In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-758V

* *	Chief Special Master Corcoran
KELLEY KACZEROWSKI, *	
*	Filed: August 28, 2025
Petitioner, *	
*	
v. *	
*	
SECRETARY OF HEALTH AND *	
HUMAN SERVICES, *	
*	
Respondent. *	
*	
* *	

Leah V. Durant, Law Offices of Leah V. Durant, Washington, DC, for Petitioner.

Nina Y. Ren, U.S. Department of Justice, Washington, DC, Respondent.

ENTITLEMENT DECISION[1]

On January 15, 2021, Kelly Kaczerowski filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program).[2] Petitioner alleges that she suffered Guillain-Barré syndrome ("GBS") as a result of receiving the tetanus-diphtheria-acellular-pertussis ("Tdap") vaccine on July 25, 2019. Petition (ECF No. 1) at 1.

I determined that this matter could be fairly resolved via ruling on the record, and both sides filed briefs in support of their positions. Petitioner's Brief, filed November 15, 2024 (ECF No. 46) ("Br."); Respondent's Opposition, filed January 17, 2025 (ECF No. 47) ("Opp."); Petitioner's Reply, filed February 28, 2025 (ECF No. 49). The matter is now ripe for resolution. For the reasons set forth in more detail below, I hereby deny entitlement. Petitioner has not preponderantly established that the Tdap vaccine can cause GBS, or did so to her.

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

I. Factual Background

Petitioner was thirty-one years old and in her third trimester of pregnancy when she received a Tdap vaccine at a routine prenatal visit on July 25, 2019. Ex. 1 at 1; Ex. 7 at 232, 381. Her medical history included systemic lupus erythematous ("SLE") and psoriasis. Ex. 7 at 384. She had received Tdap doses on three prior occasions, with the most recent one administered on May 10, 2018 (during the third trimester of a prior pregnancy). *Id.* at 378.

There is no record evidence of any immediate reaction. However, on Monday, August 19, 2019 (twenty-six days post-vaccination), Petitioner went to an Obstetric Triage reporting high blood pressure, dizziness, and sweating over the weekend, adding that she had experienced over that timeframe a fever of 101.7˚F. Ex. 3 at 475. She had also been experiencing hip and back pain, treating it with over-the-counter pain medications. *Id.* Although a physical exam produced normal results, a comprehensive metabolic panel revealed, *inter alia*, elevated liver function test results and high lactate dehydrogenase, atypical lymphocytes relative, and protein/creatine urine ratio levels. *Id.* at 478–80. Petitioner was informed to increase her hydration and discharged home. *Id.* at 481.

A few days later (August 22, 2019), Ms. Kaczerowski went to her obstetrician ("OB") and reported headaches plus "some pain and tingling" in her arms, hands, and fingers. Ex. 7 at 411. She also noted the fever she had just experienced. *Id.* The OB was unable to propose an explanation for these symptoms, and recommended continued monitoring and follow-up. *Id.*

Petitioner returned to her OB on August 29, 2019. Ex. 3 at 532. She had now been experiencing intermittent dizziness for the past week (becoming constant the day before), which was "so bad last night she was unable to walk much [as a nurse] at work so her tech pushed her around in a wheelchair." *Id.* She again noted headaches beginning two weeks prior (mostly nonresponsive to Tylenol), shortness of breath (alleviated with an inhaler she had for her asthma), and intermittent nausea (which she had experienced throughout her pregnancy). *Id.* The OB did not perform a neurologic exam, and Petitioner's physical exam was normal. *Id.* at 534–35. But laboratory results revealed, *inter alia*, many elevated readings (elevated lactate dehydrogenase, uric acid, atypical lymphocytes relative, lymphocytes absolute, and urine protein concentration, among other things). *Id.* at 535–36. After receiving an analgesic, Petitioner's headache resolved within an hour, and she was diagnosed with headache/dizziness and instructed to avoid work. *Id.* at 537.

Ms. Kaczerowski went back to her OB on September 2, 2019. Ex. 3 at 619. She was now experiencing left-sided facial numbness, increased weakness (which caused her to fall forward onto her knees and hands), numbness and paresthesia of her extremities bilaterally, and headaches that had resolved with medication. *Id.*; *see also id.* at 612 (Petitioner recalled that her paresthesia began two weeks prior). Other than left maxillary numbness, Petitioner's neurologic exam was

normal, but she was admitted to the hospital's antepartum unit for observation and a neurology consultation. *Id.* at 619, 621–22.

The next day, Petitioner had a neurology consultation. Ex. 3 at 628. The neurologist documented that her headaches had begun in early August 2019, and that she had experienced a two-day high temperature (101–102˚F) beginning on August 17th, with numbness and tingling in her bilateral upper extremities beginning on August 19th, numbness in her bilateral lower extremities with joint pain and weakness beginning on August 26th, and left facial numbness beginning on September 2nd. *Id.* On exam, petitioner displayed limb ataxia (worse on the left), right facial droop and abnormal sensation on the left side, and a lack of deep tendon reflexes in the bilateral lower extremities. *Id.* at 628–29.

During her hospitalization, Petitioner underwent additional testing, and was eventually diagnosed with GBS. Ex. 3 at 629, 640, 645–46 (unremarkable brain and c-spine MRI and head and neck MRA); *id.* at 670–71 (lumbar puncture showed albuminocytological dissociation); *id.* at 677, 690–92 (electromyography/nerve conduction study "showed generalized primary demyelinating sensory motor polyneuropathy consistent with the clinical diagnosis of GBS"). She also had positive antinuclear antibodies ("ANA") at 1:160 in a speckled pattern; positive GM2 IgG-IgM, GD1a IgG-IgM, and GD1b IgG-IgM antibodies; and equivocal GM1 IgG-IgM antibodies. *Id.* at 1028, 1031–32. Her positive GM2 antibody titer levels were deemed "supportive but not specific for GBS," but the lab work comments also noted that "[t]he role of isolated anti-GM2 antibodies is unknown." *Id.* at 799, 802.

On September 5, 2019, Petitioner delivered her baby without any complications, and started a five-dose course of plasmapheresis. Ex. 3 at 706, 739. She gradually improved and was discharged on September 12, 2019. *Id.* at 762, 917, 928, 974, 1149. Petitioner's subsequent records relate to her recovery from GBS and treatment for unrelated conditions, but do not shed light on the entitlement issues to be resolved in this action, and are therefore not discussed further.

II. Expert Reports

A. *Petitioner's Expert – Dr. Carlo Tornatore*

Dr. Tornatore is a neurologist, and he prepared four written reports in this case. *See* Report, dated February 12, 2022, filed as Ex. 13 (ECF No. 18-1) ("First Tornatore Rep."); Report, dated April 3, 2023, filed as Ex. 25 (ECF No. 27-1) ("Second Tornatore Rep."); Report, filed November 21, 2023, as Ex. 39 (ECF No. 32-1) ("Third Tornatore Rep."); Report, filed May 28, 2024, as Ex. 55 (ECF No. 41-1) ("Fourth Tornatore Rep.").

Dr. Tornatore graduated from Cornell University with a Bachelor of Arts and Sciences in Neurobiology, and attended Georgetown University Medical Center, where he received a Master

of Science in Physiology. Curriculum Vitae, filed as Ex. 14 (ECF No. 18-2) ("Tornatore CV") at 2. He subsequently graduated from medical school at Georgetown University School of Medicine, completing a residency in the Department of Neurology at Georgetown University Hospital. *Id*. Dr. Tornatore also completed a fellowship in Molecular Virology at the National Institute of Health in Bethesda, Maryland. *Id*. He has published multiple articles addressing demyelinating disorders and their pathology. *Id*. at 8–13. Currently, Dr. Tornatore serves as a Professor and Chairman of the Department of Neurology at Georgetown University Medical Center, Chairman and Neurologist-in-Chief of the Department of Neurology at Medstar Georgetown University Hospital in Washington, D.C., and Medstar Health's Regional Director Neurology. *Id.* at 3. Currently, Dr. Tornatore follows approximately 373 patients with TM, and a few hundred others with varying demyelinating disorders. Tornatore CV at 3.

First Report

The initial section of Dr. Tornatore's first report featured a lengthy review of Petitioner's medical history, consistent with what is set forth above. First Tornatore Rep. at 2–17. He did, however, glean from it some specifics factors that he deemed especially relevant to the case. In particular, he noted that the timing of Petitioner's onset of GBS as occurring within her third pregnancy trimester—which he deemed "a period of relative immunosuppression," and hence suspicious for the development of an idiopathic autoimmune disease (absent some unusual environmental signal—here, vaccination). First Tornatore Rep. at 17.

