In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 22-1354V

VANESSA GARRIS,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: June 20, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Jessica A. Olins, Maglio Christopher & Toale, Seattle, WA, for Petitioner.

Ryan P. Miller, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On April 28, 2022, Vanessa Garris filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner initially alleged that she suffered an unspecified "demyelinating neurological condition of the central nervous system" as a result of receiving the hepatitis B vaccine on February 10, 2020. Petition (ECF No. 1) at 3. She has since acknowledged that her proper diagnosis is in fact multiple sclerosis ("MS")—and thus seeks to prove MS as her vaccine-caused injury. *See* Petitioner's Memorandum in Support of Entitlement, dated November 22, 2024 (ECF No. 42) ("Second Memo") at 9.

Earlier in the case's life, I expressed reasoned doubt that MS could be caused by *any* covered vaccine. *See* Scheduling Order, dated October 12, 2023 (ECF No. 25). But because I had

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

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

not previously evaluated the possibility of a specific association between MS and the hepatitis B vaccine, I allowed Petitioner the opportunity to substantiate her claim with expert input. *Id.* at 2.

Both parties have now filed expert reports, and have also briefed their positions. Petitioner's Memorandum in Support of Entitlement, dated May 24, 2024 (ECF No. 37) ("First Memo."); Second Memo; Respondent's Brief, dated January 3, 2025 (ECF No. 45) ("Opp."). For the reasons discussed below, I hereby deny entitlement. Petitioner has not established preponderantly that the hepatitis B vaccine can cause MS. In addition, the record supports the conclusion that Petitioner's MS most likely *predated* vaccination—but Petitioner has not established that receipt of the vaccine could significantly aggravate MS, or did so to her.

I. Factual Background

Petitioner was 29 years old when she received the vaccination at issue. ECF No. 1 at 1. She was previously employed as a police officer, although at the time of vaccination was working as a firefighter. Ex. 2 at 8, 11–12. Petitioner has a medical history including some back discomfort and neck pain issues, as well as paresthesias, muscle spasms, and hypomobility. Ex. 9 at 7–10.

Vaccination and Subsequent Neurologic Symptoms

Petitioner was administered a hepatitis B vaccine on February 10, 2020. Ex. 1 at 1. There is no record evidence of any reported immediate reaction. Indeed, almost a month later (on March 7, 2020) Petitioner had a visit with Dr. Patricia Solomos at Sound Family Medicine in Puyallup, Washington, for treatment of hypothyroidism, but at that time voiced no other complaints, and her physical exam yielded normal results. Ex. 5 at 25–29.

Not long thereafter, however, Ms. Garris began to experience more alarming concerns. She visited an emergency room on March 8, 2020, complaining of dizziness and numbness in her right leg. Ex. 6 at 37. She now stated (although she had not reported any such symptoms at her visit the day before with Dr. Solomos) that for the prior three to four days she had been experiencing right, lower extremity numbness that had not gone away, plus a headache with right side tongue numbness and lightheadedness earlier that day, which mostly resolved after she took Motrin. *Id.* Petitioner also reported a feeling that she was "not in control," and that her handwriting did not look the same, and her spouse also noted that Petitioner had experienced a comparable episode the week before, including slurred speech which did not appear to have resolved. *Id.* at 37–38.

Petitioner was subsequently transferred for additional work-up to Good Samaritan Hospital in Puyallup, and while there she saw Mitchel Brown, D.O. Ex. 6 at 31; Ex. 7 at 221. The history section of these records indicates that she reported dizziness while driving the week before, followed by right leg numbness consistent with what was memorialized from her emergency department visit. Ex. 6 at 31. She also noted that she was under stress at the time. *Id.* at 32. Her

physical exam yielded normal results. *Id.* at 37–38. But a brain MRI (that had been performed the night before, during Petitioner's emergency visit) revealed "four foci of T2 hyperintensity in the right and left periventricular white matter with another focus in the left pons (pontine focus is largest). In the right parietal corona radiata, one of the abnormal signal foci demonstrate associated T1 hypointensity," leading the results to be deemed "concerning," if not proof of "active demyelination." Ex. 7 at 221. And a CT angiography of the head and neck revealed no other possible concerns (e.g., stenosis or vasculitis). Ex. 6 at 40.

Despite the serious implications of the MRI findings (especially given Petitioner's reported symptoms), Petitioner sought discharge from the hospital because of work concerns. Ex. 5 at 18; Ex. 7 at 221. Dr. Brown allowed this to occur, since at this point Petitioner's symptoms seemed largely sensory (although he noted that certain confirmatory testing, such as a lumbar puncture, remained to be performed). *Id.* But she was advised to seek emergency treatment again if necessary. Ex. 7 at 221.

That very afternoon, however (March 9th), Petitioner returned to the hospital after reporting a worsening of tingling in her legs and face, plus slurred speech and balance issues. Ex. 7 at 227. She now reported a one-week history of constant, but moderate, right foot numbness that was becoming more intense, plus headache, left facial numbness, numbness to her mouth, difficulty walking and writing, and slurred speech. *Id.* at 229, 242. But exam noted only a slight decrease in sensation in her right foot, and a thoracic MRI resulted in unremarkable findings. *Id.* at 223, 241. Petitioner was readmitted. *Id.* at 234; *see also* Ex. 5 at 67.

During her admission, Petitioner was seen by a neurologist, Seth Stankus, D.O. Ex. 5 at 76, 81. He observed that Petitioner had displayed upper respiratory symptoms, including congestion and a dry cough, with the onset of her neurologic symptoms. Ex. 7 at 246. His exam noted decreased sensation to light touch of the face and mild motor weakness in her upper and lower right-side extremities. *Id.* at 247. Dr. Stankus diagnosed Petitioner with acute disseminated encephalomyelitis ("ADEM"),[3] adding that because she reported developing symptoms four-to-five days after receipt of the hepatitis B vaccine, her ADEM was "likely secondary" to that vaccination. Ex. 5 at 76, 81; Ex. 7 at 246. (There is, however, an absence of record evidence to support an onset of symptoms on February 14–15, 2018. In fact, in the same record Petitioner reports neurologic symptoms onset "10 days ago," or no sooner than March 1st/February 28th. Thus, in either case the immediate post-vaccination symptoms that Dr. Stankus appeared to rely upon for his vaccine association opinion was inaccurate. *See* Ex. 5 at 76–77). As treatment, Dr. Stankus recommended five days of intravenous methylprednisolone, followed by a thirteen-day tapered dose of oral prednisone. *Id.* at 76.

[3] ADEM is "an acute or subacute encephalomyelitis to myelitis characterized by perivascular lymphocyte and mononuclear cell infiltration and demyelination. . . . It is believed to be a manifestation of an autoimmune attack on the myelin of the central nervous system." *Acute Disseminated Encephalomyelitis*, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=73033 (last visited June 16, 2025).

From March 9–12, 2020, Petitioner was an in-patient at St. Joseph Medical Center. *See* Ex. 7 at 241. During this timeframe, she underwent a lumbar puncture, with some results deemed inconclusive for purposes of confirming MS. *Id.* at 242, 294–95. However, her cerebrospinal fluid ("CSF") immunoglobulin G ("IgG") index was slightly elevated, and thus was consistent with inflammation/autoimmune disease (and other biomarkers for inflammation were positive). *Id.* at 294. More significantly, Petitioner displayed on CSF testing three oligoclonal bands—a recognized hallmark for MS (although it was noted that evidence of four bands are usually required to fully substantiate the diagnosis).[4] *Id.* Ms. Garris was discharged on March 12, 2020, after her symptoms were deemed to have greatly improved. *Id.* at 242.

Subsequent Treatment and Confirmation of MS Diagnosis

It was some time thereafter before Petitioner again sought specialist evaluation of her neurologic-like symptoms. In mid-October 2020 (now more than six months after her March hospitalization), Petitioner returned to Dr. Stankus reporting a recent incident of gait ataxia. Ex. 2 at 11–12. In particular, the week before she had woken up and encountered difficulty walking, plus right arm and leg weakness—and although symptoms subsided later that day, they had returned two days later (along with additional clumsiness and difficulty in writing). *Id.* at 12. She had taken herself to the emergency room, and an MRI performed at this time revealed "increased periventricular and callosal white matter lesions consistent with demyelination." *Id.* And at the time of this visit with Dr. Stankus, Petitioner was experiencing leg clumsiness. *Id.*

On exam, Petitioner revealed mild right-sided weakness with intact sensation and coordination, plus somewhat brisk right-side reflexes. Ex. 2 at 14. But Dr. Stankus (based on Petitioner's course to date—and particularly taking note of the fact that Petitioner had now experienced two neurologic episodes) deemed her to meet the "diagnostic criteria for multiple sclerosis, relapsing and remitting." *Id.* at 11. Dr. Stankus recommended a repeat MRI, proposed specific disease-modifying therapies in order to manage her course, and asked that Petitioner initiate routine follow-up visits, beginning three months later. *Id.*

Thereafter, Petitioner's MS diagnosis has been consistently maintained. In February 2021, for example, Petitioner saw Dr. Stankus, reporting another neurologic incident involving hand clumsiness (although she otherwise reported feeling better). *Id.* at 15–16. A non-contrasted head CT showed non-specific white matter changes consistent with a demyelinating diagnosis. *Id.* at

[4] Oligoclonal bands are immunoglobulin proteins "with decreased electrophoretic mobility." *Oligoclonal Bands*, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=60106 (last visited June 16, 2025). They are present in the CSF in 95 percent of MS patients, and are "considered to be the immunological hallmark of disease," since they reflect the presence of CNS inflammation. *Samuels v. Sec'y Health & Human Servs*, No. 17-071V, 2020 WL 2954953, at *3 (Fed. Cl. Spec. Mstr. May 1, 2020).

16. Her MS diagnosis was confirmed, and Dr. Stankus discussed with Petitioner drugs that had proven effective in treatment of relapsing and remitting MS. *Id.* at 15–17. Petitioner had a subsequent telemedicine visit with Dr. Stankus in October 2021, and it was noted that she had experienced two additional neurologic episodes that summer, causing her to seek emergency care. *Id.* at 18. But imaging revealed no additional lesions on her brain or spine. *Id.*

For four days in November 2021, Petitioner was readmitted to the hospital after experiencing exacerbation of her MS symptoms. Ex. 7 at 12. She specifically noted they occurred after receipt of a COVID-19 vaccine as well as an upper respiratory infection ("URI"). *Id.* A brain MRI performed not long after revealed increased demyelination and two new lesions in the right parietal and left temporal lobes. *Id.* at 37.

In early 2022, Petitioner saw a plateauing of symptoms and tolerance of a medication to control her symptoms. Ex. 2 at 20. And another brain MRI (performed in March 2022) revealed existing lesions decreasing in size and no new lesions, as well as no other concerns (such as an acute intracranial hemorrhage or infarct). *Id.* at 32–33. Comparably favorable MRI findings were obtained in November 2022. Ex. 14 at 15. Since that time, appears to have continued to realize some success in treatment of her MS.

II. Expert Opinions

A. *Petitioner's Expert – Dr. Darin Okuda*

Dr. Okuda is a neurologist, and he prepared two written reports for Petitioner. Report, dated May 21, 2024, filed as Ex. 17 (ECF No. 36-3) ("First Okuda Rep."); Report, dated October 17, 2024, filed as Ex. 42 (ECF No. 40-2) ("Second Okuda Rep.").

Dr. Okuda is a professor within the Department of Neurology at The University of Texas Southwestern Medical Center. CV, dated May 24, 2025, filed as Ex. 16 (ECF No. 36-2) ("Okuda CV"). He obtained his M.D. from the University of Hawai'i before pursuing an internship in internal medicine at St. Joseph's Hospital Medical Center and completing his residency in neurology at the Barrow Neurological Institute in Phoenix, Arizona. Okuda CV at 1. Dr. Okuda is a board-certified Diplomate by the American Board of Psychiatry and Neurology and is licensed to practice medicine in California and Texas. *Id.* at 2. In his current role as a clinician-scientist, Dr. Okuda specializes in diagnosing and treating MS and other demyelinating diseases of the central nervous system (the "CNS"). First Okuda Rep. at 1. He has also lectured and authored numerous articles on various topics regarding MS and neuroimmunology. Okuda CV at 23–33, 37–45.

