In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-802V

(to be published)

* *	Chief Special Master Corcoran
CHARLOTTE PORCH, *	
*	
Petitioner, *	Dated: December 8, 2022
*	
v. *	
*	
SECRETARY OF HEALTH AND *	
HUMAN SERVICES, *	
*	
Respondent. *	
*	
* *	

Joseph Alexander Vuckovich, Maglio Christopher & Toale, P.A., Washington, DC, for Petitioner.

Emily H. Manoso, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On June 14, 2017, Charlotte Porch filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Program").[2] ECF No. 1. Petitioner initially alleged that a measles, mumps, and rubella ("MMR") vaccine administered to her on February 11, 2015, caused her to develop transverse myelitis ("TM"), and/or caused or alternatively significantly aggravated her subsequently diagnosed multiple sclerosis ("MS"). At hearing (held in Washington, D.C. on January 13, 2022), however, Petitioner limited her claim to the allegation that the MMR vaccine caused her MS.

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[1] This Decision will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet**. As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Ruling's inclusion of certain kinds of confidential information. Specifically, 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 entire Decision will be available to the public in its current form. *Id.*

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

As discussed in greater detail below, after consideration of the overall record I deny entitlement. Petitioner's MS has not reliably been shown to have been caused by the MMR vaccine—or that the vaccine could cause MS in the first place.

I. Fact History

Vaccination and Onset of Symptoms

Petitioner was born on November 9, 1967. Ex. 1 at 5. Prior to the relevant vaccination, her medical history was significant for shingles, chest pains, and carpal tunnel syndrome. Ex. 3 at 9–12; Ex. 11 at 31; Ex. 13 at 3. Pre-vaccination medical records also memorialize instances of bilateral hand pain and numbness in 2010.[3] Ex. 11 at 11–12, 31.

On February 11, 2015, Ms. Porch received an MMR vaccine dose as a requirement of her employment. Ex. 1 at 5.[4] She was thereafter to be stationed in Kuwait for work in the military, beginning on March 7, 2015. Ex. 11 at 31. The records do not reveal any vaccine reaction or symptoms presaging or characteristic of MS between February 11th and Petitioner's travel abroad. Indeed, there are no medical records *at all* pertaining to any intervening medical concerns until early May 2015. Petitioner contends, however, that although she arrived in Kuwait symptom-free as of the start of March, by the end of that same month she was beginning to experience MS onset, in the form of TM. And some records from later in her treatment history do refer to symptoms beginning in this period.

On May 2, 2015, Petitioner underwent an x-ray of her cervical spine at the International Clinic in Kuwait. Ex. 15 at 180. The record from this encounter is silent as to why such testing was called for. The x-ray's findings were "suggestive of cervical spondylosis with diminished disc space between C4-C5 with neck muscle spasm." *Id*. Five days later (May 7, 2015), Petitioner underwent an MRI of her cervical spine as ordered by neurologist Raed Alroughani, M.D., at Al Seef Hospital in Kuwait. Ex. 12 at 23. The MRI revealed a herniated disc at C4-5, compromising the dural sac, and left-greater-than-right neuroforaminal openings, along with a longitudinal hyperintense lesion at C2-3 with no associated edema or mass effect. *Id.* It was subsequently recommended that Petitioner have a repeat study with contrast,[5] although this record is also silent

[3] Petitioner, however, was unable to locate or file the 2010 Kuwait medical records that would memorialize these medical complaints. ECF No. 60.

[4] Petitioner had received an initial MMR vaccine dose approximately five months before, in October 2014. Tr. at 18. The record does not reveal any prior reaction to that vaccination.

[5] As explained later by Respondent's expert, "contrast" (specifically gadolinium, a pharmaceutical agent) is injected into a subject undergoing MRI imaging because it is observable on the scan results, and can reveal where "leakage" across the blood-brain barrier into the central nervous system may be occurring. Tr. at 164–65. Evidence of

as to the reason for an MRI. *Id.* About two weeks later, however, on May 24, 2015, Petitioner had the recommended repeat imaging study, and it showed no enhancement of the lesion at C2-3, with the radiologist noting "no signs of activity." *Id.* at 24. The notes nevertheless indicated that MS was suspected. *Id.*

MS Diagnosis and Subsequent Treatment

Petitioner had a follow-up appointment with Dr. Alroughani on May 31, 2015, at which time she reported numbness, tingling, and pain in her hands for the past two months (or since late March 2015). Ex. 12 at 17, 55; Ex. 15 at 258. Her paresthesias were characterized as persistent and progressive, with abnormal sensations ascending from her hands to as far up as her elbows. Ex. 12 at 17, 55; Ex. 15 at 258. Dr. Alroughani opined that the symptoms could be consistent with carpel tunnel syndrome, and he ordered an Electromyograph/Nerve Conduction Study ("EMG/NCS") to confirm. Ex. 12 at 17, 55; Ex. 15 at 258. A week later, on June 6, 2015, Petitioner had a brain MRI completed, and on the same day followed up with Dr. Alroughani regarding her results. Ex. 12 at 18; Ex. 15 at 263. This MRI showed brain and cervical spinal cord lesions, indicative of demyelinating disease. Ex. 15 at 263. Two days later, on June 8, 2015, Dr. Alroughani wrote a letter addressed "to whomsoever it may concern," stating that Petitioner had undergone an NCS of her upper extremities to rule out entrapment neuropathy,[6] but her results came back normal. *Id.* at 256.

On June 20, 2015, Petitioner reported neck pain to Dr. Alroughani, who noted she was "unable to lift head," and he prescribed intravenous Solu-Medrol (a steroidal anti-inflammatory treatment) and other medication for neuropathic pain, which she received again a few days later. Ex. 12 at 18; Ex. 15 at 252. At this time, her diagnosis was "myelitis." Ex. 15 at 252.[7] The following month, on July 14, 2015, Petitioner presented to the Orthopedics Department of the Al Seef Hospital. Ex. 12 at 15. She complained of back and left ankle pain due to a fall the day prior. *Id.* Her exam showed neck and back tenderness and left ankle swelling. *Id.* X-rays also revealed narrowing of disc spaces in the cervical spine and no left ankle fracture, although a small planter calcaneal spur was observed. *Id.* at 15, 27–29. Petitioner also underwent a lumbar spine MRI that showed minor arthropathy at L5-S1 and "spasm of the back muscles." *Id.* at 30.

"enhancement," or image brightening, of a lesion on MRI suggests the existence of an ongoing and acute inflammatory process at the time of the scan. *Id.* at 165.

[6] Entrapment neuropathy is a condition in which a nerve becomes compressed, or entrapped, between two other structures in the body. *Entrapment Neuropathy,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92671&searchterm=entrapment+neuropathy (last visited Dec. 8, 2022).

[7] Records have been filed for appointments on July 4, July 11, and August 1, 2015, but all appear to be associated with insomnia. Ex. 12 at 19.

Continued Medical Treatment

Through August 2015, Petitioner continued to undergo orthopedic evaluations, receiving physical therapy and pharmaceutical treatments for her condition but without a clear consensus from treaters regarding the likely etiology for her symptoms. Then, Dr. Alroughani wrote a "Medical Report" (dated August 9, 2015) providing a summary of Petitioner's neurologic treatment which was more specific in proposing a possible explanation. Ex. 12 at 55. This document states that Petitioner had been evaluated in May 2015 for a "2-month history of paresthesia of both hands that were persistent and progressive in nature as there were ascending to the arms." *Id.* Dr. Alroughani noted that upon examination Petitioner displayed decreased sensation in her hands to mid-forearms, brisk reflexes in the upper extremities, and planter responses that were normal. *Id.* His impression was that her "presentation along with the radiological features of the cervical spine is supportive of a diagnosis of myelitis which is an autoimmune disorder usually idiopathic but can also be a presenting feature of a demyelinating disorder such as Multiple Sclerosis." *Id.* During this visit Petitioner "denied any history of recent immunization or infection." *Id.*

Thereafter, subsequent treaters began to suspect MS as the most likely explanation for Petitioner's overall symptoms. On September 4, 2015, for example, Petitioner was evaluated by neurologist Suprabha Bhat, M.D. Ex. 11 at 31. At this visit, Petitioner reported that starting in March 2015 she began to experience bilateral, progressive, ascending upper-extremity paresthesia. *Id.* She also reflected that she had "similar symptoms of bilateral hand numbness, paresthesias and pain while stationed in Iraq in 2010 . . . she was diagnosed and treated for carpal tunnel syndrome in 2010, when her symptoms resolved. The same symptoms of hand numbness, paresthesia and pain recurred in March 2015 while stationed in Kuwait." *Id.* Petitioner informed Dr. Bhat that the Solu-Medrol treatment she had received in June 2015 resolved most of her symptoms except for a burning sensation in both hands. *Id*. Dr. Bhat ordered an MRI and blood panel to further assess Petitioner for a demyelinating disease. *Id.* at 32–33.

A few days later, on September 10, 2015, Petitioner underwent another MRI of the brain and cervical spine. Ex. 4 at 22–25. MRI images of the brain now showed "[s]mall ovoid well-defined T2 signal hyperintensity . . . in the periventricular white matter at the atrium of the left lateral ventricle." *Id.* at 24. Consistent with the June 2015 MRI, this study also noted "[a] smaller lesion . . . in the periventricular white matter of the mid to posterior right temporal lobe." *Id.* These results were interpreted as two periventricular lesions that were most likely demyelinating plaques "based on location and morphology of the lesions." *Id.* at 25. Similarly, the cervical spine MRI was interpreted as showing "findings . . . most consistent with demyelinating plaques in the cervical spinal cord." *Id.* at 22.

On September 14, 2015, Petitioner followed up with Dr. Bhat to review the MRI results. Ex. 11 at 19–20. Dr. Bhat reported a suspicion of "relapsing multiple sclerosis," and ordered a cerebrospinal fluid ("CSF") study—but (inconsistent with an MS diagnosis) it did not reveal any oligoclonal bands,[8] and otherwise revealed normal levels of CSF protein electrophoresis and normal IgG synthesis rate. *Id.* at 15, 20. The CSF also contained normal levels of white blood cells and protein, and high level of red blood cells, but it did show slightly elevated CSF/serum albumin ratio "consistent with mildly increased permeability of the blood brain barrier." *Id.* at 14–15.

