In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-1601V

CRISTIAN GARCIA,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: January 3, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Ronald C. Homer, Conway, Homer, P.C., Boston, MA, for Petitioner.

Felicia Langel, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On July 22, 2021, Cristian Garcia filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petitioner alleges that he experienced anti-N-methyl-D-aspartate receptor encephalitis ("anti-NMDAR encephalitis") as a result of a tetanus-diphtheria-acellular pertussis ("Tdap") vaccine administered to him on October 2, 2020. *See generally* Petition (ECF No. 1).

A hearing in the matter was held on August 26, 2024. Now, after review of the complete medical record as filed, expert reports, medical/scientific literature, and the parties' briefs, I deny entitlement. Petitioner has not demonstrated it is likely that the Tdap vaccine can cause this specific form of encephalitis.

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. Id.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended at 42 U.S.C. §§ 300aa-10–34 (2012)) (hereinafter "Vaccine Act" or "the Act"). All subsequent references to sections of the Vaccine Act shall be to the pertinent subparagraph of 42 U.S.C. § 300aa.

I. Factual Background

Vaccination and Initial Seizure

Mr. Garcia was twenty-six years old when he received the Tdap vaccine on October 2, 2020. Ex. 1 at 4. He had no history of neurological concerns (Ex. 7 at 7–14), and there is no record evidence of any immediate post-vaccination issues or symptoms.

On October 20, 2020 (eighteen days post-vaccination), Petitioner appeared to suffer a seizure while driving, making some vocalizations before becoming unresponsive and exhibiting shaking movements. Ex. 4 at 31. The emergency medical services ("EMS") records state that "[t]he patient was disoriented and could not answer my questions appropriately, he did not know the year or the day of the week . . .. All signs pointed that the patient had experienced a seizure." Ex. 3 at 5–6. Petitioner was transported to the Layton Hospital Emergency Department ("ED") in Layton, Utah. Ex. 3 at 5–6; Ex. 4 at 31. There, he seemed alert but "a bit foggy," and he displayed normal vital signs and no neurological deficits. *Id*. Petitioner, however, reported recent headaches at the back of his head, although no fever or neck pain. *Id.* A head CT yielded normal results, and Petitioner was diagnosed with a seizure and referred to outpatient neurology. Ex. 4 at 31–33; Ex. 7 at 21.

A little less than a week later, Petitioner had a telehealth visit on October 26, 2020, with neurologist Mohamed Sadiq, M.D. Ex. 7 at 21. He informed Dr. Sadiq that since his seizure, he had been experiencing "intermittent episodes of confusion and impaired comprehension." *Id.* Dr. Sadiq diagnosed Petitioner with a generalized seizure and attempted to expedite the obtaining of a brain MRI and electroencephalogram ("EEG").[3] *Id.* at 16.

A brain MRI was performed at Layton Hospital's ED that same day. Ex. 4 at 11. Although alert, Petitioner appeared to have an "odd affect" and was moderately confused. *Id.* The imaging-yielded normal results, and a lumbar puncture revealed a normal total protein and high white blood cell count. *Id.* at 11, 16, 18. However, because of Petitioner's worsening symptoms, the ED treater opted to evaluate him for a possible infectious meningitis/encephalitis and administered Acyclovir, an antiviral medication. *Id.* at 11. To that end, Petitioner was transferred on October 26th to McKay-Dee Hospital ("McKay-Dee") in Ogden, Utah, for further evaluation and EEG monitoring. *Id.*; Ex. 3 at 8.

[3] "Electroencephalogram" is defined as "a recording of the potentials on the skull generated by currents emanating spontaneously from nerve cells in the brain. The normal dominant frequency of these potentials is about 8 to 10 cycles per second and the amplitude about 10 to 100 microvolts. Fluctuations in potential are seen in the form of waves, which correlate well with different neurologic conditions and so are used as diagnostic criteria." *Electroencephalogram*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15813&searchterm=electroencephalogram (last visited Jan. 3, 2025).

Hospitalization and Progression of Symptoms

Upon admission to McKay-Dee, Petitioner was evaluated by hospitalist Kory Anderson, M.D. Ex. 6 at 22. This record takes specific note of the fact that Petitioner had received his first dose of the Tdap vaccine three weeks before. *Id.* Dr. Anderson also noted in the contemporaneous record from this visit that "[i]t looks like [headache] is common with TDAP >10% of the time, but not sure if 3 weeks out it is. It looks like < 1% of the time [TDAP] can cause a meningitis and/or seizure—again, not sure if 2 weeks+ from the vaccine if it would cause such an issue." *Id.* at 27. Despite this vaccine-oriented speculation, Dr. Anderson diagnosed Petitioner with viral meningitis of "[u]nclear source," and a seizure disorder "likely due to acute infection." *Id.*

While at McKay-Dee, Petitioner also saw a different neurologist, Dr. John Greenert. Dr. Greenert observed that Petitioner's EEG results were normal, as well as the fact that Petitioner had experienced two episodes of confusion since admission, including one in which he "seemed to have automatisms, fidgeting with his fingers, picking at his clothes, or making slight tongue movements." Ex. 6 at 31–33. Dr. Greenert diagnosed Petitioner with "suspected viral meningoencephalitis," although he also took note of Petitioner's receipt of the Tdap vaccine in early October. *Id.* at 33, 34.

On October 28, 2020, Dr. Greenert re-evaluated Petitioner after he had become acutely agitated overnight, appearing confused and throwing things around the room. Ex. 15 at 64. Dr. Greenert noted that Petitioner's wife "continue[d] to feel this [was] all due to the Tdap shot." *Id.* Petitioner's blood testing yielded normal results, however, as was an autoimmune serum panel (which included testing for the NMDAR antibody), and a whole-body CT scan was negative for malignancy. Dr. Greenert started Petitioner on a continuous EEG to monitor for seizure activity. *Id.* at 64–67; Ex. 6 at 37–39, 44–45.

The next day (and although a repeat brain MRI had yielded normal results), Petitioner had another seizure, was persistently tachycardic, and was confused. Ex. 6 at 20, 41; Ex. 5 at 70. Petitioner was therefore transferred to the University of Utah Hospital for continued specialized workup. *Id.*; Ex. 5 at 2. Medical records from this admission confirmed the presence of ongoing seizures, and contained a diagnosis of infectious or autoimmune encephalitis, with a secondary seizure disorder and acute encephalopathy. Ex. 6 at 19, 20. In addition, Petitioner speculated that his symptoms might be attributable to work and financial stress, although his wife again expressed a concern "about the role of a Tdap vaccine he received 3 weeks prior." Ex. 8 at 147. A new neurologist—Safdar Ansari, M.D.—assessed Petitioner with "new seizure with encephalopathy/behavior changes in [an] otherwise young person [was] most concerning for an autoimmune encephalitis after a negative infectious work-up [], such as NMDA encephalitis." *Id.* at 152. Dr. Ansari proposed continuing treatments Petitioner had already been receiving (including

IVIG,[4] acyclovir, and anti-seizure medications), repeating a lumbar puncture, and placing Petitioner on a continuous EEG. *Id.* at 153.