First Report

Dr. Okuda began his first report with a short overview of the medical records. First Okuda Rep. at 2–3. He agreed that Petitioner had properly been diagnosed with MS, based on the medical record. *Id.* at 4. He noted that MRI findings from early March 2020 revealed "the presence of at least one periventricular lesion and one infratentorial lesion"—thereby meeting the diagnostic requirement of "dissemination in space" (i.e., that lesions characteristic of multiple sclerosis are seen via imaging in at least two areas of the CNS) (including different regions of the brain)). *Id.;* A. Thompson et al., *Diagnosis of Multiple Sclerosis: 2017 Revisions of the McDonald Criteria,* 17 Lancet Neurol. 162 (2018), filed as Ex. 26 (ECF No. 36-12), at 167, 68. He also opined that Petitioner's first clinical symptoms were consistent with the pontine lesion observed around the time. And although these lesions seemed to be non-enhancing (meaning not likely recent), Dr. Okuda proposed that this could be explained by other factors, and he ultimately deemed the pontine lesion to have been newer than the others observed. First Okuda Rep. at 4.

Dr. Okuda nevertheless acknowledged the likelihood that some of Petitioner's lesions predated her clinical symptoms. First Okuda Rep. at 4. Medical science understands a concept often called "radiologically isolated syndrome," where healthy individuals are inadvertently discovered (often by imaging performed for some other healthcare reason) to possess spine or brain lesions—in contrast to individuals who present with clinical symptoms and then on MRI are revealed to possess acute/active lesions if not older ones. *Id.* This was likely what had occurred for Petitioner.

Next, Dr. Okuda attempted to sketch a theory for how Petitioner's MS might have developed due to receipt of a vaccine. *See generally* First Okuda Rep. at 5–6. All vaccines, he maintained, "induce an immune response" that could become aberrant. *Id.* at 5. Although generally the hepatitis B vaccine "is safe and it should be used," additional evidence connecting the hepatitis B vaccine to MS exists. *Id.* He noted, for example, that "available data" in 1998 led France to temporarily halt a program aimed at encouraging receipt of this vaccine. M. Hernan et al., *Recombinant Hepatitis B Vaccine and the Risk of Multiple Sclerosis—A Prospective Study,* 63 Neurol. 838 (2004), filed as Ex. 33 (ECF No. 36-19) ("Hernan"), at 838 (summarizing history of cancelled hepatitis B vaccination push in France).

Dr. Okuda admitted that subsequent studies suggested "no evidence that the hepatitis B vaccine increases risk for MS exacerbations." O. Rutschmann et al., *Immunization and MS—A Summary of Published Evidence and Recommendations,* 59 Neurol. 1837 (2002), filed as Ex. 18 (ECF No. 36-4) ("Rutschmann"), at 1842 (referencing C. Confavreux et al., *Vaccinations and the Risk of Relapse in Multiple Sclerosis*, 344 (5) N. Eng. J. Med. 319 (2001), filed as Ex. B Tab. 3 (ECF No. 39-18) ("Confavreux")). But Hernan, Dr. Okuda stressed, was published a few years after Confavreux—and Hernan's findings underscored the existence of scientific validity in the

concerns about the hepatitis B vaccine. Hernan's authors expressly set out to design a more methodologically-reliable, "nested case-control approach" study (based on their view that prior studies casting doubt on an association with MS and the vaccine had been less trustworthy). Hernan at 838. To that end, they identified (for a seven-year period) UK vaccine recipients who also had received a verified MS diagnosis within the same timeframe, ultimately narrowing the pool of subjects to 163 individuals (compared to 1,604 control cases). *Id.* at 838–39. 6.7% of the MS patient pool had received a hepatitis B vaccine within three years of symptoms onset, compared to 2.4% of controls—resulting in a statistically-significant odds ratio of 3.1. *Id.* at 839. No risk, by contrast, was found with respect to influenza or tetanus-containing vaccines. *Id.*

However, Hernan's authors noted several limitations to their findings. The risk of post-vaccination MS, for example, was greater for those vaccinated two to three years before onset, as opposed to within a year (which is Petitioner's alleged experience). *Id.* at 840 tbl. 3. And Hernan did not disclose the exact onset date for the relevant subjects, using broader temporal categories of years. *Id.* In fact, Hernan only specifically identified *eleven* vaccinated individuals out of the 163 MS subjects (and expressly stated that "it is important to stress that 93% of the MS cases in our study had not been vaccinated"). *Id.* And Hernan acknowledged that the study did not allow for the conclusion that the vaccine "causes new cases of MS in susceptible individuals." *Id.*

In the wake of Hernan, Dr. Okuda maintained, "the debate regarding the association between hepatitis B vaccine exposure and MS was reopened." First Okuda Rep. at 5. But as further evidence for an association, he referenced no follow-up studies generated in the subsequent twenty-odd years. Petitioner has, however, filed with this case two articles published after Hernan. D. Geier & M. Geier, *A Case-Control Study of Serious Autoimmune Adverse Events Following Hepatitis B Immunization*,38(4) Autoimm. 295 (2005), filed as Ex. 38 (ECF No. 36-24) ("Geier"); D. LeHouezec, *Evolution of Multiple Sclerosis in France Since the Beginning of Hepatitis B Vaccination,* 60 Immun. Res. 219 (2014), filed as Ex. 39 (ECF No. 36-25) ("LeHouezec"); *see also* First Memo. at 29 nn. 21–22.

Although ostensibly these two articles provide the missing, later-in-time corroboration of Hernan, they both merit limited weight. Of the two, Geier is especially problematic. To begin with, its authors have been *thoroughly discredited in Vaccine Program cases* when offering testimony as experts, suggesting that their work merits little to no attention. *See, e.g.*, *Stricker v. Sec'y of Health & Hum. Servs*., No. 18-56V, 2024 WL 263189, at *13 (Fed. Cl. Spec. Mstr. Jan. 2, 2024), *mot. for review den'd,* 170 Fed. Cl. 701 (2024); *Piscopo v. Sec'y of Health & Hum. Servs.*, 66 Fed. Cl. 49, 55 (2005) (no error to reject expert opinion offered from Dr. Mark Geier as to hepatitis B vaccine's capacity to cause an immune-mediated injury, where Dr. Geier was shown to lack the necessary scientific credentials to render such an opinion reliably). Moreover, the Geier article itself is facially unreliable, as it relies on passively-reported instances of a variety of claimed

adverse events (including MS), without confirmation the adverse event actually occurred. Geier at 295–96.

LeHouezec attempts to retrospectively evaluate the concerns in France (reflected in articles filed in this case like Hernan) about a causal association between the hepatitis B vaccine and MS. LeHouezec at 220 (referencing Hernan). Its author looked at the number of individuals receiving the hepatitis B vaccine in France over a defined timeframe, superimposing this data over the incidence of MS derived from a database of passive surveillance adverse events reported (meaning where individuals *claim* a vaccination or drug-related adverse event). *Id.* at 221 fig. 2. (LeHouezec maintained that this data was still reliable, contending that the "real frequency" of adverse events is significantly underreported (*Id.* at 220)—although this does not cure the obvious self-selection bias problem inherent to reliance on passive surveillance data). LeHouezec concludes that there is a statistically-significant relationship between vaccination within one or two years of onset of MS, but admits that it is only a "strong signal" worthy of further study. *Id.* at 222. (LeHouezec also relies in part on the Geier study mentioned above. *See id.* at 222 n.11).

Dr. Okuda did offer case reports or case studies (some of which were published before Hernan) that he maintained showed that "MS and other demyelinating variants" could occur after receipt of this vaccine. *See, e.g.*, A. Tourbah et al., *Encephalitis After Hepatitis B Vaccination,* 53(2) Neurol. 396 (1999), filed as Ex. 19 (ECF No. 36-5) ("Tourbah"), at 401 (observing eight cases of some form of CNS inflammatory disease, beginning within ten weeks of receipt of the hepatitis B vaccine; seven experienced recurring symptoms, and two had a family history of MS); J.A. Cabrera-Gomez et al., *A Severe Episode in a Patient with Recurrent Disseminated Acute Encephalitis Due to Vaccination Against Hepatitis B—For or Against Vaccination?* 34 (4) Rev. Neurol. 358 (2002), filed as Ex. 20 (ECF No. 36-6) ("Cabrera-Gomez") (40 year-old with prior ADEM history (but who had not been found to meet diagnostic criteria for MS) developed new neurologic symptoms within six weeks of receipt of hepatitis B vaccine).[5]

Dr. Okuda also proposed a biologic mechanism for how the hepatitis B vaccine could lead to MS, relying on the standard kinds of arguments about molecular mimicry advanced in the vast majority of Program cases involving autoimmune-mediated illnesses. *See generally* First Okuda Rep. at 5–6. In particular, he proposed that a surface antigen for the hepatitis B virus has "strong homologies" with myelin basic protein ["MBP"] (one component of the myelin sheath that covers nerve axons and aids in transmission of nerve signals) and myelin oligodendrocyte glycoprotein ["MOG"]. *Id.* at 5; D. Bogdanos et al., *A Study of Molecular Mimicry and Immunological Cross-Reactivity Between Hepatitis B Surface Antigen and Myelin Mimics,* 12(3) Clin. & Dev. Immunol. 217 (2005), filed as Ex. 21 (ECF No. 36-7) ("Bogdanos"), at 222–23. Introduction of this viral antigen contained in the vaccine, he contended, could result in the immune system mistaking the

[5] The version of Gomez-Cabrera filed in this case is in Spanish, with only the introductory abstract in English, making it impossible to confirm its contents.

similar self components for the foreign pathogen, attacking the self tissues and structures indiscriminately. First Okuda Rep. at 5–6. Dr. Okuda admitted that "Bogdanos did not establish a definitive link to [MS] pathology" when observing the mimicking similarity between hepatitis B components and nerve-associated tissues, but deemed the results sufficient to at least suggest further exploration of the concept was merited. *Id.* at 6.

Dr. Okuda later contended that Bogdanos had observed better-supported cross-reactivity between the actual components of the hepatitis B vaccine and antibodies believed to be the drivers of a *distinguishable* demyelinating disease—referred to as myelin oligodendrocyte glycoprotein antibody-associated disease, or "MOGAD."[6] First Okuda Rep. at 6; Bogdanos at 220–21; *see also* B. Banwell et al., *Diagnosis of Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: International MOGAD Panel Proposed Criteria,* 22 Lancet Neurol. 268 (2023), filed as Ex. 35 (ECF No. 36-21) ("Banwell"), at 272 tbl. 1 (differentiating MOGAD features from MS). Dr. Okuda nevertheless maintained that the kinds of illnesses likely driven by MOG antibodies are "closely related" to MS—although the literature filed on this topic readily acknowledges distinctions between the two. *See, e.g.*, Banwell at 273 (discussing findings of MOG antibodies in individuals possessing "clinically silent" brain lesions (usually characteristic of MS), but also noting that the "hallmark" of such pre-clinical lesions in MS patients "might not be equally applicable to patients with a first demyelinating attack associated with [MOG antibodies]").

Nevertheless, Dr. Okuda opined that the fact that MOGAD involves "neuroinflammation in the human brain provides a strong scientific basis for the development of MS." First Okuda Rep. at 6. Indeed, Dr. Okuda opined that the kind of self-attack illustrated by research involving MOG (leading to "damaged 'transected wires' that do not regenerate") was likely contributory to the progressive form of MS (although Petitioner has not been so diagnosed, and the association of that form of MS with MOG-driven diseases is even less well-established). *Id.*; *but see* Banwell at 278 ("[t]he progressive neurological deterioration that drives worsening disability independent of relapses in patients with multiple sclerosis *does not appear to be operative in MOGAD*. Patients with MOGAD do not seem to harbour the smouldering lesions that are associated with progressive neurological decline in multiple sclerosis" (emphasis added)).

Dr. Okuda concluded his first report by addressing the other two prongs of the causation test set by the Federal Circuit's decision in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005). With respect to the second prong (which obligates claimants to prove a vaccine "did cause" their actual injury), Dr. Okuda maintained that the "gradual onset of

[6] "Myelin oligodendrocyte glycoprotein (MOG)-associated disease (MOGAD) is a rare, antibody-mediated inflammatory demyelinating disorder of the central nervous system (CNS) with various phenotypes starting from optic neuritis, via [TM] to acute demyelinating encephalomyelitis (ADEM) and cortical encephalitis." Wojciech Ambrosius et al., *Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: Current Insights into the Disease Pathophysiology, Diagnosis and Management*, 22 Int'l J. Molecular Scis. 100 (2021), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7795410/.

symptoms consistent with MS" experienced by Petitioner was consistent with the causal theory he proposed. First Okuda Rep. at 6. He also referenced the MRI results, which revealed "clear evidence of prior central nervous system involvement before the development of [Petitioner's] large pontine lesion"—demonstrating that the receipt of the vaccine had aggravated a process already underway. *Id.*

The third *Althen* prong (which requires proof that the timeframe for post-vaccination development of the alleged disease was medically acceptable) was also met, in Dr. Okuda's view. *Id*. Petitioner reported specific, neurologic-like symptoms (including "constant, moderate numbness in her legs and mouth, difficulty walking, writing, and slurred speech") beginning March 1, 2020—approximately three weeks after vaccination. *Id.* Dr. Okuda deemed this "a timeframe that is considered reasonable for the onset of an adaptive autoimmune response triggered by a vaccine." *Id.* He did not, however, cite any independent authority for this contention.