Petitioner followed up with Dr. Bhat again on September 25, 2015, complaining of urinary frequency, urgency, and incontinence, as well as ongoing upper-extremity paresthesia. Ex. 11 at 11.[9] Dr. Bhat's assessment was that since Petitioner had "multiple demyelinating lesions in her brain and cervical cord, [and] had 2 episodes of ascending numbness in both upper extremities since 2010," she could be diagnosed with "relapsing remitting multiple sclerosis." *Id.* at 11–12.

The following month, on October 22, 2015, Petitioner saw Dr. Bhat again, complaining of fatigue, hand and hip pain, numbness in the right foot, blurry vision, and ongoing urinary frequency, urgency, and incontinence. Ex. 11 at 8. Her examination revealed normal results, but Dr. Bhat noted that "fatigue and pain are the 2 most common symptoms in patients with MS." *Id.* at 9. Petitioner was advised to continue previously-prescribed medications, told not to return to work until January 2016, and started on fatigue-specific treatments. *Id.*

Petitioner sought a second opinion, and to that end presented to neurologist Julia Jones, M.D., on October 28, 2015. Ex. 4 at 26.[10] After discussion of Petitioner's medical history and a physical exam, Dr. Jones noted that Petitioner had experienced an episode of TM in the past (presumably, while in Kuwait as reported by Petitioner, though there is no additional information provided or treater support for this assertion), but that Petitioner met the criteria for remitting MS based on her current workup and history since March 2015. *Id.* at 28. Dr. Jones also observed that there was a new lesion evident from imaging, which she characterized as "typical MS changes on the MRI brain." *Id.* at 28.

[8] Oligoclonal bands, or bands of immunoglobulins, in the CSF are evidence of inflammation deemed a biomarker for MS. *Oligoclonal Bands,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=60106&searchterm=oligoclonal+bands (last visited Dec. 8, 2022).

[9] Respondent incorrectly identified the date of this visit as September 27, 2015. Respondent's Prehearing Brief at 7.

[10] Similar findings were reported in Ex. 9 at 7–9, though it appears it be formatted differently. There is no additional mention of a TM diagnosis besides this visit.

Petitioner's MS has since been confirmed by other treaters, based on her subsequent course and further exams and testing. Because disposition of this case does not turn on her diagnosis or disease progression, no further discussion of her subsequent treatment is called for.

II. Witness Testimony

A. *Fact Witness – Charlotte Renee Porch*

Ms. Porch was the only fact witness, and her testimony focused mainly on describing the state of her health both before and after receiving the MMR vaccine in February 2015. *See generally* Tr. at 5–19. She was diagnosed with carpal tunnel in 2010 that she initially treated with Tylenol, but she eventually received a single steroid injection which alleviated the symptoms. *Id.* at 6, 15–16. At that time, she was working on a military project in Iraq. *Id.* at 6, 16. Ms. Porch claimed to have received five or six vaccines, including the MMR vaccine, in October 2014, in association with her position—though the medical records *only* indicate prior receipt of an MMR dose. Ex. 1 at 5–8; Tr. at 18. She subsequently received a second MMR dose on February 11, 2015, as a requirement of her processing to go back to the Middle East. Tr. at 6–7.

Upon arrival in Kuwait on March 7, 2015, Petitioner reports having been in good health and able to complete the fitness for duty exam that was required to return to the project on which she worked. Tr. at 7, 16. By the end of March, however, she began experiencing neck pain and tingling in her forearms, hands, and feet. *Id.* at 7–8. Her symptoms progressed through the spring and summer of 2015, and she described having chronic pain and weakness in her legs. *Id.* at 8. She fell three times while in Kuwait and recalled feeling weakness in her legs and tingling in her feet when these incidents occurred. *Id.* at 9, 16. On one of those occasions, however, the fall was caused by the steps she was walking up being wet and slippery. Tr. at 17. Ms. Porch also experienced other symptoms, like bladder issues. *Id.* at 9. She returned to the United States in the fall of 2015 and saw Dr. Bhat for her chronic pain and leg weakness as well as tingling in her hands and forearms. *Id.* at 8–10. She was diagnosed with relapsing-remitting MS and was prescribed medications with unpleasant side effects. *Id.* at 10.

Since 2016, Ms. Porch has experienced several flare-ups of MS, entailing chronic pain, fatigue, and bladder issues, and which prevent her from getting up and walking. Tr. at 10–11. She reported that her flare-ups typically last between seven to eight days, and in 2021 she experienced five flare-ups, which were more than usual. *Id.* at 10–11, 17. Ms. Porch continues to have issues with falling due to the weakness in her legs and she feels fatigued all the time. *Id.* at 12–13. Steroid medication helps control her symptoms, but she nonetheless experiences chronic pain and stiffness in her legs and joints in between flare-ups. *Id.* at 12. These symptoms have profoundly impacted her life—Ms. Porch experienced depression after her diagnosis and is concerned about the long-term side effects of steroid use. *Id.* at 13–14. She has also been diagnosed with osteoarthritis in both of her knees and is going through the clearance for gastric bypass surgery. *Id.* at 14, 17.

B. *Petitioner's Expert – Jeffrey Rumbaugh, M.D., Ph.D.*

Dr. Rumbaugh, a licensed neurologist, submitted two written expert reports and testified for the Petitioner, alleging that the MMR vaccine could cause MS and did so in this case.[11] See generally Tr. at 19–144, 250–52. Report, dated May 1, 2019, filed as Ex. 22 (ECF No. 27-2) ("Rumbaugh First Rep."); Report, dated May 14, 2021, filed as Ex. 58 (ECF No. 63-2) ("Rumbaugh Second Rep.").

Dr. Rumbaugh attended Haverford College for his undergraduate degree in chemistry. Tr. at 20; *see* Curriculum Vitae, filed May 1, 2019 (ECF No. 27-3) ("Rumbaugh CV") at 1. He then attended the University of Rochester for his Master of Science in biochemistry, doctorate in biochemistry, and medical degree. Tr. at 20–21; Rumbaugh CV at 1. He completed his medicine internship and neurology residency at Johns Hopkins Hospital, where he later became the chief resident in neurology. Tr. at 20; Rumbaugh CV at 1. He most recently started a private practice in Tampa, Florida where his outpatient practice is focused on evaluating and treating patients with multiple sclerosis. Tr. at 22; Rumbaugh First Rep. at 2. Dr. Rumbaugh is also a tele-neurologist at the National Comprehensive Neurology Services in Tampa, Florida. Rumbaugh. Tr. at 22, 29; Rumbaugh CV at 1. He is board certified in neurology by the American Board of Psychiatry and Neurology, a member of the American Academy of Neurology and the American Neurological Association, and a licensed physician in Florida. Tr. at 20–21; Rumbaugh First Rep. at 2. Over the span of his career, he has treated thousands of MS patients. Tr. at 28. He has also published articles on caring for patients with MS and neurological infections. *Id.* Most of his basic science research and publications have focused on the neurological complications of human immunodeficiency virus and Lyme disease. *Id.* at 84.

Dr. Rumbaugh began with a discussion of MS. Tr. at 35–39. Although its etiology is not completely understood, MS is believed to be an autoimmune disease in which the immune system attacks the nerve myelin[12] in the central nervous system ("CNS"). *Id.* at 35, 38, 47–48, 85–87, 127; Rumbaugh First Rep. at 4, 6. Inflammatory damage to the myelin causes a disruption in the flow of signals through the nerves, leading to neurological symptoms. Tr. at 35–36.

[11] Dr. Rumbaugh's expert reports also included the contention that the MMR vaccine significantly aggravated Petitioner's pre-existing MS-like symptoms from 2010. Rumbaugh First Rep. at 1, 8–10; Rumbaugh Second Rep. at 1. However, during the hearing he maintained that Petitioner did not have any MS symptoms before arriving in Kuwait on March 8, 2015. Tr. at 114–15. Otherwise, Petitioner does not advance a significant aggravation claim.

[12] Myelin is the insulation that wraps around nerves protectively. Tr. at 35. Autoimmune responses to myelin proteins are elevated in patients with MS. *Id.* at 48.

Although there are different forms of MS,[13] the variant pertinent to this case is relapsing-remitting MS. Tr. at 36. In relapsing-remitting MS, a patient experiences intermittent episodes with associated neurological symptoms for a period of time, with symptoms appearing in peaks and waves. *Id.* The patient generally gets better between flares, but the overall continuation of flares/attacks can damage the CNS sufficiently to cause longer-term disability. *Id.* at 36, 85. There are distinct immunopathological characteristics for different forms of MS, with the relapsing-remitting form characterized by active inflammation, and it therefore calls for pharmaceutical treatments that might not be as useful in treating other MS variants. *Id.* at 37.[14]

Petitioner was correctly diagnosed, according to Dr. Rumbaugh, with relapsing-remitting MS. Tr. at 35, 39; Rumbaugh First Rep. at 3; Rumbaugh Second Rep. at 1. He briefly summarized what in the medical record supported the diagnosis. Tr. at 39–41. Petitioner developed neurological symptoms in 2015, showing symptoms of difficulty with her hands, numbness and tingling, imbalance, and bladder problems. *Id.* at 39–40. She experienced various episodes in 2015, and then five flares in 2021—all consistent with the relapsing-remitting form. *Id.* at 40, 85, 123. In addition, her June 6, 2015 MRI imaging (which revealed the presence of brain and spinal cord lesions) was strong evidence of MS. *Id.* at 40; Ex. 15 at 263; Rumbaugh First Rep. at 3. Although Petitioner did not test positive for oligoclonal bands in her CSF, Dr. Rumbaugh argued that a percentage of MS patients do not manifest this common biomarker (and there is not otherwise a specific single test to confirm an MS diagnosis in any event). Tr. at 40–41. Dr. Rumbaugh opined that Petitioner is likely to continue to experience relapsing-remitting MS. *Id.* at 81–83.