Confirmation of Anti-NMDAR Encephalitis

While at the University of Utah Hospital, Petitioner was also seen by a specialist—autoimmune neurologist Justin Abbatemarco, M.D. Dr. Abbatemarco noted in his write-up that "[Petitioner] had a TDAP vaccination on 10/2 [complicated by] severe arm pain. A few days later, he noted some generalized myalgias, insomnia, and headaches of which [were] very unusual for him. No clear fever but [his] wife noted he wanted the room cold at night. These symptoms persisted until 10/20." Ex. 8 at 178. Dr. Abbatemarco interpreted Petitioner's overall clinical presentation and laboratory findings as "concerning for [an] autoimmune encephalitis (most specifically NMDAR)," and he added plasma exchange therapy ("PLEX")[5] to the treatment regimen. *Id.* at 179.

Lab results subsequently obtained for Petitioner were consistent with Dr. Abbatemarco's suspicions. Serum testing initially had revealed for Petitioner an NMDAR antibody titer of 1:10 (reference range: < 1:10), or "high-normal." *Id.* at 406. But a repeat lumbar puncture performed on October 31, 2020, now showed a high white blood cell count and a higher NMDAR antibody titer of 1:32. *Id.* at 430–33. Additionally, the encephalopathy-autoimmune analysis of Petitioner's cerebrospinal fluid ("CSF") showed an NMDAR antibody titer of 1:4 (reference range: <1:2). *Id.* at 428–29. And the continuous EEG revealed a "delta brush"[6]—a nonspecific finding that nevertheless is sometimes seen in NMDAR encephalitis. *Id.* at 532–33.

By November 2, 2020, the University of Utah Hospital's neurology critical care unit attending physician assessed Petitioner with anti-NMDAR encephalitis with "[e]tiology unclear," adding that "[Petitioner] did receive tDAP vaccination and there is [a] case report of possible NMDA encephalitis after tDAP." Ex. 8 at 162. He was subsequently discharged on November 8, 2020. *Id.* at 132. The discharge summary noted that "[t]he etiology of [his] NMDA encephalitis

[4] "Intravenous Immunoglobulin" is defined as "a pooled antibody, and a biological agent used to manage various immunodeficiency states and a plethora of other conditions, including autoimmune, infectious, and inflammatory states." *Intravenous Immunoglobulin (IVIG)*, National Library of Medicine, https://www.ncbi.nlm.nih.gov/books/NBK554446/ (last visited Jan. 3, 2025).

[5] "Plasma Exchange" is "a procedure in which a machine is used to separate the plasma (the liquid part of the blood) from the blood cells. After the plasma is separated from the blood cells, the blood cells are mixed with a liquid to replace the plasma and are returned to the body. Plasma exchange is often done to remove extra antibodies, abnormal proteins, or other harmful substances from the blood. It may be used to treat certain types of blood disorders, autoimmune disorders, nervous system disorders, or other conditions. Also called plasmapheresis." *Plasma Exchange*, National Cancer Institute, https://www.cancer.gov/publications/dictionaries/cancer-terms/def/plasma-exchange (last visited Jan. 3, 2025).

[6] "Delta Brush" is defined as a "transient pattern[] characterized by a slow delta wave with superimposed fast activity" on an EEG. *Characteristics and Clinical Significant of Delta Brushes in the EEG of Premature Infants*, National Library of Medicine, https://pmc.ncbi.nlm.nih.gov/articles/PMC6123866/ (last visited Jan. 3, 2025).

[was] unclear," reiterating statements made by Petitioner's wife about her views of a vaccine relationship, but adding that only a single case report seemed to corroborate that possibility. *Id.* at 135.

Subsequent Treatment

Petitioner had a follow-up visit with another neurologist, Stacey Clardy, M.D., Ph.D., on November 24, 2020. Ex. 8 at 79. He was noted to be making progress in his recovery and exhibited no personality changes, but had limited recall of his hospitalization, and was apathetic and anxious. *Id.* Petitioner was still on the medication regimen previously set for him while hospitalized, and he reported that he had not had any additional seizures. *Id.* In assessing Petitioner and proposing future treatment, Dr. Clardy also commented on the possible explanation for Petitioner's illness, noting that although "[Petitioner's] TDaP vaccination may have played a role in [his] presentation given it's temporal relationship to symptom onset . . . ,[7] there is limited evidence/literature on this topic and given our current understanding of NMDA we do not believe this precipitated his illnesses. We would recommend staying up-to-date on all vaccinations including a yearly flu vaccine especially given his immunosuppressed state." *Id.* at 84.

In the early spring of 2021, Petitioner experienced a bit of a relapse, characterized by fatigue and declining cognitive function. Ex. 8 at 18–41. Dr. Abbatemarco in response ordered additional testing, but a repeat MRI and EEG yielded normal results. *Id.* Only the lumbar puncture (which otherwise revealed a normal white blood cell count and total protein) showed a somewhat-high NMDAR antibody titer of 1:5. *Id.* That May, Petitioner saw Dr. Clardy again, noting continued fatigue, apathy, and anxiety, but also that he was not experiencing seizures and otherwise was working. Ex. 13 at 101. Dr. Clardy deemed his presentation to constitute "a significant recovery" despite the residual symptoms, adding that greater improvement might take a year or more. *Id.* at 104.

The final records filed in this case are from the following year—March 2022, when Petitioner saw a different neurologist, Suzanne Liu, M.D. Ex. 13 at 4. Petitioner reported that his energy level and sleep were improved, his work was going well, and he was functioning normally, although his wife felt his personality and emotions had not recovered. *Id.* at 5. Further medication and follow-up with treaters were proposed. *Id.* at 8–9.

[7] Other treaters Petitioner saw around this time also acknowledged the temporal association between Petitioner's vaccination and his development of anti-NMDAR encephalitis. *See, e.g.*, Ex. 12 at 21 (documenting December 18, 2020, visit with seizure clinic neurologist Tawnya Constantino, M.D.).

II. Hearing Testimony

A. Petitioner's Expert — Dr. Kristen Babinski, M.D., Ph.D.

Dr. Babinski, a neurologist, prepared a single written report and testified at hearing. *See generally* Tr. at 5–55; Report, dated Apr. 9, 2023, filed as Ex. 18 (ECF No. 32-1) ("Babinski Rep.").

