex. 2 at 10, Ex. 3 at 13 (notes residual neuropathy from gunshot wound), Ex. 4 at 13–14, Ex. 7 at 11.

On September 18, 2018 (approximately three weeks before receiving the flu vaccine at issue), Petitioner visited an emergency room with complaints of leg pain, lower back pain, and neck pain that he reported had begun around 10 days prior. Ex. 4 at 11. He also complained of a “running sensation” in both thighs. *Id.* After an examination (but no imaging) yielded normal results, Petitioner was prescribed muscle relaxers and pain medication and discharged. *Id.* at 14. Then, about a week later (September 24, 2024), Mr. Hock saw a provider at Mian Family Medicine in Rosedale, Maryland, for a sore throat and ear pain. Ex. 3 at 19. He reported that his neck and back pain had improved since the ER visit. *Id.* He was diagnosed with an unspecified upper respiratory infection, and prescribed amoxicillin and a cough suppressant. *Id.* at 21.

Vaccination and Initial Evidence of Neurologic Symptoms

Petitioner received the flu vaccine during a routine appointment at Mian Family Medicine on October 8, 2018. Ex. 3 at 18. He did not report any complaints during this visit (and thus did not reference the seemingly infection-related symptoms he had recently experienced—or his earlier September issues). *Id.* at 17. There is no record evidence of any immediate vaccine reaction, and no symptoms were reported in the period between vaccination and Petitioner’s temporally-next medical record.

Three weeks later, on October 29, 2018, Petitioner presented to the Franklin Square emergency room in Baltimore, Maryland, with complaints of constant neck and back pain. Ex. 4 at 65. He did not report numbness or tingling, but had difficulty with motor skills and picking up objects like his phone. *Id.* at 74. Petitioner also at this time noted some urination difficulties, and that he had not urinated since the day before. *Id.* at 65, 76.

A neurological exam and head CT were normal. Ex. 4 at 75–76. An MRI showed a disc protrusion at C4-5, and disc bulges at L3-4 and L5-5. *Id.* There were no other significant spinal abnormalities (including lesions) identified, but the MRI lumbar imaging revealed that Petitioner’s bladder was distended to the middle of the lowest lumbar vertebrae. *Id.* Lab work showed elevated levels of white blood cells, and mildly elevated C-reactive protein (an

inflammation biomarker). *Id.* at 76–77. Given all of the above, treater impressions centered on neck pain as the primary concern, and Petitioner was treated with steroids, discharged, and told to follow up with a neurologist. *Id.*

The next day, Petitioner saw his primary care provider, Dr. Jamshid Mian. Ex. 3 at 13. Dr. Mian discussed Petitioner’s ER visit the day prior, and Petitioner now reported that he had bladder sensitivity, sinus pressure, and postnasal drip. *Id.* Petitioner was diagnosed with acute sinusitis, inflammatory spondylopathy, cervical arthritis, white blood cell disorder, and bladder disorder. *Id.* at 13–14. He was prescribed Naproxen. *Id.*

Petitioner thereafter saw a urologist, Dr. Kannan Manickam, on November 1, 2018. Ex. 10 at 13. He specifically complained of “urine problems” (difficulty urinating). *Id.* His post-void urine residuals measured 228 ml—an abnormal finding.³ *Id.* at 15. Dr. Manickam concluded that Petitioner’s urinary issues were most likely neurologic in nature. *Id.* Several days later, on November 5, 2018, Petitioner saw neurologist Dr. Ali Kooshkabadi, with complaints of hand weakness and neck pain that he now reported (as the face of this record indicates) had begun “six months earlier,” but had dramatically worsened in the more recent months. Ex. 11 at 7. A neurological exam was normal, however, and Petitioner made no mention of the bladder issues that he had been reporting to other treaters. *Id.* at 8. Dr. Kooshkabadi diagnosed Petitioner with cervical spondylosis, left arm weakness, and cervical spine stenosis, and prescribed physical therapy and anti-inflammatories. *Id.*

Petitioner saw Dr. Manickam again on November 7, 2018, for an urgent visit, as he was still unable to urinate. Ex. 10 at 10. On repeat testing, his post-void urine residuals measured more than 1000 ml. *Id.* He was prescribed self-catheterization two to three times daily. *Id.* at 12. Later that day, Petitioner presented to the MedStar Harbor Hospital ER for his ongoing issues (neck and back pain plus inability to urinate). Ex. 5 at 12–13. The treating physician, Dr. Neil Majmundar, noted that a neurological exam was unremarkable, and did not order imaging. *Id.* at 16–17. Petitioner’s urine tested positive for “rare bacteria, but no other signs of infection.” *Id.* at 18. Dr. Majmundar attributed Petitioner’s urinary issues to hygiene, but told him to follow up with a neurologist and PCP. *Id.*

Hospitalization

Petitioner was hospitalized at Johns Hopkins Hospital in Baltimore, Maryland from November 9-14, 2018, after going to the ER again on November 9th. Ex. 6 at 8. He reported to

³ A post-void residual volume over 200 mL indicates inadequate emptying. A volume over 300 mL is suggests urinary retention, and a volume over 400 mL confirms urinary retention. L. Ballstaedt et al., *Bladder Post Void Residual Volume*, NCBI Bookshelf at <https://www.ncbi.nlm.nih.gov/books/NBK539839/#:~:text=Less%20than%20100%20mL%20PVR,is%20suggestive%20of%20urinary%20retention>. (last accessed June 24, 2024).

initial emergency treaters the same symptoms of neck and back pain and urinary retention, but also a new symptom of progressive numbness. *Id.* at 11. Preliminary exams showed sensation “intact, but diminished distal to the T4 level,” but normal gait and motor strength. *Id.* at 12–13. Further neurological exams by neurologist Dr. Carlos Pardo-Villamizar showed hyperreflexia, sensory loss around T10/11, and pain with eye movement. Ex. 6 at 26.

An MRI performed on November 10, 2018, revealed “multiple small foci of faint enhancement in the bilateral frontal and temporal lobes, and T2/FLAIR hyper-intensity in the frontal lobe without cranial nerve enhancement.” Ex. 6 at 29. Lab work showed elevated proteins in the cerebrospinal fluid, and an elevated white blood cell count. *Id.* at 18, 49, 53. A neurosurgeon evaluating these results deemed the imaging to be concerning for a neuro-inflammatory or infectious process. *Id.* at 27–30. Dr. Pardo characterized the imaging as “highly suspicious for a demyelinating myelopathy,” and considered neuromyelitis optica (“NMO”), MOGAD, and rheumatic myelopathies as possible diagnostic explanations, to be explored through further testing. *Id.* at 21. Petitioner was started on a three-day course of methylprednisolone. *Id.*

Petitioner was later examined by a neurology fellow on November 12th and 13th. Ex. 6 at 32. He still reported eye tenderness, neck pain, and urinary retention, but added that his numbness had improved. *Id.* at 32–33. An exam showed decreased fine touch and pin prick sensation on his left leg up to the T12 level. *Id.* at 35. He continued to receive methylprednisolone. *Id.* The next day, Petitioner was able to urinate on his own and have a bowel movement, and no longer was experiencing numbness. *Id.* at 39.

Mr. Hock was subsequently discharged, but without a final diagnosis. Ex. 6 at 48–49. Lab work for NMO and MOG antibodies was still pending at this time. *Id.* However, an optical coherence tomography (“OCT”) test⁴ performed while Petitioner was hospitalized showed chronic injury to *both* optic nerves—supportive of the existence of a demyelinating process that might have been present for some time. *Id.* at 49. Petitioner was referred to outpatient neurology and prescribed a slow prednisone taper. *Id.*

December 2018 Testing and Treater Opinions on Nature of Presentation

Petitioner’s first post-discharge treater visit occurred on December 5, 2018, when he saw Dr. Pardo and a neuroimmunology fellow at the Johns Hopkins Outpatient Transverse Myelitis Center (“Johns Hopkins OTMC”). Ex. 6 at 287–88. Petitioner’s lab work taken during the hospitalization had resulted in a positive MOG antibody test, with high titers at 1:1000, while

⁴ An OCT is a non-invasive imaging test that uses light waves to take cross-section pictures of the retina. This allows ophthalmologists to measure the thickness of the retina, helping to diagnose illnesses like glaucoma and other retinal diseases. *What is Optical Coherence Tomography?*, American Academy of Ophthalmology, at <https://www.aao.org/eye-health/treatments/what-is-optical-coherence-tomography> (last accessed June 24, 2024).