Dr. Rumbaugh then discussed TM, which appeared early in Petitioner's medical records (around spring and summer of 2015) as a potential diagnosis. Tr. at 41–42; Rumbaugh First Rep. at 3. He defined TM as inflammation of the spinal cord, noting that it often is a presenting clinical symptom for MS.[15] Tr. at 41–42. He explained that MS affects three parts of the nervous system: the brain, optic nerves, and spinal cord. *Id.* at 42. When MS initially affects the spinal cord, it can appear to be TM. *Id.*; Rumbaugh First Rep. at 3. In Dr. Rumbaugh's view, when Petitioner was initially diagnosed with TM, her treaters were *actually* referring to inflammation of the spinal cord

[13] Other types of MS include primary progressive, which is a persistent neurological condition or symptom, and secondary progressive, which begins with a pattern of relapsing-remitting MS, but over a number of years becomes more progressive, with less evidence of improvement after an attack. Tr. at 36–37. Why some patients develop one form of MS over another is unknown, although Dr. Rumbaugh opined that a person's individual genetic makeup likely explained the differences. *Id.* at 38–39; Rumbaugh Second Rep. at 4.

[14] Thus, in relapsing-remitting MS, medications are aimed at reducing inflammation and addressing the imbalance in the immune system. Tr. at 37. Progressive MS, by contrast, is more neurodegenerative without active inflammation, lessening the effectiveness of inflammation-targeting drugs and treatments. *Id.* at 37–38.

[15] There are other possible causes of TM when it is a single, self-limiting occurrence, such as conditions that cause inflammation on the spinal cord or other organs that secondarily impacts the spinal cord, although it can also be idiopathic. Tr. at 42.

as a part of MS. Tr. at 42, 94; Rumbaugh First Rep. at 3. He thus did not propose that the TM diagnosis was ultimately accurate, even if Petitioner's *initial* presentation could have involved the kind of spinal cord damage associated with a single case of TM. Tr. at 42, 94; Rumbaugh First Rep. at 3.

Dr. Rumbaugh also addressed the chronology of Petitioner's symptoms, pre- and post-vaccination. Tr. at 42–44. Petitioner was told in 2010 that she had carpal tunnel syndrome, which sounded reasonable to Dr. Rumbaugh, although he admitted he could not know definitively what caused these symptoms or how she was evaluated, because the relevant medical records were never located. *Id.* at 43, 114. More evidence of her course was found in a letter written by one of Petitioner's treating physicians while in Kuwait (Dr. Alroughani), addressed "to whomsoever it may concern." Tr. at 43; Ex. 12 at 55. The letter stated that Petitioner was initially seen in May 2015, when she presented with a two-month history of paresthesias in both hands that were ascending to her arms. Tr. at 43; Ex. 12 at 55. This suggested that her onset began in March 2015. Tr. at 44.

Next, Dr. Rumbaugh explained the causal theory at issue. He first noted that vaccines can cause injuries in rare instances. Tr. at 44–45; Rumbaugh First Rep. at 4. Because the immune system is usually adept at distinguishing between self-proteins and foreign agents, vaccines do not generally pose autoimmune risk, but occasionally internal safeguards against cross-reactivity break down. Tr. at 44–45; Rumbaugh First Rep. at 4. For this kind of self-protective "failure" to occur, a patient must possess some underlying susceptibility. Tr. at 46, 59–60, 87; Rumbaugh Second Rep. at 4. Under such circumstances, medical science agrees that an aberrant autoimmune process can lead to "neurological complications" (even if not every detail of the process involved is understood). Tr. at 44–46; Rumbaugh Second Rep. at 3–4.

Second, Dr. Rumbaugh highlighted the different external/environmental triggers thought to possibly incite MS. Science has proposed that a variety of external factors (e.g., surgery, insect bites, allergy immunotherapy, or certain forms of chemotherapy) can provoke MS in a susceptible individual. Tr. at 48; Rumbaugh First Rep. at 6–7. Recent research has particularly focused on viral infections as causal of autoimmune disease generally. Tr. at 48, 87; Rumbaugh First Rep. at 6-7; Second Rep. at 1–2; M. van Gemeren et al., *Vaccine-Related Autoimmune Hepatitis: The Same Disease as Idiopathic Autoimmune Hepatitis? Two Clinical Reports and Review,* 52 Scandinavian J. Gastroenterology 18, 20 (2017), filed as Ex. 29 (ECF No. 27-9) ("van Gemeren").

Since vaccines are engineered in part to invoke an immune response comparable (if more controlled) to what a wild virus or bacterium would prompt, they too should be capable of triggering MS. Tr. at 48–50; Rumbaugh Second Rep. at 1–2. Here, the MMR vaccine was in Dr. Rumbaugh's view a credible MS trigger. Tr. at 48–50; Rumbaugh Second Rep. at 1–2. The vaccine's measles antigens are equivalent to wild type measles viral antigens—the vaccine itself

is a live attenuated virus vaccine, in which live but weakened viral components are included to spark immune memory without also causing disease. Tr. at 49.[16]

Medical literature was offered by Dr. Rumbaugh to support his vaccine-MS association conclusions—although not all items proved supportive of his opinion. Tr. at 50–56, 58. First, he discussed a meta-analysis of case studies of CNS demyelinating disorders secondary to vaccination. *Id.* at 50–52, 93; Rumbaugh Second Rep. at 2; D. Karussis & P. Petrou, *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, Autoimmunity Rev.'s 215, 221 (2014), filed as Ex. 64 (ECF No. 63-8) ("Karussis"). Karussis conducted a literature search of instances between 1979 and 2013 of reports of post-vaccination CNS demyelinating diseases—including nine instances of vaccines against measles or rubella (although none specifically involving MMR vaccine). Karussis at 218–19. Although Karussis observed instances of demyelinating disease (most commonly optic neuritis), and discussed other findings relating MS to vaccination, MS was not one of the five clinical syndromes focused on by its authors.[17] Tr. at 93–94; Rumbaugh Second Rep. at 2; Karussis at 215, 220. And Karussis ultimately concluded that the overall risk of post-vaccination demyelinating disease was "relatively low," especially in comparison to the greater risk posed by a wild infection. Karussis at 221.

Second, Dr. Rumbaugh referenced an epidemiological study looking at several vaccines, but heavily focused on the Hepatitis B and HPV vaccines. Tr. at 95–96; A. Langer-Gould et al., *Vaccines and the Risk of Multiple Sclerosis and Other Central Nervous System Demyelinating Diseases*, 71 JAMA Neurology 1506, 1512 (2014), filed as Ex. 65 (ECF No. 63-9) ("Langer-Gould"). Langer-Gould performed a retrospective, case-control study of 780 patients who had been properly diagnosed with a CNS-specific demyelinating syndrome (including MS) after receiving *some* type of vaccination. Langer-Gould at 1506, 1508. It observed a greater risk for MS in the sample vaccinated group within 30 days of vaccination, in particular for patients younger than 50 years old. Tr. at 52–53; Rumbaugh Second Rep. at 2; Langer-Gould at 1508–10.

Yet Langer-Gould also admitted that its data did not establish a causal link between the current vaccines and the risk of MS (or other CNS demyelinating syndromes for that matter). Tr. at 97; Langer-Gould at 1509–10. Indeed, to the extent there was any observed short-term post-

[16] Dr. Rumbaugh also addressed (albeit more in passing) the possibility that the vaccine's other two antigens—mumps and rubella—could be causal. Tr. at 69–70. Mumps and rubella antigens are also live attenuated viruses, like the measles antigen. *Id.* at 69. For the same reasons the measles virus could cause relapsing-remitting MS, Dr. Rumbaugh opined, the mumps and rubella antigens could have the same effect. *Id.* He also found it significant that these three different viruses in the MMR vaccine are "viewed" by the immune system simultaneously, increasing the chance that something could go wrong and cross-react with a self-antigen. *Id.* at 69–70.

[17] There was only one CNS condition of myelitis following the measles reported in Karussis, but Dr. Rumbaugh agreed that the TM referenced in Petitioner's medical records was a manifestation of her MS and not an isolated event—thus reducing the probative value of this item of evidence. Tr. at 94; Karussis at 219.

vaccination association, Langer-Gould's authors speculated that it was likely attributable to the fact that "vaccines are redundant enhancers of preexisting autoimmunity" (meaning the disease process had begun *pre*-vaccination) as opposed to initiating triggers. Langer-Gould at 1510. And its authors (noting that the MMR vaccine is more often than not administered to children, rather than adults who would bear an MS diagnosis) could not even distinguish between MMR boosters and single-doses of the MMR vaccine in reviewing the relevant data, further diminishing what, if anything, could be said about that vaccine's causal potentiality. *Id.* at 1507.

Dr. Rumbaugh, however, sought to minimize the conclusions to be drawn from the overall paucity of evidence involving the MMR vaccine. Tr. at 56–58. He noted that the vaccine is typically administered to young children, whereas MS is a disease of adults, so it would be rare that an adult would receive the MMR vaccine at a time in their life when it is likely to trigger MS—simply stating that meaningful data on the topic would not exist. *Id.* at 56. He also more generally maintained that larger-scale epidemiologic studies (which he admitted are useful for purposes of causation) would be difficult to perform, given MS's rarity. *Id.* at 56–57, 124–25; A. Steelman, *Infection as an Environmental Trigger of Multiple Sclerosis Disease Exacerbation,* 6 Frontiers in Immunology 1, 2 (2015), filed as Ex. 34 (ECF No. 28-5). Because MS is likely multi-factorial in cause, especially given the role individual susceptibility plays, it would be difficult to isolate a specific trigger through an epidemiological study. Tr. at 57–58.