Dr. Babinski received her undergraduate degree in Biochemistry from Colgate University, her Ph.D. in Biochemistry from Duke University, followed by her medical degree from the University of North Carolina School of Medicine. *Curriculum Vitae*, filed as Ex. 19 (ECF No. 32-2) ("Babinski CV") at 1; Tr. at 5–6. She then completed an internship at Lenox Hill Hospital, followed by her residency in Neurology at New York University School of Medicine. Babinski CV at 1; Tr. at 6. Thereafter, Dr. Babinski completed a fellowship in Multiple Sclerosis (Neurology) at New York University School of Medicine. *Id.* She is currently the Director of Multiple Sclerosis and an Assistant Professor of Neurology at Tufts Medical Center, as well as a Neurohospitalist at MetroWest Medical Center. Babinski CV at 1–2; Tr. at 6. Over the course of her career, Dr. Babinski has treated and managed approximately 5,000 patients, a majority of whom suffer from a central nervous system disorder. Tr. at 7. She is board certified by the American Board of Neurology and Psychiatry. Babinski CV at 2. Dr. Babinksi admitted, however, that she has no direct expertise in the topic of immunology. Tr. at 39.

Dr. Babinksi began her testimony with a review of Petitioner's relevant medical history. *See generally* Tr. at 11–15. Based on those records (and more specifically the testing results and views of contemporaneous treaters), Dr. Babinski opined that Petitioner had been properly diagnosed with anti-NMDAR encephalitis, according to accepted diagnostic criteria. *Id.* at 16, 19–20.

Anti-NMDAR encephalitis, Dr. Babinski maintained, features neuroinflammation in the brain, and manifests with such symptoms as "seizures, confusion, agitation, speech disturbances, depressed consciousness, [and] autonomic instability." Tr. at 16, 18–19; J. Dalmau et al., *Paraneoplastic Anti-N-methyl-d-aspartate Receptor Encephalitis Associated with Ovarian Teratoma*, 61 Ann Neurol 25 (2007), filed as Ex. 57 (ECF No. 55-8). It is rare and largely impacts younger individuals, and is most often (*i.e.*, 60 percent of the time) associated with coincident neoplasms/tumors (which instigate the production of the antibodies responsible for the resulting encephalitic cross-reaction). Tr. at 17–18. Anti-NMDAR encephalitis can often begin with nonspecific symptoms such as headache or flu-like malaise, progressing (especially in children and young men) to seizures. *Id.* at 18–19. Testing for the specific antibodies to the NMDA receptor is the "only test" to confirm its existence. *Id.* at 19.

Dr. Babinski went on to explain anti-NMDAR encephalitis's pathophysiology. It is typically characterized as "an immune-mediated disease" propagated by its characteristic antibodies that attack a specific cortical neuron glutamate receptor. *Id.*; J. Dalmau et al., *An Update*

on Anti-NMDA Receptor Encephalitis for neurologists and Psychiatrists: Mechanisms and Models, 18 Lancet Neurol 1045, 1051 (2019), filed as Ex. 25 (ECF No. 32-8) (stating that "[p]athological and immunological evidence exists [showing] that NMDAR antibodies are synthesized systemically and within the CNS by antibody-producing cells that are able to cross the blood-brain barrier"). The treatments most effective against it, such as steroids or drug therapies that impact B cell antibody production, all underscore its inflammatory nature. Tr. at 27–28. The specific antibodies that drive the disease process (assuming they are generated systemically) cross the "blood brain barrier" ("BBB") that protects the central nervous system, binding to NMDAR and disrupting that receptor's function. *Id.* at 20–21, 26. This is what in turn causes the clinical symptoms associated with this form of encephalitis. *Id.* For the BBB to be permeable enough to cross requires the influence of factors sufficient to weaken it, such as cytokines (immune system messenger cells). *Id.* at 27.

Vaccination, Dr. Babinski proposed, has been reliably associated with anti-NMDAR encephalitis. Tr. at 22;[8] S. Martin et al., *Anti-NMDA Receptor Encephalitis and Vaccination: A Disproportionality Analysis*, 13 Front Pharmocol 1 (2022), filed as Ex. 36 (ECF No. 32-19) ("Martin"). Martin, for example, revealed that the World Health Organization's "pharmacovigilance database"[9] (akin to VAERS[10] reporting in the United States) had revealed a number of complaints of anti-NMDAR encephalitis after receipt of various vaccines—with a statistically significant association for the Tdap vaccine in particular. Tr. at 30–31; Martin at 1. Martin also considered a number of case reports underscoring the purported association. Tr. at 31. Dr. Babinski contrasted the evidentiary value of case reports with the purported difficulty of obtaining a more scientifically-reliable large-scale epidemiologic study, maintaining that such a study would not be feasible given the number of subjects required. Tr. at 33 (stating "you'd probably had to develop a study with a few hundred million patients, which would be an impossible task"). On cross, however, Dr. Babinski admitted that Martin's showing of an association did not involve the form of the Tdap vaccine at issue in this case. *Id.* at 51–52. And she accepted that its

[8] On cross-examination, Dr. Babinski admitted that the package insert for Tdap did not include anti-NMDAR encephalitis as a contemplated adverse event. Tr. at 41–44. Of course, the Program does not typically give great weight to package inserts, *pro* or *con* causation, since marketing licensure testing results are not meaningful evidence of causation either way. *Zumwalt v. Sec'y of Health & Hum. Servs.*, No. 16-994V, 2019 WL 1953739, at *17 (Fed. Cl. Spec. Mstr. Mar. 21, 2019)) (" …package inserts are generally afforded very little weight in Vaccine Program cases as proof of causation"), *mot. for review den'd*, 146 Fed. Cl. 525 (2019).

[9] "VigiBase" is "the WHO global database of adverse event reports for medicines and vaccines. It is the largest database of its kind in the world, with around 40 million reports of suspected adverse effects of medicines submitted by member countries of the WHO PIDM since 1968. It is continuously updated with incoming reports." *About VigiBase*, Uppsala Monitoring Centre, https://who-umc.org/vigibase/ (last visited Jan. 3, 2025).

[10] VAERS "is a national early warning system to detect possible safety problems in U.S.-licensed vaccines" and "accepts and analyzes reports of adverse events (possible side effects) after a person has received a vaccination." About VAERS, Vaccine Adverse Event Reporting System, https://vaers.hhs.gov/index.html (last visited Jan. 3, 2025).

findings were better at identifying a "signal" pointing toward a possible causal relationship than as constituting reliable proof of causation. *Id.* at 52.