Petitioner tested negative for NMO antibodies. *Id.* Dr. Pardo also reviewed his OCT results from the November hospitalization, which (as noted above) “suggested there was already some *established* damage to both optic nerves” at the time of this testing. *Id.* at 292 (emphasis added). Petitioner reported that he was still unable to urinate, and had to self-catheterize five to eight times a day. *Id.* at 288.

Based upon the clinical exam, symptoms, as well as recent test results, Dr. Pardo characterized Mr. Hock’s overall presentation as consistent with a “MOG-related neuroinflammatory disorder.” Ex. 6 at 287. However, although Dr. Pardo identified onset as having occurred in mid-October 2018 (manifesting “with inflammation of the spinal cord, nerve roots, meninges (patchy) and probably optic nerve”) his comment about the results of the OCT testing suggested a relapsing disease course, which could have begun prior to Petitioner’s initial complaints. *Id.* at 292 (“anti-MOG antibodies can be associated with either monophasic or relapsing disease”). Mr. Hock was prescribed another course of prednisone, and told to come back for a repeat MRI, OCT, and anti-MOG testing in six weeks, so that it could be determined if the disease course had subsided (since “there is some evidence that patients who revert to a negative antibody after an attack are less likely to experience a relapsing disease course”). *Id.*

The notes from Petitioner’s December 5th visit (prepared principally by Olwen Cait Murphy, MBChB)⁵ also memorialize the fact that Petitioner’s case was subsequently discussed during a neuroimmunology case conference at Johns Hopkins on December 6, 2018. Ex. 6 at 292. Treeters who at this time collectively evaluated Mr. Hock’s overall history since October “felt that the OCT supports prior inflammation, *suggesting that disease may already be considered relapsing rather than monophasic.*” *Id.* (emphasis added).

Petitioner visited the Johns Hopkins OTMC on December 30, 2018, for a follow-up OCT. Ex. 6 at 302–03. This second OCT revealed that the “mean peripapillary retinal nerve fiber layer thickness was normal for each eye,” but “the retinal nerve fiber superior quadrant on the left,” “average macular thickness on the right,” and both GCIP (ganglion cell inner plexiform layers) were thinned. *Id.* The ophthalmologist who performed the OCT testing noted that its results established “the presence of bilateral optic neuropathies,” and recommended yearly exams. *Id.* No comment was made as to how long the damage might have existed, however.

Subsequent Treatment

Despite his somewhat acute post-vaccination presentation and the concerning nature of his symptoms, Petitioner’s treatment diminished from the start of 2019 thereafter. For example,

⁵ The MBChB is the equivalent of an MD for countries that adhere to the UK’s higher education system. NEIL M. DAVIS, MEDICAL ABBREVIATIONS (16th ed. 2020)

Mr. Hock saw his PCP on January 28, 2019. Ex. 3 at 7. He reported feeling better, but had coughing and post-nasal drip. *Id.* He also reported that a neurologist had told him not to get the flu vaccine in the future, asking that this be noted on his chart. *Id.*⁶ He saw the same PCP again on February 25, 2019, but reported no concerns besides asking for a Cialis prescription. *Id.*

Petitioner underwent MRI imaging again on February 26, 2019. Ex. 9 at 8. Treater notes in connection with this event stated the existence of “prior episodes of upper and lower extremity weakness with incontinence. Effects from flu shot.” *Id.* The brain MRI showed signal abnormalities in the periventricular and deep white matter of the right parietotemporal area, in the periventricular white matter of the left periarterial region, and also a tiny signal abnormality in the deep white matter of the left frontal region. *Id.* His spinal imaging was unremarkable. *Id.* at 9–11. Treater noted that this might be evidence of CSF suppression, or sequelae of a prior demyelination or infection, although no active demyelination was observed. *Id.* at 8.

Petitioner visited the Johns Hopkins OTMC again on March 13, 2019. Ex. 7 at 96. He reported that he still self-catheterized several times a day, but could sometimes urinate with a “strange sensation.” *Id.* at 101. He had no more neck or back pain, but had issues ejaculating. *Id.* at 97. He also reported some peripheral vision blurring when looking from left to right. *Id.* The neurologist recommended continued monitoring and follow-up MOG testing. *Id.* at 101.

Petitioner’s most recent records, based on the materials filed in this case, are from a visit with Dr. Pardo on September 18, 2019. Ex. 7 at 120. He was now stable in terms of symptoms, but his urinary issues remained. *Id.* Dr. Pardo recommended repeat MRI and MOG antibody tests, as well as a test for Sjogren’s antibodies. *Id.* He also recommended placement of a bladder stimulation device if Petitioner remained stable. *Id.* This appears to be the last time Petitioner received neurologic-specific treatment, and no records from any subsequent temporal period have been filed.

II. Hearing Witnesses

A. Petitioner’s Witnesses and Experts

1. *Justin Hock* - Mr. Hock (the sole fact witness) testified that, a few weeks prior to receiving the vaccine at issue, he had visited the Franklin Square Medical Center Emergency Room with complaints of back and neck pain, with a running sensation in the thighs. Tr. at 8. This pain had lasted for around ten days, and intensified when he was sitting or laying down. *Id.* at 9. He was prescribed ibuprofen and Flexeril, and the pain subsided shortly after. *Id.* at 10. He attributed the pain to long periods of sitting while driving for work, and possibly from keeping his wallet in his back pocket while sitting. *Id.* at 9. He noted that he had thereafter

⁶ I have not been able to identify a record in which this recommendation was made to Petitioner.

stopped sitting on his wallet after the ER visit, and speculated that this may have contributed to his symptoms resolving. *Id.* at 10. Mr. Hock also saw a primary care physician for a sore throat and ear pain on September 24, 2018, and was prescribed antibiotics, but remembered nothing else about this incident. *Id.* at 10–11.

On October 8, 2018, Mr. Hock visited the same PCP and received the flu vaccine. Tr. at 11. He testified that he did not typically get flu shots, but was “trying to be responsible” after the birth of his daughter. *Id.* at 11–12. Toward the end of October, he went to the Franklin Square Medical Center ER. *Id.* at 12. He was in “excruciating pain” around his head, neck, and shoulders. *Id.* He had been experiencing great difficulty urinating for two days, and had deteriorating motor skills, with difficulty grabbing objects and picking them up. *Id.* at 13. At the ER, he had MRI and CAT scans performed. *Id.* He was sent home with Motrin (or a similar pain medication) and steroids. *Id.*

Thereafter, however, Petitioner continued having “extreme urinary retention,” and visited a urologist twice during November 2018. Tr. at 14–15. A sonogram showed an extremely full bladder, and he could not urinate to complete a stream test the doctor requested. *Id.* at 15. He was prescribed self-catheterization. *Id.*

During the same time period, Mr. Hock experienced “excruciating” pain in his neck, pain in his legs, and continuing loss of motor function in his hands. Tr. at 16. He saw a neurologist with a referral from his prior hospitalization. *Id.* Treaters continued prescribing him steroids, but his symptoms did not improve. *Id.* at 17. He tried going to a different emergency room where a relative worked, hoping to get more attentive treatment. *Id.* at 18. This facility also prescribed him steroids. *Id.* at 18. Two days after this visit, he was admitted at Johns Hopkins. *Id.* at 18. Treaters ran various tests, such as scans and a spinal tap. *Id.* at 19. At this point, his peripheral vision was compromised, and he had very poor balance. *Id.* at 20.

Mr. Hock expressed some recollection of treaters discussing the neurological root of his symptoms during his hospitalization. Tr. at 21. He recalls they told him “the sheathing on my brain stem and my spine was deteriorated,” and cited a possible “infection” as the cause. *Id.* They also said his condition was “similar to MS [multiple sclerosis] and that [he] would be continuously treated as an MS patient.” *Id.* He was given an IV of high-dose steroids, which allowed him to make a bowel movement (which he had trouble doing previously, along with urinating). *Id.* Some numbness in his feet subsided, but his poor balance and neck pain did not. *Id.* at 21–22. He was eventually discharged, and instructed to continue self-catheterization and follow up with a neurologist at Johns Hopkins. *Id.* at 22.

After discharge, Petitioner followed up at the Johns Hopkins Transverse Myelitis Center on December 5, 2018, seeing the same neurologist he saw during his hospitalization. Tr. at 22.