Second Report

Dr. Okuda offered a brief supplemental report reacting to some of Respondents' experts' criticisms (although he focused more on the comments of Respondent's immunology expert, Dr. William Hawes). First, he deemed Dr. Hawes's rejection of Hernan's findings "perplexing." Second Okuda Rep. at 2. Hernan, he argued, employed a particularly-reliable methodology, especially in comparison to case control or larger cohort studies. *Id.* In so maintaining, he stressed the fact that the concept of a vaccine injury presented a rare outcome, making larger studies too "inefficient." *Id.* He also contended that the finding of only three instances of post-vaccination MS in the period of "0–1 years" after vaccination was consistent with Petitioner's experience (although as noted above Hernan provided no more granular data as to *when* within the one year interval a vaccinated subject developed MS).

Second, Dr. Okuda defended his invocation of Bogdanos as demonstrating a hepatitis B vaccine-MS association. Second Okuda Rep. at 2–3. In fact (and contrary to Dr. Hawes's argument), Bogdanos did observe a "meaningful amount" of response among studied vaccinated patients when exposed to MOG of hepatitis B surface antigen antibodies. *Id.* at 3; Bogdanos at 220–21. While the cross-reactivity was not universal or consistent among the studied patients, Bogdanos's authors deemed that attributable to the general rarity of "adverse post-vaccination autoimmune reactions." Bogdanos at 223. Thus, Bogdanos was reliable evidence of not only the possibility of a cross-reaction, but its pathogenic impact. Second Okuda Rep. at 3.

Studies referenced by Respondent's experts that purportedly undercut a vaccine-MS association were in fact unreliable, in Dr. Okuda's estimation. Confavreux, for example, made what Dr. Okuda deemed incorrect statistical assumptions. *Id*. Confavreux's authors had analyzed a pool of 643 patients who had experienced an MS relapse sometime within a four-year period

(1993–97). Confavreux at 319. Applying a version of a case-control methodology, Confavreux noted that 15 percent of the sample had received a vaccine within the preceding twelve months, but only approximately two percent had been vaccinated within two months of relapse—a lower percentage than the control groups. *Id.* at 319, 321–24. Dr. Okuda maintained, however, that Confavreux (a) based its conclusions on too small a sample (likely again due to the rarity of a vaccine adverse event), and (b) involved a cohort with an average older age than Petitioner. Second Okuda Rep. at 3; Confavreux at 323 tbl. 1. Dr. Okuda also noted that a 2012 Institute of Medicine ("IOM") report on vaccine adverse events had allowed that the alleged hepatitis B-MS association lacked adequate evidence to accept or reject (leaving the door open for a possible association). *See Adverse Effects of Vaccines: Evidence and Causality* (Kathleen Stratton et al. eds., 2012), filed as Ex. 49 (ECF No. 43-3) (the "2012 IOM Report"), at 319 (noting methodologic deficiencies in Confavreux), 450.[7]

Dr. Okuda also attempted to distinguish another article offered by Respondent's experts. *See* Y. Mikaeloff et al., *Hepatitis B Vaccination and the Risk of Childhood-Onset Multiple Sclerosis*, 161 (12) Arch. Pediatr. Adolesc. Med. 1176 (2007), filed as Ex. A Tab 6 (ECF No. 39-7) ("Mikaeloff I"). Mikaeloff I looked at a sample of 143 French children experiencing MS onset before the age of 16, identifying a third of this group that had received the hepatitis B vaccine within three years of onset, comparing this subset against a control group. *Id.* at 1177–79. Mikaeloff I's authors found no association between onset of MS and receipt of the vaccine. *Id.* at 1181.

But Dr. Okuda discounted these findings, given that they were derived from a cohort of individuals with personal characteristics distinguishable from Ms. Garris. Second Okuda Rep. at 4. He also noted that Mikaeloff I's authors had produced a follow-up article in 2009 that observed an "increased trend" of post-vaccination MS risk in a subgroup taken from the original studied cohort (although the risk was over a particularly *large* time interval—more than three years post-vaccination). Y. Mikaeloff et al., *Hepatitis B Vaccine and the Risk of CNS Inflammatory Demyelination in Childhood*, 72 Neurol. 873 (2009), filed as Ex. 43 (ECF No. 40-3) ("Mikaeloff II"), at 873, 878–79**.** Dr. Okuda added that the risk of MS seemed somewhat dependent as well on the formulation of hepatitis B vaccine used. *Id.* at 878–79 ("Engerix" form of vaccine more associated with higher risk).

Dr. Okuda concluded his supplemental report attempting to rebut the argument of Respondent's neurology expert, Dr. Michael Sweeney, that no MS "antigenic target" had yet been

[7] Astoundingly, Petitioner's filing of this 2012 IOM report includes *every single page* of the work—all 895—rather than merely the portions specifically relevant to Dr. Okuda's report. *See generally* ECF No. 43-3. But this report discusses numerous vaccines and illnesses having nothing to do at all with this matter. This typifies the overfilling of medical literature that is so rampant in the Vaccine Program, and the concomitant, wholly-unnecessary burden placed on special masters to search through oceans of literature in an effort to find the single relevant portion applicable to a given contention.

identified (thus rendering contentions about MOG antibodies generated via molecular mimicry as inapposite). Second Okuda Rep. at 4. He deemed three self antigens to be "well-known" as relevant to MS: MBP, MOG, and proteolipid protein. *Id.* at 4–5; R. Hohlfeld et al., *The Search for Target Antigens of Multiple Sclerosis, Part 1: Autoreactive CD4+ T Lymphocytes as Pathogenic Effectors and Therapeutic Targets*, 15 Lancet Neurol. 198 (2016), filed as Ex. 44 (ECF No. 40-4) ("Hohlfeld"). Hohlfeld, however, underscores the fact that "the target antigens of multiple sclerosis have been difficult to identify," and mainly attempts to illuminate the inquiry by evaluating the role of T helper cells in MS's pathogenesis. Hohlfeld at 198. At most, the article considers MOG, MBP, and proteolipid protein as target antigens relevant to experimental autoimmune encephalomyelitis ("EAE") animal studies,[8] and help to flesh out the role T helper cells may play in regard to these antigenic targets. *Id.* at 200. Hohlfeld thus does not stand for the proposition that any of the three identified targets are the *subject* of antibody attack leading to MS (let alone reflecting antibodies produced in response to vaccination).[9]

Dr. Okuda stressed that MBP was particularly likely to constitute an antigenic target for patients with a demonstrated genetic susceptibility (although that is not known to be the case for Ms. Garris). Second Okuda Rep. at 4. MOG antibody levels were also observed in higher titers of individuals presenting with "clinical and radiological characteristics typical of multiple sclerosis," thus in Dr. Okuda's view "supporting the pathogenicity" of such antibodies. *Id* (referencing Banwell). Notably, however, Banwell does not support Dr. Okuda' conclusion. Banwell clearly recognized important differences between MOGAD and MS, and was focused on delineating "formal consensus diagnostic criteria for MOGAD *as a distinct entity*." Banwell at 268 (emphasis added). In addition, it found a very low frequency of the presence of the anti-MOG antibodies in patients fitting the diagnostic criteria for MS, adding that because "the absolute number of false-positive results" for testing for this antibody in MS patients would likely be high if routinely performed, it is "not recommended." *Id.* at 275 Panel 2, 277. Accordingly, findings specific to the pathogenic role of this kind of autoantibody were not pertinent in the context of MS.

B. *Respondent's Experts*

1. Dr. Michael Sweeney – Dr. Sweeney is a pediatric neurologist, and he prepared two written reports in opposition to the opinions of Dr. Okuda. Report, dated August 14,

[8] Experimental allergic encephalomyelitis or "EAE" refers to "animal models for acute disseminated encephalomyelitis in which the characteristic pathophysiology and clinical signs of this disease are produced by immunization of an animal with extracts of brain tissue or with myelin basic protein together with Freund adjuvant" studies aimed at understanding the causal connection between neurological damage and viral infections. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 607 (33d ed. 2020).

[9] In fact, Hohlfeld references a "part 2" article by the same authors that looked specifically at the role of B cell responses in MS's pathogenesis (which would include B cell production of antibodies akin to what is alleged to occur herein due to vaccination). Hohlfeld at 198 n.17. But that counterpart article was not filed in this case.

2024, filed as Ex. A (ECF No. 39-1) ("First Sweeney Rep."); Report, dated December 24, 2024, filed as Ex. C (ECF No. 44-1) ("Second Sweeney Rep.").

Dr. Michael Sweeney is a pediatric neurologist with board certification in neurology with special qualifications in child neurology. CV, dated August 19, 2024, filed as Ex. A Tab 13 (ECF No. 39-14) ("Sweeney CV"). Dr. Sweeney obtained his bachelor's degree from Purdue University and his M.D. from the Medical College of Wisconsin. Sweeney CV at 1. After medical school, Dr. Sweeney completed his residency training at Cincinnati Children's Hospital Medical Center and the University of Cincinnati. *Id.* He then completed a fellowship in autoimmune neurology at the University of Utah. *Id.* Dr. Sweeney is currently an associate professor at the University of Louisville's Norton Children's Hospital in the Department of Pediatrics where he engages in clinical and academic work specifically geared towards treating autoimmune or inflammatory conditions that affect the nervous system, such as MS. First Sweeney Rep. at 1; Ex. A at 1. He has authored twelve peer reviewed publications on topics concerning neurology and has collaborated on another three neurology publications. Sweeney CV at 1–2.

First Report

Dr. Sweeney provided his own summary of Petitioner's medical history, and in so doing embraced her diagnosis of relapsing-remitting MS. First Sweeney Rep. at 1–2, 5. But he denied that the hepatitis B vaccine could cause MS, and provided a more expansive discussion of the alleged vaccine association maintained in Dr. Okuda's initial report. *Id.* at 3–4.

Consistent with Dr. Okuda, Dr. Sweeney noted that there had been a time more than 20 years ago when (in reaction to the French pullback of its hepatitis B vaccination campaign) medical science had been concerned enough about a possible link between the hepatitis B vaccine and MS to evaluate it closely. First Sweeney Rep. at 3. Indeed, it had been the publication of case reports cited by Dr. Okuda (such as Tourbah or Cabrera-Gomez) that had resulted partially in the decision in France to curtail its vaccination drive for the hepatitis B vaccine.

But subsequent studies performed not long after had found no link between the hepatitis B vaccine and MS. First Sweeney Rep. at 3. Rutschmann, for example, was a review article that looked at all then-existing studies on the subject, pooling their data into a single "meta-analysis."[10] Rutschmann at 1837–38, 1841. Based on the totality of the amassed study findings, Rutschmann's authors deemed the risk of wild infections to outpace the risk posed by vaccination (and therefore

[10] A "meta-analysis" is a quantitative statistical analysis of several separate but similar studies. It is conducted in order to test the pooled data for statistical significance. *Meta-analysis*, Merriam-Webster Online, https://www.merriam-webster.com/dictionary/meta-analysis (last visited June 16, 2025). While a meta-analysis is not necessarily more persuasive simply because it aggregates the findings of smaller studies, it is considered to have a valid methodology—although the studies it obtains its aggregated data from may be subject to reliability issues, undermining the value of the larger study. *See e.g.*, *Dinh v. Sec'y of Health & Hum. Servs.*, No. 16-171V, 2022 WL 730258, at *11–12 (Fed. Cl. Spec. Mstr. Feb. 14, 2022) (discussing reliability issues raised by meta-analyses).

the latter's ability to prevent the former *favored* vaccination of MS patients). *Id.* at 1840–42. And they noted as well that the risk of exacerbation after receipt of the hepatitis B vaccine was deemed low by Confavreux, while adding that overall the evidence with respect to vaccines other than the flu vaccine was "much spottier." *Id.* at 1842.

In addition, studies and articles issued since the publication of Confavreux only bolstered the conclusion that the hepatitis B vaccine did not pose a risk of MS. *See, e.g.*, Mikaeloff I; F. DeStefano et al. *Vaccinations and Risk of Central Nervous System Demyelinating Diseases in Adults*, 60(4) Arch. Neurol. 504 (2003), filed as Ex. A Tab 7 (ECF No. 39-8) ("DeStefano"), at 507–08 (case-control study of 400 adults who experienced MS after vaccination with hepatitis B and other vaccines revealed no increased MS risk).