Dr. Babinski further proposed that vaccines might be capable of stimulating existing, but nonspecific, T and B cell lymphocyte clones, encouraging the production of the relevant antibodies. Tr. at 23–24; D. Endres et al., *Psychiatric Presentation of Anti-NMDA Receptor Encephalitis*, 10 Frontiers Neurology 1, 7 (2019), filed as Ex. 27 (ECF No. 32-10) ("Endres"). Endres is a case report involving a 22-year-old female patient who developed an acute polymorphic psychotic episode three days after receiving a booster Tdap-IPV vaccination. Its authors suggested the vaccination physiologically led to stimulation of "[p]reexisting specific T- and B-lymphocyte clones . . . ., causing them to proliferate and leading to excessive antibody synthesis." Endres at 7. The relevant autoantibodies can also be detected in a meaningful minority number of healthy subjects, as well as individuals with distinguishable neurological conditions like Parkinson's disease. Tr. at 25–26; C. Hammer et al., *Neuropsychiatric Disease Relevance of Circulating Anti-NMDA Receptor Autoantibodies depends on Blood-Brain Barrier Integrity*, 19 Molecular Psychiatry 1 (2013), filed as Ex. 60 (ECF No. 55-11); L. Dahm et al., *Seroprevalence of Autoantibodies against Brain Antigens in Health and Disease*, 76 Ann Neurol 82 (2014), filed as Ex. 56 (ECF No. 55-7). It is theorized that an inflammatory trigger akin to what vaccination represents (due to its encouragement of cytokine production) would be sufficient to "restimulate the B cells" responsible for the anti-NMDAR antibodies. Tr. at 28. The same cytokines could (and at the same time) weaken the BBB sufficient for the autoantibodies to cross it, resulting in anti-NMDAR encephalitis. *Id.*

As an alternative mechanism, Dr. Babinski offered the possibility that microRNA[11] found in vaccines might also be a driver of anti-NMDAR encephalitis. Tr. at 29; H. Wang, *Anti-NMDA Receptor Encephalitis and Vaccination*, 18 Int J Mol Sci 1 (2017), filed as Ex. 40 (ECF No.49-21). In effect, Dr. Babinski maintained, certain microRNA particles can play a role in the receptor binding that result in NMDAR-encephalitis—and a "phylogenetic relationship" had been shown between one type of microRNA and "the viruses or bacteria specifically used for vaccinations," thus suggesting that receipt of a vaccine could lead to the illness in this alternative manner. Tr. at 29.

The same medical record that supported the anti-NMDAR encephalitis diagnosis also, in Dr. Babinski's view, corroborated the contention that Petitioner's disease was due to vaccination. That record showed that Petitioner had no pre-vaccination medical issues, developing disease only in the wake of vaccination. Tr. at 36. Contemporaneous treaters also seemed to take note of at least

[11] "microRNA" is defined as "any of a number of very small (20–22 nucleotides) RNA molecules that act as negative regulators of gene expression by binding to specific segments of messenger RNA, thus interfering with translation. MicroRNAs are important in the regulation of cellular development, differentiation, proliferation, apoptosis, and the stress response; abnormalities of miRNA expression are involved in the development of malignancies." *microRNA*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=31439&searchterm=microRNA (last visited Jan. 3, 2025).

the temporal relationship between vaccination and disease here, and there was no other explanation for Petitioner's illness (like a detected teratoma/tumor or known infectious cause). *Id.* at 36–37. And the timeframe in which Petitioner's initial, non-specific symptoms (myalgia, headaches) first appeared—a few days post-vaccination—was consistent with literature (again, largely case reports finding onset after vaccination). *Id.* at 37.

Besides setting forth her opinion, Dr. Babinski took some time to identify deficiencies she saw in the contentions of Respondent's expert (Dr. Stephen Hedrick). For example, she disputed the argument that data about predicted number of instances of anti-NMDAR encephalitis (when compared to the number of Tdap vaccines administered) was meaningful. Tr. at 34–36. She proposed that Dr. Hedrick had actually miscalculated the predicted incidence of Tdap vaccine-caused anti-NMDAR encephalitis—and when done correctly (especially given the rarity of the injury), the adverse events reporting data relied upon confirmed an association. *Id.* at 35. At the same time, Dr. Babinski accepted that VAERS reporting (from which these data were derived) was causally unreliable, at least since not all adverse events were likely reported. *Id.* at 36.[12]

On cross, Dr. Babinski admitted that she had mainly relied on case reports to bulwark her proposed mechanism of vaccines restimulating B-cells to produce autoantibodies (and hence in the context of an "underlying systemic autoimmune disease"), but that none of those reports directly involved the Tdap vaccine and anti-NMDAR encephalitis. Tr. at 46. Only one case report, Endres, involved an individual with preexisting antibodies to the NMDA receptors, but the antibodies were only detected in her blood and not her cerebrospinal fluid. *Id.* at 47. Thus, the pathogenic antibodies had to come from *outside* the BBB—but Dr. Babinski could not identify independent evidence establishing the capacity of the Tdap vaccine to cause weakening of the BBB in the first place. *Id.* at 48.

Dr. Babinski further acknowledged that it could not be ascertained from the record whether Petitioner himself had possessed the relevant antibodies before onset of his disease (and hence whether he was one of the low percentage of individuals in that category). Tr, at 48–49. Indeed—Petitioner had initially tested *negative* for the anti-NMDAR antibodies in his blood on October 28, 2020—after he had manifested initial symptoms. *Id.*; Ex. 6 at 37–39. But Dr. Babinski emphasized in response that a test performed two days later was positive—and in any event the titer levels overall were low, suggesting merely to her that "some of those tests might not have captured the antibodies," even though they were likely present (and thus the negative test result was not necessarily reliable). Tr. at 50; M. Guasp et al., *Clinical Features of Seronegative, but CSF Antibody-Positive, Anti-NMDA Receptor Encephalitis*, 3 Neurol. Neuroinflamm. 1 (2020), filed as

[12] Of course, Dr. Babinski relied on comparable, semi-self-reported data from WHO databases to support her contention that there was a Tdap vaccine association. *See generally* Martin. Petitioner has not demonstrated why one set of data could produce a reliable determination, whereas another could not, when both collections of data comparably involve reports of adverse events as opposed to confirmed events.

Ex. 59 (ECF No. 55-10) (concluding that "NMDAR antibodies are not detected in the serum of 15% of the patients with anti-NMDAR encephalitis").

Finally, when asked why Petitioner tested negative initially for the anti-NMDAR antibodies at the outset of his late-October hospitalization (when his disease process was likely already in swing), Dr. Babinski maintained it was possible damage from the antibodies was attributable to their production "intrathecally" (meaning within the space between the spinal cord and its covering membranes), with antibodies thereafter leaking systemically into the blood from there, but in titers too low to be measured. Tr. at 57.[13] (Of course, this would mean a disease process initiated *non-systemically* was driving the disease, inconsistent with Petitioner's theory of vaccine-caused antibodies generated in the periphery, and then finding their way into the central nervous system).

B. Respondent's Expert — Dr. Stephen Hendrick, Ph.D.[14]

Dr. Hedrick is a molecular biologist and chemist with expertise in immunology, and he offered a single written report along with his testimony. *See generally* Tr. at 61–130; Report, dated Feb. 29, 2024, filed as Ex. B (ECF No. 41-1) ("Hedrick Rep."). Although he accepted Petitioner's anti-NMDAR diagnosis, he denied the illness had been vaccine-caused (or could be).