At this point, he still had pain, but was experiencing improvement in using the restroom. *Id.* at 23. He received a formal diagnosis of MOGAD at this visit, and the neurologist said that he could not rule out the flu vaccine as the cause of his symptoms. *Id.* at 24–25. He recommended that Mr. Hock not receive the vaccine again, but that there was no issue with his children receiving it. *Id.* at 25. He also advised Mr. Hock to have his eyes monitored regularly, since it was possible his vision could continue to deteriorate. *Id.*

Petitioner had follow-up visits with his PCP, the Transverse Myelitis Center, and the Wilmer Eye Institute over the coming months. Tr. at 26–27. He believes he was tapered off from steroids during this time, but was not given a comprehensive treatment plan, and was instructed instead to follow up if symptoms flared. *Id.* at 27. By September 2019, Petitioner’s neck pain was gone, although he still had erectile dysfunction, and usually had to self-catheterize to urinate. *Id.* at 27–28. He felt as if his body was “adapting” with different muscles, improving his motor skills and allowing him to urinate normally at times. *Id.* He had returned to work by that point, after being out for an unspecified amount of time previously. *Id.* at 29.

By the time of the hearing, Mr. Hock testified, he no longer needed to self-catheterize, but still finds urination difficult, and requires medication to treat erectile dysfunction. Tr. at 30, 32. He otherwise does not feel significant lower extremity weakness beyond his legs occasionally feeling numb, which he proposed may be from sitting too long. *Id.* He characterized himself as healthy, and stated that he did not take any medications. *Id.*

2. *Syed Rizvi, M.D.* - Dr. Syed Rizvi prepared two reports in this case. Rizvi First Report, dated April 13, 2022, filed as Ex. 12 (ECF No. 20); Rizvi Second Report, dated December 15, 2022, filed as Ex. 68 (ECF No. 26). He also testified at the hearing.

Dr. Rizvi is a neuro-immunologist in practice at Brown Neurology, and a Professor of Clinical Neurology at Alpert School of Medicine at Brown University. Rizvi CV, dated April 13, 2022, filed as Ex. 13 (ECF No. 20-3). He routinely cares for patients with a wide variety of autoimmune conditions, including but not limited to MS and related disorders such as MOGAD and NMO. Rizvi First Report at 1. He is board-certified by the American Board of Psychiatry and Neurology to practice neurology. *Id.* He received his MD from Dow Medical College in Karachi, Pakistan, and completed his residency at Stony Brook University Hospital. Rizvi CV at 1. He received post-graduate training in MS and clinical neuro-immunology, and has practiced in that field since 2000. Rizvi First Report at 1. He teaches medical students, residents, and fellows about the diagnosis and management of MS and related disorders, and has authored over 20 journal articles in his field. *Id.*

Dr. Rizvi began his testimony discussing his professional background and experience seeing MOGAD patients. Tr. at 41–46. He explained that he sees primarily MS patients, with only about “10 percent” of his patients have related disorders like MOGAD and NMO. *Id.* at 43.

He deemed MOGAD a newer diagnosis, although “[e]very year you see a lot more” MOGAD patients in practice. *Id.* at 45. Testing for the disorder did not become commercial until 2017. *Id.* at 49. Some patients he treated for MS previously were later diagnosed as having MOGAD after new testing became available. *Id.*

Dr. Rizvi then briefly explained the nature of MOGAD. Tr. at 49–50. MOG protein makes up a small percentage of the myelin sheath and is found on oligodendrocytes (cells responsible for the creation of myelin in the central nervous system). *Id.* at 50. When autoantibodies mistakenly attack the MOG protein, demyelination occurs, leading to the clinical symptoms of disorders associated with MOGAD. *Id.* As Dr. Rizvi explained, a “spectrum of diseases...fall into the MOGAD category,” including optic neuritis, ADEM, and transverse myelitis (“TM”). *Id.* at 49. MOGAD patients have a “variety of presentations,” including seizures and meningitis. *Id.* at 52. MOGAD is diagnosed with a combination of imaging and testing - optic neuritis and transverse myelitis may appear on scans, and a MOG titer test confirms the diagnosis. *Id.* at 53. Patients are typically treated with steroids, but may also require IVIG and immunosuppressant drugs if they have relapsing symptoms. *Id.* at 55.

Dr. Rizvi then testified about a number of the studies cited in his reports. These articles came to varying conclusions he deemed applicable to Petitioner’s case, either reflecting a similar timeframe to Petitioner’s symptom onset, or establishing an association between demyelinating conditions and the flu vaccine more generally (albeit not specifically in connection with MOGAD itself). Tr. at 59-64; J. Roszkiewicz & Y. Shoenfeld, *Vaccines and Optic Neuritis: Consequence or Coincidence?*, 17 *Immunome Research* 1, 4 (2021) (15-30 days as the most common time of onset between vaccination and optic neuritis), filed on Nov. 11, 2022, as Ex. 61 (ECF No. 23-25); D. Karussis & P. Petrou, *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, 13 *Autoimmunity Reviews* 215, 216 (2014) (mean of 14.2 days between vaccination and CNS demyelination), filed on Nov. 11, 2022, as Ex. 59 (ECF No. 23-23) (“Karussis”). He also discussed a few case reports in which authors associated different demyelinating illnesses with vaccines. *Id.* at 65, 73–76; C. Alicino et al., *Acute Disseminated Encephalomyelitis with Severe Neurological Outcomes Following Viroosomal Seasonal Influenza Vaccine*, 10 *Human Vaccines & Immunotherapeutics* 1969 (case study of patient with ADEM after flu vaccine), filed on December 15, 2022, as Ex. 72 (ECF No. 26-6); W. Huynh et al., *Post-Vaccination Encephalomyelitis: Literature Review and Illustrative Case*, 15 *Journal of Clinical Neuroscience* 1315, 1321 (2008) (case study of patient with optic neuritis and encephalomyelitis, attributed by authors to flu vaccine), filed on April 13, 2022, as Ex. 17 (ECF No. 20-7).

Many of these studies proposed that molecular mimicry constituted a likely mechanistic process for autoimmune disease pathology. Tr. at 63–64, 70–72; N. Kumar et al., *Postvaccination Anti-Myelin Oligodendrocyte Glycoprotein Neuromyelitis Optica Spectrum Disorder: A Case Report and Literature Review of Postvaccination Demyelination*, 22

International Journal of MS Care 85, 88 (2020) (discussing molecular mimicry as a mechanism for autoimmune demyelination), filed on April 13, 2022, as Ex. 23 (ECF No. 20-13). Dr. Rizvi briefly explained molecular mimicry as a theory, admitting that it is “tough to prove experimentally.” *Id.* at 66.

Based on his review of Petitioner’s records, Dr. Rizvi opined that Petitioner had developed MOGAD as a result of receiving the flu vaccine. He first endeavored to distinguish evidence of a possible pre-vaccination onset for Petitioner’s symptoms. Petitioner’s reports of back and neck pain around September 2018, for example, were deemed by Dr. Rizvi to be musculoskeletal rather than “something more central”—perhaps attributable to a pinched nerve. *Id.* at 77. By contrast, he felt that Petitioner first displayed MOGAD symptoms around his ER visit on October 29, 2018. *Id.* at 81. At that point, Petitioner had headaches (along with the preexisting neck pain), was having trouble picking up objects, and could not urinate. *Id.*

This, Dr. Rizvi stated, was “a sign of something going on neurologically.” Tr. at 82. He acknowledged that Petitioner’s MRI was normal at this time, with no signs of lesions. *Id.* at 83. However, Dr. Rizvi deemed this to *undercut* the conclusion that Petitioner’s condition predated vaccination. Had Petitioner been at that point in a MOGAD progression that had begun in September, lesions would not have resolved by October, and thus would definitely have appeared in this scan. *Id.* at 84.

Petitioner’s November 2018 hospitalization and the findings made at this time were further support for MOGAD beginning after vaccination. Tr. at 86. By this time, Petitioner had significant neurological symptoms, including urinary retention, gait problems, and sensory abnormalities. *Id.* Most significantly, an MRI performed in mid-November now showed the existence of enhancing lesions—a clear indicator of demyelination. *Id.* at 88. By this time, Petitioner was reasonably suspected by treaters to have “a myelitis,” and medical professionals were trying to determine why, through testing such as a spinal tap. *Id.* at 88. Confirmation was received by MOG antibody test results from early December. *Id.* at 89. Petitioner’s ongoing bladder issues were also typical of a MOGAD disease course. *Id.* at 91.