Besides the above, Dr. Rumbaugh referenced some case reports. *See* N. Agmon-Levin et al., *Transverse Myelitis and Vaccines: A Multi-Analysis,* 18 Lupus 1198 (2009), filed as Ex. 63 (ECF No. 63-7) ("Agmon-Levin"). Agmon-Levin collected 37 case reports (based on a 39-year review of relevant literature) in which post-vaccination TM was reported. Agmon-Levin at 1199. Putting aside the fact that the article does not involve MS, however, Agmon-Levin observed only *six* instances of TM after receipt of the MMR or rubella vaccination. Tr. at 92–93; Agmon-Levin at 1200. And Dr. Rumbaugh agreed generally that case reports are not particularly strong proof in support of causation (although he defended their use herein, invoking the rarity of MS as a justification). Tr. at 95, 137–38.[18]

Dr. Rumbaugh next proposed a mechanism for how vaccination could result in MS, offering the theory of molecular mimicry. Tr. at 58–60, 89; Rumbaugh First Rep. at 6; Rumbaugh Second Rep. at 2. Molecular mimicry, he explained, occurs when the immune system confuses a

[18] I also note that Agmon-Levin (which is often cited by Vaccine Program claimants as evidence of CNS-oriented autoimmune demyelinating diseases after vaccination) has been criticized in many other cases, since its limited findings had to be dredged from a lengthy reporting period of nearly 40 years, thus revealing very few instances of post-vaccination adverse events. *See Pearson v. Sec'y of Health & Hum. Servs.*, No. 16-9V, 2019 WL 3852633, at *14 (Fed. Cl Spec. Mstr. July 31, 2019) (giving limited weight to Agmon-Levin in a case alleging that flu vaccine caused TM, since Agmon-Levin referenced only two post-flu vaccine TM cases, despite the number of years of data considered).

self-protein for a foreign antigen (because the two appear similar, structurally[19] or in molecular composition), and attacks itself via antibodies or T cells that were generated in response to the mimic foreign antigen. Tr. at 58–59, 89, 90–91; Rumbaugh First Rep. at 4–5 (describing molecular mimicry as a cross-reaction mediated by similarities in chemical structure between the body's own protein and then a pathogen); *see also* van Gemeren at 20 (identifying molecular mimicry as one of the mechanisms "postulated or . . . confirmed to cause development of an autoimmune disease"). Molecular mimicry is in fact widely accepted among neuroimmunologists as a potential mechanism of autoimmunity—and Dr. Rumbaugh maintained the MMR vaccine could result in MS (understood to be an autoimmune-mediated disease) through this biologic mechanism. Tr. at 59–67.

In support, Dr. Rumbaugh referenced literature suggesting that antibodies generated in response to the measles virus could erroneously cross-react with vimentin, a cytoskeletal/structural intermedial filament protein.[20] Although it is found in cells throughout the body (and thus not only in the CNS), vimentin is also expressed in the CNS astrocyte cells[21] that (among other things) assist the oligodendrocytes[22]—another specialized CNS cell that generates and maintains nerve myelin. Tr. at 65–66, 101, 105. But (as purportedly established in one item of literature) antibody attacks on the vimentin found in the astrocytes could have a downstream, deleterious effect on oligodendrocyte myelin maintenance, resulting in MS. *Id.* at 61–67, 102–07; J. Srinivasappa et al.,

[19] In his second expert report, Dr. Rumbaugh noted that molecular mimicry could occur even where the self and foreign antigens lacked sequential similarity (in amino acids or other building blocks), but there was a structural/three-dimensional similarity, arising from the manner in which the linear peptide components of a protein might "fold." Tr. at 90–91, 126; Rumbaugh Second Rep. at 4. However, he acknowledged that he could cite no evidence supporting the concept that structural similarity between MMR vaccine components and CNS-myelin-related structures is actually linked to pathogenic autoimmunity. Tr. at 91–92, 126–28; Rumbaugh Second Rep. at 4.

[20] Intermediate filament proteins are involved in the structural integrity of a cell, according to Dr. Rumbaugh. Tr. at 63–64; *Intermediate Filaments,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=75869 (last visited Dec. 8, 2022). They help maintain the cell membrane, organelles, and subcomponents of the cell. Tr. at 63–64. Dr. Rumbaugh acknowledged, however, that he had not addressed vimentin as a proposed target for autoimmune cross-reaction leading to MS in his written reports. *Id.* at 89–90, 101. He also acknowledged a debate involving the distinction between intracellular and extracellular antigens as targets of antibodies produced in response to the measles vaccine, but maintained that there was a growing body of evidence that intracellular antigens (like vimentin) can also trigger an antibody response despite their location. *Id.* at 250–51.

[21] Astrocytes cells are a major component of the CNS. *Astrocyte,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4587&searchterm=astrocyte (last visited Dec. 8, 2022). They are specialized support cells that assist many processes in the CNS—including providing nutrients and other inputs needed by the oligodendrocytes to produce myelin. Tr. at 65–66.

[22] Oligodendroglia is "the non-neural cells of ectodermal origin forming part of the adventitial structure (neuroglia) of the central nervous system; projections of the surface membrane of each of these cells (oligodendrocytes) fan out and coil around the axon of many neurons to form myelin sheaths in the white matter. . . ." *Oligodendroglia,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=34894 (last visited Dec. 8, 2022).

Molecular Mimicry: Frequency of Reactivity of Monoclonal Antiviral Antibodies with Normal Tissues, 57 J. Virology 397, 397 (1986), filed as Ex. 66 (ECF No. 66-2) ("Srinivasappa").

In Srinivasappa (an animal study published 36 years ago), researchers generated more than 600 monoclonal (meaning lab-created)[23] antibodies against 11 different viruses, including 39 against the measles virus, and then screened them for reactivity against 14 different organs in healthy mice. Tr. at 61, 110; Srinivasappa at 397, 400. Generally, Srinivasappa's authors found that 3.5 percent of the antibodies in the study reacted with uninfected healthy tissue, leading to the conclusion that a cross-reaction was common. Tr. at 61; Srinivasappa at 397, 400. Five of the antibodies generated in response to the measles virus cross-reacted (a higher number than any group of antibodies at issue save one)—although only one[24] had any CNS involvement, and Srinivasappa's authors noted that disease pathogenesis could not be assumed simply on the basis of a single instance of cross-reactivity. Tr. at 113; Srinivasappa at 400. Indeed, Dr. Rumbaugh admitted that there was no way of knowing whether the five measles-specific monoclonal antibodies *would* be produced in response to the wild measles virus itself, let alone to the vaccine. Tr. at 111, 137. And Srinivasappa said nothing about vimentin in CNS cells as being the likely situs of a measles-responsive cross-reaction capable of resulting in MS. *Id.* at 110.

Dr. Rumbaugh also discussed two other articles. *See* R. Fujinami et al., *Molecular Mimicry in Virus Infection: Crossreaction of Measles Virus Phosphoprotein or of Herpes Simplex Virus Protein with Human Intermediate Filaments*, 80 Proceedings. Nat'l Acad. Sci.'s 2346, 2348–49 (1983), filed as Ex. 67 (ECF No. 66-3) ("1983 Fujinami"). This article (even older than Srinivasappa) observed that phosphoprotein[25] antibodies to the measles virus (and to a lesser extent the herpes simplex virus protein)[26] could cross-react with an intermediate filament protein—likely vimentin, although the article did not formally confirm this).[27] Tr. at 63; 1983 Fujinami at 2346. During cross examination, however, Dr. Rumbaugh acknowledged that the measles strain that was tested in 1983 Fujinami had been *discontinued* in 1970. Tr. at 103–04; 1983 Fujinami at 2346. Dr.

[23] Dr. Rumbaugh proposed that it was reasonable to conclude that the monoclonal antibodies would be similar in their chemical structure to antibodies generated *in vivo* and in reaction to a wild virus. Tr. at 136.

[24] Specifically, only the monoclonal antibody 3 (MV) was identified as reacting with the cerebellum and the hippocampus. Tr. at 11; Srinivasappa at 399.

[25] Dr. Rumbaugh defined a phosphoprotein as a protein that was modified by the body to contain a phosphate group. Tr. at 63.

[26] In July 2012, Petitioner's lab work was positive for herpes simplex virus 1 and 2 antibodies—the same virus that the authors in this study theorized as reacting with the intermediate filament protein vimentin. Tr. at 105–07; Ex. 8 at 3. However, Dr. Rumbaugh did not believe this likely-resolved infection could suddenly trigger an autoimmune reaction in 2015. Tr. at 135–36.

[27] Although 1983 Fujinami's authors theorized it was vimentin, based on a similar molecular weight and staining pattern, they never directly confirmed it was vimentin in the body of the article. Tr. at 102. And Dr. Rumbaugh admitted that 1983 Fujinami only said that vimentin *may* have certain sequences or service configurations in common with the measles virus phosphoprotein. *Id.* at 105; 1983 Fujinami at 2349.

Rumbaugh did not know any details about this original strain, or about the strain used today in the MMR vaccine (Enders attenuated Edmonston strain), and thus he could not provide insight into whether the results specific to the version considered in 1983 Fujinami still had applicability to the version of the vaccine likely received by Petitioner. Tr. at 104–05. However, he did not deem the difference to be significant, because the measles virus was used in both scenarios, and though differing strains can have individual characteristics and properties, the virus *overall* has similar proteins, and should therefore elicit the same autoimmune response.[28] *Id.* at 132–33.

A second article was cited more to shore up those aspects of Petitioner's causation theory specific to the putative target for the autoimmune cross-reactive CNS attack leading to MS. R. Fujinami et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 Clinical Microbiology Rev.'s 80 (2006), filed as Ex. 31 (ECF No. 28-2) ("2006 Fujinami"). 2006 Fujinami is largely a review article summarizing its authors' views (published 15 years ago) on the state of medical and scientific knowledge regarding "three potential mechanisms for viral-induced autoimmune disease." 2006 Fujinami at 80. The article specifically states that "[i]n MS the target for immune mediated damage is the myelin producing cell, the oligodendrocyte, and the axon. Loss of oligodendrocytes either by direct viral infection or immune attack can lead to large areas of demyelination, since an oligodendrocyte can myelinate multiple axons with myelin." *Id.* at 84.

2006 Fujinami was not specific to MS, however, and Dr. Rumbaugh agreed that its authors did not embrace the view that *all* cross-reactivity was likely pathogenic. Tr. at 108–10; 2006 Fujinami at 81.[29] In fact, 2006 Fujinami seems to propose that MS could be triggered in association with an infection via a "fertile field" model, in which the immune system's response to an infection "sets up or tills the field," immunologically speaking, allowing MS to occur, rather than directly causing the demyelination and nerve damage associated with it. 2006 Fujinami at 84–85. But Dr. Rumbaugh did not in this case propose such a pathogenic mechanism, nor is there evidence that Petitioner possessed a pre-vaccination infection in any event with which vaccination could have interacted.

Dr. Rumbaugh otherwise acknowledged that these items of literature did not stand as particularly robust support for his theory. Only 1983 Fujinami discussed vimentin, for example, as a putative cross-reactive target—unlike 2006 Fujinami, which discussed myelin basic protein, and Srinivasappa, which mentioned *none* of the above. Tr. at 101, 108–12. He also agreed that 23 years separated the 1983 Fujinami and 2006 Fujinami articles—ample time for advancement in scientific understanding of the vimentin-oriented theory he outlined (although he offered no more

[28] As a comparison, Dr. Rumbaugh invoked the seasonal flu vaccine, which is formulated with different strains every year but can still trigger the same immune response or neurological complications. Tr. at 134–35.