Dr. Hedrick received his undergraduate degree in Biology and his Ph.D. in Molecular Biology and Biochemistry at the University of California, Irvine. *Curriculum Vitae*, filed as Ex. B-1 (ECF No. 41-2) ("Hedrick CV") at 1; Tr. at 61. Thereafter he completed a postdoctoral fellowship at the national Institutes of Health. *Id.*; Hedrick Rep. at 1. He is currently a distinguished Professor, Emeritus at the University of California, San Diego, having retired from the University in 2021. Tr. at 61; Hedrick Rep. at 1. Over the course of his career, Dr. Hedrick lectured on immunology, virology, and the history and biology of epidemic diseases, as well as an immunology course for the American Association of Immunology. Tr. at 62–63; Hedrick Rep. at 1. He has published approximately 178 peer-reviewed articles, with a particular focus on T cell immunology. *Id.*; Hedrick CV at 2–11. Dr. Hedrick, however, does not have the requisite training in central nervous system illnesses or epidemiology. Tr. at 104.

[13] In fact, Dr. Babinski acknowledged that some studies suggested that in the context of anti-NMDAR encephalitis, "antibody values are usually much higher in the CNS than they are in the serum" (Tr. at 58)—although this could mean *either* that it should be expected that blood tests for antibodies will be somewhat equivocal, *or* that the disease is more likely to originate in a non-systemic manner (making it less likely to be propagated by vaccine-driven pathology starting in the periphery).

[14] Respondent also filed an expert report from a neurologist, Dr. Jagannadha Avasarala, who provided an opinion largely specific to the propriety of the proposed diagnosis. *See* Report, dated Aug. 28, 2023, filed as Ex. A (ECF No. 35-1). But the parties do not dispute that Petitioner experienced anti-NMDAR encephalitis, and Respondent therefore did not call Dr. Avasarala to testify. As a result, I do not include in this Decision a summary of Dr. Avasarala's opinion.

Dr. Hedrick characterized anti-NMDAR encephalitis as a chronic autoimmune disease with certain known etiologies, such as tumors or other neoplasias, or "neurotropic viruses" capable of causing nerve inflammation, like the herpes virus. Tr. at 67, 128.[15] Many cases, however, have no known explanation. *Id.* at 68. In either case, antigens to the NMDA receptors would be generated and interfere with those same receptors. *Id.* In addition, certain T-helper cells would assist in the process of generating these antibodies, releasing cytokines that could also "loosen[]" the BBB. *Id.* If these processes become chronic, then encephalitis will occur. *Id.* Dr. Hedrick acknowledged that anti-NMDAR encephalitis is antibody-mediated and hence autoimmune in nature. *Id*. at 103, 107, 122. And he accepted that genetic susceptibility and environmental factors could play a role in its development. *Id.* at 107.

It made sense to Dr. Hedrick that Petitioner tested positive for the anti-NMDA antibodies. Indeed (and echoing Dr. Babinski) he noted that even healthy people have tested positive for them (although he explained that the presence of the antibodies was likely an artifact of the innate immune response, and could therefore be differentiated from the kinds of antibodies that would become pathogenic in driving anti-NMDAR encephalitis). Tr. at 71, 72. Dr. Hedrick also deemed these preexisting antibodies to have limited pathogenic potential, if any. *Id.* at 121.[16] But he maintained that scientific and medical literature showed these antibodies would more often than not be seen in the cerebrospinal fluid as opposed to the blood serum. *Id.* at 69. In fact, "the most pathogenic way of the disease progressing" would be where "secondary lymph node-like structures, organs, in the central nervous system produce an immune response" localized to the central nervous system. *Id.* at 70, 122 (noting the pathogenic character of these kinds of antibodies when found in the CSF); Hedrick Rep. at 3.

Vaccines, Dr. Hedrick maintained, like Tdap were unlikely to result in anti-NMDAR encephalitis.[17] He referenced his own review of VAERS data, which revealed only four reported

[15] This, Dr. Hedrick maintained, explained why Petitioner was immediately administered an antiviral drug during his October 26, 2020, Layton Hospital ED visit. Ex. 4 at 11.

[16] On cross-examination, Petitioner noted that some literature actually supported the pathogenic potential of these existing autoantibodies. Tr. at 121–22; E. Castillo-Gómez et al., *All Naturally Occurring Autoantibodies against the NMDA Receptor Subunit NR1 have Pathogenic Potential Irrespective of Epitope and Immunoglobulin Class*, 22 Mol Psychiatry 1776 (2017), filed as Ex. 55 (ECF No. 55-6). Dr. Hedrick in response admitted he had not reviewed that article, but he maintained that IgM-type antibodies (meaning ones recently generated) would not themselves likely be able to penetrate the BBB. Tr. at 122.

[17] Petitioner attempted on cross to establish Dr. Hedrick's bias against causation, and also to uncover an overly-strict view of what level of evidence would be sufficient to "prove" a causal relationship in this case. *See generally* Tr. at 110–13. I do not give great weight to this aspect of his testimony, however, since whether the legal standard for causation is met is ultimately the product of *a special master's analysis* and not merely derived from an expert's say-so (meaning the expert's personal standards for proof do not bind my determinations, or necessarily discredit the expert). As I have noted in other cases, while an expert may have views about what kinds or amounts of evidence are needed to convince them personally of a causal relationship, those views are not a substitute for the Program's "more likely than not" standard—and therefore experts are not discredited merely because they demand a higher level of proof than the Program does. At worst, restrictive views on such matters might lead to giving the expert's opinion less

cases of anti-NMDAR encephalitis after receipt of the Tdap vaccine, derived from thirty years of data. Tr. at 91–92.[18] The "signal" of a relationship should have shown up if there were even a possible association. *Id.* at 92, 97.[19] Dr. Hedrick acknowledged, however, that Respondent's own diagnostic expert (who did not testify at hearing) had seemed to concede that anti-NMDAR encephalitis could be vaccine-associated (or at least a factor increasing the risk of encephalitis), although Dr. Hedrick attributed this view to an overreliance on temporality with vaccination. *Id.* at 119–20.

Dr. Babinski's reference to Martin was deemed unpersuasive by Dr. Hedrick. He argued that the sample considered in Martin consisted of self-reported adverse events (equivalent to VAERS reports), and was therefore not likely representative of the total body of individuals with anti-NMDAR encephalitis, since the percentage of individuals considered to possess the relevant antibody did not conform to the expected proportion. Tr. at 93–95; Martin at 3. Martin's actual findings relevant to this claim (development of anti-NMDAR encephalitis with DTP-Polio vaccination occurring in only 8 out of 51 individuals) was also likely incorrect, or too small overall to be meaningful. Tr. at 94. And Martin's authors themselves had disclaimed that their findings had causal significance. Martin at 5. Dr. Hedrick allowed, however, that Martin noted the likely under-reporting of an anti-NMDAR diagnosis (although he maintained that this actually meant that the likelihood of vaccine-induced encephalitis was even lower). Tr. at 116–18.