Dr. Rizvi rejected the possibility of other explanations for Petitioner’s MOGAD. He acknowledged that the medical record allowed for the possibility that Mr. Hock had experienced respiratory symptoms in September 2018, immediately before vaccination, and therefore a post-infectious autoimmune reaction could not be ruled out. Tr. at 106. But Dr. Rizvi emphasized the fact that the yellow mucus and treatment with antibiotics for that infection suggested that it was likely bacterial, rather than viral, and thus would (in his view) have been less likely to cause an autoimmune reaction. *Id.* at 107.⁷

⁷ Dr. Rizvi also, however, cited literature involving Guillain-Barré syndrome, a peripheral neuropathy thought to be autoimmune in mechanistic pathology—but known to have bacterial *and* viral triggers (reducing the value of his distinguishing between the two in this context). See, e.g., *Harris v. Sec’y of Health & Hum. Servs.*, No. 18-944V,

Dr. Rizvi then discussed the timing of Petitioner’s disease onset. Tr. at 93. He specifically opined that Petitioner’s MOGAD likely first manifested around October 27th, two days before he visited an ER, and within three weeks of vaccination. *Id.* He deemed that timeframe medically acceptable, noting that a typical onset for demyelinating disorders following vaccines is thought to be 3-42 days, although he derived this timeframe from articles specific to a distinguishable peripheral neuropathy, Guillain-Barré syndrome (“GBS”). *Id.* at 93-94; L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 *American Journal of Epidemiology* 105 (1979), filed on November 18, 2022, as Ex. 66 (ECF No. 23-30). But other studies involving central nervous system demyelinating conditions also supported a 19-day onset. Tr. at 94–96; A. Langer-Gould et al., *Vaccines and the Risk of Multiple Sclerosis and Other Central Nervous System Demyelinating Diseases*, 71 *JAMA Neurology* 1506, 1510 (2014) (timeline of up to 30 days for the first symptoms of CNS demyelinating disorders after vaccination), filed on Nov. 11, 2022, as Ex. 64 (ECF No. 23-28) (“Langer-Gould”); A. Rowhani-Rabar et al., *Biologically Plausible and Evidence-Based Risk Intervals in Immunization Safety Research*, 31 *Vaccine* 271, 273 (2012) (mean onset of 17 days between vaccination and ADEM), filed on Nov. 11, 2022, as Ex. 67 (ECF No. 23-31) (“Rowhani-Rabar”).⁸

At the same time, however, Dr. Rizvi admitted that physicians at Johns Hopkins (taking into account evidence of chronic optic nerve damage) had classified Petitioner’s illness course as possibly relapsing, rather than monophasic—thereby allowing for the possibility that Petitioner’s MOGAD *predated* vaccination. Tr. at 89, 112. Indeed, Dr. Rizvi admitted that Petitioner had complained of peripheral vision difficulties during his hospitalization, adding that there was no way to tell if the optic nerve damage was in fact pre-existing. *Id.* at 90.

On a brief rebuttal, Dr. Rizvi reiterated his prior opinion that Petitioner’s September 2018 back and neck pain complaints were distinguishable from his post-vaccine complaints. Tr. at 338. Petitioner had complained of tenderness in his back and neck at these visits, but displayed full range of movement. *Id.* at 339. Dr. Rizvi deemed this to indicate a musculoskeletal problem rather than a neurological one, which would be characterized by weakness, numbness, and reflex changes. *Id.* This difference, coupled with the lack of lesions on his October 2018 MRIs, suggested that Petitioner had not been experiencing MOGAD prior to vaccination. *Id.* at 340. A course that relapsed and healed so rapidly within a short period of time would be “very, very unusual.” *Id.* at 341.

Dr. Rizvi also deemed it unlikely that medication Petitioner received in September for his neck and back pain would have masked ongoing neurologic symptoms. Tr. at 341. Otherwise,

2023 WL 2583393, at *22 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (“GBS is well accepted as an autoimmune condition with a wide variety of suspected antigenic triggers, inclusive of antigens from both infection and vaccination...”).

⁸ Petitioner’s counsel inadvertently quoted the wrong sentence from this study during the hearing, citing the mean time between natural infection and ADEM (range of 6.2-17.8 days) rather than with regard to vaccination (mean time 17 days, with a range of 9-30).

Dr. Rizvi proposed the timeframe from Petitioner’s late-October onset to nadir in November was consistent with what was known about MOGAD. Tr. at 348, 350; S. Jarius et al., *MOG-IgG in NMO and Related Disorders: A Multicenter Study of 50 Patients. Part 2: Epidemiology, Clinical Presentation, Radiological and Laboratory Features, Treatment Responses, and Long-Term Outcome*, 13 *Journal of Neuroinflammation* 280 (2016), filed on April 13, 2022, as Ex. 25 (ECF No. 20-15) (“Jarius”).

Dr. Rizvi acknowledged that Petitioner’s OCT scans from November-December 2018 showed prior optic nerve damage. Tr. at 354. He agreed that some literature placed the minimum timeframe for this damage to occur at three months, which would imply that Petitioner’s onset was *earlier* than October. *Id.* And he admitted that other post-vaccination records could indicate a relapsing course. *Id.* at 358–64.

3. *S. Michael Phillips, M.D.* - Dr. Phillips prepared one expert report in this case, and testified at the hearing. Report, filed November 18, 2022, as Ex. 31 (ECF No. 23).

Dr. Phillips is a Professor of Medicine in the Pulmonary, Allergy, Critical Care Division of The University of Pennsylvania School of Medicine and a Lead Clinical Physician at the University of Pennsylvania, Department of Medicine. Phillips CV, filed on Nov. 18, 2022, as Ex. 32 (ECF No. 23-2). He received his medical degree from the University of Wisconsin, and completed his residency at the University of Pennsylvania. Phillips CV at 1. He is Director of Allergy and Immunology Services at the University of Pennsylvania and a Consultant in Medicine and Immunology to the Philadelphia Veterans Administration Hospital. *Id.* at 1-2. He is Board Certified in Allergy and Immunology and Internal Medicine. *Id.* at 2. He is also a Professor of Neurology in the Neurology Department of the University of Pennsylvania. *Id.* He has over 30 years of clinical and scientific experience in the field of internal medicine, allergy medicine, immunology, and epidemiology. Phillips Report at 2. During his career, he has treated over 200 cases of GBS and CIDP, as well as over 20 patients with confirmed or suspected MOGAD. *Id.* at 3.⁹

Dr. Phillips explained at the beginning of his testimony that he had in the past regularly acted as an expert for Respondent for several years—making this the first instance in which he offered an opinion for a Program claimant. Tr. at 130. He explained, however, that he felt comfortable with the change, noting that his views about vaccine injury theories had evolved with “increasing scientific sophistication”—“I’ve becoming increasingly convinced that through a number of mechanisms, the vaccines can cause some of these problems.” *Id.* at 133. In

⁹ Cross examination identified several issues with Dr. Phillips’s cited credentials as set forth in his previously-filed 2019 CV. Tr. at 168. For example, he is no longer an emeritus professor at the University of Pennsylvania School of Medicine, and has scaled back his teaching duties to clinicals twice a week. *Id.* at 169-170. He presently only sees patients “in a teaching context with interns and residents and fellows.” *Id.* at 170. And he is no longer a professor of neurology, going so far to clarify that he does not hold himself out as a “neurology specialist.” *Id.* at 173. He was also unaware that his Pennsylvania medical license expired in 2022. *Id.* at 170. Counsel requested permission during the hearing to file an updated CV, but did not do so. *Id.* at 207.

addition, Petitioner's case had "unique" aspects, leading him to "support the causal relationship," and they dovetailed with "advances in understanding of molecular mimicry, a newer diagnosis with limited research available, and timing that matched with established 'autoimmune models of demyelination.'" *Id.* at 133–35.