Dr. Tornatore next provided a brief explanation of GBS. He deemed it an "autoimmune demyelinating neuropathy" impacting the peripheral nervous system, and understood to be instigated by antigens from either a viral/bacterial infection or (in some limited cases) vaccination. *Id.* at 18. An autoimmune attack on nerve myelin, due to cross-reactivity with antibodies produced in response to the stimulating antigens, would drive GBS. Its symptomatic features include extremity weakness and numbness, and could also feature facial weakness. *Id.* at 18.

That cross-reactivity would occur as a result of molecular mimicry between the presenting foreign antigens and self-tissue myelin structures. First Tornatore Rep. at 18–19; R. Hughes & D. Cornblath, *Guillain-Barré Syndrome,* 366 Lancet 1653 (2005), filed as Ex. 15 (ECF No. 20-1) ("Hughes & Cornblath"), at 1660. The general concept of mimicry, or shared homology between amino acid sequences in viral/bacterial antigens and host structures, was well accepted in science—along with the fact that this could result in antibodies produced in reaction to a foreign antigen to mistakenly attacking self. *Id.* at 19. But Dr. Tornatore acknowledged that medical science had not identified a cross-reactive antibody likely causal for *most* patients suffering from

the common form of GBS, acute inflammatory demyelinating polyneuropathy ("AIDP"). First Tornatore Rep. at 19; Hughes & Cornblath at 1659.[3]

Vaccines, Dr. Tornatore maintained, could also likely trigger an autoimmune process leading to GBS from molecular mimicry. First Tornatore Rep. at 19–20. It was known already that the swine flu vaccine had been associated with inflammatory demyelinating polyneuropathies like GBS. L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977,* 110 Am. J. Epid. 2:105 (1979), filed as Ex. 18 (ECF No. 20-4) ("Schonberger"). There was also some support for a relationship between GBS and tetanus-containing vaccines. J. Pollard & G. Selby, *Relapsing Neuropathy Due to Tetanus Toxoid: Report of a Case,* 37 J. Neurol. Sci. 113 (1978), filed as Ex. 19 (ECF No. 20-5) ("Pollard & Selby"). Pollard & Selby, however, is merely a 47 year-old case report—and it involved an individual who experienced three documented instances of relapse of neuropathic symptoms after receipt of a tetanus toxoid vaccine—a fact pattern distinguishable from this Petitioner's experience (and as noted herein, there are other reasons to question Pollard & Selby's probative value in this case).

In addition, Dr. Tornatore noted the existence of case reports and series observing a temporal association between different vaccines (other than the flu vaccine) and GBS. First Tornatore Rep. at 19–20; N. Souayah et al., *Guillain-Barré Syndrome After Vaccination in United States: Data from the Centers for Disease Control and Prevention/Food and Drug Administration Vaccine Adverse Event Reporting System (1990-2005),* 11 Neuromusc. Dis. 1:1 (2009), filed as Ex. 22 (ECF No. 20-8) ("Souayah"). Relying on VAERS[4] passive surveillance data, Souayah noted a number of other vaccines that might also be associated with GBS, although (a) a flu vaccine association was the most commonly-observed, and (b) tetanus-containing vaccines came in third place in term of how often post-vaccination GBS was reported. Souayah at 2 (Table 1), 3.

[3] Dr. Tornatore also briefly referenced another kind of molecular mechanism that could explain an autoimmune process prompted by a foreign antigen—"degeneracy" of T and B cells nonspecific to the presenting antigen. First Tornatore Rep. at 20; D. Mason, *A Very High Level of Crossreactivity is an Essential Feature of the T Cell Receptor,* 19 Immunol. Today 9:395 (Sept. 1998), filed as Ex. 23 (ECF No. 20-9) ("Mason"). But he did not provide details as to how this kind of nonspecific activation was likely vaccine-sparked, and overall relied more heavily on the standard kind of molecular mimicry mechanism (cross-reactivity driven by autoantibodies generated by antigenic mimics) usually featured in Program cases.

[4] The Vaccine Adverse Event Reporting System ("VAERS") is a national warning system designed to detect safety problems in U.S.-licensed vaccines. *See About VAERS*, VAERS, https://vaers.hhs.gov/about.html (last visited August 20, 2025). It is managed by both the CDC and the FDA. VAERS monitors and analyzes reports of vaccine related injuries and side effects from both healthcare professionals and individuals. But it has been observed in the Program that VAERS data is not particularly probative of causation, unless supplemented with other reliable evidence—since a VAERS report only establishes a temporal, post-vaccination occurrence, and does not independently confirm the reported adverse event either. *See also Vig v. Sec'y of Health & Human Servs.*, No. 01–198V, 2013 WL 6596683, at *17 (Fed. Cl. Spec. Mstr. Nov. 14, 2013) ("VAERS is a stocked pond, containing only reports of adverse events after vaccinations but no data about the number of vaccines administered or the occurrence of the same adverse event in individuals who have not been vaccinated").

Souayah's authors also acknowledged the limitations on reliance on uncorroborated VAERS reports. *Id.* at 5.

Dr. Tornatore also contended that Petitioner's onset (within four to five weeks of vaccination) was medically acceptable, when measured from the date she received the Tdap vaccine. First Tornatore Rep. at 20–21. To support this opinion, he relied on Schonberger—a study more than forty years old involving the swine flu vaccine, which had observed an increased risk of a demyelinating polyneuropathy "within the 5-week period after vaccination but extended out to 10 weeks." Schonberger at 112. He also referenced an article commenting on the epidemiology connecting the swine flu vaccine to GBS, noting the extent to which it confirmed the capacity of the vaccine to result in the production of the anti-ganglioside antibodies (known from research involving bacterial infections to cross-react via molecular mimicry and cause demyelination), adding that the article "may be relevant to the Dtap vaccine and [Petitioner]," since she had tested positive for the same antibodies. I. Nachamkin et al., *Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome,* 198 J. Infect. Dis. 15:226 (July 2008), filed as Ex. 24 (ECF No. 20-10) ("Nachamkin"), at 230 (swine flu vaccine induced antiganglioside antibodies in animal study). But Nachamkin does not address the capacity of the *Tdap vaccine* to cause the creation of these same antibodies.

Second Report

Dr. Tornatore devoted his second written report to attempting to rebut the points raised by Respondent's two experts—Drs. Dara Jamieson (a neurologist) and Marcela Pasetti (an immunologist).

First, Dr. Tornatore denied that he had proposed that autoimmune disease could *not* occur in pregnancy, but instead repeated his prior contention that because GBS in that context would be "distinctly unusual," it was reasonable to suspect some kind of external, aberrant immune stimulation (here, receipt of the Tdap vaccine) as causal. Second Tornatore Rep. at 2, 11. He emphasized that "the cellular and humoral elements during pregnancy are distinctly different than the non-pregnant state," although he acknowledged that "the extent is highly variable," as studies were inconsistent in what they observed. *Id.* at 12–13.

In so maintaining, Dr. Tornatore challenged the relevance of literature cited by Dr. Jamieson that was specific to neuromyelitis optica spectrum disorder ("NMOSD")—a central nervous system ("CNS") demyelinating condition. *Id.* Studies involving NMOSD conditions had in fact observed higher relapse rates in pregnant subjects (who already had been diagnosed with an NMOSD) during their third trimester (when compared to patients with multiple sclerosis)—but with all groups displaying an increased risk of relapse in the post-partum phase. *Id.* at 2–3; B. Bourre et al., *Neuromyelitis Optica and Pregnancy,* 78 Neurol. 875 (March 2012), filed as Ex. 26

(ECF No. 28-1) ("Bourre"), at 878. (Bourre also, however, emphasized that the immunologic impacts of pregnancy on different disease processes could vary by disease, and was not purely negative or positive. Bourre at 878). Other articles reached similar findings. Second Tornatore Rep. at 3–5 (citations omitted).

One of these studies had speculated that a tapering off of immunosuppressive medications prior to pregnancy in patients with a preexisting NMOSD might explain a higher relapse rate while pregnant (in the first trimester). E. Klawiter et al., *High Risk of Postpartum Relapses in Neuromyelitis Optica Spectrum Disorder,* 89 Neurol. 2238 (Nov. 2017), filed as Ex. 29 (ECF No. 28-4), at 2242. Dr. Tornatore deemed this significant, because it suggested that *natural* immunosuppression (which would be heightened as pregnancy advanced) would prevent onset of relapse in an autoimmune disease context. Second Tornatore Rep. at 5–8. Accordingly, Dr. Tornatore reasoned, pregnancy likely afforded some kind of protections against autoimmune diseases—highlighting the receipt of the Tdap vaccine as a notable likely trigger. *Id.* at 8.