There was alternative evidence also suggesting a non-vaccine-encephalitis relationship, in Dr. Hedrick's view. Tr. at 98–99; N. Klein et al., *Post-Marketing Safety Evaluation of a Tetanus Toxoid, Reduced Diphtheria Toxoid and 3-Component Acellular Pertussis Vaccine Administered to a Cohort of Adolescents in a United States Health maintenance Organization*, 29 Pediatric Infectious Disease Journal 613, 617 (2010), filed as Ex. A-4 (ECF No. 35-5) ("Klein"). Klein studied 13,427 individuals, aged ten to eighteen-years old, who received the Tdap vaccine as a part of their routine health care, but observed no increased risk for medically-attended neurologic events, medically-attended hematologic events, or allergic reactions, as well as no increased risk for new onset chronic illnesses. Klein at 617. On cross-examination, however, Dr. Hedrick admitted that Klein might not be sufficiently powered to detect the kind of rare occurrence that a vaccine injury would represent (especially with regard to the background rate for anti-NMDAR encephalitis in the general population). Tr. at 114–16.

---

weight (just as an overly-generous view of what is needed to establish causation—for example, an over-reliance on a vaccine's temporal relationship with an injury—would also be discounted).

[18] On cross-examination, Dr. Hedrick admitted that anti-NMDAR encephalitis was not described by medical science before 2007—meaning that earlier-in-time VAERS reports logically would *not have referenced it* as an adverse event. Tr. at 109. But he maintained that his conclusions about the limited VAERS reporting of an association were still reliable, since his search had focused merely on the claim of a post-vaccine "encephalitis." *Id.*

[19] Dr. Hedrick did also admit, however, that VAERS data itself is limited by its self-reporting quality, as well as the fact that not all medical providers are aware of this reporting system. Tr. at 96.

Dr. Hedrick also questioned whether Petitioner had offered sufficient reliable evidence to establish cytokines upregulated solely due to vaccination could weaken the BBB. Tr. at 74; W. Banks, *Blood-Brain Barrier Transport of Cytokines: A Mechanism for Neuropathology*, 11 Curr Pharm Des. 973 (2005), filed as Ex. 53 (ECF No. 55-4); J. Roth et al., *Signaling the Brain in Systemic Inflammation: Role of Sensory Circumventricular Organs*, 1 Front Biosci 290 (2004), filed as Ex. 67 (ECF No. 55-18). At most, certain cytokine-encouraging T cells (T helper cells in particular) might cause the production of the type of cytokines capable of allowing weakening of the BBB, but their activation would most likely occur in the presence of "acute viral infections." Tr. at 75. Otherwise, the BBB was not subject to being "broken down routinely on a systemic basis," and not in reaction to a "mild immune insult" attributable to a vaccination." *Id.* at 75–76.

More specifically, the Tdap vaccine was not *itself* likely to cause the production of the specific kinds of cytokines implicated in BBB breakdown. The T-helper cells most associated with BBB weaking, he maintained, would not be stimulated by the Tdap vaccine's aluminum adjuvant. Tr. at 76. Rather, the T-helper cell this adjuvant stimulates leads to the production of other cytokines "not thought to be inflammatory to break down the [BBB]." *Id.*; Hedrick Rep. at 3. The Tdap vaccine's actual antigenic components would not themselves cause this cytokine production. Tr. at 76. And Dr. Hedrick could identify no other reliable evidence supporting the conclusion that a routine immunization would likely lead to BBB breakdown. *Id.* at 84–85.

Dr. Hedrick agreed on cross-examination that pro-inflammatory cytokines were otherwise involved in BBB breakdown, but denied that a systemic source for them would result in this occurrence. Tr. at 125; A. Iwasaki, *Immune Regulation of Antibody Access to Neuronal Tissues*, 23 Trends Molecular Medicine 227 (2017), filed as Ex. B-15 (ECF No. 41-16). He also allowed that even if vaccines did not themselves directly stimulate production of the relevant cytokines, the cytokines they did stimulate could promote T-helper cells responsible for manufacture of other cytokines associated with BBB breakdown. Tr. at 126–27; H. Li et al., *Aluminum Hydroxide Adjuvants Activate Caspase-1 and Induce IL-1ß and IL-18 Release*, 178 J. Immunology 5271, 5275 (2007), filed as Ex. B-18 (ECF No. 41-19) (finding IL-1ß to specifically promote $CD4^+$ T-cell responses).

The same distinction in cytokines capable of BBB attack, Dr. Hedrick maintained, explained why other vaccines allegedly associated with anti-NMDAR encephalitis could not be analogized to the Tdap vaccine. For example, Dr. Babinski had proposed in her report that the inactivated polio virus vaccine had been so associated (pointing to Endres as evidence of it), but Dr. Hedrick noted that the immune response such a vaccine elicits could not be compared to the Tdap vaccine. Babinski Rep. at 5; Tr. at 78–79. Vaccines containing inactivated viral particles were fare more likely to prompt a more comprehensive innate response (since the viruses would be recognized by existing innate immune system complexes), resulting in the kind of inflammatory cytokine production proposed to be capable of BBB weakening. *Id.* (Dr. Hedrick later noted that Endres did not identify the presence of CSF antibodies in the individual case it discussed—making

it an outlier, since literature specific to anti-NMDAR encephalitis suggested the relevant antibodies would nearly *always* be identified in CSF if also present in the blood serum. *Id.* at 83–84).

Dr. Hedrick then set forth his understanding of Petitioner's causation theory and its different components. One version of the theory, he maintained, assumed that Petitioner had some "preexisting autoimmunity" systemwide that the Tdap vaccine had exacerbated or re-stimulated. Tr. at 65, 80. But he deemed testing for the antibody performed on Petitioner was too inconclusive to conclude that he had in fact likely possessed the anti-NMDAR antibody prior to vaccination. *Id.* at 73.