Because of his prior work as an expert for Respondent, Dr. Phillips was cross-examined closely on opinions he had offered in the past that seemed to contradict his testimony in this case. Indeed, in past cases Dr. Phillips had harshly criticized molecular mimicry theories. Tr. at 194–96; see *Kelly v. Sec'y of Health & Hum. Servs.*, No. 16-878V, 2021 WL 5276373, at *14 (Fed. Cl. Spec. Mstr. Oct. 18, 2021). One of these cases involved MS, a disease Dr. Phillips compared to MOGAD several times during his testimony. Tr. at 199; *Townsend v. Sec'y of Health & Hum. Servs.*, No. 14-266V, 2023 WL 6212496 (Fed. Cl. Spec. Mstr. Aug. 29, 2023), *mot. for review den'd*, 170 Fed. Cl. 130 (2024). In fact, the *Townsend* Special Master had described him as the expert most critical of molecular mimicry theories. Tr. at 201; *Townsend*, 2023 WL 6212496 at n.32. It was also noted that Dr. Phillips had cited some of the same literature herein in cases where he *criticized* molecular mimicry theories as scientifically unreliable. Tr. at 200; see B. Trost et al., *No Human Protein is Exempt From Bacterial Motifs, Not Even One*, 1 Self/Nonsel 328 (2010), filed on Nov. 30, 2022, as Ex. 54 (ECF No. 25-6); B. Trost et al., *Bacterial Peptides are Intensively Present Throughout the Human Proteome*, 1 Self/Nonsel 71 (2010), filed on November 30, 2022, as Ex. 55 (ECF No. 25-7) ("Trost 2"); M. Sospedra & R. Martin, *Molecular Mimicry in Multiple Sclerosis*, 39 *Autoimmunity* 3 (2006), filed on Nov. 30, 2022, as Ex. 51 (ECF No. 25-5).

The initial part of Dr. Phillips's testimony involved an explanation of molecular mimicry as a pathologic mechanism for autoimmune disease. Tr. at 137–39. He also explained the demyelination process, and how myelin loss interrupts electrical transmission in nerves, producing neurological symptoms such as paralysis, weakness, and loss of motor skills. *Id.* at 146. However, Dr. Phillips emphasized that molecular mimicry was only the first step in a complex autoimmune disease process that was also reliant upon an imbalance of T-regulatory and T-effector cells. *Id.* at 143. This creates a destructive autoimmune reaction, where cytotoxic chemicals damage the myelin sheath. *Id.* at 149. And Dr. Phillips briefly touched on bystander activation as an alternative mechanistic explanation leading to autoimmune cross-attack, but deemed bystander immune cells "only really important as they interfere with the regulatory pathways which are dependent ultimately on the presence of molecular mimicry." *Id.* at 155.

MOGAD, Dr. Phillips emphasized, overlaps with optic neuritis, ADEM, and TM, as all are central nervous system inflammatory conditions characterized by damaging demyelination. Tr. at 155. Like Dr. Rizvi, however, Dr. Phillips deemed MOGAD a newer diagnosis, noting that many cases previously diagnosed as other demyelinating illnesses, like TM, could have actually been MOGAD. *Id.* at 157. Thus, studies on the connection between vaccines and these illnesses

cited by Dr. Rizvi were relevant to the case at hand, since MOGAD as a diagnostic classification is still too new to be the basis of large epidemiological studies. *Id.* at 155–156; see also L. Mumoli et al., *ADEM Anti-MOG Antibody-Positive After SARS-CoV2 Vaccination*, 43 *Neurological Sciences* 763 (2022), filed on April 13, 2022, as Ex. 22 (ECF No. 20-12).

Dr. Phillips then discussed Petitioner’s clinical course. Tr. at 159. He disputed the possibility that Petitioner’s September, pre-vaccination back pain could have been an initial presentation of MOGAD, opining that it was more likely due to his disc disease and strenuous physical activity. *Id.* At the same time, several treaters had mentioned the flu vaccine in their notes as possibly causal, with Dr. Phillips stating that “[t]he doctors who are most able to interpret what’s going on with a patient generally are the doctors who are taking care of that patient at that point in time.” *Id.* at 162.

Dr. Phillips further embraced the onset timeframe of Petitioner’s MOGAD as medically acceptable. Petitioner’s initial symptoms of clumsiness and neck pain were the “first obvious neurological symptoms,” although the disease process could have started a few days before (but nevertheless still after vaccination). A 19-day, post-vaccination onset timeframe was, in his opinion, “perfect” for vaccine-induced MOGAD, as supported by studies cited in both expert reports. *See* Karussis at 216 (mean of 14.2 days between vaccination and CNS demyelination); Rowhani-Rabar at 273 (17-day mean onset for ADEM after vaccination). Dr. Phillips denied that Petitioner’s upper respiratory infection during that time period could have triggered his MOGAD. *Id.* at 161. In his view, it was “very unlikely” that Petitioner would have had an MRI scan (like the one he had during his initial hospitalization) without signs of demyelination, had the disease onset already begun prior to vaccination. *Id.* at 160.

Dr. Phillips was then asked about several studies that seemed to potentially contradict his conclusions. Trost 2, for example, concluded that the high amount of homologies between human and viral and bacterial peptides made it difficult to support molecular mimicry as a theory in autoimmunity, given how common it was (but without leading to disease). Tr. at 178; Trost 2 at 73. Dr. Phillips contended, however, that Trost 2 (authored in 2010) was outdated, and that he no longer accepted its conclusions about molecular mimicry. *Id.* He was also asked about a study finding that there was no statistically significant relationship between vaccination and MS, a demyelinating disease Dr. Phillips compared to MOGAD several times in his testimony. *Id.* at 185; Langer-Gould at 1509 (“In this nested case-control study, we found no long-term association between vaccines and MS or other CNS [diseases].”).

Respondent’s cross-examination of Dr. Phillips further delved into certain record evidence—in particular, the November treatment record from Dr. Kooshkabadi (Ex. 11 at 7)—and whether it suggested that Petitioner’s MOGAD-related symptoms actually began well before vaccination. Tr. at 190, 211. Dr. Phillips did not gainsay what the record indicated Petitioner had

told Dr. Kooshkabadi, but allowed only that it was “theoretically possible,” not likely, that Petitioner had experienced a relapsing form of MOGAD that began pre-vaccination. *Id.* at 212. In so doing, he emphasized again that molecular mimicry was just the beginning of the autoimmune process, with an imbalance of T-cells thereafter “ultimately responsible for the problem.” *Id.* at 217. (This contention would, however, only explain why Petitioner’s course might have been erratic or relapsing *after* vaccination—and not whether it could have been based on a pre-vaccination onset).

B. Respondent’s Experts

1. *Brian Callaghan, M.D.* - Dr. Callaghan authored one report in this case, and testified at the hearing. Callaghan Report, filed on July 29, 2022, as Ex. A (ECF No. 21-1) (“Callaghan Rep.”).

Dr. Callaghan is an Associate Professor of Neurology at the University of Michigan. Callaghan CV, filed on July 29, 2022, as Ex. C (ECF No. 21-7). He received his medical degree from the University of Pennsylvania, and completed his neurology residency there as well. Callaghan CV at 1. He is American Board of Psychiatry and Neurology (ABPN) and American Board of Electrodiagnostic Medicine (ABEM) certified. *Id.* He has published more than 120 articles on neurologic diseases. *Id.* at 14–24. In his clinical practice, he has encountered and treated at least 50 patients with CNS demyelinating disorders. He has testified in four vaccine injury compensation program hearings to date. Callaghan Report at 1.

Although Dr. Callaghan accepted Petitioner’s MOGAD diagnosis, he disputed that the flu vaccine had caused it. Tr. at 227, 257. He started by discussing several of the articles filed on both sides in the case. *Id.* at 232. One, which discusses the research landscape on MOGAD, does not mention vaccine causation. *Id.* at 233; R. Marignier et al., *Myelin-Oligodendrocyte Glycoprotein Antibody-Associated Disease*, 20 *Lancet Neurology* 762 (2021), filed on July 29, 2022, as Ex. A1 (ECF No. 21-2) (“Marignier”). Two others concluded that there was no significant association between the flu vaccine and MS (a parallel central nervous system demyelinating disease). *Id.* at 233-35; M. Mailand & J. Frederiksen, *Vaccines and Multiple Sclerosis: A Systematic Review*, 264 *Journal of Neurology* 1035 (2017), filed on July 29, 2022, as Ex. A2 (ECF No. 21-3); L. Belbasis et al., *Environmental Risk Factors and Multiple Sclerosis: An Umbrella Review of Systematic Reviews and Meta-Analysis*, 14 *Lancet Neurology* 263 (2015), filed on July 29, 2022 as Ex. A3 (ECF No. 21-4). Rather, filed literature far better associated *infections* with MOGAD, especially since a live infection would have much more of a stimulative impact on the immune system. Tr. at 258; *see, e.g.,* D. Dubey et al., *Clinical, Radiologic, and Prognostic Features of Myelitis Associated with Myelin Oligodendrocyte Glycoprotein Autoantibody*, 76 *JAMA Neurology* 301 (2019), filed on Nov. 18, 2022, as Ex. 40 (ECF No. 23-9). Case reports purporting to make a more direct association deserved less weight,

in Dr. Callaghan's view, and he deemed them "the lowest levels of evidence." Tr. at 236. Dr. Callaghan also stated that he did not find literature studying GBS or other central nervous system conditions to be particularly helpful, due to the differences in those conditions and MOGAD. *Id.* at 228–29.