[29] Dr. Rumbaugh also observed that 2006 Fujinami cited another article in which specific viruses were isolated from the brain of MS patients—suggesting (in Dr. Rumbaugh's opinion) that the measles virus may also be present. Tr. at 68; 2006 Fujinami at 84. However, Petitioner did not offer this article as evidence.

recent evidence suggesting any further research or developments into vimentin as an MS autoimmune target). *Id.* at 107–08. And none of the articles involved the MMR vaccine itself—although Dr. Rumbaugh insisted their virus-oriented findings were applicable, emphasizing that because the measles vaccine is a live attenuated vaccine, "it's basically the same virus." *Id.* at 62–63, 68, 103.

Petitioner's medical record was also, in Dr. Rumbaugh's view, supportive of the conclusion that the vaccine was causal of her MS. Tr. at 72–74, 80–81, 89; Rumbaugh First Rep. at 3, 6; Rumbaugh Second Rep. at 5. Prior to vaccination, Petitioner was in her usual state of health and without any known neurological symptoms (other than her 2010 carpal tunnel diagnosis). Tr. at 72. There was no evidence of any other known non-vaccine triggers for demyelinating diseases—viral infection, bacterial infection, surgery, insect bites, allergy, immunotherapy. *Id.* at 72–73; Rumbaugh First Rep. at 6–7. There were also no other risk factors for Petitioner's lesion activity,[30] evidenced by her June and September 2015 MRI results. Tr. at 75–79; Ex. 4 at 22–23; Ex. 15 at 263. Dr. Rumbaugh specifically proposed that the pattern of demyelination observed in such imaging studies was consistent with the theory of an autoimmune cross-reaction causing damage and dysfunction to oligodendrocytes in the CNS. Tr. at 77–79; 2006 Fujinami at 84. But he agreed on cross examination that more broadly this pattern was merely consistent with what is expected in *any* individual experiencing relapsing-remitting MS, and thus is not specific to any suspected cause. Tr. at 114.

The fact that Petitioner's February 2015 MMR dose was her second in less than six months was significant to Dr. Rumbaugh. Tr. at 79–80. Children alone were thought to receive lifelong immunity from a similar two-dose course, he noted, but with a longer period of time between doses.[31] *Id.* at 118. Since Petitioner's immune system had encountered the antigen in a far tighter timeframe, the immune system response to the second dose had to have occurred fairly fast, increasing the likelihood of an aberrant cross-reaction. *Id.* at 79–80, 118; Rumbaugh Second Rep. at 3. However, Dr. Rumbaugh acknowledged that a two-dose MMR regimen was recommended for adults as well (suggesting that science did not perceive much of a risk from such a dosage).[32]

[30] Dr. Rumbaugh embraced the idea that Petitioner's non-enhancing lesions (appearing as early as May 2015) were in reaction to the vaccine and had resolved by the time they were detected in later MRIs. Tr. at 141–44. This was not an event of radiological isolated syndrome ("RIS"), where there were lesions consistent with MS, but imaging discovered them before clinical symptoms had manifested. *Id.* at 143–44; Rumbaugh Second Rep. at 1.

[31] The MMR vaccine is typically given to children in a two-dose series, with the first administered at the age of 12-15 months, and the second at age 4-6 years. Rumbaugh Second Rep. at 3.

[32] On cross examination, Respondent referenced the CDC's "Pink Book"—a collection of information on vaccines and viruses—for these points, although it was not filed. Tr. at 119. Dr. Rumbaugh did not deem it surprising, however, that the Pink Book recommended two doses of the MMR vaccine for colleges and other post-high school educational facilities. *Id.* at 120, 139–41. He also admitted he did not find it difficult to accept that the Pink Book recommended people receive an additional dose before traveling outside the United States—as Petitioner had in her travel to Kuwait. *Id.* at 120.

Tr. at 118–19. And he offered no evidence establishing that the timeframe of Petitioner's dosage was out of the ordinary, or more truncated than medically appropriate.

Dr. Rumbaugh concluded his testimony by discussing Petitioner's likely onset date for her first MS symptoms, and whether it was consistent with his causation theory. He deemed Petitioner's testimony and medical records supportive of an onset date after her arrival in Kuwait but before the end of March 2015—most likely with her symptoms beginning between 30 to 45 days post-vaccination (or between March 13th and March 28th).[33] Tr. at 74; Rumbaugh Second Rep. at 2. Notably, however, the first medical record in which Petitioner mentioned an onset date for her initial numbness and pain in her hands is from May 31, 2015—at which time she stated her symptoms had been present for two months, or no sooner than the *end* of March 2015. Tr. at 115–16; Ex. 12 at 17.

In Dr. Rumbaugh's view, anything between two weeks to a few months from vaccination to onset would be a medically acceptable timeframe for stimulation of the immune system sufficient to produce CNS demyelination, depending on variables (the susceptibility of the individual, the strength of the stimuli, etc.). Tr. at 70–71. Thus, a mid to late-March 2015 onset was consistent with his theory, as well as relevant literature about other timeframes of other post-vaccination neurologic injuries.[34] *Id.* at 74, 80–81; *see generally* R. Bakshi & J. Mazziotta, *Acute Transverse Myelitis After Influenza Vaccination: Magnetic Resonance Imaging Findings*, J. Neuroimaging 248, 249 (1996), filed as Ex. 32 (ECF No. 28-3) (reporting neurological complications in a case report of the flu vaccine and appear with a latency of 1 to 63 days (with an average of 16.5 days)).

In support of this contention, Dr. Rumbaugh referenced Langer-Gould, which found the risk of CNS demyelinating disease following vaccination was elevated in patients under the age of 50 (and Petitioner was 47 at the time of her receipt of the second MMR dose). Langer-Gould at 1511; Tr. at 74–75. Dr. Rumbaugh admitted, however, that it was unclear in Langer-Gould how age was cross-referenced against timeframe, making it difficult to ascertain specifically when onset was more or less likely. Tr. at 97–98. This was not accurate for other literature he had offered. Agmon-Levin, for example, referenced six reports of TM following some combination of either MMR, measles or rubella, but with the oldest patient experiencing disease in a shorter timeframe (20-year-old and two-week onset) than the youngest (newborn infant experiencing onset a year

[33] Dr. Rumbaugh's first expert report proposed a broader onset timeframe around 18-48 days post-vaccination. Tr. at 115; Rumbaugh First Rep. at 7. But he deemed Petitioner's testimony to support a narrower timeframe. Tr. at 115.

[34] Dr. Rumbaugh also opined that the onset date would not differ if the initial immune stimulus were an infection or vaccination—the process to get from stimulus to neurological disease were basically the same. Tr. at 71–72.

post-vaccination).[35] *Id.* at 116–17, 138; Agmon-Levin at 1200. Karussis, by contrast, observed symptoms beginning in an average period of 14.2 days post-vaccination, although in some instances symptoms were delayed. Tr. at 117; Karussis at 215. However, the most relevant case study it cited involved a 19-year-old experiencing TM merely *four days* after measles vaccination—hardly comparable to Petitioner's circumstances. Tr. at 117–18; Karussis at 219.

Moreover, Dr. Rumbaugh acknowledged, Langer-Gould actually showed a *decreased* risk of post-vaccination adverse events as time passed. Tr. at 54–56, 75; Langer-Gould at 1511–12. Regarding MS, Langer-Gould observed that at roughly 14 days post-vaccination there was 2.58 times increased relative risk, slipping to 1.45 relative risk at 30 days, and 1.01 at 42 days.[36] Tr. at 53–56, 75, 98–99, 128–30; Rumbaugh Second Rep. at 2; Langer-Gould at 1511. Langer-Gould's authors actually found *no* increased risk of CNS demyelinating syndromes beyond 30 days, undermining the assertion of a causal link where onset was not close in time to the purportedly-causal vaccination. Tr. at 99–100, 130–31; Langer-Gould at 1509–10, 1512.

C. *Respondent's Expert – Jeffrey Gelfand, M.D., M.A.S.*

Dr. Gelfand, a board-certified neurologist specializing in caring for patients with a wide range of neurological and neuroinflammatory disorders, testified on behalf of Respondent, and submitted a single expert report. *See generally* Tr. at 145–249; Report, dated Sept. 16, 2019, filed as Ex. A (ECF No. 30-1) ("Gelfand Rep."). Dr. Gelfand discussed Petitioner's disease presentation and medical history, as well as other aspects of the evidence offered to support causation.

Dr. Gelfand attended Princeton University for his undergraduate degree. *See* Curriculum Vitae, filed as Ex. B (ECF No. 30-2) ("Gelfand CV") at 1. He then attended Harvard Medical School for his medical degree. Tr. at 146; Gelfand CV at 1; Gelfand Rep. at 2. He is currently an Associate Professor of Clinical Neurology at University of California, San Francisco ("UCSF"), where he also completed his medical residency with a subspeciality fellowship training in MS and neuroimmunology, and master's in advanced study in clinical research. Tr. at 145–48; Gelfand CV at 1; Gelfand Rep. at 2. He is also the Clinical Fellowship Program and Assistant Medical Director at the UCSF MS and Neuroinflammation Clinic. Tr. at 146; Gelfand Rep. at 2. He is board certified in neurology by the American Board of Neurology and Psychiatry and has a medical license in California. Tr. at 151; Gelfand Rep. at 2. He has also published several articles and editorials on

[35] Dr. Rumbaugh found it relevant that middle aged and older adults seldom get the MMR vaccine. Tr. at 138–39. This would explain why there is more of an association between younger people, as they are more likely to receive the vaccine. *Id.*

[36] Relative risk was explained by Dr. Rumbaugh as involving a comparison of two groups. If the groups have the same risk of getting a condition, the relative risk is 1.0. Tr. at 54. A relative risk of two, however, would mean that the observed group is twice as likely to get the condition. *Id.* A relative risk of 1.1 would thus indicate that an affected individual has a ten percent higher chance of developing the adverse event at issue than someone not similarly impacted. *Id.*

neurology, neuroimmunology, and MS. Tr. at 149. Dr. Gelfand has treated several hundred MS patients, seeing some annually, and has also treated patients with TM. *Id.* at 153–55.