In addition, Dr. Hedrick maintained that literature offered to support this part of the causation theory was unpersuasive. One article, for example, did not involve the Tdap vaccine, but instead a straight comparison of certain antibody levels pre- and post-vaccination, without any baseline comparison to a non-vaccinated sample—thus not permitting the conclusion that the vaccine was responsible. E. Wiesik-Szewczyk et al., *Anti-Influenza Vaccination in Systemic Lupus Erythematosus Patients: An Analysis of Specific Humoral Response and Vaccination Safety*, 29 Clin Rheumatol 605 (2010), filed as Ex. 42 (ECF No. 49-23) ("Wiesik"). The studied sample in Wiesik, moreover, consisted of lupus patients already *known* to have "clearly dysregulated immune systems," as opposed to the Petitioner in this case. Tr. at 81; Wiesik at 607. And nothing in the record allowed the conclusion that Petitioner possessed a preexisting autoimmune disease in any event. Tr. at 88. Another such article involved a live attenuated vaccine not comparable to Tdap. *Id.* at 82; M. Farez & J. Correale, *Yellow Fever Vaccination and Increased Relapse Rate in Travelers with Multiple Sclerosis*, 68 Arch Neurol 1267 (2011), filed as Ex. 28 (ECF No. 49-9) ("Farez"). Such a vaccine would inherently have a greater immune-stimulative capacity. Tr. at 82. And Farez also involved a distinguishable disease as well. Farez at 1267.

Preexisting nonspecific immune cell activation was also, in Dr. Hedrick's view, not likely to lead to anti-NMDAR encephalitis after vaccination. He deemed this part of Dr. Babinski's theory to involve the concept of "bystander activation," in which an immune insult (such as infection—or arguably a vaccine) can cause other existing, nonspecific but autoreactive immune cells to activate secondarily to an immune stimulation, causing a process that can exacerbate the effects of the initial antigenic stimulus and thereby breaking tolerance to the antigen. Tr. at 86–87. While the subsequent process would be autoimmune, bystander activation would not involve *preexisting* autoimmune disease. *Id.* at 88. Dr. Hedrick acknowledged that a variety of cytokines could encourage secondary bystander activation, but stressed that "you have to have an ongoing memory response to the antigen you're talking about" for the process to occur. *Id.* at 128.

Dr. Hedrick deemed other aspects of Petitioner's theory lacking. For example, Petitioner's prior exposure to components of the Tdap vaccine (say, in receipt of the comparable "DTaP" version administered to children) did not make it more likely he would have suffered an aberrant reaction from immune memory, since individuals "get reimmunized all the time" but do not

routinely experience subsequent reactions after their immune system again confronts a previously-administered vaccine). *Id.* at 91; Hedrick Rep. at 4.

On cross-examination, Petitioner asked Dr. Hedrick about an article Respondent had filed establishing the pathogenic nature of the NMDAR antibodies via an animal model. Tr. at 122; Y. Ding et al., *Anti-NMDAR Encephalitis Induced in Mice by Active Immunization with a Peptide from the Amino-Terminal Domain of the GluN1 Subunit*, 18 J. Neuroinflammation 1 (2021), filed as Ex. B-8 (ECF No. 41-9) ("Ding"). The researchers in Ding had immunized mice directly with the relevant antibodies—but also used pertussis toxin to "stimulate the encephalitis." Tr. at 123; Ding at 2. Dr. Hedrick noted in response, however, that the results Ding's authors obtained had to be attributed more to the use of a special and particularly-strong adjuvant only used in experimental settings (although he also admitted the symptoms manifested after use of the pertussis toxin). Tr. at 124. And he stressed that the Tdap vaccine does not contain pertussis toxin, but instead a less immunogenic *toxoid* only. *Id.* at 131.

III. Procedural History

This claim was initiated in the summer of 2021. After completion of "pre-assignment review" (performed to ensure that sufficient records to analyze the claim have been filed), the matter was assigned to my docket a year later. Respondent filed his Rule 4(c) Report contesting entitlement in November 2022, and the parties then obtained and filed expert reports through March 2024. Prior to then, I set the case for hearing, to be held in August 2024. Trial proceeded as scheduled, and the claim is now ripe for resolution.

IV. Relevant Legal Standards

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

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[20] Petitioner advances a causation-in-fact claim, since anti-NMDAR encephalitis is not an injury listed on the Vaccine Injury Table.

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

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

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

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

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" with respect to the first *Althen* prong deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V,

2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

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

B. *Legal Standards Governing Factual Determinations*

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

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs.*, 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those

professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything

reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

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

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

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

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

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

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

D. *Consideration of Medical Literature*

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

ANALYSIS

I. Treatment of Anti-NMDAR Claims in the Vaccine Program

Several other special masters have been tasked with determining if anti-NMDAR encephalitis (or some form of autoimmune encephalitis comparable to it) is attributable to a vaccine. In the majority of instances, causation has not been found. *See, e.g.*, *Faulkenberry v. Sec'y of Health & Hum. Servs.*, No. 19-238V, 2024 WL 4892507 (Fed. Cl. Spec. Mstr. Nov. 1, 2024) (Moran, S.M.); *Baxter v. Sec'y of Health & Hum. Servs.*, No. 16-922V, 2024 WL 1912575 (Fed. Cl. Spec. Mstr. Mar. 28, 2024) (Oler, S.M.); *Baron v. Sec'y of Health & Hum. Servs.*, No. 14-341V, 2019 WL 2273484 (Fed. Cl. Spec. Mstr. Mar. 18, 2019) (Young, S.M.); *Lehner v. Sec'y of*

Health & Hum. Servs., No. 08-554V, 2015 WL 5443461 (Fed. Cl. Spec. Mstr. July 22, 2015) (Vowell, C.S.M.). In most such cases, the existence of post-vaccination anti-NMDAR encephalitis was not disputed, but the claimants could not demonstrate the vaccine was causal.

In *Lehner*, the petitioners argued that their minor daughter developed an autoimmune encephalopathy as a result of her receipt of the flu vaccine. *Lehner,* 2015 WL 5443461, at *1. Their causation theory involved two parts—first, that voltage-gated potassium channel ("VGKC") antibodies are pathogenic, or a marker for autoimmune neurologic disease; and second, that VGKC antibodies can be caused by an autoimmune response to the flu vaccine. *Id.* at *42. To bulwark the notion that the flu vaccine could cause these specific antibodies to occur, Petitioners relied on several items of medical literature—most of which, however, discussed different types of autoimmune encephalitis, or different vaccinations than at issue therein. *Id.* at *48. Moreover, the *Lehner* special master noted that none of the child's treating physicians attributed her receipt of the flu vaccine as causal of her purported autoimmune encephalopathy, but merely relied on a temporal relationship between the vaccination and her sudden loss of language and social skills. Thus, the special master determined that the petitioners had failed to meet their burden under *Althen*. *Id.* at *51.

In *Baron*, petitioners alleged that their minor daughter developed anti-NMDAR encephalitis following receipt of the Hepatitis A and flu vaccines. *Baron,* 2019 WL 2273484, at *1. These petitioners' proposed causation theory—"an increase in proinflammatory cytokines caused by the vaccinations resulted in a breakdown of [the] BBB," and thus NMDA receptor antibodies were created as a result of the vaccine-induced immune response—was found to have failed to meet *Althen* prong one, because there was insufficient evidence to support the notion that the vaccines at issue could cause the creation of the specific antibody necessary for anti-NMDAR encephalitis to occur. *Id.* at *17. Moreover, contentions regarding the overall role of pro-inflammatory cytokines in the breakdown of the BBB were not only too vague in delineating how the mechanism applied to the vaccinee specifically, but were unsupported by any medical literature demonstrating the relevant vaccines produce the necessary pro-inflammatory cytokines in sufficient levels to even permeate the BBB. *Id.* at *19.