Second, Dr. Tornatore questioned the proper interpretation of evidence relied upon by Dr. Jamieson to suggest that GBS incidence in pregnant women was no different than the general population. Q. Cheng et al., *Increased Incidence of Guillain-Barré Syndrome Postpartum,* 9 Epidem. 601, 603 (1998), filed as Ex. A Tab 6 (ECF No. 22-7) ("Cheng") ("[t]he risk for GBS during pregnancy appears to be similar to that in non-pregnant women"). In fact, Dr. Tornatore emphasized, Cheng recognized an increased GBS incidence within a month of delivery—which corroborated his argument that "pregnancy is indeed a state of immunosuppression." Second Tornatore Rep. at 8; Cheng at 604 (noting an increased risk immediately after delivery). This, plus Petitioner's prior exposure to the Tdap vaccine, made it likely that its receipt was responsible for triggering her GBS.

Next, Dr. Tornatore emphasized again his view that Petitioner's initial, pre-neurologic symptoms were likely early precursors of her GBS. Second Tornatore Rep. at 9–10. He characterized these symptoms (intermittent fevers, sweating, dizziness) as "dysautonomia," and noted that literature supported the conclusion that GBS often impacted the autonomic nervous system.[5] Z. Zaeem et al., *Autonomic Involvement in Guillain-Barré Syndrome: An Update*, 29 Clin. Autonom. Rs. 289 (2019), filed as Ex. 32 (ECF No. 28-7) ("Zaeem"). However, Zaeem seems to embrace the overall concept that these kinds of symptoms would be *secondary* to GBS's classic presenting symptoms, rather than occur first. Thus, Zaeem notes that because GBS patients "are often stable on initial presentation," it is critical that individuals diagnosed with GBS be monitored

[5] Autonomic Nervous System is defined as "the portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glandular epithelium; usually restricted to the two visceral efferent peripheral components, the sympathetic nervous system, and the parasympathetic nervous system." *Autonomic Nervous System*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111779&searchterm=autonomic+nervous+system (last visited August 20, 2025).

later for dysautonomic conditions that may subsequently arise. Zaeem at 289, 293. In fact, Zaeem reviews the kind of autonomic-related concerns that could *follow* GBS already caused by other factors, like a bacterial *Campylobacter jejuni* infection. *Id.* at 292 ("[t]he intimate supply of the GI track with autonomic parasympathetic and sympathetic nerves *can be targeted by GBS*," resulting in secondary concerns).

Dr. Tornatore nevertheless insisted that dysautonomic issues could precede neurologic symptoms—and even cause a fever, as Petitioner had experienced. He even deemed fever "quite common" in GBS. Second Tornatore Rep. at 16; T. Chakroborty et al., *Dysautonomia in Guillain-Barré Syndrome: Prevalence, Clinical Spectrum, and Outcomes,* 32 Neurocrit. Care 113 (2020), filed as Ex. 33 (ECF No. 28-8) ("Chakroborty"). Chakroborty is a retrospective study of 187 patients admitted to the Mayo Clinic over a 17-year period and who had been diagnosed with GBS. Dysautonomic symptoms or conditions were observed in approximately 40 percent of the sample, including fever (although the study ruled out fevers "attributable to superimposed infection," likely meaning infections leading to fever *prior* to onset of GBS-like neurologic symptoms). *Id.* at 114, 116–17. But Chakroborty is silent as to whether these symptoms *predated* neurologic symptoms—and again, its authors' concerns seem focused on dysautonomia as a secondary complication arising from GBS, rather than as a pre-neurologic presenting concern. *Id.* at 119 ("dysautonomia is seen in over one-third of patients *admitted for GBS*") (emphasis added).

Dr. Tornatore endeavored to defend his reliance on certain literature to establish a vaccine risk incidence for GBS—while attacking studies offered by Respondent's experts that purportedly undermined the vaccine-GBS relationship. Souayah, for example, had revealed a "striking spike in GBS" in a two-week, post-vaccination interval, and determined this risk to be applicable to vaccines other than the flu vaccine. Souayah at 2. He noted that Pollard & Selby stood as good evidence of the challenge-rechallenge risk posed by tetanus-containing vaccines. And he also proposed another mechanism for an autoimmune injury—the concept of the "fertile field," in which multiple overlapping factors (an environmental trigger, a person's age and immunologic health, and then the "anatomical location" in which pathogens interact with the host) can provoke an autoimmune response. Second Tornatore Rep. at 15–16; M. von Herrath et al., *Microorganisms and Autoimmunity: Making the Barren Field Fertile?* 1 Nature 151 (20023), filed as Ex. 37 (ECF No. 28-12) ("von Herrath").[6] This framework could be utilized in understanding how the Tdap vaccine could stimulate a susceptible person's immune system to in turn cause an autoimmune disease.

[6] Dr. Tornatore only glancingly referenced von Herrath, putting more emphasis on molecular mimicry as the explanatory mechanism for Tdap vaccine-caused GBS. I have in prior cases, however, noted that von Herrath has better applicability to circumstances in which an individual's exposure to a prior *infection* is deemed to have the capacity to encourage an autoimmune reaction to a second immune stimuli (occurring within the already-existing "fertile field")—*not* that vaccination itself accomplishes both steps in one fell swoop. *Howard v. Sec'y of Health & Hum. Servs*., No. 16-1592V, 2022 WL 4869354, at *25 (Fed. Cl. Spec. Mstr. Aug. 31, 2022), *mot. for review den'd,* 2023 WL 4117370 (Cl. Ct. May 18, 2023), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024).

Studies offered by Respondent to undermine a vaccine association, by contrast, were deemed unreliable by Dr. Tornatore. But in so arguing, he solely maintained that Respondent's experts had forgotten what Dr. Tornatore deemed "one of the fundamental principles of epistemology"—that "epidemiologic studies cannot rule out a rare event." Second Tornatore Rep. at 14. He thus did not in this report attempt to establish specific grounds for doubting the conclusions of any such studies that either expert had offered.

Otherwise, Dr. Tornatore deemed treater views of some likely association to reflect their sound judgment, even if they were unspecific analytically. Second Tornatore Rep. at 11. He felt that the evidence that Petitioner had previously experienced other autoimmune diseases only underscored her risk of incurring GBS. *Id.* at 14. He maintained that testing results establishing "atypical lymphocytosis" were not inconsistent with a short-lived vaccine reaction, referencing an old article that in turn cited a *different* (but unfiled) article over 50 years old to support the contention. Second Tornatore Rep. at 16–17; T. Shiftan & J. Menderlsohn, *The Circulating 'Atypical' Lymphocyte*, 9 Human Pathol. 1:51 (1978), filed as Ex. 38 (ECF No. 28-13), at 53. And he noted (without citation in support) that atypical lymphocytes had been observed in the context of autoimmune disease relapse—comparable to Petitioner's likely autoimmune predisposition. Second Tornatore Rep. at 17.

Third Report

The third written report prepared by Dr. Tornatore was submitted in the Fall of 2023—a few months before the claim's then-scheduled hearing date of February 2024. This report did not respond to arguments made by Respondent's experts, but instead reflected what Dr. Tornatore deemed "new information" bearing on the causation opinion he had offered. (It is not, however, facially evident what new findings are featured in this supplemental report. Of its nine cited reference articles or studies, three had already been submitted in the case; at least three were authored before 1990; and none of the remainder had been published later than 2018—*before* Petitioner even experienced her alleged vaccine injury).

This supplemental report begins with consideration of two articles from the 1980s, one of which had demonstrated in an animal study the existence of amino acid sequential homology between components of an influenza A virus and myelin basic protein ("MBP")[7]—and that the situs for the self mimic was significant as a target antigen for an experimentally-induced form of MS, "experimental autoimmune encephalitis" ("EAE"). Third Tornatore Rep. at 1–2; U. Jahnke et

[7] Myelin Basic Protein is "a basic protein (MW 18,000) that constitutes about 30 per cent of myelin proteins; elevated levels of MBP occur in acute exacerbation of multiple sclerosis and acute cerebral infarction." *Myelin Basic Protein,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100535&searchterm=myelin+basic+protein (last visited Aug. 20, 2025).

al., *Sequence Homology Between Certain Viral Proteins and Proteins Related to Encephalomyelitis and Neuritis,* 229 Science 282 (July 1985), filed as Ex. 41 (ECF No. 32-3) ("Jahnke"). Later studies found comparable homologies for peptide sequences from "different viral origins." Third Tornatore Rep. at 2–3; K. Wucherpfennig & J. Strominger, *Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activate Human T Cell Clones Specific for Myelin Basic Protein,* 80 Cell 695 (March 1995), filed as Ex. 42 (ECF No. 32-4) ("Wucherpfennig"), at 701 (MS patients). Dr. Tornatore thus concluded that it was "unlikely that a single viral peptide" would be the primary cause of at least the form of CNS autoimmune inflammatory diseases that these studies focused upon (but could be extrapolated to apply to other kinds of demyelinating conditions also involving MBP).