Dr. Sweeney acknowledged (consistent with Dr. Okuda's contention) that Hernan had seemed to breathe new life into the possibility of a hepatitis B vaccine-MS association. But Hernan's findings were not long-after evaluated by the World Health Organization's Global Advisory Committee on Vaccine Safety, and that entity concluded that Hernan "did not provide convincing support for the hypothesis that the hepatitis immunization is associated with an increased risk of [MS]." First Sweeney Rep. at 4; *World Health Organization Global Advisory Committee on Vaccine Safety: Response to the paper by M.A. Hernan and Others, Neurology 14th September 2004 issue entitled "Recombinant Hepatitis B Vaccine and the Risk of Multiple Sclerosis"* (Sept. 2004), available online at https://www.who.int/groups/global-advisory-committee-on-vaccine-safety/topics/hepatitis-b-vaccines/multiple-sclerosis (last visited June 16, 2025) filed as Ex. A Tab 5 (ECF No. 39-6) ("2004 WHO Article"). In particular, the 2004 WHO Article noted that Hernan had only identified a heightened post-vaccination risk of MS in 11 patients; had relied upon overly-lengthy post-vaccination timeframes (up to three years); and had derived its sample from a pool of high risk individuals (since the practice at the time was only to vaccine such individuals with hepatitis B vaccine), but without explaining well why the studied subjects were ultimately selected. First Sweeney Rep. at 4; 2004 WHO Article at 3–4.

Dr. Sweeney also questioned Dr. Okuda's proposal that molecular mimicry could provide a mechanistic explanation for how the hepatitis B vaccine might initiate an autoimmune process leading to MS. First Sweeney Rep. at 4–5. Even though Bogdanos had observed some sequence homologic similarity between hepatitis B surface antigen and MBP or MOG, it could not be read as support for the proposition that cross-reactivity *due* to this homology was the cause of an autoimmune attack. *Id*. at 4. In fact, to date (and despite "extensive research") "no antigen has emerged as a target for the immune system in [MS]." *Id.* at 5.

MOGAD, and associated research linking anti-MOG antibodies to hepatitis B, was of low relevance in this context, Dr. Sweeney maintained. *Id.* The process by which such anti-MOG antibodies might trigger CNS demyelination is "not described" for MS, a chronic condition in

which CNS lesions can long predate clinical manifestations. *Id*. Moreover, the MOG antibodies are only associated with a subset of symptomatic CNS presentations (ADEM, transverse myelitis) that are wholly distinguishable from MS in numerous regards. In fact, MS patients are not commonly found to possess anti-MOG antibodies in the first place. *Id.;* A. Cobo-Calvo et al., *Frequency of Myelin Oligodendrocyte Glycoprotein Antibody in Multiple Sclerosis*, 7 Neurol. Neuroimmunol. Neuroinflamm. e649:1 (2020), filed as Ex. A Tab 8 (ECF No. 39-9) ("Cobo-Calvo") at 3 (blood serum of 0.3 percent of sample of 685 MS patients possessed anti-MOG antibodies, leading researchers to recommend against testing of MS patients for this antibody).

In addition to addressing general causation issues, Dr. Sweeney commented on Petitioner's history, and whether it was consistent with the proposed theory for how the hepatitis B vaccine could have caused, or aggravated, her MS. He noted that a brain MRI performed in March 2020 (when Petitioner first complained of post-vaccination symptoms) yielded findings that were "concerning for demyelination," albeit with possible evidence of enhancement. First Sweeney Rep. at 2. Later cerebrospinal fluid testing produced results more confirmatory of MS, such as the presence of oligoclonal bands. *Id.*

Although Petitioner was ultimately diagnosed more formally with relapsing-remitting MS, Dr. Sweeney proposed that she likely experienced her first relapse in March 2020, since she reported being under personal stress around that time, and stress was understood to be able to trigger symptoms in MS. Frist Sweeney Rep. at 5; J. Jiang et al., *The Relationship Between Stress and Disease Onset and Relapse in Multiple Sclerosis*, 67 Mult. Scl. Rel. Disorders 104142:1 (2022), filed as Ex. A Tab 10 (ECF No. 39-11) ("Jiang") at 4 (review article noting "consistent evidence supporting an association between stress and MS relapse"). Dr. Sweeney did not explicitly identify *when* he believed Petitioner's MS onset occurred, although he seemed to accept Dr. Okuda's determination that Petitioner "likely had brain lesions prior to her first clinical episode," and therefore the first noted instance in the medical record of a clinical response was properly deemed a relapse. First Sweeney Rep. at 3.

By contrast, Dr. Sweeney denied that vaccination could cause MS flares, citing several items of literature in support. *See, e.g.*, Confavreux at 325; L. Grimaldi et al., *Vaccines and the Risk of Hospitalization for Multiple Sclerosis Flare-Ups*, 80(10) JAMA Neurol. 1098 (2023), filed as Ex. A. Tab 11 (ECF No. 39-12) ("Grimaldi"), at 1101–2 (French national study of more than 35,000 MS patients who had symptoms flare within 60 days of receipt of several different vaccines (including a combination vaccine containing hepatitis B) but finding no vaccine association). By contrast, Dr. Sweeney maintained, there was a total absence of medical literature connecting the hepatitis B vaccine to MS relapse. Rather, MS relapses occurred at a general rate of one per year, and likely as a result of "the nature history" of the disease course overall. First Sweeney Rep. at 5.

Second Report

Dr. Sweeney prepared a two-page supplemental report in reaction to Dr. Okuda's second report. In it, Dr. Sweeney flatly disagreed with Dr. Okuda's argument that the target antigens for MS were "well-known." Second Sweeney Rep. at 1. Hohlfeld (which Dr. Okuda cited for that proposition) only established a *possible* target antigen (myelin-specific T helper cells) relevant to the artificial and controlled setting of EAE. Hohlfeld at 198. But the article also acknowledged that in the context of "human" MS, such antigens had not been identified. *Id.* at 205–06. The target antigens mentioned by Dr. Okuda were, Dr. Sweeney admitted, properly considered in studies as "potentially involved" in MS's pathophysiology, but "their roles remain unresolved," and they had not been shown to be *likely* susceptible to pathogenic attack leading to MS. Second Sweeney Rep. at 1; Hohlfeld at 199–200 (noting that target antigen-reactive T cells are evident even in healthy subjects). Ultimately, Hohlfeld expressly noted that "our ignorance of the target antigens of [MS]" was an obstacle to development of effective MS therapies specific to such antigens, underscoring the extent to which medical science did not actually yet "know" much about MS's target antigens. Hohlfeld at 201.

Dr. Sweeney further emphasized the limits of scientific understanding regarding the proposed pathogenic impacts of anti-MBP or MOG antibodies in causing MS. Second Sweeney Rep. at 1–2. For example, a cross-reactive process involving MBP (in which foreign antigens would bind to protein mimics in human nerve tissue) had only been demonstrated experimentally, rather than *in vivo*. *Id.* at 1. Meanwhile, other studies intended to evaluate the effectiveness of MS treatments (which functioning by mimicking MBP, cross-reacting with it in a way that inhibits pathologic cross-reactivity) have established that immunosuppressive autoantigens degrade peripherally, and thus are unable reach the CNS in humans. R. Arnon & R. Aharoni, *Mechanism of Action of Glatimer Acetate in Multiple Sclerosis and its Potential for the Development of New Applications,* 101(2) PNAS 14593 (Oct. 5, 2024), filed as Ex. 48 (ECF No. 43-2) ("Arnon & Aharoni") at 14593 (noting that the effectiveness of the MS treatment, as evaluated in EAE studies, cannot be assumed to have the same function *in vivo,* "because [the treatment] is degraded in the periphery and not likely to reach the brain and compete with the relevant myelin antigens *in situ*. It is therefore likely that, in CNS diseases, additional immunomodulatory mechanisms factors that can access the blood-brain barrier mediate the therapeutic activity of [the treatment]"). If a treatment intended to cross-react *beneficially* cannot reach the CNS, there is no reason to believe allegedly-pathogenic antibodies produced in response to a vaccine could do so.

MOG was even less likely to be associated with MS's pathogenesis and course. MOGAD is a clinically-distinct disease entity, as articles filed by Dr. Okuda clearly established. *See, e.g.*, Banwell at 272 tbl. 1. The putative pathogenic role of anti-MOG antibodies in MS is insufficiently established, since testing for theses antibodies is often based on peripheral blood samples, but

without corroborative proof that individuals with "MOGAD-consistent presentations" also possess these antibodies in their cerebrospinal fluid (and hence are not found at the direct situs of CNS attacks relevant to MS). Second Sweeney Rep. at 2. And testing that reveals the presence of MOG antibodies is often deemed a "false-positive" finding of demyelinating diseases for patients whose clinical presentations were more likely attributable to neurological diseases attributable to other causes, such as vitamin deficiency or stroke. E. Sechi et al., *Positive Predictive Value of Myelin Oligodendrocyte Glycoprotein Autoantibody Testing*, 78(6) JAMA Neurol. 741 (2021), filed as Ex. C Tab 1 (ECF No. 44-2) ("Sechi") at 742. Finding the presence of these MOG-specific antibodies thus says nothing about MS. *Id.* at 744 ("Multiple sclerosis was overrepresented among patients with false-positive results, *which should dissuade clinicians from uniform ordering of [MOG antibody] testing in patients with typical MS*") (emphasis added).

2. Dr. William Hawse – An academic immunologist, Dr. Hawes offered two written expert reports on Respondent's behalf. Report, dated August 13, 2024, filed as Ex. B (ECF No. 39-15) ("First Hawes Rep."); Report, dated December 22, 2024, filed as Ex. D (ECF No. 44-3) ("Second Hawes Rep.").

Dr. William Hawse obtained his Ph.D. in biophysical chemistry from Johns Hopkins in 2009. CV, dated August 19, 2024, filed as Ex. B Tab 9 (ECF No. 39-24) ("Hawse CV"). After graduation, he pursued a postdoctoral fellowship in structural immunology at the University of Notre Dame. Hawse CV at 1. Since then, Dr. Hawse has been an assistant professor at the University of Pittsburgh where he established an immunology laboratory. First Hawes Rep. at 1. His current research involves identifying therapeutic strategies for autoimmune diseases, like MS, using preclinical murine autoimmune models. First Hawse Rep. at 1–2. Not only has Dr. Hawse taught students and conducted research, but he has also authored or co-authored over twenty peer reviewed articles on various topics regarding immunology. Hawes CV at 2–5. Dr. Hawse is not, however, a medical doctor.

First Report

Dr. Hawes acknowledged that his expertise as a "cellular and molecular immunologist" meant his opinion was inherently limited to whether there was a demonstrated association between the hepatitis B vaccine and MS, "through molecular mimicry or another immunological process." First Hawes Rep. at 2. He thus did not comment on Petitioner's clinical history (and I disregard any statements he might have made that could be construed as offering an opinion akin to what only a medical doctor and/or neurologist would be competent to propose).

Dr. Hawes opined that the initial concerns (prompted by the case reports cited in Dr. Okuda's report) that had resulted in the cessation of the push in France to obtain universal hepatitis B vaccination in the late 1990s had ultimately been discredited—as recognized by literature

Petitioner filed (and which in one case was co-authored by Dr. Okuda himself). First Hawse Rep. at 3–4; Rutschmann at 1840. Dr. Hawes allowed that Hernan subsequently seemed to substantiate a connection between the vaccine and MS, but he deemed the study to be "rather underpowered," since it relied on findings of only three MS cases post-vaccination in the relevant time period (within one year of vaccination), compared to only 17 controls. First Hawes Rep. at 4; Hernan at 840 tbl. 3. He otherwise maintained that this yearlong timeframe was too broad to be applied to a proposed adaptive immune response causing or exacerbating MS within a few weeks of vaccination. First Hawes Rep. at 4.

By contrast, Dr. Hawes argued, other more reliable medical/scientific studies had debunked the possibility of an association between the hepatitis B vaccine and MS. *See, e.g.*, C. Sestili et al., *HBV Vaccine and Risk of Developing Multiple Sclerosis: A Systematic Review and Meta-Analysis*, 17(7) Hum. Vacc. & Immunother. 2273 (2021), filed as Ex. B Tab 2 (ECF No. 39-17) ("Sestili"). Sestili is another meta-analysis that evaluated the findings and underlying data from seven articles (out of an initial pool of more than 400 relevant items) published between 2001 and 2015, including some of the articles filed in this case, such as Hernan and Mikaeloff I. *Id.* at 2274 tbl. 1. The results were deemed unsupportive of any association between the hepatitis B vaccine and MS. *Id.* at 2277.