Dr. Callaghan then discussed Petitioner's clinical course, and what it suggested to him about possible onset timeframe. Tr. at 237. He characterized Petitioner's MOGAD as relapsing in nature, with onset likely in early September 2018, when he visited an emergency room with complaints of neck and back pain. Callaghan Rep. at 6; Tr. at 238. He did not agree with Dr. Phillips that Petitioner had disc issues, as this was not indicated on the MRI. Tr. at 239. When asked why Petitioner had reported that his neck and back pain had improved at the October 8, 2018 appointment (when he received the vaccine), Dr. Callaghan proposed that Petitioner likely had MOGAD lesions that "came and went." *Id.* at 241, 263. And Dr. Callaghan did not deem the absence of lesion evidence in late-October to be significant, opining that "MRIs are not perfect," and could well have been missed by the reviewing radiologist. *Id.* at 242. He otherwise stated that he had seen patients where lesions were suspected but whose imaging showed no lesions, only appearing on later scans. *Id.*

Dr. Callaghan further emphasized the record of Petitioner's November 5, 2018 neurologist visit, at which time Petitioner reported that his symptoms began six months prior. Tr. at 243. Dr. Callaghan characterized this to be possibly "when it started," in contrast to the October symptoms (such as pain, numbness, and loss of motor skills), which occurred "when it became dramatically worse." *Id.* at 243–44. The fact that Petitioner's MRI during the November 2018 hospitalization showed spinal cord lesions did not impact his contention of a pre-vaccine disease onset. *Id.* at 245–46.

Another significant factor suggesting to Dr. Callaghan that Petitioner's MOGAD predated onset were the results of the OCT testing from November-December 2018. Tr. at 248. As Dr. Callaghan explained, it takes three to six months for optic neuropathy to manifest—meaning the OCT findings were inconsistent with a more recent October onset. *Id.* at 248–49. In fact, the Johns Hopkins neuroimmunology case conference notes from December 6, 2018, concluded that Petitioner's course was likely relapsing over time, as opposed to recent and monophasic, based on the OCT findings. Tr. at 250; *see also* Ex. 6 at 292. Thus, while the optical nerve findings "aren't typical in MOGAD," they did "indicate that this has been going on for at least three to six months." Tr. at 249.

On cross, Dr. Callaghan was confronted with his conflicting statements that Petitioner's MOGAD may have been caused by infection (which the record suggested had possibly occurred in two or so weeks before vaccination), versus an idiopathic onset of months before (a conflict also seemingly present when comparing Drs. Callaghan's and He's opinions). Tr. at 259–60. He

agreed that the record had “more obvious” evidence of neurologic-like symptoms in September than earlier. *Id.* But Dr. Callaghan would not state that he outrightly disagreed with Dr. He’s theory that Petitioner’s MOGAD was caused by an upper respiratory infection. *Id.* at 269. At best, Dr. Callaghan opined that he “would place the onset before the vaccine and the infection. But if one was to say that [onset] was in late October, then I pointed to multiple exhibits that show infections are way more common than vaccines prior to the onset of MOGAD.” *Id.*

In addition, Dr. Callaghan could not opine as to what length of time would typically separate MOGAD symptoms flares, stating only that it “could be all over the place. You can, you know, have relapses that happen in short order. It can happen months and years later.” Tr. at 262. He otherwise agreed, however, that Petitioner’s November 2018 hospitalization was the “nadir” of his illness. *Id.* at 269. He also suggested the possibility that Petitioner’s MOGAD was continuous even in times when he had no symptoms, but that he was “taking symptomatic treatments that were masking some of his symptoms.” *Id.* at 271.

2. *You-Wen He, M.D., Ph.D.* - Dr. He wrote one report in this case, and testified at the hearing. Report, filed July 29, 2022, as Ex. B (ECF No. 22). Dr. He denied the flu vaccine could have caused Petitioner’s MOGAD.

Dr. He is a Professor of Immunology in the Department of Immunology at Duke University School of Medicine. Curriculum Vitae of You-Wen He, M.D., Ph.D., filed July 29, 2022, as Ex. D (ECF No. 22-19) (“He CV”). He earned his medical degree in 1986 from the Fourth Military Medical University in Xian, China. He CV at 1. He went on to earn a Ph.D. in Microbiology and Immunology from the University of Miami School of Medicine in 1996. *Id.* He has been conducting research in immunology since 1986 after completing his MD training. *Id.* His research areas include both innate and adaptive immunity against viral and bacterial infections as well as tumors. He Report at 1. He has directed research on human immune responses to viral infections including influenza, HIV, HBV and HCV. *Id.* He has been the Director for an advanced immunology course (IMM601), Immunology of Human Diseases, at Duke University Medical Center for 10 years. *Id.* He has also served as a co-Principal Investigator for four clinical trials focusing on cancer immunotherapy using personalized cancer vaccine. *Id.* He is a reviewer for more than 30 scientific journals, and an editor for several more. *Id.* at 2.

Dr. He started by explaining the difference in immune system stimulation caused by vaccines versus wild-type viruses. Tr. at 284. He defined “PAMPs” (pathogen-associated molecular patterns) as “the individual component from microbe pathogens, and our host use[s] the receptor called a pattern recognition receptors” in reacting to PAMPs. *Id.* at 288. A flu vaccine, however, is much “simpler” than a wild-type virus, as it has been stripped down to contain only one PAMP. *Id.* at 286, 292. This allows it to create a “controlled immune process.”

Id. at 291. In contrast, a wild-type virus contains many more PAMPS; the flu virus, for example, contains at least “five different categories” of PAMP. *Id.* at 290. Thus, “[t]he magnitude, the breadth, the depth of the immune response is fundamentally different.” *Id.* at 292. Though no testing revealed the cause of Petitioner’s upper respiratory symptoms, *any* wild bacteria or virus would have stimulated his immune system with more PAMPs than a vaccine. *Id.* at 294.

Dr. He next criticized molecular mimicry as a theory of autoimmune injury. Tr. at 295. He noted that molecular mimicry theories had initially emerged before the existence of technology sufficient to uncover massive sequence sharing in genomes. *Id.* at 296. When this genome sequencing became possible on a large scale, evidence emerged weakening the concept that sequence sharing would likely be pathogenic. *Id.* Instead, Dr. He explained, “self-reactive lymphocytes [are] the nature of our immune system,” constantly communicating with peripheral immune cells to function.” *Id.* at 298. Thus, “the majority of the time, you know, [cross-reactivity]’s not pathogenic at all, and actually most cases are protective even.” *Id.* at 299. In support, Dr. He referenced three studies discussing significant sequence similarities between viral and human genomes. *Id.* at 299-302; Trost 2 at 71; A. Kusalik et al., *Widespread and Ample Peptide Overlapping Between HCV and Homo Sapiens Proteomes*, 28 *Peptides* 1260 (2007), filed on July 29, 2022, as Ex. B6 (ECF No. 22-7); D. Li & M. Wu, *Pattern Recognition Receptors in Health and Diseases*, 6 *Signal Transduction and Targeted Therapy* 291 (2021), filed on July 29, 2022, as Ex. 7 (ECF No. 22-8).