Next, Dr. Tornatore noted that one particular protein (comparable to a component of the influenza vaccine) was "almost identical in sequence" to MBP found in the CNS—allowing it to "stimulate the same T cell clones from patients with central demyelination." Third Tornatore Rep. at 3. And Dr. Tornatore referenced Pollard & Selby for this contention. Although that case report—questionable for many other evidentiary reasons—involved *peripheral* demyelination, Dr. Tornatore suggested that if the homologic sequence in the wild flu virus could lead to demyelinating injury, a similar sequence could also do so peripherally (presumably since in either case MBP would be implicated). *Id.*

Dr. Tornatore continued on with his analogy to findings specific to the wild flu virus or flu vaccine. Nachamkin, he stated, had noted that anti-ganglioside antibodies were likely drivers of GBS (via their targeting of ganglioside structures on the MBP), and had confirmed that the 1970s-era swine flu vaccine could induce these kinds of antibodies, via molecular mimicry leading to GBS. Third Tornatore Rep. at 5–6; Nachamkin at 5–6. A more recent study had provided more granular support for the conclusion that this occurred. Y. Li et al., *Identification and Characterization of Epitopes from Influenza A Virus Hemagglutinin that Induce Broadly Cross-Reactive Antibodies,* 41 Int'l. J. Molec. Med. 1673 (2018), filed as Ex. 45 (ECF No. 32-2).

Relying on the foregoing, Dr. Tornatore contended that molecular mimicry had equal reliability as the likely mechanism for driving Tdap vaccine-induced GBS. Third Tornatore Rep. at 6. But he again posited that this was so almost exclusively on the basis of studies there *were not at all specific to Tdap components*. *Id.* at 6–7; H. Inglis et al., *Antibody Responses to Peptides of Peripheral Nerve Myelin Proteins P0 and P2 in Patients with Inflammatory Demyelinating Neuropathy,* 78 J. Neurol. Neurosurg. Psychiatry 419 (2007), filed as Ex. 46 (ECF No. 32-8) (Sample of GBS patients displayed increased antibody reactivity to "P2" peripheral myelin protein). Dr. Tornatore nevertheless maintained that "significant homology" could be observed between amino acid peptides used in animal models to induce neuritis (more comparable to GBS

than EAE) and the tetanus toxin, relying on his own BLAST search[8] to substantiate the contention. Third Tornatore Rep. at 7–8. In effect, because "epitopes from tetanus toxin bear resemblance" to a peptide relevant to experimentally-induced forms of CNS and peripheral neuropathies, then it was likely the same kind of mimicry could occur, and drive GBS, in the context of a Tdap vaccine's administration. *Id.* at 8.

Fourth Report

Dr. Tornatore's final report was filed after the parties had determined that the matter should be decided on the papers, rather than at hearing. He accordingly endeavored to summarize the most salient points of his opinion, focusing in particular on evidence that he felt persuasively associated the Tdap vaccine itself with GBS.

First, Dr. Tornatore referenced a number of Government publications that he maintained recognized the association. A 1994 version of the Institute of Medicine's (the "IOM") publication on vaccine-associated adverse events, for example, expressly noted that there was evidence favoring a tetanus toxoid-GBS association. Fourth Tornatore Rep. at 1 (*citing Adverse Effects of Vaccines: Evidence and Causality*, Institute of Medicine (K. Stratton et al., eds. 1994), filed as Ex. 56 (ECF No. 42-1), 19 ("1994 IOM Rep."), at 109. Dr. Tornatore admitted that in an updated version of this report (published almost 20 years later), the IOM adopted a more equivocal view, noting that "[t]he evidence is inadequate to accept or reject a causal relationship between diphtheria toxoid–,tetanus toxoid–,or acellular pertussis–containing vaccines and GBS." *Adverse Effects of Vaccines: Evidence and Causality*, Institute of Medicine (K. Stratton et al., eds. 2012), filed as Ex. 57 (ECF No. 42-2) ("2012 IOM Rep."), at 558. But the 2012 IOM Report had generally observed that overall limited evidence existed pertinent to this subject (which informed their neutral position on causality), and thus expressly did not *reject* its prior views reflected in the earlier report. Fourth Tornatore Rep. at 2–3; 2012 IOM Rep. at 557 (noting only consideration of four epidemiologic studies, but rejecting three—including Souayah—on the grounds that they were based on VAERS data, and did not otherwise compare vaccinated populations against controls of unvaccinated individuals).

Other Governmental entities had more expressly deemed a causal association demonstrated, in Dr. Tornatore's view. Fourth Tornatore Rep. at 7–8. A Centers for Disease Control ("CDC") website, for example, notes the risk of GBS after vaccination (although the screenshot provided in Dr. Tornatore's report says nothing about Tdap specifically, let alone GBS

[8] According to its own website, the "Basic Local Alignment Search Tool" (BLAST) "finds regions of local similarity between sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance of matches. BLAST can be used to infer functional and evolutionary relationships between sequences as well as help identify members of gene families." https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Aug. 20, 2025). It is common in the Program for immunology experts to utilize BLAST searches when arguing about whether a vaccine's protein components mimic self-structures.

risk due to any of its wild infectious components). Fourth Tornatore Rep. at 7 (referencing Centers for Disease Control and Prevention, *Guillain-Barré Syndrome,* (May 15, 2024) https://www.cdc.gov/campylobacter/guillain-barre.html [https://web.archive.org/web/20240515063152/https://www.cdc.gov/campylobacter/guillain-barre.html]. The "Advisory Committee on Immunization Practices" ("ACIP"), moreover, specifically identified in a 2017 publication (as a "precaution") that treaters should take care in administering tetanus-containing vaccines to individuals who had previously experienced GBS within six weeks of receipt of a tetanus-containing vaccine. Fourth Tornatore Rep. at 8, *citing* E. Ezeanolue et al., "Best Practices Guidance of the Advisory Committee on Immunization Practices (ACIP)," *General Best Practice Guidelines for Immunization,* https://www.cdc.gov/vaccines/hcp/acip-recs/general-recs/downloads/general-recs.pdf (2017) (last visited Aug. 25, 2025), filed as Ex. 72 (ECF No. 42-17) ("Ezeanolue"), at 54.

Second, independent studies had also observed a Tdap vaccine—GBS association, Dr. Tornatore contended. Fourth Tornatore Rep. at 6–7. Some studies in particular suggested that there was risk of relapse after receipt of a tetanus-containing vaccine of a chronic inflammatory demyelinating polyneuropathy ("CIDP"), a peripheral, autoimmune neuropathic condition somewhat comparable to GBS. *See, e.g.*, J. Pritchard et al., *Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculopathy Following Immunization,* 73 J. Neurol. Neurosurg. Psych. 343 (2002), filed as Ex. 68 (ECF No. 42-13) ("Pritchard") (Retrospective study in which 1,114 patients with GBS or CIDP were surveyed about relapse of symptoms they experienced after receipt of vaccines; relapse after receipt of tetanus was second most-common-reported occurrence in patients); R. Hughes et al., *Immunization and Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculopathy,* Muscle & Nerve 1230 (Sept. 1996), filed as Ex. 71 (ECF No. 42-16) ("Hughes"), at 1231 (Case series report of 110 patients with GBS or CIDP over 12-year period, identifying only two instances of a temporal relationship to vaccination, but stressing (from an "overcautious" position) the risk of receiving a second vaccine if a person experienced a symptoms relapse within 12 weeks of a prior dose).[9]

Dr. Tornatore further highlighted case reports or case series articles observing the causal association he contends exists. Contrary to Dr. Pasetti's argument, for example, Dr. Tornatore deemed one study supportive of an increased incidence of GBS after vaccination. Fourth Tornatore Rep. at 3–4; W. Yih et al., *An Assessment of the Safety of Adolescent and Adult Tetanus-*

[9] In discussing the risk of CIDP relapse (as indirect proof that Tdap could trigger GBS directly), Dr. Tornatore also provided a more detailed review of Pollard & Selby, which he had cited in his initial report. Fourth Tornatore Rep. at 6 ("the authors were very clear that the tetanus toxoid resulted in multiple relapses of autoimmune peripheral neuropathy in this individual"). As explained below, however, Pollard & Selby is not only dated, but was a primary reason the IOM later determined the evidence associating tetanus-containing vaccines and GBS was *inadequate*—in part because Pollard & Selby involved CIDP, as well as the fact that it only showed "challenge-rechallenge" with respect to the unique experience of the subject patient. It is simply not a useful case report (and case reports *generally* merit little weight in assessing causation).