Dr. Okuda had also invoked case reports, like Cabrera-Gomez and Tourbah, as circumstantial evidence of a causal relationship. But Dr. Hawes noted that this kind of evidence was not itself particularly robust proof of causation. First Hawes Rep. at 5. Rather, it only served as a "launching point for potential study"—but here, subsequent studies had not corroborated the hypothesis of a causal relationship. *Id.*

Dr. Hawes moved on to the issue of whether support existed for molecular mimicry as the mechanism for how a vaccine-triggered autoimmune cross-reaction against nerve tissues in the CNS might occur. *See generally* First Hawse Rep. at 6–7. He noted that immunologists had proposed some criteria for evaluating when molecular mimicry could reasonably be applied to explain a disease process. *See, e.g.*, C. Benoist & D. Mathis, *Autoimmunity Provoked by Infection: How Good is the Case for T Cell Epitope Mimicry?* 2(9) Nat Immunol. 797 (2001), filed as Ex. B Tab 5 (ECF No. 39-20) ("Benoist & Mathis"). Benoist & Mathis specifically proposed five criteria, including (a) whether there is some association/correlation between an infection (or here, infectious analog) and the inflammatory state, with evidence that it can persist even in the absence of the inciting event; (b) what the triggering and target antigens are; (c) their relevance to the putative autoimmune attack; (d) evidence that the homologic antigens are involved in the autoimmune disease's development; and (e) that immune cells elicited by the homologic similarity can provoke the disease. Benoist & Mathis at 797–98.

But the criteria set forth in Benoist & Mathis could not be met in this matter, Dr. Hawes contended. In particular, there was no reliably-established MS-hepatitis B vaccine association, and no evidence that the specific immune reaction to the vaccine likely causes the production of cross-reactive antibodies capable of driving a pathogenic disease process leading to MS. First Hawes Rep. at 6–7.

In addition, Dr. Hawes addressed the literature offered by Dr. Okuda that purported to support amino acid sequence homology between the hepatitis B vaccine and MS. Bogdanos's authors, he maintained, had expressly admitted that their homology findings suggesting the *possibility* of a molecular mimicry-provoking autoimmune response had not been confirmed scientifically. First Hawse Rep. at 5–6; Bogdanos at 222–23. In fact, Bogdanos could be more persuasively read to *discount* the possibility that antibodies generated in response to the vaccine could cause MS or something also involving demyelination, since the article did not observe that antibodies generated as a result of the homologic similarities *resulted* in MS in the relevant patients. First Hawes Rep. at 6; Bogdanos at 222–23 ("[a]ll of the vaccinees were free of autoimmune phenomena before vaccination and remain free of any adverse reactions during the follow up. *Clues as to the pathogenic link between the vaccine and specific autoimmune disorders, therefore, could not be established*") (emphasis added).[11]

Second Report

Dr. Hawes's final report responded to points made by Dr. Okuda in a supplemental report—primarily focusing on three items of literature. For example, Dr. Okuda had attempted to justify his invocation of Hernan, which he purported showed instances of MS after receipt of the hepatitis B vaccine in a timeframe comparable to what Petitioner experienced. But Dr. Hawes emphasized that Hernan employed very broad timeframe categories—"0–1" years, for example—and therefore it could not be said to have useful applicability to Petitioner's circumstances, since her onset timeframe occurred with a much shorter span (a month post-vaccination). Hawes Second Rep. at 1–2; Hernan at 840 tbl. 3. (Hernan itself does not specify *when* within the one year timeframe the three subjects who experienced MS post-vaccination precisely saw symptoms onset).

[11] Dr. Hawes also briefly addressed statements in Dr. Okuda's report suggesting other means by which the hepatitis B vaccine might trigger an aberrant response—for example, because of its alum adjuvant content (included in vaccines to increase their immunogenicity). First Hawes Rep. at 7. Dr. Hawes characterized this specific theory as "autoimmune/inflammatory syndrome induced by adjuvants" ("ASIA"), and noted that it was "no longer accepted." *Id*. The Program has uniformly rejected ASIA as a scientifically-*unreliable* causation theory. *See, e.g.*, *Rowan v. Sec'y of Health & Hum. Servs*., No. 10-272V, 2014 WL 7465661, at *12 (Fed. Cl Spec. Mstr. Dec. 8, 2014) (rejecting the ASIA theory because it "is not a proven theory" and no "persuasive or reliable evidence" supports it), *aff'd* WL 35 62409 (Fed. Cl. 2015)). But because Dr. Okuda only referenced this concept off-handedly, and does not otherwise seem to embrace it as his primary theory for causation, I will give it no further attention.

Next, Dr. Hawes defended his reading of Bogdanos. Second Hawes Rep. at 2–3. He noted that Bogdanos's authors had expressly acknowledged that despite their findings of molecular mimics in comparing hepatitis B surface antigen and MBP components, they had not been able to show any pathologic response to the cross-reactivity. *Id.;* Bogdanos at 222–23. Thus, Dr. Hawes maintained his contention that there was no "evidence that hepatitis B vaccine can cause pathogenic antibodies to cause MS," or that the antibodies themselves were likely pathogenic, even if they could *in theory* arise in connection with the homologic similarity between foreign antigen and self. Second Hawes Rep. at 3.

Finally, Dr. Hawes pointed out inconsistencies in Dr. Okuda's criticisms of some epidemiologic evidence. Dr. Okuda had, for example, attempted to discount Confavreux by stressing that the patients in the studied cohort were younger than the Petitioner (and thus its findings were less pertinent to her circumstances). Second Okuda Rep. at 3. But Bogdanos—a study Dr. Okuda embraced as *reliable* evidence supporting causation—involved almost the same youthful cohort. Bogdanos at 218. In the end, Dr. Hawes maintained, the balance of all such evidence suggested it was unlikely the hepatitis B vaccine can cause MS, even if individual items of evidence did not conclusively establish the point, or were not exactly on all fours with the facts of this case. Second Hawes Rep. at 3.

III. Procedural History

The Petition was filed in September 2022, activated in January 2023, and was initially assigned to a different special master before transfer to me in May 2023. Respondent filed his Rule 4(c) Report opposing entitlement in June 2023 (ECF No. 20), and simultaneously moved to dismiss the matter, relying on the fact that MS appeared to be the proper diagnosis, but that in prior cases I had not deemed MS to be a proper vaccine injury. That fall, I determined that I would allow Petitioner the opportunity to substantiate her claim with expert input, and the parties subsequently filed the aforementioned reports. The parties thereafter briefed the claim based on MS as the injury, and the matter is now ripe for resolution.

IV. Parties' Arguments

Petitioner

In defending her claim, Petitioner had the opportunity to file two briefs, and while they overlap to some extent, they also are not identical.

In her initial brief (filed in reaction to Respondent's then-pending motion to dismiss), Petitioner set forth a lengthy legal defense of her claim's viability based on the Federal Circuit's *Althen* test. First Memo. at 11–51. First, she argues that she can demonstrate that the hepatitis B

vaccine "can cause" MS.[12] *Id.* at 27–37, 38–45. She notes (consistent with Dr. Okuda's opinion) that case reports of an association between the vaccine and MS later caused France to cease its hepatitis B vaccination campaign, and that in later studies like Hernan the association was substantiated. *Id.* at 28–30.

In addition, Petitioner observed that the Program had previously litigated (via an omnibus proceeding) whether the hepatitis vaccine generally could cause certain CNS demyelinating conditions, answering that questions in the affirmative. First Memo. at 30–31, (citing *Stevens v. Sec'y of Health & Hum. Servs.,* No. 99-594V, 2006 WL 659525, at *1–3 (Fed. Cl. Spec. Mstr. Feb. 24, 2006)). Indeed, *Stevens* was later explicitly relied upon by another special master, who found the hepatitis B vaccine could be linked to MS. First Memo. at 31–33; *Werderitsh v. Sec'y of Health & Hum. Servs.,* No. 99-310V, 2006 WL 1672884 (Fed. Cl. Spec. Mstr. 2006). Since that time, many prior Program decisions concluded that causation under such circumstances could be established. First Memo. at 34 n.25 (identifying 20 successful cases, and contrasting them against cases where fact issues prevented a favorable determination).

Petitioner further explained the basis for her theory. Molecular mimicry, she maintained, is a reliable mechanistic explanation for how autoimmune diseases can occur, because the body produces autoantibodies in response to a vaccine's antigens that then (due to similarity between the antigens and self-tissue structures or components) erroneously cross-react, attacking the body. *See* First Okuda Rep. at 5–6. Evidence filed by Dr. Okuda (although not necessarily discussed in his reports) alleges that CNS diseases are possibly related to an inappropriate autoimmune reaction to myelin antigens contained in a vaccine or virus. First Memo. at 42–43. Petitioner points to studies where patients developed CNS inflammation within weeks of receiving the hepatitis B vaccine and Dr. Okuda's assertion that hepatitis surface antigens share "strong homologies with myelin basic protein and myelin oligodendrocyte glycoprotein" to support the theory that the hepatitis B vaccine could cause inflammatory conditions using molecular mimicry."]. *See id.* (discussing Tourbah at 396–401 (1999)); and First Okuda Rep. at 5. While the studies put forward by Petitioner do not assert or find a direct association between the hepatitis B vaccine and CNS disease development, they hypothesize that molecular mimicry could be the reason for the vaccine's role as a possible trigger for autoimmune adverse reactions. First Memo. at 44 (quoting Bogdanos at 223). It is because of these studies that question the understanding of the hepatitis B

[12] Petitioner also suggests that a "biologically plausible" causation theory is sufficient under *Althen* (although agrees it must be demonstrated on the basis of reliable evidence). First Memo. at 12–17. Of course (and as noted below), the Circuit has repeatedly stressed that given the obligation to prove each *Althen* element by a "more likely than not" standard, a claimant *cannot* evade her evidentiary obligation by merely proposing a plausible theory that falls short of the preponderant standard. *See, e.g., Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly v. Sec'y of Health & Hum. Servs*., 592 F.3d 1315, 1322 (Fed. Cir. 2010)). I concur that an expert's mere utilization of the term "plausible" does not rule out a proposed theory – but also note that numerous theories *plausibly* proposing how the immune-stimulating effects of vaccination could cause an autoimmune injury are not sufficiently supported by enough evidence reliably bulwarking the theory to deem the theory preponderantly established. Plausibility cannot be used a shield by claimants to avoid meeting their preponderant obligations.

vaccine's relationship with MS and offer molecular mimicry as a possible link that Petitioner puts forward her theory that the vaccine could have caused or aggravated her MS by a preponderance of the evidence.

In the case where a special master disagreed that molecular mimicry can cause MS, Petitioner briefly raises the argument that other components of the vaccine—the "adjuvants, preservatives, or contaminants associated with unique batches"—may also be responsible for the Petitioner's damages, but gives no studies or scientific support to this claim. First Memo. at 44 (quoting First Okuda Rep. at 5). Petitioner is confident that either of these theories satisfies *Althen* prong one. *Id.* at 45.

The other *Althen* prongs are also met, in Petitioner's view. First Memo. at 45–48. Petitioner claims prong two is met because the facts from her medical records show a logical sequence of cause and effect. Specifically, Dr. Okuda believes that the mechanism of molecular mimicry is plausible between the hepatitis B vaccine and MOG to trigger demyelination in CNS when the immune system mistakes MOG for a foreign invader. *Id.* at 46. Further support for satisfying prong two comes from treating-physician attributions where Petitioner's neurologist, Dr. Stankus, noted that the Patient had ADEM and that the petitioner did have a hepatitis B immunization roughly 4 to 5 days before the onset of her symptoms. Ex. 5 at 76. Lastly, Petitioner claims that prong three is satisfied because the twenty-one days from her vaccine to the onset of symptoms is consistent and reasonable for symptoms to appear based on an adaptive autoimmune response triggered by a vaccine. *Id.* at 47–48.

Petitioner filed her Supplemental Memorandum in Support of Entitlement after reviewing Respondent's Experts' Reports. This Memorandum greatly overlaps with the Petitioner's First Memo, but offers an in depth explanation of supporting evidence for general and specific causation. Petitioner's *Althen* three argument remains unchanged between her two briefs.

Petitioner's argument that the hepatitis B vaccine can cause MS remains mostly unchanged between the first and second brief. Petitioner takes the time to lay out both Petitioner's and Respondent's experts' arguments to establish molecular mimicry as a plausible theory for Petitioner's MS. Second Memo. at 13–24. Petitioner specifically highlights studies where findings, or lack of explanation for results, open the door for the possibility of a relationship between the hepatitis B vaccine, antibodies generated via molecular mimicry, and the pathogenesis for MS. *Id.* at 13–14, 16–18, 22–23 (discussing findings in Bogdanos (mimicking similarity between hepatitis B components and associated nerve tissues does not establish a definitive link to MS, but merit further studies to explore relationship); Hernan (suggesting the existence of scientific validity in the concerns about the hepatitis B vaccine); and Mikaeloff II (showing an increasing trend of MS developing three years after receiving Engerix B vaccine, but conceding preparation of the vaccine could explain the trend)).