Dr. Gelfand accepted Petitioner's relapsing-remitting MS diagnosis. Tr. at 156, 216; Gelfand Rep. at 5–6, 8.[37] He defined MS as a chronic inflammatory autoimmune condition thought to be caused by a mixture of environmental exposures and genetic susceptibility, but with no clear originating etiology. Tr. at 158, 176, 217–20; Gelfand Rep. at 6; D. Reich et al., *Multiple Sclerosis*, 378 New Eng. J. Med. 169, 172 (2018), filed as Ex. A, Tab 1 (ECF No. 31-1) ("Reich") (listing examples of environmental exposures, including the Epstein-Barr virus infection and mononucleosis, UV exposure, which may relate to the latitude effect, low vitamin D status, and smoking). Its "attacks" are acute inflammatory demyelinating events involving the CNS. Tr. at 157–59, 217. An attack manifests as a neurologic symptom often correlated with lesions observable on MRI. *Id.* at 157. MS is clinically diagnosed based on an appropriate history of symptoms, typically with supportive findings and features on MRIs, a rigorous exclusion of other potential causes, and possibly by additional evidence derived from spinal fluid or paraclinical tests. *Id.* at 169. Overall, a diagnosis of relapsing-remitting MS requires evidence of "dissemination and space and time," with clinical symptoms and/or lesions impacting distinguishable parts of the nervous system and in a chronic rather than monophasic process. *Id.* at 170.

Dr. Gelfand as a general matter disputed the contention that MS could be caused by *any* vaccine. Tr. at 198–202, 222–23; Gelfand Rep. at 6; Reich at 173–74 (not identifying vaccines as an environmental risk factor for MS, but acknowledging that not all causes of MS are known, and viruses are thought to be one cause). Although he agreed with Dr. Rumbaugh that vaccines trigger an immune response somewhat comparable to what foreign viruses or bacteria elicit, and that sometimes viruses and vaccines can be associated with specific autoimmune demyelinating diseases, he denied that the same association was true for MS. Tr. at 220–21. On the contrary, the weight of scientific evidence was strongest for associations with other forms of *acute* inflammatory demyelinating events which are often monophasic (e.g., Guillain-Barré syndrome ("GBS"))—not something like MS, which is chronic. *Id.* at 248; Langer-Gould at 1512 (finding the strongest association for monophasic conditions).

In addition, Dr. Gelfand noted, medical practice guidelines for patients with MS encourage vaccination (further undermining arguments about the danger that a vaccine could trigger a flare). Tr. at 208–11, 232–33; O. Rutschmann et al., *Immunization and MS: A Summary of Published Evidence and Recommendations*, Am. Acad. Neurology 1837, 1842–43 (2002), filed as Ex. A, Tab 7 (ECF No. 31-7) ("Rutschmann"); M. Farez et al., *Practice Guideline Update Summary: Vaccine Preventable Infections and Immunization in Multiple Sclerosis*, 93 Am. Acad. Neurology 1, 5, 8 (2019), filed as Ex. A, Tab 8 (ECF No. 31-8) ("Farez"). At most, vaccines containing live

[37] Dr. Gelfand described in detail the nature of Petitioner's MS, but because his diagnosis is not at issue, I will not summarize Dr. Gelfand's commentary or the records referenced therein. *See generally* Gelfand Rep. at 2–5; Tr. at 157–58; 161–75, 177–182.

components[38] are contraindicated for people who are actively immune-suppressed—but MS does not feature immunosuppression, and so the benefits of preventing intercurrent infections in MS patients far outweigh any negligible risk. Tr. at 208–10, 231–33; Gelfand Rep. at 7; Rutschmann at 1839 (reviewing the published literature, and unable to provide evidence that MMR causes either MS risk or an increased risk of relapse in people with MS, and concluding there was insufficient evidence to support or reject an association between the measles vaccine and MS exacerbation); Farez at 3, 5 (noting insufficient data to support or refute an association between MS and history of vaccinations generally and including MMR); *see also* C. Confavreux et al., *Vaccinations and the Risk of Relapse in Multiple Sclerosis*, 334 New Eng. J. Med. 319, 319, 324–25 (2001), filed as Ex. A, Tab 6 (ECF No. 31-6) ("Confavreux") (finding that a person with MS who gets vaccinated is not at an increased risk of another attack).[39]

Dr. Gelfand attempted to go further than simply opining that vaccination was safe for MS patients, offering literature he maintained rebutted *any* connection between the MMR vaccine and MS's instigation. *See, e.g.*, M. Mailand & J. Frederiksen, *Vaccines and Multiple Sclerosis: A Systematic Review*, 264 J. Neurology 1035 (2017), filed as Ex. A, Tab 2 (ECF No. 31-2) ("Mailand"). Mailand, a review article, considered the findings of nine studies examining the risk of developing MS following vaccination against measles, mumps and or rubella, with only one of the cited studies supporting an increased risk. Mailand at 1036, 1044, and 1048. Petitioner noted on cross examination that Mailand's authors had admitted they were motivated in part to bulwark the "public attitude towards vaccination" (based on instances in which vaccination coverage dropped when there appeared to be a temporal association between vaccination and certain adverse events), and thus might have been biased in their effort, but Dr. Gelfand accepted its authors' statement that they merely hoped to summarize what reliable and existing epidemiologic studies showed. Tr. at 240; Mailand at 1035. And he emphasized the fact that the article's methodology excluded numerous other articles and case reports. Tr. at 240–41; Mailand at 1035–36.

In addition, it was pointed out on cross examination (and in some detail as well) that most of the items of literature considered in Mailand had been criticized for methodologic reasons[40] by

[38] The MMR vaccine is a live attenuated vaccine. *Vaccinations,* Nat'l Multiple Sclerosis Society, https://www nationalmssociety.org/Living-Well-With-MS/Diet-Exercise-Healthy-Behaviors/Vaccinations, filed as Ex. A, Tab 5 (ECF No. 31-5).

[39] At worst, vaccination is associated with transient systemic reactions—and for MS patients this can encourage a response akin to a symptoms flare. Tr. at 215–16. But Dr. Gelfand noted there was inadequate research into whether such a reaction constituted a flare or was merely a pseudo relapse. *Id.*

[40] Respondent did not file any of these questioned items of literature, however, even if he indirectly relied on them through Dr. Gelfand's favorable citation to Mailand. Respondent did file Confavreux, by contrast, which Petitioner also pointed out had been methodologically questioned by the IOM. Tr. at 233–35, 242. But Confavreux (observing that vaccination is unlikely to cause MS symptom flares) only indirectly supported Dr. Gelfand's larger opinion, and in any event the IOM's criticism of Confavreux referenced on cross-examination is not otherwise set forth in the filed IOM Report (even if it is contained in that work elsewhere).

the Institute of Medicine (the "IOM")—and in an article *offered by Respondent*. Tr. at 202–03, 236–39, 243–44 (references omitted); *Evidence and Causality* 159 (K. Stratton et al. eds., 2011), filed as Ex. A, Tab 3 (ECF No. 31-3) ("IOM Report"), 159–61. Dr. Gelfand did not contest the legitimacy of these criticisms, but maintained they merely underscored overall limitations in what could be concluded from the "state of the literature." Tr. at 245. He also observed that Mailand considered at least one item of literature he *had* filed (along with Petitioner) that was not criticized by the IOM—Langer-Gould. Tr. at 245–47; Mailand at 1046.

Another piece of literature offered by Dr. Gelfand was a large population-based retrospective case-control study. Tr. at 203–08, 227–29; A. Hapfelmeier et al., *A Large Case-Control Study on Vaccination as Risk Factor for Multiple Sclerosis*, 93 Am. Acad. Neurology e908 (2019), filed as Ex. A, Tab 4 (ECF No. 31-4) ("Hapfelmeier"). Hapfelmeier's authors considered the relationship between an MS diagnosis and vaccinations (in the five years prior to diagnosis) of 12,000 German patients, including the MMR vaccine. Hapfelmeier at e909–10. Focusing on vaccination as the sole tested risk factor, Hapfelmeier observed no increased risk of MS post-vaccination, and found the odds of MS were actually *lower* after receipt of some vaccines (although the MMR vaccine was not identified in this group). *Id.* at e914–15. Dr. Gelfand also disagreed with Petitioner's reading of Hapfelmeier—that it deemed the vaccine-MS relationship "uncertain"—noting that in fact the article was explicit in finding otherwise, and only characterized the "uncertain" state of thinking on the subject as a threshold statement regarding the determination of a "large number of studies" *prior* to Hapfelmeier's findings. *Id.* at e912; Tr. at 228.

The IOM report also invited attack from Petitioner. The IOM Report looked closely at two of six epidemiologic studies involving MS and the MMR vaccine, rejecting four at the outset as having facial methodologic issues. IOM Report at 159. The remaining two were deemed not supportive of causation, but the IOM Report expressed "limited confidence" in their findings due to identified issues in the respective studies. *Id.* at 161. The IOM Report (although again on the basis of limited evidence) was, however, somewhat more definitive in finding a lack of "mechanistic evidence" showing how immune cells are stimulated by the MMR vaccine, which could lead to "onset of MS in adults." *Id.* Dr. Gelfand stressed this finding over the IOM's ultimate determination that there was insufficient evidence overall to accept or reject a causal relationship between the vaccine and the onset of MS in adults. Tr. at 202–03; Gelfand Rep. at 6; IOM Report at 164.

In addition to challenging the existence of studies supporting an MMR vaccine-MS link, Dr. Gelfand more specifically questioned the logic of Petitioner's theory of causation, disputing that it (with molecular mimicry as its mechanism) reliably explained how the MMR vaccine could cause MS. Tr. at 183, 222. He defined the theory of molecular mimicry as when the human immune system responds to a foreign antigen in a manner sufficient to overcome the typical self tolerance

that prevents autoimmunity, attacking the host at the situs having molecular similarity with the original antigen (due to mimicry between the two). Tr. at 183–84, 222. Dr. Gelfand acknowledged examples of particular bacterial infections that may have shared antigens with self myelin structures sufficient to cause GBS, a peripheral neuropathy also involving harm to nerve myelin. *Id*. at 185. To establish that molecular mimicry explains an autoimmune process, there must be evidence of sufficient similarity (molecularly, functionally, and immunologically) between the antigen and the target tissue in the host. *Id*. at 184. But additional steps are necessary for this kind of mimicry to have any consequential significance, for not all cross-reactivity attributable to molecular mimicry is pathogenic. *Id*. at 184–87.