Diphtheria-Acellular Pertussis (Tdap) Vaccine, Using Active Surveillance for Adverse Events in the Vaccine Safety Datalink, 27 Vaccine 4257 (2009), filed as Ex. 60 (ECF No. 42-5) ("Yih"). Yih had expressly noted not only that the risk of Tdap vaccine adverse events was no higher than the risk from older formulations, but also that the study's power to detect relative risk was underpowered for GBS specifically. Yih at 4262. Dr. Tornatore nevertheless referenced a table in Yih which he maintained still established a heightened risk. Yih at 4260 (Table 3). In addition, a study surveying Canadian children over a 16-year period who received vaccines revealed 24 cases of GBS occurring within a month of vaccination—and nine of the 24 instances involved a tetanus-containing vaccine. K. Top et al., *Guillain-Barré Syndrome After Immunization in Canadian Children (1996-2012),* 34 Ped. Infect. Dis. J. 12:1411 (2015), filed as Ex. 61 (ECF No. 42-6) ("Top"), at 1412.

Dr. Tornatore further reference a number of smaller surveys or individual case reports observing a comparable association. *See, e.g*., K. Kongbunkiat et al., *Clinical Manifestations and Outcomes of Guillain-Barré Syndrome After Diphtheria and Tetanus Vaccine (dT) During a Diphtheria Outbreak in Thailand: A Case Series,* 19 Neurol. Asia 2:137 (2014), filed as Ex. 62 (ECF No. 42-7) ("Kongbunkiat") (discussing four cases of GBS that occurred after receipt of tetanus-containing vaccine; vaccinations were part of drive to vaccinate during a wild diphtheria bacterial infection outbreak; approximately 2.2 million individuals vaccinated); N. Newton & A. Janati, *Guillain-Barré Syndrome After Vaccination with Purified Tetanus Toxoid,* 80 South Med. J. 1053 (1987), filed as Ex. 64 (ECF No. 42-9) ("Newton & Janati") (single adult developed GBS within nine days of vaccination with tetanus); R. Bakshi & M. Graves, *Guillain-Barré Syndrome After Combined Tetanus-Diphtheria Toxoid Vaccination,* 147 J. Neurol. Sci 201 (1997), filed as Ex. 65 (ECF No. 42-10) ("Bakshi & Graves") (young adult developed GBS within four days of receipt of tetanus toxoid-containing vaccine); H. Ammar, *Guillain-Barré Syndrome After a Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccine: A Case Report,* 5 J. Med. Case Reps. 502 (2011), filed as Ex. 66 (ECF No. 42-11) ("Ammar") (recipient of Tdap vaccine develops GBS seven-plus days after vaccination).

Studies offered by Respondent's experts were, by contrast, challenged by Dr. Tornatore as unreliable or deficient methodologically. One such epidemiologic study, for example, was cited by Dr. Jamieson to undermine a causal relationship. *See generally* J. Tuttle et al., *The Risk of Guillain-Barré Syndrome After Tetanus Toxoid-Containing Vaccines in Adults and Children in the United States,* 87 Am. J. Public Health 2045 (1997), filed as Ex. A Tab 21 (ECF No. 22-22) ("Tuttle"). Relying on surveillance study data obtained in prior retrospective studies, Tuttle expressly found no enhanced risk of GBS within six weeks of receipt of tetanus toxoid-containing vaccines. Tuttle at 2047. But, Dr. Tornatore noted, the 2012 IOM Report had *rejected* Tuttle, reasoning that its reliance on this kind of passive surveillance data reduced its probative value when assessing whether the Tdap vaccine carried risk of GBS. Fourth Tornatore Rep. at 3; 2012

IOM Rep. at 557. (Of course, the 2012 IOM Report also rejected Souayah—which Dr. Tornatore cited in his first report as proof of a causal association—for the same reason).

Dr. Tornatore offered several additional points intended to bulwark or add detail to his previously-voiced opinions. For example, he directly maintained that the wild tetanus infection is in fact associated with GBS. Fourth Tornatore Rep. at 8–9. For support, however, he referenced statements from the 2012 IOM Report, noting only in a general sense that "the effects of natural infection" might constitute a "minor" type of evidence supporting certain mechanisms. 2012 IOM Rep. at 13. He also contended (citing another dated case report in support) that a specific bacterial infection other than tetanus had been observed to be temporally associated with a secondary case of GBS. W. Sonnabend et al., *Intestinal Toxicoinfection by Clostridium Botulinum Type F in an Adult – Case Associated with Guillain-Barré Syndrome,* 329 The Lancet 357 (1987), filed as Ex. 74 (ECF No. 42-19) ("Sonnabend"). But all Sonnabend notes (based on a single case report, moreover) is that a person might erroneously present with GBS-like symptoms that are later understood to be evidence of a form of botulism[10] capable of generating *in vivo*. Sonnabend at 360 ("[w]e propose that patients with *suspected* Guillain-Barré syndrome should be tested for BT in serum and Cl botulinum in stool") (emphasis added).

Dr. Tornatore also referenced a more recent article discussing how a tetanus infection could be *complicated* by GBS. J. Lee et al., *Generalized Tetanus Could be Complicated with Guillain-Barré Syndrome,* 48 J. Infect. Dis. 20 (2016), filed as Ex. 75 (ECF No. 42-20) ("Lee"). But Lee (actually only a letter to the editor of a journal) does not find one to be causal of the other. Lee at 20 (observing that an association between tetanus infection and GBS is "unknown," but noting (based on a small pool of 13 patients in Korea diagnosed with a tetanus infection) only two instances of GBS occurring in the context of treatment of the initial tetanus infection).

Finally, Dr. Tornatore endeavored to rebut the concept that (assuming some underlying genetic susceptibility to incur autoimmune diseases) Petitioner should have developed GBS after receipt of an earlier Tdap booster. Fourth Tornatore Rep. at 9–10. He stressed in response that Petitioner had received *two* booster doses within a span of 14 months—one in May 2018 (during an earlier pregnancy) and then the second in July 2019. *Id.* at 9. Antibodies to tetanus toxin have a long half-life, and research had therefore proposed that a person's protection to it after vaccination would last potentially for more than 30 years, with no need for a booster. E. Hammarlund et al., *Durability of Vaccine-Induced Immunity Against Tetanus and Diphtheria Toxins: A Cross-Sectional Analysis,* 62 Clin. Infect. Dis. 1: 1111 (May 2016), filed as Ex. 76 (ECF

[10] Botulism is defined as "any poisoning caused by Clostridium botulinum in the body; it produces a neurotoxin called botulinum toxin. . . . [S]pecifically, in humans, food poisoning with neurotoxicity resulting from the eating of spoiled food contaminated with Clostridium botulinum. Characteristics include central nervous system symptoms with motor disturbances; visual and oculomotor difficulties; and disturbances of secretion such as dryness of the mouth and pharynx with coughing." *Botulism*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=6698&searchterm=botulism (last visited Aug. 20, 2025).

No. 42-41) ("Hammarlund") at 1114–16 (there may be no need to receive Tdap boosters every 10 years to maintain protective immunity, given fact of long-lasting immunity). Accordingly, the "kinetics of tetanus toxoid antibodies" suggested to Dr. Tornatore that "her antibody level following the third 2019 Tdap vaccination had to be markedly higher than that following the 2018 vaccination," in turn increasing the odds that a susceptible person (presumably like the Petitioner) would experience a reaction. Fourth Tornatore Rep. at 10.

B. *Respondent's Experts*

1. Marcela Pasetti, Ph.D.– Dr. Pasetti is a pediatric immunologist, and she prepared three written reports in this case. Report, dated November 18, 2022, filed as Ex. C (ECF No. 24-1) ("First Pasetti Rep."); Report, dated January 10, 2024, filed as Ex. E (ECF No. 37-1) ("Second Pasetti Rep."); Report, dated August 27, 2024, filed as Ex. G (ECF No. 44-5) ("Third Pasetti Rep.").