No evidence put forward by the Petitioner offers a conclusory showing that the hepatitis B vaccine causes MS through molecular mimicry. But Petitioner emphasizes that she does not need to prove epidemiological certainty to satisfy her *Althen* one burden; rather, she argues, she needs only show general causation by a preponderance of the evidence. And after reviewing epidemiological evidence, the IOM agreed that it was unable "to accept or reject a causal relationship between the hepatitis B vaccine and relapse of MS in adults." 2012 IOM Report at 83. "[S]ome issues simply cannot be resolved with currently available epidemiologic data." Second Memo. at 23 (citing 2012 IOM Report). Petitioner believes the evidence put forward (and discussed above) suggests the possibility of a connection between the hepatitis B vaccine and MS via molecular mimicry that satisfies the preponderance burden of *Althen* one.

For *Althen* prong two, Petitioner offers Dr. Okuda's explanation of the Banwell article as further support that her hepatitis vaccine did cause her MS. *See* Second Memo. at 26. Specifically, Dr. Okuda's assertion that the article found elevated MOG-IgG levels in those with CNS demyelinating syndromes, including MS. *Id.* (quoting Second Okuda Rep. at 4). Dr. Okuda reasoned Banwell's proposal to measure MOG-IgG levels to assist in diagnosis of MOGAD supports a theory of pathogenicity of MOG-IgG antibodies in those with MS. *Id.*; Second Okuda Rep. at 4 (citing Banwell). Petitioner believes this theory of pathogenicity bolsters her argument that a sequence of cause and effect between receiving her vaccine and the onset of her MS symptoms is logical and probable.

Respondent

Respondent filed one brief in opposition to Petitioner's First and Second Memo that argued for the denial of compensation and dismissal.

Respondent argues that the record does not support a significant aggravation or causation-in-fact claim, and Petitioner has failed to make a showing for either claim.[13] The six-prong test to satisfy a significant aggravation claim is described in *Loving v. Sec'y of Health & Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009).[14] The first three *Loving* prongs are unique to a significant aggravation claim, while the last three prongs are identical to the *Althen* requirements of a causation-in-fact claim. Respondent asserts that—regardless of whether Petitioner intends to argue significant aggravation—Petitioner has not satisfied the first three *Loving* prongs required of a significant aggravation claim. Moreover, Respondent also argues that even if Petitioner could satisfy the first three *Loving* prongs, Petitioner has failed to satisfy general causation, specific causation, and

[13] Petitioner has not in her Petition explicitly argued that the hepatitis B vaccine she received significantly aggravated pre-existing MS, but alludes to the possibility of such a claim in both her briefs. First Memo at 45; Second Memo at 1. However, even if a significant aggravation claim was intended, it should be noted that the Petitioner fails to address the elements of significant aggravation claim in either her First or Second Memo. *See generally* First Memo; Second Memo.

[14] *See infra* pp. 40–41.

proximate temporal relationship required of both significant aggravation and causation-in-fact claims, thereby failing to bring a successful claim in this matter.

Respondent observes that the Petitioner's records did not show an MS diagnosis, or that the Petitioner suffered MS symptoms, before receiving the vaccine at issue. Opp. at 23. However, Dr. Okuda noted that Petitioners post-vaccine MRIs showed lesions that likely developed before the vaccination. First Okuda Rep. at 4. These pre-vaccination lesions could have been indicative of MS. Opp. at 23. But even if it is assumed the Petitioner had MS before the vaccination, neither Dr. Okuda nor any of the medical records produced indicate or suggest how the vaccination adversely affected her ultimate condition beyond what is expected of relapsing-remitting MS. *Id.* Respondent believes this lack of evidence does not satisfy the first three *Loving* prongs by a preponderance of the evidence, and why Petitioner cannot prevail on a significant aggravation claim. *Id.* at 23–24.

Respondent next attacks Petitioner's causation-in-fact claim. Respondent argues that Petitioner cannot support her claim because she cannot establish either general or specific causation required under *Althen* prong one and two.

First, Respondent argues that it has not been demonstrated that the hepatitis B vaccine can cause MS via molecular mimicry. Opp. at 25. Petitioner provided purportedly significant homologies between the hepatitis B vaccine and the MOG protein in the development of experimental allergic encephalomyelitis and acute disseminated encephalomyelitis. *Id.* Dr. Okuda's theory relies on the inference that, because allergic encephalomyelitis and ADEM are "closely related" to MS, there is a possibility that MS can be caused from an immune response to MOG from the hepatitis B vaccine. *Id.* (quoting First Okuda Rep. at 6). But after reviewing the literature, Respondent concludes that Dr. Okuda has not provided any corroborative evidence that suggests these homologies likely result in MS.

Respondent notes that Petitioner does not put forward any epidemiological evidence that supports a causal connection between the hepatitis B vaccine and MS. Opp. at 26. And the literature put forward by Dr. Okuda suggesting a causal connection was undermined or dismissed by Respondent's experts. *Id.* at 25; *see* Ex. A–D. In fact (and contrary to Petitioner's contentions that the association has Program support), multiple Vaccine Program cases have found that MS is not vaccine-caused, precisely due to a lack of reliable scientific evidence. *Id.* at 26 (citing *Porch v. Sec'y of Health & Hum. Servs.*, No. 17-802V, 2023 WL 3301420 (Fed. Cl. Spec. Mstr. May 8, 2023); *Samuels v. Sec'y Health & Human Servs,* No. 17-071V, 2020 WL 2954953 (Fed. Cl. Spec. Mstr. May 1, 2020); *Pek v. Sec'y of Health & Hum. Servs.*, 16-0736V, 2020 WL 1062959 (Fed. Cl. Spec. Mstr. Jan. 31, 2020)). Prior Vaccine Program decisions are not binding, but can be used to inform decisions before the Court. *See Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 167 Fed. Cl. 127 (2023). The Petitioner's offered *Werderitsh* to do just that. But although

the Petitioner in *Werderitsh* prevailed on a hepatitis B/progressive MS claim, this case involves relapsing/remitting MS, and a claimant who had received only received one hepatitis B vaccine dose. Opp. at 29–30. Respondent also notes that the present case is void of evidence supporting a challenge-rechallenge, while the Court in *Werderitsh* felt the disease course "*reminiscent* of [a] challenge/rechallenge." Opp. at 30 (quoting *Werderitsh*, 2006 WL 1672884, at *26) (emphasis added).[15]

Respondent also offers four other prior decisions where the Respondent was successful on vaccine caused MS claims. Opp. at 29-30 (citing *Ross v. Sec'y of Health & Hum. Servs.*, No. 06-412V, 2007 WL 5171598, at *5 (Fed. Cl. Spec. Mstr. Oct. 4, 2007) (denying compensation in this hepatitis B/MS case, in which petitioner alleged the vaccine caused or significantly aggravated MS, as petitioner failed to prove Althen prongs one or two); *Davis v. Sec'y of Health & Hum. Servs.*, No. 99-510V, 2007 WL 5177468, at *16 (Fed. Cl. Spec. Mstr. May 30, 2007) (denying compensation in this hepatitis B/MS case); *Mann v. Sec'y of Health & Hum. Servs.*, No. 99-577V, 2007 WL 5180507, at *4 (Fed. Cl. Spec. Mstr. July 30, 2007) (denying compensation in this hepatitis B/MS case based on uncertain diagnosis and delayed onset); *Rodriguez v. Sec'y of Health & Hum. Servs.*, No. 99-307V, 2009 WL 3824733, at *27 (Fed. Cl. Spec. Mstr. Oct. 30, 2009) (denying compensation in this hepatitis B/MS case on Althen prong three)).

Based on a what Respondent believes to be a lack of reliable evidence, Respondent concludes that molecular mimicry is only a *possible* causal link between the vaccine and the injury—insufficient to prove general causation. Opp. at 25–26 (citing *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1357 (Fed. Cir. 2013)). Based on the above, Respondent believes Petitioner failed to satisfy *Althen* one, and, therefore, failed to satisfy her causation-in-fact claim.

If a Petitioner fails to satisfy *Althen* one, then Petitioner cannot satisfy *Althen* prong two. *McDaniel v. Sec'y of Health & Hum. Servs.*, No. 17-1322V, 2023 WL 4678688, at *33 (Fed. Cl. June 26, 2023). But Respondent still engaged in a case-specific analysis to establish why Petitioner fails a *prima facie* showing of specific causation. Opp. at 31. To satisfy *Althen* prong two, the petitioner must put forward preponderant evidence of a logical sequence of cause and effect between her hepatitis B vaccine and her MS. *See Althen*, 418 F.3d at 1278. Petitioner's onset of symptoms was temporally proximate to the hepatitis vaccine, but also to a URI that was noted by Petitioner's neurologist when determining the cause of her symptoms. Opp. at 33. In addition, none of Petitioner's doctors embraced a vaccination cause for Petitioner's MS, nor do Petitioner's medical records support a causation theory. *Id.* at 31–32. At best, Dr. Stankus initially noted a possible connection between the hepatitis vaccine and ADEM, but never put forward a theory of causation for Petitioner's ailment. *Id.* at 31. This lack of evidence along with Dr. Okuda's sparse

[15] As noted in *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006), 440 F.3d at 1322, "[a] rechallenge event occurs when a patient who had an adverse reaction to a vaccine suffers worsened symptoms after an additional injection of the vaccine."

argument for specific causation do not accumulate to a *prima facie* showing by a preponderance of the evidence. *Id*. at 33.

Respondent's last argues that Petitioner failed to demonstrate a medically appropriate temporal relationship between the vaccine and onset of MS symptoms. *Id*. at 34. Dr. Okuda makes a conclusory statement that the twenty-one days between the vaccine and Petitioner's onset of symptoms is reasonable, without offering any scientific support for the contention. *Id.*

V. Applicable Law

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

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that her illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly*, 592 F.3d at 1321; *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[16] There is no Table claim for MS as an injury after the receipt of *any* covered vaccine—so such a claim can only sound in causation-in-fact.

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

[16] 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).

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

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

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

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs.*, 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C.*, 704 F.3d at 1356 (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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

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

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

B. *Legal Standards Governing Factual Determinations*

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

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd sub nom. Rickett v. Sec'y of Health & Hum. Servs*., 468 F. Appx. 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd*, 968 F.2d 1226 (Fed. Cir.), *cert. denied*, 506 U.S. 974, 113 S. Ct. 463, 121 L.Ed.2d 371 (1992) (citing *United States v. United States*

Gypsum Co., 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

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

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

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

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

D. *Review of Medical Literature*

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

E. *Resolution of Case on Papers*

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck*, 945 F.3d at 1366; *see also Hooker v. Sec'y of Health & Hum. Servs*., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs*., 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

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

A. *Medical Characteristics of MS*

The parties agree that Petitioner was accurately diagnosed with relapsing-remitting MS. First Memo. at 37–38; Opp. at 9, 31. As noted in *P.M. v. Sec'y of Health & Hum. Servs.*, No. 16-949V, 2019 WL 5608859, at *21 (Fed. Cl. Spec. Mstr. Oct. 31, 2019):

> MS is a demyelinating CNS disease. *See Taylor v. Sec'y of Health & Human Servs.*, No. 13-700V, 2018 WL 2050857, at *21 (Fed. Cl. Spec. Mstr. Mar. 9, 2018). It likely has an autoimmune pathogenesis. *W.C. v. Sec'y of Health & Human Servs.,* No. 07-456V, 2011 WL 4537877, at *3 (Fed. Cl. Spec. Mstr. Feb. 22, 2011), *mot. for review den'd*, 100 Fed. Cl. 440 (2011), *aff'd,* 704 F.3d 1352 (Fed. Cir. 2013). Patients diagnosed with MS typically experience multiple episodes of CNS demyelination separated in time and space (meaning throughout the CNS), concurrent with a progressive, if remitting and relapsing, decline in their overall health course. *Taylor*, 2018 WL 2050857, at *21. An MRI can be used to corroborate the dissemination in space and time requirement, and often reveals old lesions as well as enhancing/new lesions. *Id.* Symptoms can include numbness or weakness in the body, loss of vision, tremors, unsteady gait, slurred speech, and dizziness. *Id.*

Significantly, medical science still does not reliably understand *why* MS begins, or later recurs—and has generated no study supporting the proposition that a one-time neurologic "hit" can thereafter initiate what becomes a chronic injury (although the research into the MS-Epstein Barr virus ("EBV") connection is promising).[17] There are thus no widely-accepted specific causes of MS—and (contrary to Dr. Okuda's contentions) no particular antibodies associated with it either. Yet many other demyelinating diseases—MOGAD, for example—*do* have associated antibodies, standing at least as biomarkers of the disease even if medical science is not precisely confident even in those cases that such antibodies drive the disease, as opposed to arise in the circumstances of the disease.