Dr. Rumbaugh's literature used to support his causation theory was not, in Dr. Gelfand's view, persuasive on a fundamental point: how antigens in the MMR vaccine could trigger pathogenic autoimmunity leading to MS. Gelfand Rep. at 6. 1983 Fujinami, for example, suggested the possibility of antibodies that could target vimentin—a protein found *inside* the cell—but Dr. Rumbaugh had not provided *other* supportive links required by his theory to establish that putative cross-reactivity of this kind could in fact become pathogenic. Tr. at 189. For an autoimmune process to cause disease, purportedly cross-reacting antibodies must target a self antigen *in vivo*, thereby causing damage or interfering with other processes. *Id.* at 187–88. When proteins are on the *outside* of the cell, it is far easier to model how an antibody might directly cause pathology. Tr. at 188. But when antigens are intracellular, or *inside* the cell, there is a question as to how the allegedly-causal antibody would directly access the mimicked antigen. *Id.* at 188. In addition, 1983 Fujinami was published 35-40 years ago, but in the ensuing timeframe medical science never embraced an MMR virus cross-reaction with vimentin as a reliable hypothesis for a potential cause of MS. *Id.* at 189. And even if it were assumed that vimentin *could* constitute a target antigen, 1983 Fujinami (along with Srinivasappa and 2006 Fujinami) did not explain how such an initial response would lead to a chronic and persistent process culminating in MS. *Id.* at 189–90.

Dr. Gelfand also discussed the use of case reports, and the related argument that since MS is a rare disease case report evidence merits greater weight. Tr. at 197–98, 205–07. In Dr. Gelfand's view, case reports may provide clinical observations suggesting the need for more rigorous scientific exploration and study, but cannot substantiate causation on their own. *Id.* at 197–98. Nor did he deem persuasive the argument that it is not possible to design larger-scale studies to detect rare events like MS. *Id.* In Dr. Gelfand's understanding approximately 750,000 people in the United States have been diagnosed with MS, making it one of the most common neurologic conditions seen in clinical practice. *Id.* at 205–06. Otherwise, it was always possible to study rare diseases as long as an appropriate study design was employed. *Id.* at 206–07. Dr. Gelfand did not contest, however, that for many diseases and conditions, existing epidemiologic "tools" might be inadequate. *Id.* at 224–26.

Besides offering an opinion on the evidence pertaining to causation, Dr. Gelfand also expressed the view that Petitioner's medical record did not support a finding that the MMR vaccine she received in February 2015 had caused her MS. Tr. at 159, 211–12, 221. For example, he saw nothing in the record indicating that any treaters attributed her MS to the vaccine. Tr. at 183; Gelfand Rep at 6, 8. He also deemed the lesions viewed on MRI as typical in MS generally, rather than somehow consistent with a vaccine-induced form of autoimmune condition. Tr. at 77–79, 175. Nor was it out of the ordinary that Petitioner was in her usual state of health prior to vaccination, since it was very common for MS patients to suddenly experience clinical symptoms with no prior warning. *Id.* at 176.

Dr. Gelfand concluded by offering an opinion on onset. Based on his reading of the record, Dr. Gelfand proposed (consistent with other treaters) that Petitioner's MS began in mid to late March 2015 (or around five to six weeks following vaccination).[41] Tr. at 216–17; Gelfand Rep. at 5–6; Ex. 15 at 63, 258. But new MS lesions (which would likely predate clinical manifestations) typically enhance on imaging for a few weeks to months (with one to two months being the average),[42] and the lesions observed in May 2015 were not enhancing, meaning they had already resolved. Tr. at 165–66. The lack of MRI results from before May 2015 made it difficult to date Petitioner's progression or pinpoint her actual onset more carefully. *Id.*; Gelfand Rep. at 5–6.

Dr. Gelfand did not, however, accept a five to six-week post-vaccination onset to be medically reasonable. Tr. at 190. In support, he referenced Langer-Gould. *Id.* at 190–97. That article suggested an increased risk of post-vaccination MS at most very early after vaccination, and only for CNS *acute* demyelinating events—not chronic ones like MS.[43] *Id.* at 197. And MMR was not a common vaccine in this data set (in fact, Langer-Gould's focus was on Hepatitis B and HPV), so Dr. Gelfand did not find that the study showed the MMR vaccine could cause MS in this specific timeframe. *Id.* at 190–91, 197; Langer-Gould at 1511–12.

III. Procedural History

The matter was initially assigned to another special master after its filing in 2017. ECF No. 1. By June 2018, Petitioner had filed all relevant medical records, an affidavit, and a Statement of Completion. ECF No. 22. Respondent filed a Rule 4(c) Report on August 28, 2018, contesting

[41] Dr. Gelfand acknowledged Petitioner's 2010 hand-related symptoms might have been a first presenting MS symptom of some kind, but stated that without additional information there was not enough evidence to connect these earlier symptoms to her subsequently-diagnosed MS. Tr. at 216–17; Gelfand Rep. at 6.

[42] Dr. Gelfand did not substantiate this assertion with literature.

[43] Dr. Gelfand went into detail describing the use of the odds ratios, confidence intervals, and statistical significance to further explain Langer-Gould. Tr. at 193–97. Ultimately, he opined that the larger the sample size (so long as the data quality is good), the more power there is when observing an effect. Tr. at 213–14.

Petitioner's right to compensation. ECF No. 23. Expert reports were filed over the next two years. ECF Nos. 27, 30, 63. The matter was transferred to me on January 26, 2021, and I subsequently scheduled a hearing to be held on January 13-14, 2022. ECF Nos. 56, 62. With the submission of post hearing briefs (ECF Nos. 82, 86–87), the claim is now 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).[44] Petitioner does not assert a Table 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 & Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a

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

medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

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

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

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 Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)). 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 denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including

"any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (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, in comparison to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations

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

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

C. *Analysis of Expert Testimony*

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

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for

controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

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

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

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

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical

literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. Treatment of MS in Prior Program Cases

The parties agree that Ms. Porch was properly diagnosed with the relapsing-remitting form of MS (with her TM simply representing the first MS-associated flare rather than a distinguishable, single instance of disease). But some discussion of what is known about MS, and also how claims involving it in the Vaccine Program have been analyzed, will be useful in ascertaining whether entitlement is appropriate herein.

MS is a chronic disease process impacting the CNS that (regardless of its initial presentation) is likely to recur and/or progress. Reich at 169. In addition, MS can be subclinical and symptomatically silent for a long period of time, with MS-characteristic brain lesions often discovered in the absence of symptoms, or non-recent lesions discovered only after clinical manifestations. *Id.* at 169–72. Significantly (and of course independent of the burden of proof petitioners bear in Program cases), there is little to no direct scientific knowledge as to *why* MS recurs—and no similar evidence that a one-time neurologic "hit" can explain subsequent symptoms months or years later. *Id.* at 169, 172; Tr. at 74.

MS thus must be considered distinct from other demyelinating CNS injuries that the Program often considers (and awards damages for), *but* which are acute and monophasic, like TM or ADEM. *Compare Raymo v. Sec'y of Health & Human Servs*., No. 11-654V, 2014 WL 1092274, at *23 (Fed. Cl. Spec. Mstr. Feb. 24, 2014) (finding causal relationship between flu vaccine and TM) *with Wei-Ti Chen v. Sec'y of Health & Human Servs*., No. 16-634V, 2019 WL 2121208, at *22 (Fed. Cl. Spec. Mstr. Apr. 19, 2019) (determining there was insufficient evidence provided to support a causal connection between the flu vaccine and petitioner's subsequent development of neuromyelitis optica spectrum disorder, which is chronic and relapsing/remitting, like MS). I have noted the importance of this distinction in several prior decisions. *See, e.g.*, *Morgan v. Sec'y of Health & Human Servs*., No. 15-1137V, 2019 WL 7498665, at *16 (Fed. Cl. Spec. Mstr. Dec. 4, 2019), *mot. for review den'd*, 148 Fed. Cl. 454 (2020), *aff'd*, 850 F. App'x 775 (Fed. Cir. 2021); *Taylor v. Sec'y of Health & Human Servs.*, No. 13-700V, 2018 WL 2050857, at *28–29 (Fed. Cl.

Spec. Mstr. Mar. 9, 2018); *Caruso v. Sec'y of Health & Human Servs.*, No. 15-200V, 2017 WL 5381154, at *12–13 (Fed. Cl. Spec. Mstr. Oct. 18, 2017), *mot. for review den'd*, 137 Fed. Cl. 386 (2018).[45]

Because of the above, special masters have more often than not denied compensation in cases alleging MS as a vaccine-caused injury. *See, e.g.*, *Samuels v. Sec'y of Health & Hum. Servs.*, No. 17-071V, 2020 WL 2954953, at *18–19 (Fed. Cl. Spec. Mstr. May 1, 2020) (finding petitioner's actual injury was MS, an illness far less associated with vaccination than one-time acute CNS demyelinating events like ADEM); *Pek v. Sec'y of Health & Hum. Servs.*, No. 16-0736V, 2020 WL 1062959, at *17 (Fed. Cl. Spec. Mstr. Jan. 31, 2020) (determining that evidence and expert reports did not provide sufficient proof that a progressive, chronic demyelinating condition like MS could be initiated by the flu and Tdap vaccines); *Chen,* 2019 WL 2121208, at *22; *Hunt v. Sec'y of Health & Human Servs.*, No. 12-232V, 2015 WL 1263356, *15 (Fed. Cl. Spec. Mstr. Feb. 23, 2015) (denying entitlement where MS was the alleged injury, but the literature offered only discussed a causal relationship between vaccines and ADEM).

Admittedly, some special masters have gone in the opposite direction, and granted compensation in MS cases—but my review of those decisions does not reveal any reasoned efforts to grapple with the distinctions highlighted above. *See, e.g., Robinson v. Sec'y of Health & Hum. Servs.*, No. 14-952V, 2021 WL 2371721, at *25 (Fed. Cl. Spec. Mstr. Apr. 12, 2021); *Hitt v. Sec'y of Health & Human Servs.*, No. 15-1283V, 2020 WL 831822, at *9–10 (Fed. Cl. Spec. Mstr. Jan. 24, 2020). Rather, their assumption appears to have been that if a vaccine can cause one kind of CNS autoimmune demyelinating injury, it can cause another.