Petitioner’s cited studies did not, in Dr. He’s reading, suggest molecular mimicry was a likely driver of most instances of autoimmune disease. Some studies, for example, involved an animal model—experimental autoimmune encephalomyelitis (“EAE”)—applicable mainly to MS, and which requires use of a highly-stimulative laboratory-specific additive, “complete Freund’s adjuvant” (“CFA”). Tr. at 303; K. O’Connor et al., *Self-Antigen Tetramers Discriminate Between Myelin Autoantibodies to Native or Denatured Protein*, 13 *Nature Medicine* 211 (2007), filed on Nov. 18, 2022, as Ex. 34 (ECF No. 23-5) (“O’Connor”). But there is currently no animal model for MOGAD, and CFA is a “nasty adjuvant” not approved for use on humans (while the version of the flu vaccine at issue contains no adjuvant at all). *Id.* at 303, 304. Accordingly, EAE studies were not helpful in establishing causation. He also criticized the use of case studies, stating that while a series of case reports may be useful, a single case report is not reliable evidence for establishing causality. *Id.* at 307.

While denying the vaccine was causal, Dr. He (although not a neurologist or treating expert) deemed infection a far more likely explanation for Petitioner’s MOGAD. Tr. at 309. He opined that antibodies (such as the MOG antibody Petitioner unquestionably had) would take up to seven days to develop post-infection. *Id.* at 310. But at a primary care visit on October 30, 2018, Petitioner complained of sinus pressure and post-nasal drip. *Id.* at 312; Ex. 3 at 13. Petitioner’s hospitalization in November 2018 began nine days after this visit, matching the

timeline in which an adaptive immune response. *Id.* at 313. This timeframe was not consistent with the longer period separating Petitioner’s vaccination and likely MOGAD onset. *Id.* at 315.

On cross, Dr. He refused to disagree with Dr. Callaghan’s assertions that Petitioner’s MOGAD onset likely predated vaccination. Tr. at 318. Dr. He in fact argued that their somewhat competing etiologic explanations were not contradictory, while adding that he could not comment on Dr. Callaghan’s opinion overall because he was not a neurologist. *Id.* at 319. He was then asked about two of his cited studies, which expressly mentioned vaccines as a potential causes for MOGAD or other central nervous system demyelinating injuries. *Id.* at 323–27; E. Flanagan et al., *Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD): Clinical Features and Diagnosis*, Wolters Kluwer (2022), filed on July 29, 2022, as Ex. B1 (ECF No. 22-2) (“Flanagan”); O’Connor at 211. In response, Dr. He only pointed out that O’Connor involved EAE, reiterating his prior contentions that results from this animal stud would not translate to humans. Tr. at 326. He also acknowledged that several articles filed in this case did discuss molecular mimicry as a viable mechanism for at least *some* autoimmune diseases. *Id.* at 328–29. And Dr. He conceded that cross-reactivity could serve as the first step in a far more complex autoimmune process. *Id.* at 330.

III. Procedural History

Although the Petition originally alleged that Mr. Hock had developed “demyelinating disease” as a result of receiving the flu vaccine, he later amended his petition to specify MOGAD as his injury. Amended Petition, dated March 30, 2022 (ECF No. 19). Respondent filed his Rule 4(c) Report on December 16, 2021, recommending against compensation. (ECF No. 17). Expert reports were filed through the end of 2022, and then trial was set in the matter for December 2023. The parties opted not to submit post-hearing briefs, and the claim is ripe for resolution.

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).¹⁰ There is no Table injury for MOGAD, so Petitioner can only proceed herein with a causation-in-fact claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). 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 and 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 *Althen* prong requires a different showing. Under *Althen* prong one, petitioners must provide a “reputable medical theory,” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec’y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be “legally probable, not medically or scientifically certain.” *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec’y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009)

¹⁰ 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. App’x. 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).

(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*, 121 Fed. Cl. at 245 (“[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one” (emphasis in original)).

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 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, 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 as a Putative Vaccine Injury

In keeping with the fact that medical science has only recently identified the MOGAD-associated autoantibodies, and in turn proposed their possible link to certain CNS-oriented demyelinating injuries, there are few Program decisions addressing MOGAD. I awarded entitlement in one such case, however. *L.C. v. Sec’y of Health & Hum. Servs.*, No. 17-722V, 2021 WL 3630315 (Fed. Cl. Spec. Mstr. July 2, 2021) (Tdap vaccine found to have caused MOGAD in minor). The overall evidence offered therein was limited, with only neurologic treaters testifying, allowing me to conclude that *in that case* preponderant evidence was presented in favor of causation (albeit barely). Another petitioner, however, was previously able to demonstrate that the anti-MOG antibody was vaccine-induced, causing an individual to experience a CNS demyelinating injury. *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).

Thus, while there is nascent Program recognition that vaccination *might* play a role in the propagation of these antibodies, the question is far from resolved. It cannot be said based on these two decisions that the Program *favors* a vaccine-MOGAD connection. And I am not otherwise prepared to adopt the flu-GBS “template” as my analysis, applying it rotely simply because MOGAD also involves demyelination and is believed in part to be associated with certain autoimmune neuroinflammatory conditions.

All experts in this case agreed that Petitioner had MOGAD, and the record establishes a persuasive basis for that conclusion. Petitioner tested positive for MOG antibodies after experiencing a cluster of neurological symptoms characteristic of MOGAD—pain, weakness, loss of motor function, bladder dysfunction, and visual loss. Tr. at 53; Ex. 6 at 8. Imaging showed spinal cord lesions and evidence of optic neuritis, while laboratory testing showed the presence of the MOG antibodies. Ex. 6 at 287. The diagnosis has solid evidentiary support.

Literature specific to MOGAD is very limited, given that widespread commercial testing for the autoantibody has only been available for less than a decade. Experts in this case agreed that there are no large epidemiological studies focusing on MOGAD (though some experts discussed MS studies, a condition that I have *not* generally found to be credibly linked to any covered vaccine). However, a few findings regarding MOGAD can be gleaned from what does exist. Importantly, expert witnesses and cited articles note that the MOG antibody itself has not

been *proven* to be pathogenic—hence the name “MOG-antibody-associated disease (MOGAD).” Tr. at 306; Marignier at 762.

There is also some evidence about the nature of MOGAD that might distinguish it from other central nervous system demyelinating conditions. For example, one 50-patient study on MOG-IgG positive patients found that 80% of the sample experienced a relapsing disease course (rather than monophasic). Jarius at 3. In addition, the median time between the first and second attacks in these patients with a relapsing course was five months, and the median annualized relapse rate was 0.83. *Id.* at 8. The most common symptom at onset was optic neuritis (74%), followed by myelitis (34%). *Id.* at 6. Two patients in this study received different vaccines shortly before onset—one 12 days prior, and one two weeks prior. *Id.* at 14. In addition, tissue studies have shown a CD4+ T-helper cell-driven reaction with granulocytic inflammation is the likely pathogenesis of the disease. Flanagan at 2–3.

II. Petitioner Has Carried His Burden of Proof

A. Althen Prong One

Petitioner’s “can cause” showing was thin, but nevertheless sufficiently preponderant to carry his burden—and the balance of factors that lead me to so conclude were not rebutted by Respondent.

An overarching consideration underlying my analysis is the fact that the flu vaccine has often been found in past Program cases to be associated with *comparable* central nervous system inflammatory demyelinating diseases, like ADEM, TM, or optic neuritis. *See, e.g., Schmidt v. Sec’y of Dep’t of Health & Hum. Servs.*, No. 07-20V, 2009 WL 5196169 (Fed. Cl. Spec. Mstr. Dec. 17, 2009) (finding that flu vaccine caused acute transverse myelitis); *Daniels v. Sec’y of the Dep’t of Health & Hum. Servs.*, No. 07-462V, 2012 WL 763175 (Fed. Cl. Spec. Mstr. Feb. 16, 2012) (finding that flu vaccine caused ADEM); *Calise v. Sec’y of Dep’t of Health & Hum. Servs.*, No. 08-865V, 2011 WL 1230155 (Fed. Cl. Spec. Mstr. Mar. 14, 2011) (finding that flu vaccine caused neuromyelitis optica). As noted at hearing, many of these diseases might today be categorized as MOGAD instead, even if MOGAD is not wholly congruent (or even yet fully understood).

I am of course not bound herein to find that the flu vaccine likely causes MOGAD simply on the basis of these prior determinations. But I am reluctant to ignore them at the same time. I have noted in other contexts that relevant prior determinations should be taken seriously, especially in the absence of compelling proof counseling a different outcome. *See, e.g., Nieves v. Sec’y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148, at *36 (Fed. Cl. May 22, 2023), *mot. for review den’d*, 167 Fed. Cl. 422 (2023) (prior cases with favorable results relevant to CIDP-flu vaccine association deemed “persuasive guidance,” despite issues with causation

theory overall).¹¹ It is reasonable to consider and be guided by a history of relevant reasoned decisions involving the same theory and vaccine, especially when the injury at hand is consistent with one that has been repeatedly deemed vaccine-caused in prior cases. *Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39 (2007) (“[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is *most probative of a claim*”) (emphasis added).