B. *Program Decisions Involving MS Generally*

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

[17] Some recent studies have posited a reliable link between MS and an EBV infection. *See, e.g.*, Grimaldi at 1099 (citations omitted). But claimants cannot persuasively invoke these studies in the context of other vaccines – especially since there is *no* EBV vaccine to begin with.

Other CNS demyelinating diseases that manifest in an acute and monophasic manner, like TM or ADEM, have been reasonably associated with vaccines in many prior Program matters, based on sound medical science. MS is not the same, however—and there is compelling logic to treat it differently from somewhat-related demyelinating neuropathic injuries that also impact the CNS. *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).

MS is *chronic and persistent*, and often can be subclinical for lengthy periods of time (as reflected in Dr. Okuda's discussion of "radiologically isolated syndrome," in which demyelinating lesions are observed by imaging, in the absence of clinical symptoms). And its symptomatic flares (common to relapsing/remitting MS) can be triggered by a wide array of external environmental factors. *See* First Sweeney Rep. at 5 (citing R. F. Brown et al., *Relationship Between Stress and Relapse in Multiple Sclerosis: Part II. Direct and Indirect Relationships*, 12 Multiple Sclerosis J. 465, (2006), filed as Ex. A Tab 9 (ECF No. 39-10), at 465; Jiang at 1). But the trigger for such a flare, whatever its nature, cannot be said to have "started" the underlying disease process *ab initio*.[18] By contrast, more self-limiting neurologic injuries are understood to on occasion be triggered by the single impact of an external stimulus, such as a wild infection—and hence reasoning that the same kind of monophasic illness could also begin with the single instance of vaccine exposure has far greater power.

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

[18] I address whether Petitioner's vaccination could have aggravated an existing but sub-clinical case of MS below.

[19] I have also dismissed several cases where MS proves to be the evidentiarily-supported diagnosis, even though the petitioner's initial symptoms seemed to first-responding treaters to reflect TM, ADEM, or a comparable acute demyelinating neuropathy. *See, e.g.*, *Morgan v. Sec'y of Health & Human Servs*., No. 15-1137V, 2019 WL 7498665, at *16 (Fed. Cl. Spec. Mstr. Dec. 4, 2019), *mot. for review den'd*, 148 Fed. Cl. 454 (2020), *aff'd*, 850 F. App'x 775

Based on such reasoning, I have repeatedly rejected causation theories that many different covered vaccines are capable of causing (or worsening) MS. *See, e.g.*, *Porch v. Sec'y of Health & Hum. Servs.*, No. 17-802V, 2023 WL 21875 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (MMR vaccine did not cause MS); *P.M.*, 2019 WL 5608859 (flu vaccine not shown to be capable of worsening MS); *L.Z. v. Sec'y of Health & Hum. Servs.*, No. 14-920V, 2018 WL 5784525 (Fed. Cl. Spec. Mstr. Aug. 24, 2018) (same). Other special masters have also so concluded. *See, e.g.*, *Juranek v. Sec'y of Health & Hum. Servs.*, No. 19-226V, 2025 WL 399501, at *33–34 (Fed. Cl. Spec. Mstr. Jan. 8, 2025) (flu vaccine not shown to be capable of aggravating MS).

C. *Hepatitis B Vaccine and MS*

I have not previously had the occasion to evaluate whether the *hepatitis B vaccine* can cause MS. Other special masters have found an association—in particular in *Werderitsh.* But that determination (a decision rendered nearly 20 years ago—and hence based on the then-available science, as opposed to what prevails today) is limited to its unique facts, and thus does not provide helpful guidance in this case.

In *Werderitsh*, a petitioner received *two* doses of the hepatitis B vaccine (in contrast to the facts of this case) over a period of five weeks, and then began to experience neurologic symptoms approximately one month after the second dose. *Werderitsh*, 2006 WL 1672884, at *2. The individual was subsequently diagnosed with MS. *Id.* at *3–4. Although there was an absence of record evidence of any initial reaction to the first dose, there was other secondary evidence that the petitioner had experienced "visual disturbances" close in time to it, with more clinically-obvious neurologic dysfunction within a month of the second dose. *Id.* at *26–27.

In finding causation met, the special master relied on several items of literature also filed in this case, pro and con, including Hernan, Confavreux, and DeStefano—in effect, what was known about the putative association between the vaccine and MS *up to around 2005*. *Werderitsh*, 2006 WL 1672884, at *8–15. The special master gave weight as well to the findings of the "Hepatitis B-Neurological Demyelinating Omnibus Proceeding" (as described in *Stevens*, 2006 WL 659525, at *1–3), which had determined that a number of acute demyelinating conditions could be caused by the hepatitis B vaccine.[20]

(Fed. Cir. 2021); *Caruso v. Sec'y of Health & Human Servs.*, No. 15-200V, 2017 WL 5381154, at *12–13 (Fed. Cl. Spec. Mstr. Oct. 18, 2017), *mot. for review den'd*, 137 Fed. Cl. 386 (2018).

[20] I note that I would have far less difficulty relying on *Stevens* in a case alleging that the hepatitis B vaccine caused a one-time, acute and monophasic vaccine reaction, leading to GBS or a CNS-oriented demyelinating neuropathy.

The *Werderitsh* special master also deemed significant another factor in finding causation that is not present herein. That claimant had experienced a *progressive* form of MS, as opposed to the relapsing-remitting form at issue in this case. *Werderitsh*, 2006 WL 1672884, at *24. The petitioner's medical history was effectively consistent with the conclusion that the two doses of vaccine caused that progressive form to manifest. The special master acknowledged, however, that if the phenotype of relapsing/remitting MS had been at issue, it would have been "impossible to determine whether the vaccine played any role *in the course of an illness that, by itself, is episodic*." *Id.* (emphasis added). And (as Respondent has noted), the *Werderitsh* special master also found that the concept of "challenge-rechallenge" (where a person experiences a demonstrated reaction to an antigenic stimulus—then a more pronounced or faster reaction to re-exposure, thus suggesting that the stimulus had some causal effect) seemed present based on Petitioner's two-dose experience (and even though the symptoms after each dose were not precisely the same). *Id.* at *26.

All of the foregoing establishes why *Werderitsh* has limited guidance value in resolving the present claim. This case involves relapsing-remitting MS (a context which even the *Werderitsh* special master acknowledged made causation unlikely), and only one dose of vaccine instead of two. Moreover, a significant period of time has passed since *Werderitsh* was issued—giving medical science ample opportunity to corroborate the evidence supporting causation or rebut it. As will be shown below, however, it largely appears the hypothesis of a causal relationship between the hepatitis B vaccine and MS *has not been strengthened* in the past twenty-plus years.

A more recent decision underscores my rationale for giving *Werderitsh* limited weight. *See generally Hodge v. Sec'y of Health & Hum. Servs.,* No. 9-453V, 2023 WL 4186513 (Fed. Cl. Spec. Mstr. May 24, 2023), *mot. for review den'd,* 168 Fed. Cl. 117 (2023). *Hodge* involved the claim that a distinguishable CNS disease (neuroborreliosis) was aggravated by the hepatitis B vaccine. *Id*, at *1. *Hodge* discussed prior Program treatment of claims involving the hepatitis B vaccine, including *Werderitsh* and the *Stevens* decisions (which mostly involved acute CNS demyelinating injuries), but did not find them to be helpful guidance. *Id.* at *55. In so determining, the *Hodge* special master invoked the general principle that special masters are not bound formally by the prior decisions of their colleagues—but he also observed that (a) these prior decisions favoring a causal relationship were never appealed, and therefore the strength of their reasoning had not been tested, and (b) other decisions had not only called into question the persuasiveness of molecular mimicry as a mechanism for certain CNS demyelinating injuries, but *had* been appealed—and were affirmed. *Id.* at *55–56.

A contrary recent determination—*Jane Doe*, 2023 WL 4741993—embraced causation in this context, and some of the logic of *Werderitsh*. But that petitioner had begun experiencing symptoms suspicious for optic neuritis *within a week* of vaccination—a much faster timeframe than at issue here, and one deemed supportive of causation by the *Jane Doe* special master. 2023

WL 4741993, at *4, 36. Hence, the record evidence that the hepatitis vaccine had sparked some kind of close-in-time reaction was far more compelling than what is seen in this case.

The *Jane Doe* special master also concluded that the hepatitis B vaccine can significantly aggravate "asymptomatic" MS,[21] applying the test for that alternative kind of causation claim set forth in *Loving*, 86 Fed. Cl. 135, 144 (2009). *Id.* While this decision does not expressly mention *Werderitsh*, it discusses the state of medical science on the question of the hepatitis B vaccine's relationship to MS prior to 2005, and mentions articles also offered in this case, like Bogdanos, as well as the 2012 IOM Report. *Id.* at *30–31. Ultimately, *Jane Doe* (relying in part on a finding that infections could trigger MS *relapses*—distinguishable from causing MS) deemed significant the fact that the proposed relationship continues to be "studied with the theory that a hepatitis B vaccine-induced immune response can lead to demyelination"—and that fact, coupled with MS's rarity,[22] and the Program's preponderant standard of proof, meant the theory was established.

I do not deem the reasoning of *Jane Doe* to be compelling. It assumes that the science on the purported MS-hepatitis B vaccine association from twenty or more years ago to have remained static, when that is not actually the case. To the contrary—a possible association between the two has, if anything, *weakened* over time. It also incorrectly invokes the lack of existing evidence on the vaccine-MS association as a justification for demanding *less* of a preponderant showing from the claimant. *Caves v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 143 (2011) ("the standard of proof does not operate as a sliding scale that varies depending upon the quantity and quality of the scientific evidence that is available"), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012).

[21] I do not find the characterization of MS as "asymptomatic" (meaning no clinical symptoms have been observed at the time a patient's CNS lesions are radiologically discovered) to be well-taken. It certainly could be determined by a neurologist, based on retrospective evaluation, that a patient was *at risk* to develop outwardly-clinical MS later, based on radiologic lesion evidence uncovered prior to when symptoms manifested. This may in part be why the phrase "radiologic isolated syndrome" is used to describe such cases (which otherwise would not yet meet the diagnostic criteria for MS).

But to call MS "asymptomatic" in the context of a Vaccine Program claim seems reflective of an effort to reverse-engineer a means of compensation—evading the fact that a vaccine could not be blamed for lesions *already present* at the time of vaccination by recasting the claim as one of significant aggravation. An actual significant aggravation claim seems mostly intended to address circumstances in which a claimant is already known to suffer from an existing illness that is believed to have been *worsened* by the impact of vaccination. It thus is not evident that this framing of the circumstances is a proper invocation of significant aggravation. Indeed—given that it is known that autoimmune diseases like MS often occur in part due to a person's genetic susceptibility, *when* could a petitioner not claim that they possessed an "asymptomatic" form of the disease that was aggravated thereafter?

[22] MS actually is *not* considered to be a particularly rare disease. *See, e.g.*, Grimaldi at 1099 ("Multiple sclerosis [], the most common chronic inflammatory demyelinating disease of the central nervous system [], affects over 2.8 million patients worldwide and is the leading cause of permanent nontraumatic neurological disability in young adults").

Thus, there *is* prior Program support for the claim that the hepatitis B vaccine can cause, or worsen, MS. But the science itself underlying the purported causal relationship is dated and somewhat limp—and should not be assumed sufficient to cross the preponderant "line" simply because the theory has been embraced in prior cases, but without much in the way of persuasive reasoned analysis. Otherwise (as in all Vaccine Act cases), favorable entitlement determinations turn on the specific facts at issue—meaning that not every instance of post-hepatitis B vaccine MS will result in compensation, even when causation is deemed to have been established.

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

Petitioner's claim founders primarily on *Althen* prongs two and one (and therefore I do not address the third prong, since all three must be satisfied for entitlement to be found).[23]

Prong Two

Petitioner did not preponderantly establish that the hepatitis B vaccine she received "did cause" her MS, because the record supports the conclusion that her MS likely *preceded* vaccination—meaning the vaccine *could not have caused it*. *McDaniel*, 2023 WL 4678688 at *33 (finding that Petitioner could not satisfy Althen prong two where onset of immune-mediated myopathy occurred before vaccination).