Dr. Pasetti earned her bachelors in biochemistry at the University of Buenos Aires, where she then went on to earn her Ph.D. in immunology. Curriculum Vitae, filed as Ex. D (ECF No. 24-2) ("Pasetti CV"). Dr. Pasetti is not a medical doctor, but rather an academic. *See id.* Dr. Pasetti has twenty-five years of experience lecturing on and working with vaccine immunology. *Id.* at 1–2. Currently, Dr. Pasetti serves as a Professor in the Departments of Pediatrics and Microbiology and Immunology and faculty member of the Center for Vaccine Development and Global Health (CVD) at the University of Maryland (UMB), School of Medicine. *Id.* at 2. Her research centers on identifying biomarkers that predict successful immunization against infectious diseases and vaccine efficacy and new tools that can prevent and treat infection. First Pasetti Rep. at 1.

First Report

After a summary of Petitioner's medical history, Dr. Pasetti addressed what she proposed were flaws in Dr. Tornatore's causation opinion. She noted at the outset that purported treater support for an association was based more on the observed temporal association with vaccination than reasoned analysis of a causal relationship. First Pasetti Rep. at 3 (referencing comments made in treatment records by neurologist in October 2019 and February 2020 (Ex. 5 at 14 and Ex. 8 at 1). Dr. Pasetti thus deemed this unsupportive of a vaccine relationship.

Next, Dr. Pasetti questioned whether Petitioner's pregnancy (especially in the third trimester) created a milieu conducive for an autoimmune disease like GBS. She noted in this regard that views that pregnancy was immunosuppressive had been revisited by medical science in recent years. First Pasetti Rep. at 3; I. Vojtek et al., *Maternal Immunization: Where Are We Now and How to Move Forward?* 50 Annals. Med. 3:193 (2018), filed as Ex. C Tab 4 (ECF No. 34-4), at 203 ("[a] growing body of evidence suggests maternal immunization is a well-tolerated and efficient approach to provide protection to pregnant women, foetuses and new-borns against

vaccine-preventable diseases"). In Dr. Pasetti's view, current thinking on the topic had concluded that the "immune system is exceptionally modulated" during pregnancy, with "fine-tuned physiological adaptations" occurring throughout (and not just limited to the final stages). First Pasetti Rep. at 3.

As a result, Dr. Pasetti disputed Dr. Tornatore's contention that because of presumed third-trimester immune suppression, it was notable Petitioner had developed GBS at that time (thus allowing for the inference that immunization had played a role). In fact, she maintained, GBS did often occur later in pregnancy (although she admitted its incidence was rare)—and vaccination responses in pregnant women later in their pregnancies was robust, allowing them to confer immunity on the infants they carried and thereby prevent disease. R. Baxter et al., *Effectiveness of Vaccination During Pregnancy to Prevent Infant Pertussis,* 139 Pediatrics 5:1 (2017), filed as Ex. C Tab 6 (ECF No. 34-6) ("Baxter I"), at 2, 6 (maternal receipt of Tdap vaccine during pregnancy effective in protecting infants from pertussis infection, and superior in protection than "cocooning" method (vaccinating mother post-partum, while mother and newborn child are in close contact).

Accordingly, Dr. Pasetti argued, "any exposure event" during the third trimester would be enough to stimulate an immune response (and therefore the impact of receipt of a vaccine at that time was not especially notable). First Pasetti Rep. at 3. And studies of pregnant women specifically were unsupportive of the conclusion that the Tdap vaccine posed risk of autoimmune diseases like GBS (although Dr. Pasetti accepted that smaller sample size limited the weight the conclusions of such studies should be given). *See, e.g.*, H. Tseng et al., *Safety of Tetanus, Diphtheria, Acellular Pertussis (Tdap) Vaccination During Pregnancy,* 40 Vaccine 4503 (2022), filed as Ex. C Tab 18 (ECF No. 34-18) ("Tseng"), at 4503, 4507–08 (comparison of cohort of women receiving Tdap vaccine during third trimester versus unvaccinated women at same stage of pregnancy revealed no increases risk of a number of adverse events, with some focus on newborn outcomes; no consideration of risk of GBS or comparable neurologic injuries); L. Sukumaran et al., *Association of Tdap Vaccination with Acute Events and Adverse Birth Outcomes Among Pregnant Women with Prior Tetanus-Containing Immunizations,* 314 JAMA 15:1581 (2015), filed as Ex. C Tab 19 (ECF No. 34-19) ("Sukumaran"), at 1585 ("[w]e did not find any differences in acute events in the mothers or adverse birth outcomes in neonates when comparing women who were vaccinated with Tdap during pregnancy *regardless of the length of time since a prior tetanus-containing vaccine*") (emphasis added).

Dr. Pasetti also disputed the significance of an absence of any alternative explanation in the medical record for Petitioner's GBS. On the contrary, she maintained, there was evidence that Petitioner had a pre-vaccination history of two autoimmune conditions (SLE and psoriasis), thus suggesting some propensity for this kind of immune-mediated injury. In addition, in September 2019 Petitioner had tested positive for ANA—an antibody associated with SLE. First Pasetti Rep. at 7. GBS was a possible, secondary outcome attributable to such conditions. Petitioner also

experienced a high fever in mid-August 2019, not long before onset of her neurologic symptoms—and although Dr. Tornatore attempted to include these more immediate post-vaccination symptoms as part of Petitioner's GBS, Dr. Pasetti noted that a febrile episode is not compellingly associated with the syndrome. *Id.* S. Leonhard et al., *Diagnosis and Management of Guillain-Barré Syndrome in Ten Steps,* 15 Nature Reviews (Neurol.) 671 (2019), filed as Ex. C Tab 1 (ECF No. 34-1) ("Leonhard"), at 676 Box 1 (identifying "fever at onset" as one of the "Features that cast doubt on [GBS] diagnosis").

Dr. Pasetti similarly disagreed with how Dr. Tornatore interpreted other aspects of Petitioner's early presentation. For example, although Dr. Tornatore deemed the August 2019 WBC count measures as demonstrating the presence of normal levels, in fact the lab report in question deemed them high. Ex. 3 at 481. This would, she proposed, be consistent with an existing infection, like Epstein-Barr virus ("EBV"), although testing for that infection was never performed. First Pasetti Rep. at 7. And "atypical lymphocytes" observed in Petitioner's labs were attributed by Dr. Tornatore to be likely non-infectious, therefore consistent with vaccination—yet Dr. Pasetti characterized them as "not usually expected following vaccination," and in fact associated with infections like EBV. *Id;* W. van der Meer et al., *The Divergent Morphological Classification of Variant Lymphocytes in Blood Smears,* J. Clin. Pathol. 838 (2007), filed as Ex. C Tab 22 (ECF No. 34-22), at 838 (deeming atypical lymphocytes to reflect "a non-specific response to stress from a variety of disorders," but noting their association (if in large enough quantities) with mononucleosis—which is known to be caused often by an EBV infection).[11] This, plus other concerning lab results (like liver enzyme levels), suggested some greater and more damaging process was at work, that could include GBS as a secondary concern. First Pasetti Rep. at 7.

Dr. Pasetti then engaged in a discussion of what is known about the pathogenesis of GBS, and how that might relate to vaccination as a disease trigger. *See generally* First Pasetti Rep. at 4–6. She agreed with Dr. Tornatore that reliable scientific studies established that certain bacterial infections could result, via molecular mimicry, in the production of autoantibodies directed to ganglioside structures on nerve myelin, resulting in damage to the myelin characteristic of GBS. First Pasetti Rep. at 4. But the Tdap vaccine *itself* does not contain such microbial components, and "there is not evidence of concrete similarities or shared homology between Tdap antigen and human ganglioside antigens." *Id.* In fact, literature cited by Dr. Tornatore noted there were four scientific criteria for application of molecular mimicry as an explanatory mechanism for autoimmune diseases—but only one was met herein. N. Yuki, *Ganglioside Mimicry and Peripheral Nerve Disease,* 35 Muscle & Nerve 691 (2007), filed as Ex. 17 (ECF No. 20-3) ("Yuki") at 691–92 (listing the four criteria as "establishment of an epidemiological association between the infectious agent and the immune-mediated disease; identification of T cells or

[11] Epstein-Barr Virus is "a virus of the genus Lymphocryptovirus that causes infectious mononucleosis and is associated with Burkitt lymphoma and nasopharyngeal carcinoma." *Human Herpesvirus 4,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=80849 (last visited Aug. 26, 2025).

antibodies directed against the patient's target antigens; identification of microbial mimics of the target antigen; and reproduction of the disease in an animal model").