II. Petitioner Has Not Carried Her Burden of Proof

A. *Althen Prong One*

The evidence offered in this case (mainly via the testimony of Dr. Rumbaugh) does not preponderantly support the conclusion that the MMR vaccine can cause MS. At the outset, I note that the wild viral versions of the vaccine's primary components are not themselves associated with MS, thus diminishing the value of the general argument (which Dr. Rumbaugh embraced) that because vaccines mimic a wild viral infection, if the latter is causal the former can plausibly be, as well. This also means that the MMR's inclusion of actual measles viral particles is not especially persuasive proof of causation by itself. Nor is it compelling that other vaccines have been deemed causal of distinguishable demyelinating diseases, like GBS, or other CNS-oriented demyelinating diseases more acute in nature, as reviewed in Karussis (which did not even focus

[45] I also have yet to see, or preside over, a case in which a claimant successfully explained, through expert testimony or literature, how a purportedly vaccine-caused acute demyelinating event could subsequently evolve into a chronic condition (although it is wholly conceivable that this could be done).

on MS). The mere fact that MS is believed to be autoimmune in nature is only a starting point for a causal theory based on a vaccine instigating the illness.

Petitioner needed instead to establish something specific about the MMR vaccine that could be causal of MS. His primary argument was that the vaccine's antigenic components could (via the mechanism of molecular mimicry) result in an autoimmune cross-reaction against self tissues, with Dr. Rumbaugh specifically identifying vimentin as a potential antigenic target. But this causal theory lacked reliable support for many of its sub-elements. And that support was needed. It is the case that molecular mimicry is a reliable theory for how autoimmune cross-reactions might *sometimes* occur—but it is hardly the case that they *always* occur, and therefore the concept's core scientific reliability (which I accept) is not enough for it to render a causation theory preponderantly established simply by invoking it, as I have noted on many prior occasions. *See, e.g.*, *McKown v. Sec'y of Health & Hum. Servs.,* No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (citing *Devonshire v. Sec'y of Health & Hum. Servs.,* No. 99-031V, 2006 WL 2970418, at *15 (Fed. Cl. Spec. Mstr. Sept. 2006) ("[b]ut merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the mechanism to the injury and/or vaccine in question") (emphasis in original), *mot. for review den'd,* 76 Fed. Cl. 452 (2007)).

First, Dr. Rumbaugh did not preponderantly demonstrate what element of the MMR vaccine was likely to serve as a potentially mimicking antigen, causing the production of cross-reacting antibodies. At best, he referenced items like Srinivasappa, which stands as somewhat-stale support for the potentiality of a cross-reaction between antibodies produced (in the environment of a lab) against the measles virus and host tissue—*not* that the cross-reaction was reliably demonstrated to be pathogenic, and without anything additional to reliably suggest that the same process was likely *in vivo*. Tr. at 111, 137; Srinivasappa at 397, 400. And unlike in other cases (where experts attempt to show a specific amino acid peptide sequence or structure in common between the vaccine antigens and a putative self-tissue structure serving as the antigenic target for the cross-reactive attack), Dr. Rumbaugh did not similarly strive. Of course, even when experts *do* attempt to show homology, this is insufficient to stand as preponderant evidence of causality by itself—again because cross-reactivity is not congruent with pathogenicity. *Schultz v. Sec'y of Health & Hum. Servs.,* No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020).

Second, Dr. Rumbaugh's effort to establish a possible target for the proposed autoimmune antibody-driven attack—vimentin—was equally bulwarked with thin support. He certainly did not demonstrate that an intracellular structural protein like vimentin would necessarily be accessible to antibodies in the first place. But even if that hurdle is ignored, his support for the contention that this could be the situs for an MS-inducing cross-reaction was based on essentially two items of literature, both (like Srinivasappa) fairly old and not updated or reevaluated: 1983 Fujinami and

2006 Fujinami. The findings of the two are only tenuously linked, moreover, with 1983 Fujinami observing primarily only the *possibility* of a cross-reaction between antibodies produced in response to an outdated measles virus strain and a vimentin-like protein, while 2006 Fujinami suggests that MS's damage begins with harm to oligodendrocytes in the brain. The idea that vimentin *in* brain cells specific to CNS myelination can be reached by measles-reactive antibodies is thus no more than a speculative theory at this point—despite ample opportunities of medical science to explore it.

Petitioner also invoked a number of studies for indirect support of an MMR vaccine-MS link, but they only provided limited assistance to his theory. Langer-Gould, for example, observed *some* temporal association between vaccination and MS in at least a more immediate timeframe, although its showing loses force when applied to the MMR vaccine—and its own authors largely disclaimed the conclusion that Dr. Rumbaugh felt was compelled by the article. In fact, Langer-Gould's authors felt it *more* likely that vaccination might stoke an existing, if subclinical, autoimmune process—a theory consistent with 2006 Fujinami's "fertile field" concept, but not with Petitioner's theory that the MMR vaccine was directly causal, and in the absence of existing but undetected MS.[46] 2006 Fujinami at 84–85. And it observed little to no risk outside of a 30-day window (shorter than the five to six-week timeframe for onset in this case).[47] The other case report evidence (long understood in the Program to merit limited probative weight as a general matter) was also mostly unhelpful. *See, e.g.*, *Campbell v. Sec'y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) ("[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value"). Agmon-Levin did not at all involve MS.

In addition, Petitioner's effort to establish causation must be viewed in the context of the relevant injury. As discussed above, MS is chronic and persistent in nature, waxing and waning over time for reasons not fully understood. It is simply more difficult to preponderantly show how a single vaccination event can put into motion such a long-lasting disease process, as opposed to causing an acute, monophasic injury that may have secondary sequelae but *itself* eventually ceases. The fact that MS features demyelination, like more acute autoimmune disease counterparts, does not eliminate this foundational issue.

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[46] Petitioner has not alleged a claim of significant aggravation of preexisting MS. To the contrary—she alleges her MS *began* in late March 2015, and not prior to the February vaccination. I could not otherwise find on this record that it is likely her MS was subclinical at the time she received the vaccine (despite the evidence of seemingly-neurologic symptoms in 2010, believed at the time to be carpal tunnel syndrome), and the record also does not suggest that after she received it, she began to experience some kind of immune-oriented response. On the contrary, she alleges no symptoms at all a few weeks after vaccination, at the time of her arrival in Kuwait. Tr. at 7, 16.

[47] I do not include a detailed analysis of Petitioner's success in demonstrating the third *Althen* prong—for the simple reason that all three prongs must be established, and my findings as to the first two therefore prohibit recovery for Petitioner regardless of her success on the timeframe prong. *Althen*, 418 F.3d at 1278. The medical acceptability of a five or six-week post-vaccination MS onset is *first* dependent on the conclusion that the vaccine can cause MS—a conclusion I do not reach.

I reach my determination despite the fact that Petitioner made several persuasive points rebutting some of Respondent's contentions. In particular, Petitioner effectively demonstrated that several of the epidemiologic articles or studies offered in this case had methodologic deficiencies (as the IOM Report itself noted) that prevented me from giving such evidence significant weight. Petitioners often (erroneously) argue that epidemiologic evidence should be given no weight at all because it is never a "required" class of evidence that must be marshaled in favor of causation. In fact, it can be very probative of causation when it exists, *and* if it is reliable. *King v. Sec'y of Health & Hum. Servs.*, No. 03-584V, 2010 WL 892296, at *74 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) ("special masters have routinely found that epidemiologic evidence, and/or other medical journal articles, while not *dispositive,* should be *considered* in evaluating scientific theories") (emphasis in original). But if the latter is not demonstrated, epidemiologic evidence can reasonably be drained of its probative value. And here, Petitioner effectively and persuasively established that the IOM Report not only *itself* equivocated as to its findings on an MMR vaccine-MS association, but called into question *most* of the articles relied upon by the Mailand review article, as well—killing two birds with one stone.

Of course, simply because some items of literature relied upon by Respondent did not move the needle *against* causation does not also mean that Petitioner's showing *in favor of* causation was any better. And in fact, Respondent did offer one item of epidemiologic evidence, Hapfelmeier, that was not only very recent but was *not* subject to the same reliability attacks that Petitioner convincingly launched against the other articles previously mentioned. Accordingly, while this is not a case where epidemiologic evidence robustly rebuts causation, the limited items of reliable proof offered on the topic were not overall *supportive* of Petitioner's claim.

This is also not a case in which one expert's demonstrated command of the disputed medical or scientific issues predominated over the other. Both experts possessed neurologic expertise (although it was largely not needed in this case, since Petitioner's diagnosis was not disputed), coupled with just enough immunologic knowledge to testify intelligently on the topic, but without being able to establish enough specialized, real-world grasp of the subject to make their proposed opinion more credible. The case instead turned mostly on the more fundamental fact that Petitioner, via her expert, simply could not carry her burden of establishing the MMR vaccine can likely cause MS—a determination that I reached based on consideration mostly of Petitioner's expert reports along with the literature offered in support. Dr. Rumbaugh otherwise did not have the demonstrated expertise in immunologic issues to give his opinion weight that the literature he filed lacked.

B. *Althen Prong Two*

The record also does not preponderantly establish that the MMR vaccine likely caused Petitioner's MS. First, no treaters have ever proposed her MS was vaccine-caused. Second, the record is devoid of any evidence that Petitioner experienced any kind of reaction or immune-oriented symptoms that might link the vaccine to injury; instead, the record is silent as to Petitioner's condition for almost the entire month of March 2015. There is also no proof that Petitioner possessed even some of the putative cross-reacting antibodies. All the above is amplified by the absence of evidence of any prior reaction by Petitioner to her earlier MMR dose (negating the possibility of a demonstrated "rechallenge"[48] to the second dose—despite Dr. Rumbaugh's suggestions that the dosage schedule increased the chance of an aberrant immune reaction). The record does not provide more than a temporal association between Petitioner's onset and prior vaccination.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such as showing. Petitioner is therefore not entitled to compensation.

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

IT IS SO ORDERED.

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

[48] *See generally Nussman v. Sec'y of Health & Hum. Servs.*, No. 99-500V, 2008 WL 449656, at *9 (Fed. Cl. Spec. Mstr. Jan. 31, 2008), *mot. for review den'd,* 83 Fed. Cl. 111 (2008) (defining challenge-rechallenge as "when a person (1) is exposed to one antigen, (2) reacts to that antigen in a particular way, (3) is given the same antigen again, and (4) reacts to that antigen similarly").