Dr. Okuda largely conceded that when Petitioner first underwent an MRI on March 8, 2020, the resulting imaging revealed the existence of some lesions that were non-enhancing—meaning not active or new. First Okuda Rep. at 3–4. And he did not try to establish that all of these lesions could have come into being within a month of vaccination. At most, he contended that there existed one *possibly* enhancing lesion, and seemed to opine that it could have been instigated or encouraged by the impact of vaccination—but this would have represented worsening rather than initiation of Petitioner's MS (given the presence of the other lesions).

Accordingly, this is not a case where a claimant's MS can be established to have *begun* post-vaccination (even if Petitioner's clinical symptoms only manifested in that post-vaccination timeframe).[24] The fact that Petitioner was only *diagnosed* with MS later, after treaters were able to factor in a combination of clinical evidence with test results, does not mean it began post-vaccination as well. *Flowers v. Sec'y of Health & Hum. Servs.*, No. 20-285V, 2024 WL 2828211,

[23] *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 566 Fed. App'x 976, 980 (Fed. Cir. 2014).

[24] Some of Petitioner's initial treaters, like Dr. Stankus, opined a vaccine relationship—but based on the assumption that her presenting symptoms reflected only ADEM (and that onset occurred closer-in-time to vaccination). Ex. 5 at 76, 81; Ex. 7 at 246. But as her illness progressed, and more clinical and testing results were obtained, he shifted his diagnosis to MS. Ex. 2 at 11. And I do not discern in this record evidence that treaters ever later associated Petitioner's MS with her vaccination.

at *12 (Fed. Cl. Spec. Mstr. May 8, 2024) ("[i]t is a foundational matter of Vaccine Program law that onset occurs at first manifestation of a symptom, *regardless* of whether the disease it foretells could be diagnosed at that time—and thus whether the onset symptoms would be clearly understood to reflect the start of the illness"), *mot. for review den'd,* 173 Fed. Cl. 613 (2024).

Prong One

Petitioner did not offer sufficient reliable evidence for the contention that the hepatitis B vaccine *can* cause MS. Rather, this was yet another case in which a Petitioner attempts to leverage the otherwise-acceptable medical theory of molecular mimicry as an explanation for how a vaccine could spark an autoimmune disease, but without linking up all elements of the theory with sufficient reliable scientific or medical evidence. *McKown v. Sec'y of Health & Hum. Servs*., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (explaining that "merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or vaccine in question" (emphasis omitted)).

Petitioner's theory meets some, but not all, milestones[25] necessary to fit into a reliable chain of propositions sufficient to meet the preponderant standard of proof. She does, for example, successfully identify some possible homologic similarities between components of the vaccine and nerve molecular structures, relying on Bogdanos to do so. But Bogdanos itself admits it says nothing about whether the identified mimics *result* in autoimmune disease-driving cross-reactions. Bogdanos at 223. Accordingly, the mere existence of homologic similarity does not mean that disease is a likely outcome.

After this point, the theory breaks down—with no good evidence suggesting *what* antibodies would likely drive MS, through mistaken attack on a target self-antigen in the CNS. Dr. Okuda in fact somewhat-glibly contended that such antibodies were known to exist for MS, when this is not the case. Moreover, he proposed that antibodies associated with *distinguishable* demyelinating conditions, such as MOGAD, are relevant to MS—despite literature suggesting that they clearly are not (evidenced by items of literature concluding that the associated antibodies need not even be tested for in MS patients). *See* Cobo-Calvo at 3; Banwell at 275, 277. Research specific to anti-MBP antibodies suggests that this kind of antibody would *not* easily cross the blood-brain

[25] In so reasoning, I take into account—but *do not apply as a separate test*—the Benoist & Mather criteria embraced by Dr. Hawse. *See* First Hawse Rep. at 6–7. As I have noted in other decisions, the standards that medical science might rely upon (in the interests of a certainty-level confirmation) when evaluating whether molecular mimicry is a likely mechanism for an autoimmune disease are not congruent with the preponderant evidentiary test applied in the Vaccine Program. *Cerrone v. Sec'y of Health & Hum. Servs*., No. 17-1158V 2023 WL 3816718, at *27 (Fed. Cl. June 1, 2023), *review denied, decision aff'd*, 168 Fed. Cl. 745 (2023), *appeal docketed*, No. 24-1281 (Fed. Cir. Dec. 22,2023). But this does not deprive such criteria of *all* value, since they reflect the sort of considerations that a proposed theory *should* take into account. I can loosely keep in mind those factors in analyzing causation herein—what I cannot do is demand that a claimant *conclusively meet them all*.

barrier (in order to cause damage in the CNS and produce the lesions indicative of MS and which often drive clinical symptoms) in the first place. Arnon & Aharoni at 14593.

Another important limitation to Petitioner's contentions is the extent to which the "march of science" has gradually undercut some of the tentative conclusions set forth in the literature Dr. Okuda expressly relied upon for his contentions. As articles filed in this case illustrate, there was *some* reasoned concern over 25 years ago that the hepatitis B vaccine might be associated with MS—albeit largely on the basis of case reports. Thereafter, and as science investigated the concept, studies like Hernan seemed potentially to add ballast to the putative association. And Bogdanos provides the kinds of homologic evidence often relied upon in Program cases, adding some ballast to the theory.

But since the publication of such articles, medical science has had ample time to explore further the associations they suggest. Did science confirm these nascent proposals? No. Rather, and as many other items of literature establishes, the earlier, nascent view of an association has not been confirmed (and was not even deemed wholly persuasive at the time). *See generally* Sestili, Rutschmann, DeStefano, 2004 WHO Article. And the absence of subsequent studies exploring and corroborating the contention that the hepatitis B vaccine can cause MS undermines the conclusion that the conception has the same soundness it might once have possessed. *Tompkins v. Sec'y of Health & Hum. Servs*., No. 10-261V, 2013 WL 3498652, at *26 (Fed. Cl. Spec. Mstr. June 21, 2013) (noting that there was no evidence in the years after article was published corroborating its suggestions about a vaccine-injury association), *mot. for review den'd*, 117 Fed. Cl. 713 (2014).

Dr. Okuda made reasonable points about the weight to be given certain items of literature offered against causation. Confavreux, for example, was admittedly criticized in the 2012 IOM Report for methodologic issues (as I have noted in other decisions). *See, e.g.*, *Porch*, 2023 WL 21875, at *13–14, 24). I also acknowledge that questions about the hepatitis B vaccine's possible association with MS not only arose reasonably, but *at that time* possessed some degree of reliable scientific support. The contention of an association remains *plausible* if nothing else, and it is not surprising that special masters would disagree on where the science stands today on the question, given the existence of such older evidence (although I am no more bound by their determinations than they would be by my conclusions in this case, or others like it).

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

studies about a possible vaccine-MS association that have not since been corroborated, despite ample opportunities to do so. He assumed that showings of homology in articles like Bogdanos mean a likely autoimmune cross-reaction, without offering preponderant proof for the latter proposition—with no direct or indirect evidence offered specific to MS (as opposed to distinguishable CNS demyelinating diseases that are more typically acute and monophasic). And he proposed antibody mediators of disease that have not been shown to be associated with MS, or to have the pathogenic capacity to drive its initiation.

III. Petitioner's Vaccination did not Likely Significantly Aggravate her MS

Petitioner appears to have attempted to prove only that the hepatitis B vaccine could directly cause MS, relying upon *Althen* as the framework for her claim. *See, e.g.*, Second Memo. at 9–27. Thus (and as Respondent has noted), she made no effort in briefing to address the *Loving* elements for a claim of significant aggravation—arguably a waiver of such a claim. Opp. at 23–24.

But Dr. Okuda's opinion could be read to support the proposition that the hepatitis B vaccine Ms. Garris received worsened what was in effect an existing, subclinical case of MS that had not yet become clinically manifest—whether by "unmasking" what was to that point a subacute condition, or causing at least one new lesion capable of resulting in initial symptoms—in either case kick-starting the disease process into more fulminant action. *See* First Okuda Rep. at 6 ("[t]he MRI data provide clear evidence of prior central nervous system involvement before the development of her large pontine lesion that represented *aggravation of her existing condition, resulting in her initial presenting symptoms*") (emphasis added). Thus, it is important herein to evaluate whether the evidence of this case could be read to preponderantly support a claim of significant aggravation.

Where a petitioner alleges significant aggravation of a preexisting condition, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Servs.*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a 'significant aggravation' of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason

> for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the *Loving* test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.*, 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, a Petitioner need not demonstrate an expected outcome, but merely that her current-post vaccination condition was worse than what it was pre-vaccination. *Id.* at 1081. And a claimant may make out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish a "factor unrelated" cause, after the burden of proof has shifted). *Id.* at 1083.

As noted above, I have (in prior detailed reasoned decisions) previously rejected claims that MS can be exacerbated by vaccination. *P.M.*, 2019 WL 5608859; *L.Z.*, 2018 WL 5784525. Other special masters have reached the same conclusion. *See, e.g.*, *Townsend v. Sec'y of Health & Hum. Servs.*, No. 14-266V, 2023 WL 6212496 (Fed. Cl. Spec. Mstr. Aug. 29, 2023), at *26 (evidence did not support conclusion that the flu vaccine can exacerbate MS), *mot. for review den'd*, 170 Fed. Cl. 130 (2024). I arrived at this conclusion in part because the most persuasive medical and scientific articles on the topic deem MS symptoms flares *intrinsic* to the natural course of relapsing-remitting MS—not dangerous tipping points for magnifying the potentially-harmful impact of the illness's course. *See, e.g.*, *P.M.*, 2019 WL 5608859, at *22–24; *see also* B. Weinshenker, *Natural History of Multiple Sclerosis*, 36 Ann. Neurol. S6 (1994), filed as Ex. A Tab 2 (ECF No. 39-13), at S8 (noting average attack rate in MS over a three-year period of 1.2 flares per year). And there is a distinction between the immediate/proximate *cause* of a flare (and a wide variety of external factors can cause one) and the underlying etiology for a person's MS in the first place – as recognized in literature filed in this action. *See, e.g.*, Jiang at 4 (contrasting among of consistent prior evidence of an association between MS *relapse* and stress with less, more conflicting evidence that stress could trigger MS onset outright).

I do not find that the showing that the hepatitis B vaccine could aggravate a subclinical case of MS—or specifically did so to Petitioner—was made any better herein than in such past matters. In fact, reliable items of medical literature filed in this case—Grimaldi in particular—undermine the conclusion that vaccines likely provoke MS flares leading to worsening of an individual's overall course. Grimaldi at 1102. This is a topic important to medical science, since MS patients are vulnerable to infection (a far worse thing to experience than vaccination as a general rule), and therefore studies have aimed to discern if vaccination against a possible infection

poses risks comparable to the infection the vaccine aims to prevent in the first place. *Id.* at 1099 ("infections are knowns to increase the risk of MS flare-ups, and exacerbate the severity of symptoms"). Petitioner's own course, moreover, was inconsistent with the conclusion that her vaccination provoked a damaging flare; almost a month passed between her February 2020 vaccination and initial symptoms, with no intervening evidence of any change, and (other than Dr. Stankus's initial, but incorrect, impression that she had experienced ADEM in the wake of vaccination—and based on the wrong onset date as well) her treaters do not appear to have deemed the vaccine to have had an relationship with her illness.

As noted above, there are other Program decisions reaching an alternative finding, such as *Jane Doe* or *Werderitsh.* But those determinations involve distinguishable fact patterns that made it more likely vaccine involvement was a possibility. Otherwise, they are not binding upon me – and I do not deem them to constitute persuasive reasoning in any event, for the reasons stated.

I thus find that (to the extent any significant aggravation claim was advanced at all) this version of a causation claim would founder on *Loving* prongs four and five. Petitioner did not preponderantly establish that receipt of the hepatitis B vaccine *can* worsen existing MS even if it has not yet manifested clinically. And she did not show this occurred in her own case either. He course was otherwise consistent with what would be expected for the initial presentation of relapsing-remitting MS.

IV. This Matter was Appropriately Dismissed Without Trial

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

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

In addition, there is another overarching consideration at play. Special masters are intended to develop "on the job" expertise from deciding entitlement in the numerous Vaccine Act claims that exist at the Court of Federal Claims. *Hodges*, 9 F.3d at 961 ("Congress assigned

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

My review of the record here plus Petitioner's arguments have convinced me that she cannot preponderantly establish that the hepatitis B vaccine can cause, or aggravate, MS. I reach that determination on the basis of Dr. Okuda's reports, which were succinct and clear in their arguments. I did not need to litigate the matter via live proceeding to reach the determination I have. The inquisitorial function of special masters in the Vaccine Program means that they must steer cases in the most sensible direction, based on the facts presented as well as the special master's experience with comparable claims.

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

CONCLUSION

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

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[26]

IT IS SO ORDERED.

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

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