In addition, Petitioner has offered just enough *other* evidence in support of causation to find his burden of proof was met (although barely). Dr. Phillips made several reasonable points about the potential pathologic results of an aberrant immune process triggered by molecular mimicry between a vaccine’s antigens and self-structures. He explained that the flu vaccine could stimulate production of anti-MOG antibodies through molecular mimicry—a theory that has been accepted in other Program cases as a scientifically-valid mechanistic explanation for how some autoimmune-mediated demyelinating illnesses might unfold. *Raymo v. Sec'y of Health & Hum. Servs.*, No. 11-0654V, 2014 WL 1092274 (Fed. Cl. Spec. Mstr. Feb. 24, 2014), *mot. for review den'd*, 129 Fed.Cl. 691 (Fed. Cl. 2016). And it can be the instigating factor leading to an autoimmune disease process. Tr. at 143, 149.

Respondent’s primary immunology expert, Dr. He, raised valid arguments about the limits of molecular mimicry as explanatory of all autoimmune injuries, and the more stimulative effects of a wild-type virus. I certainly *never* rule for petitioners simply because they have invoked molecular mimicry, but without other corroborative evidence. *McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (“But 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.”). But Dr. He’s criticisms, while reasonable in a broad sense, were themselves not specific enough to the context at hand to rebut Petitioner’s showing. And impeachment of Dr. Phillips and his late-career “team switching” did not persuasively call into question his expertise or the specific opinion he offered. (It would actually be to the Program’s benefit overall if experts more commonly worked for both sides in Vaccine Act cases).

Thus, and as in *L.C.*, I find that Petitioner’s showing on prong one was just preponderant enough to make this a “close” case, presenting the kind of circumstances in which special masters usually resolve the disputed question in the petitioner’s favor. *L.C.*, 2021 WL 3630315 at 20; *Andreu*, 569 F.3d at 1378. My resolution of this aspect of Petitioner’s overall burden, however, does not in any regard resolve the question of whether MOGAD is, or is not, likely

¹¹ By the same token, I frequently follow prior reasoned decisions when *dismissing* claims that repeat theories also aired in the dismissed matters, while not offering new evidentiary bases for taking a fourth or fifth look at the theory. See, e.g., *Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 17-792V, 2022 WL 2678877 (Fed. Cl. Spec. Mstr. June 17, 2022), *mot. for review den'd*, Dkt. No. 79 (Fed. Cl. Oct. 27, 2022), *aff'd*, No. 2023-1321, 2024 WL 3064398 (Fed. Cir. June 20, 2024) (rejecting theory that flu vaccine caused type II narcolepsy, where theory was largely identical to one offered in prior cases that had also been dismissed).

vaccine-caused. It is very possible the anti-MOG antibody *does not* pathogenically drive the disease—and if so, a vaccine’s encouragement of this antibody’s production could not be causal. Indeed—it is far from established with any degree of certainty that vaccines cause the production of this antibody at all. MOGAD’s novelty as a diagnosis, coupled with the extremely limited number of Program decisions addressing the topic, mean that the subject remains open to debate—and additional evidence on the topic (not offered in this case) will be required before special masters can more confidently state a MOGAD-vaccine association exists. But for present purposes, what matters is the evidence adduced *in this case*.

B. Althen Prong Two

The record preponderates in favor of the conclusion that Petitioner’s MOGAD was likely vaccine-caused. Petitioner began to experience symptoms that were largely neurologic in character shortly before his ER visit in October 2018. Ex. 4 at 65. At this time, he presented with headaches, pain in his back and neck, and trouble urinating. *Id.* at 65, 74, 76. He was prescribed steroids and discharged with instructions to follow up with a neurologist. *Id.* at 76–77. In the weeks following, his symptoms continued, and his bladder issues worsened. He visited a urologist multiple times, and was eventually prescribed self-catheterization as he could not urinate. Ex. 10 at 12–15. Petitioner attempted to visit another ER to address these issues, but was again discharged and advised to follow up with a neurologist. Ex. 5 at 18. His symptoms progressed, he obtained more focused neurologic treatment, and then was diagnosed with MOGAD on the basis of a number of reliable and relevant tests. Ex. 6 at 8, 11, 21, 26, 287–88. And Petitioner’s medical treaters attributed his symptoms to the flu vaccine at least once. Ex. 9 at 8.

Respondent did identify some record evidence that suggested onset of Petitioner’s MOGAD could have predated vaccination, but it was ultimately too inconclusive when balanced against the overall record. For example, symptoms reported at Petitioner’s pre-vaccination medical visit (back and neck pain) were convincingly distinguished by Dr. Rizvi from Petitioner’s post-vaccination constellation of concerns (which were overall more neurologic in character). Tr. at 77. Given that Petitioner had evidence of disc disease on MRIs, and worked a physical job that required several hours of driving per day, it is not difficult to see how he could have experienced musculoskeletal issues resulting in back and neck pain. His symptoms after vaccination, in contrast, were distinctly neurologic—Petitioner displayed loss of motor function, significant pain, gait problems, and sensory abnormalities. And importantly, he could not urinate on his own, and his bladder symptoms were noted to be neurological by several treaters.

In addition, the reference in a record to Petitioner reporting symptoms predating vaccination was a one-time occurrence, never corroborated with other proof suggesting he *had* been suffering from neurologic concerns for so long. Proposals of an infectious cause for MOGAD were based on evidence that the Petitioner likely experienced an upper respiratory infection in September and October 2018, but were never later confirmed during Petitioner’s

extensive treatment.¹² And imaging performed in October did not identify active demyelinating lesions—which if present would have been consistent with a disease process that likely started before vaccination. Tr. at 84. By contrast, Petitioner’s scan in mid-November revealed enhancing lesions—a clear indicator of active demyelination that would have begun in October. *Id.* at 88. This lends further support to the theory that Petitioner’s MOGAD onset occurred after vaccination in October.

A stronger argument marshalled by Respondent in favor of an earlier onset was rooted in the OCT test results. Respondent correctly noted that treaters concluded that these tests established “chronic” damage to Petitioner’s optic nerves, suggesting that a neuroinflammatory condition may have been present for some time. Ex. 6 at 48–49, 292. But Dr. Callaghan’s repeated assertion that this meant Petitioner’s MOGAD had been going on “at least three to six months” is unsupported. Tr. at 249. And a further look into these results (to the extent the medical record allows that) finds that they are less clear and robust than at first glance. No explanation is provided, for example, of what “chronic” really means—or how long the process causing the optic nerve damage was thought to have predated its discovery.

Literature cited by Petitioner raised additional questions about these findings. Flanagan, for example, notes that the optic nerves contain two different tissue layers (the peripapillary retinal nerve fiber layer (pRNFL) and inner plexiform layer (mGICPL)) that thin at different rates—“mGICPL thinning emerges within a few weeks, while pRNFL takes longer to develop, possibly due to the optic nerve head edema, which masks initial thinning in the pRNFL.” Flanagan at 16. A treater note from his December 2018 scan called Petitioner’s pRNFL normal, but noted that his mGICPL was thinned (“[m]ean peripapillary retinal nerve fiber layer thickness was normal for each eye”). Ex. 6 at 302–03. And Petitioner never even complained of any vision associated symptoms until his November hospitalization. Thus, the evidence from this testing of a long-existing condition is ultimately too inconclusive to support the determination that Petitioner’s MOGAD likely preceded vaccination.

C. Althen Prong Three/Onset

Petitioner offered persuasive and reliable evidence that a post-vaccination onset of 19 days was medically acceptable. That timeframe would be sufficient for the production of cross-reactive autoantibodies to occur and thereafter cause the MOG antibody-driven demyelination characteristic of MOGAD. *See* Karussis at 216; Rowhani-Rabar at 273. Petitioner’s experience of acute, neurologic symptoms in late October, progressing to nadir in November, was consistent with such an onset timeframe.

¹² Respondent’s experts’ disagreement on etiology also undermined the strength of arguments that an infection was causal.

CONCLUSION

Petitioner has established entitlement to a damages award. An order setting forth a schedule for the resolution of damages in this matter shall shortly follow.

IT IS SO ORDERED.

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