The *Baxter* petitioners argued that a child's receipt of the MMR and/or varicella vaccines caused him to develop anti-NMDAR encephalitis via molecular mimicry. *Baxter,* 2024 WL 1912575, at *18. Petitioners' expert opined that amino acid peptide homology could be shown between components of the MMR and varicella vaccines and the NMDA receptor, meaning that the vaccines could cause the generation of antibodies that would also cross-react against the NMDA receptors due to similarity. *Id.* However, the special master found unpersuasive petitioners' proffered sequence homologies—noting that much of the medical literature offered in support of sequence homology between components of the relevant vaccinations and the NMDA

receptor did not involve anti-NMDAR encephalitis. *Id.* at *19–21. Thus, Petitioners could not meet their burden of proof on prong one. *Id.* at *21.

Finally, in *Faulkenberry*, a petitioner alleged her minor son had developed anti-NMDAR encephalitis after receiving the hepatitis A and/or flu vaccines. *Faulkenberry,* 2024 WL 4892507, at *1. The petitioner's expert relied on molecular mimicry as in *Baxter,* noting that it "is a reliable and accepted theory within the medical community that persuasively explains how the vaccines . . . more probably than not could cause the onset of anti-NMDARE." *Id.* at *9. However, the special master found the theory to be too general, and with little to no applicability to the vaccinee's case, as well as lacking any specific citations for how the vaccines at issue could lead to the development of anti-NMDAR encephalitis. *Id.* Absent any evidence suggesting that the hepatitis A and/or flu vaccine could generate the necessary antibodies to cause anti-NMDAR encephalitis, causation could not be preponderantly established. *Id.* at *16.

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

I deem Petitioner's showing in this case on the first *Althen* prong[21] to be inadequate, for reasons similar to why the special masters in the aforementioned cases denied entitlement. It simply has not been preponderantly demonstrated that the Tdap vaccine can likely produce the specific anti-NMDAR antibodies thought to cause this form of encephalitis. Dr. Babinski did not purport to show homology even though she invoked molecular mimicry as a mechanism, and while she displayed good command of the subject of anti-NMDAR encephalitis, her expertise did not extend to matters involving purportedly pathogenic immunologic responses that would result in such an injury. In addition, her reliance on case report associations (a weak form of causation proof) was not enough to show the specific vaccine at issue could result in production of the relevant autoantibodies. And overall, testimony from both experts about the nature of this form of encephalitis (which seems in some cases to more likely begin *within* the CNS) is not consistent with systemically-driven creation of autoantibodies finding their way to the relevant receptors.

Moreover, even if the Tdap vaccine *could* cause the production of the relevant antibodies in the periphery—*i.e.*, outside the central nervous system (due to the locus of vaccine administration in Petitioner's arm)—it has not been preponderantly established that cytokines upregulated by vaccination would also *likely* breach the BBB in the same way cytokines attributable to an active infection or tumor could. Dr. Hedrick persuasively established that the type of cytokines likely to increase BBB permeability were not likely vaccine-associated—and that in fact, vaccination generally was not the kind of immune stimulative event sufficient at all to raise this as an actual risk. Tr. at 75–76, 84–85, 125.

[21] Because petitioners must establish all three *Althen* prongs to be deemed entitled to compensation, the failure to meet any single prong is fatal to a claim—and therefore all three need not be addressed in an entitlement decision. *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 566 Fed. Appx. 976, 980 (Fed. Cir. 2014).

In many respects, Petitioner's theory amounts more to an effort to retroactively explain how a vaccine-caused process leading to anti-NMDAR encephalitis might occur than a scientifically-reliable theory for how it likely *does* occur. The manner of the proposed BBB-breakdown, for example, attempts to leverage what is known about vaccine-induced cytokine production into an after-the-fact theory—but without evidence suggesting that vaccination affirmatively is *likely* to promote BBB weakening. I often am confronted with similar theories, but find them wanting in their effort to conflate what is expected from vaccination with a pathologic process. *Osenbach v. Sec'y of Health & Hum. Servs.*, No. 16-419V, 2023 WL 5714809, at *29 (Fed. Cl. Spec. Mstr. Aug. 8, 2023), *appeal docketed* No. 2024-1663 (Fed. Cir. Apr. 8, 2024). Here, too, Petitioner's argument founders.

This is not a case where Respondent's expert strongly rebutted the entirety of Petitioner's evidentiary showing. Dr. Hedrick, for example, offered a number of arguments about predicted incidence of vaccine-caused anti-NMDAR encephalitis that were not particularly robust, or relied on statistical comparisons that did not in turn derive from sound epidemiologic evidence. But it is a *petitioner's* initial burden to make a prima facie showing of causation—and that showing must be preponderant and scientifically-medically reliable. Petitioner's showing was simply too general and vague to succeed. Indeed, it would arguably apply to *any* vaccination that preceded manifestation of encephalitic symptoms.

I also cannot find on this record that the Tdap vaccine likely "did cause" Petitioner's anti-NMDAR encephalitis (assuming the first *Althen* prong were met). There is little preponderant record evidence that Petitioner experienced any unusual levels of inflammation after his early October vaccination that would be consistent with the alleged BBB-breaching cytokine reaction, with over two weeks passing before Petitioner's October 20, 2020 seizure while driving. Then, Petitioner did not even immediately test positive for the relevant autoantibodies (based on blood testing—which would have picked up the existence of these antibodies had they been systemically generated) when he first sought hospitalization on October 28, 2020—now more than *three weeks* post-vaccination. If the Tdap vaccine had in fact been instigating the production of harmful autoantibodies, why were they not then detected—and after Petitioner had manifested a number of concerning symptoms? Dr. Babinski did not credibly explain these findings away.

I do acknowledge Petitioner can on this matter point to some instances of treater speculation that the temporal association with onset and vaccination was suspicious. But more holistic considerations of Petitioner's medical history, generated after his onset and subsequent hospitalization, discounted a vaccination cause. *See, e.g.*, Ex. 8 ay 79, 84 (Dr. Clardy' late-November 2020 assessment). I give that evidence greater weight.

CONCLUSION

Claimants must carry their burden of proof—here, by preponderantly establishing, via an offering of sufficient evidence, how the Tdap vaccine could cause anti-NMDAR encephalitis. This has not been accomplished in this case. Accordingly, I deny entitlement.

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.[22]

IT IS SO ORDERED.

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

[22] 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.