Dr. Tornatore had provided an alternative explanation for how different vaccines might result in GBS, but Dr. Pasetti disputed its reliability or persuasiveness. Dr. Tornatore proposed that certain immune cell receptors might recognize foreign antigens even in the absence of amino acid sequential or structural homology, via the concept of "degeneracy." First Pasetti Rep. at 5; Mason. But the literature he cited, Dr. Pasetti contended, was specific to T cells, and did not stand for the proposition that antibodies produced by *B cells* would cross-react due to degeneracy. *See, e.g.*, Mason at 403. Thus, this alternative mechanism "would not explain the *antibody-mediated* GBS form" relevant to this case (where vaccine-induced antibodies are the pathogenic drivers of disease). First Pasetti Rep. at 5. At most, Dr. Pasetti allowed for a "remote possibility" that non-specific activation of autoreactive immune cells could occur due to vaccination—but the same was possible after exposure to many foreign pathogens. *Id.* And this kind of nonspecific cross-reaction could occur as a secondary response to an existing autoimmune illness in any event (but in that case would not be the primary disease driver). *Id.*

In addition, Dr. Tornatore had cited a number of items of literature connecting *other* vaccines with GBS—but Dr. Pasetti deemed them irrelevant to Tdap. First Pasetti Rep. at 5. Some of them, moreover, relied on passive surveillance data (such as from VAERS) that would not have value in assessing causation. F. Varricchio et al., *Understanding Vaccine Safety Information From the Vaccine Adverse Event Reporting System,* 23 Pediatr. Infect. Dis. J. 287 (2004), filed as Ex. C Tab 15 (ECF No. 34-15), at 290 ("it is crucial to appreciate that data in VAERS cannot support a determination of whether a vaccine was more likely than not to have caused an adverse event"). At most, a consensus statement by an "international consortia" of scientists had confirmed the fact that (at least as of the time of its publication several years ago) *only* the swine flu-oriented version of the flu vaccine administered in the 1970s had been "convincingly linked" with GBS. Leonhard at 675. That view continued to prevail. N. Principi & S. Esposito, *Vaccine-Preventable Diseases, Vaccines and Guillain-Barré Syndrome,* 37 Vaccine 5544 (2019), filed as Ex. C Tab 16 (ECF No. 34-16), at 5546 (noting lack of support associating Tdap with GBS), 5548.

By contrast, the connection between the Tdap vaccine and GBS had been shown to be poorly substantiated. First Pasetti Rep. at 4–5. One study (relying in surveillance data) that included millions of suspects determined that there was no greater risk of incurring GBS within six weeks of receipt of a tetanus-containing vaccine. Tuttle at 3. Another, which relied on Vaccine Safety Datalink data, looked specifically at GBS as a Tdap risk on the basis of prior determinations in the Institute of Medicine's vaccine adverse event reports. Yih at 1. But based on a pool of subjects reflecting receipt of over 650,000 vaccine doses, no statistically significant relationship was detected. *Id.* at 5.

To corroborate Yih's findings for purposes of evaluating their epidemiologic reliability, a second analysis was performed based on a larger sample (two million doses)—and again, no increased risk within six weeks of receipt of the vaccine was observed. J. Nordin et al., *Tdap and GBS Letter,* 29 Vaccine 1122 (2011), filed as Ex. C Tab 10 (ECF No. 34-10) ("Nordin") (Authors of Yih re-considered their findings based on larger sample, but reached statistically-significant conclusions that the risk of GBS post-receipt of Tdap vaccine (within 42 days of vaccination) was not higher than the background incidence). A subsequent study involving a pool of 200,000 children produced the same results. M. Daley et al., *Safety of Diphtheria, Tetanus, Acellular Pertussis and Inactivated poliovirus (DTaP-IPV) Vaccine,* 32 Vaccine 3019 (2014), filed as Ex. C Tab 11 (ECF No. 34-11) ("Daley"), at 3023 Table 2 (GBS included among eight pre-specified adverse events).

In addition, an "extensive retrospective case-center study" involving all individuals within a massive California health maintenance organization over a 12-year period also found the risk due to the Tdap vaccine was exceedingly low. R. Baxter et al., *Lack of Association of Guillain-Barré Syndrome With Vaccinations,* 57 Clin. Infect. Dis. 2:197 (2013), filed as Ex. C Tab 12 (ECF No. 34-12) ("Baxter II"). Baxter II's authors confirmed GBS diagnoses for patients in a large, northern California integrated healthcare system, identifying 415 properly-diagnosed individuals and comparing their risk against a control population. Baxter II at 199. The incidence of GBS occurring in the wake of several different vaccines, including Tdap, was noted, with only 25 of the 415 subjects having received *any* such vaccine within six weeks of GBS onset. *Id.* at 200. Although Baxter II's authors noted that causation could not be excluded, they ultimately concluded no real post-vaccination risk was evident. *Id*. at 200, 203.

Dr. Pasetti concluded by reviewing Dr. Tornatore's contentions as to the timeframe in which Petitioner's GBS manifested, and whether it was medically acceptable when measured from the date of vaccination. She agreed that a purely-temporal association had been demonstrated (although she read the medical record to support an onset of August 12, 2019, rather than almost a week later). First Pasetti Rep. at 6. But Dr. Tornatore's arguments as to the propriety of post-vaccination onset were based upon animal studies involving GBS onset after the 1970s swine flu vaccine (and based on a supposition that bacterial exposure may have played a role in disease propagation)—but protested that *this* case involved a wholly-different vaccine. *Id.* Thus, this evidence was not helpful in evaluating how long it would take to induce GBS after receipt of the Tdap vaccine (let alone how that would occur). She also noted the relevance of Petitioner's past receipt of Tdap boosters (with one received in 2018, a year prior to the vaccination at issue). First Pasetti Rep. at 4. Given the likelihood of a "strong anamnestic antibody response" to another booster (due to immunologic memory), Petitioner's first neurologic symptoms should have been clinically evident much sooner than three-plus weeks after receipt of the vaccine. *Id.*

Second Report

Dr. Pasetti's second report was filed in the early winter of 2024—in reaction to Dr. Tornatore's third report, and at a time when this matter was still scheduled for trial. This report thus addresses the relatively-short report Dr. Tornatore had offered, which mostly sought to establish how the mechanism of molecular mimicry might be invoked to explain Tdap vaccine-instigated an autoimmune, antibody-driven attack on myelin.

Dr. Pasetti first reemphasized the obvious point that the flu and Tdap vaccines are not congruent. Second Pasetti Rep. at 1. In particular, "[t]he former consists of inactivated viruses (as applicable for this response) and the latter consists of purified bacterial proteins/toxoids." *Id.* As a result, "experimental observations derived from one cannot reliably be extrapolated to the other," and hence Dr. Pasetti gave little weight to invocation of influenza virus or vaccine studies from the 1980s. *Id.*

Dr. Pasetti also highlighted specific aspects of some of the articles Dr. Tornatore had identified, in an effort to rebut their significance or relevance. Jahnke's authors, for example, openly acknowledged the limits of a mere sequential demonstration of homology in establishing a likelihood of mimicking cross-reactivity. Second Pasetti Rep. at 1–2; Jahnke at 2. Wucherpfennig identified only one instance of peptide homology (and in an experimental context only), while also noting that autoimmune cross-reactivity could not be predicted on the basis of mere homology. Second Pasetti Rep. at 2; Wucherpfennig at 7. And this study involved MS, not GBS, nor did it predict or evaluate mimicry relating to any Tdap vaccine components. Second Pasetti Rep. at 2.

Dr. Tornatore's extensive discussion of a peptide found in a myelin protein, P2, and his effort to analogize studies involving it to the context of this case was criticized by Dr. Pasetti. *See generally* Second Pasetti Rep. at 2. She acknowledged that a significant number of GBS patients often tested positive for antibodies to P2. But she disputed Dr. Tornatore's argument that his own BLAST search showing homologies in amino acid sequences between tetanus toxin and P2 was reliable proof that a cross-reaction was likely based on receipt of a tetanus-containing vaccine. *Id.* at 2–3. She did not detect in his analysis evidence that the results could be deemed scientifically reliable, or that even enough sequential homology had been demonstrated. *Id.* at 2. In fact, "[t]he large gaps in the sequence alignments and multiple non-identical amino acids argue against substantial homology." *Id.*

In addition, Dr. Pasetti noted, homologous cross-reactivity often required "conformational" similarity, rather than just linear, sequential similarity, if it was to be a credible hypothesis for an autoimmune process. Second Pasetti Rep. at 2; A. Silvanovich et al., *The Value of Short Amino Acid Sequence Matches for Prediction of Protein Allergenicity,* 90 Toxicol. Sci. 1:252 (2005), filed as Ex. E Tab 2 (ECF No. 37-3), at 252, 256–58. Moreover, Dr. Tornatore's homology showing compared not the actual tetanus *toxoid* component included in Tdap, but the toxin—and

the inactivation process by which the more dangerous toxin would be rendered into a toxoid had a capacity to change the epitope entirely—meaning that "there is no guarantee the P2-shared amino acids remain intact." Second Pasetti Rep. at 2; Z. Long et al., *Effects of Detoxification Process on Toxicity and Foreign Protein of Tetanus Toxoid and Diphtheria Toxoid,* J. Chromatog. B 1207 (2022), filed as Ex. E. Tab 3) (ECF No. 37-4). It thus could not be assumed that the immune system's processing of tetanus toxoid contained in a Tdap vaccine would likely result in an autoimmune attack on myelin, simply on the basis of Dr. Tornatore's showing. Second Pasetti Rep. at 3.

Third Report

Dr. Pasetti prepared a final report in the late-summer of 2024, reacting to the contentions in Dr. Tornatore's last supplemental report. First, with respect to Dr. Tornatore's contentions about Government endorsement of a Tdap vaccine-GBS association, Dr. Pasetti noted that the 1994 IOM report conclusions relied solely on Pollard & Selby—but by the time of the 2012 IOM Report, it had been recognized that this case report applied to the more limited circumstances of a "spontaneous relapsing remitting neuropathy," seen as well in the context of existing and acute viral infections, and thus reducing any perceived causal relationship with tetanus toxoid-containing vaccines." Third Pasetti Rep. at 1; 2012 IOM Rep. at 95–96. And the versions of the tetanus-oriented vaccines being used in 1994 were not comparable to the Tdap vaccine, first licensed for U.S. use in 2005. Third Pasetti Rep. at 1. Dr. Pasetti thus gave more weight to the equivocal statements about causation set forth in the later iteration of the IOM Report (which she emphasized did not *support* causation, even if they did not also reject wholesale an association).

Similarly, Dr. Pasetti discounted the value of statements in CDC or ACIP-related publications advising against Tdap vaccine administration to individuals who had experienced GBS after a prior dose. As she noted, "the CDC and ACIP vaccine recommendations neither explicitly discuss nor endorse a causal direct relationship between tetanus containing vaccines and GBS," and instead merely noted precautions that would only apply in narrow and specific circumstances (GBS six weeks of the prior vaccination). Third Pasetti Rep. at 4, 5. A precaution, moreover, was not the same as a contraindication, which involved much greater risk—and in any event, the circumstances of the relevant precaution had no applicability to Petitioner, who never experienced GBS before. *Id.* at 5.

Dr. Pasetti went on to question Dr. Tornatore's argument that one of the Tdap vaccine's wild infectious analogs, tetanus, was also associated with GBS or other peripheral neuropathies. Third Pasetti Rep. at 5. Dr. Tornatore offered Sonnabend (a single case report more than 40 years old) or Lee for this proposition. But she pointed out that neither addressed the causation association he presumed, as well as the obvious point that the vaccine was designed to make a wild bacterial tetanus infection less likely. *Id.*

Meanwhile, more recent, large-scale studies found no association between Tdap and GBS. *See, e.g.*, Baxter II. Reacting to Dr. Tornatore's many literature references in his final report, Dr. Pasetti engaged in a point-by-point effort to refute their purported holdings. *See generally* Third Pasetti Rep. at 2–4. Yih, she noted, explicitly found no association between Tdap and any of the adverse events it considered, including GBS, despite Dr. Tornatore's construction of some of its data. *Id.* at 2; Yih at 5. At most, Yih as a study was a bit underpowered to deem it strong evidence against causation—and yet follow-up studies like Nordin (which included more reliably robust samples) confirmed Yih's findings, concluding that "there was no evidence that Tdap is associated with increased risk of GBS within 6 weeks of vaccination." Third Pasetti Rep. at 2; Nordin at 1.

Top was referenced by Dr. Tornatore to show some instances of post-Tdap vaccine GBS in a population of Canadian children, but Dr. Pasetti highlighted the fact that the majority of instances had at least one non-neurologic symptom, and 21 percent of them displayed evidence of infection—all of which supported some other causal agent. Third Pasetti Rep. at 2; Top at 1412. Top also acknowledged the risk of GBS posed by different kinds of infection. *See* Top at 1411. And Top itself concluded that any vaccine association was more likely temporally coincidental, especially when concurrent with an infection. *Id.* at 1413.

Kongbunkiat, Dr. Pasetti maintained, involved only four collected case reports and different versions of the tetanus-containing vaccines that could not credibly be analogized to the version of Tdap Petitioner received. Third Pasetti Rep. at 3; Kongbunkiat at 1. Another was a surveillance study that included none of the kinds of methodologic guardrails needed to yield a reliable causation determination, such as a comparative background rate for the incidence of GBS in the absence of vaccination or corroboration of the reported GBS cases that the data provided, as well as allowed for the possibility of a preexisting infection (which was reported in three of the ten cases noted by Dr. Tornatore). S. Chang et al., *U.S. Postlicensure Safety Surveillance for Adolescent and Adult Tetanus, Diphtheria and Acellular Pertussis Vaccines: 2005-2007,* 31 Vaccine 1447 (2013), filed as Ex. 63 (ECF No. 42-8) ("Chang"), at 1448. And he offered a number of case reports—some of which (like Bakshi & Graves) were *rejected* as good evidence of causation by the 2012 IOM Report. 2012 IOM Report at 93–94. The same kinds of criticisms were leveled by Dr. Pasetti at other items of literature referenced by Dr. Tornatore. *See generally* Third Pasetti Rep. at 4.

Petitioner's vaccination history did not make causation more likely, in Dr. Pasetti's view. She emphasized again that Petitioner had not developed GBS after receiving prior Tdap boosters—even once when pregnant, as here. Third Pasetti Rep. at 6. In response, Dr. Tornatore had referenced "the kinetics of tetanus antibodies," maintaining that the cumulative receipt of two boosters within 14 months (2018 to 2019) increased the impact (and risk of aberrant response) of vaccination, given the known half life of the relevant antibodies. But Dr. Pasetti deemed the

argument unclear when applied to instigation of GBS. *Id.* She also observed that in fact, the timeframe between Petitioner's 2009 booster and the one she received in 2018 rendered the *latter* the more-likely instance that would have featured over-robust immune response and concomitant dysregulation (although Petitioner did not then experience GBS). *Id.*

Dr. Pasetti concluded by again noting Petitioner's history of autoimmune disease prior to vaccination. And she stressed the medical record evidence of a likely infection (as indicated by fever) before onset of neurologic symptoms. Third Pasetti Rep. at 6. Vaccination was far less likely to have caused her GBS.

2. Dara Jamieson, M.D. – Dr. Jamieson is a neurologist and medical academic, and she prepared two written reports on Respondent's behalf. *See* Report, dated October 6, 2022, filed as Ex. A (ECF No. 22-1) ("First Jamieson Rep."); Report, dated August 28, 2024, filed as Ex. F (ECF No. 44-1) ("Second Jamieson Rep.").

Dr. Jamieson received her M.D. from the University of Pennsylvania. Curriculum Vitae, filed as Ex. B (ECF No. 22-23) ("Jamieson CV"). She followed her M.D. with a neurology residency and a cerebrovascular fellowship at the Hospital of the University of Pennsylvania. *Id.* at 1. She later taught and participated in clinical practice in Philadelphia at multiple facilities, including Temple University hospital; Thomas Jefferson University, University of Pennsylvania hospital; and Pennsylvania Hospital where she gained close to twenty years of experience. *Id.* at 1–2. Dr. Jamieson is currently a Neurology lecturer at Weill Cornell Medicine where she teaches medical students and residents. *Id.* at 1. She has authored twenty-two peer reviewed medical articles and contributed to approximately fifty other pieces of medical literature regarding neurology. *Id.* at 10–14.

First Report

Like Dr. Tornatore, Dr. Jamieson provided an overview of Petitioner's medical history before offering her reaction to the Petitioner's theory. First Jamieson Rep. at 2–8. She also provided a summary of GBS and its features consistent with Dr. Tornatore's report. *Id.* at 10–11. But Dr. Jamieson stressed that although certain kinds of antibodies were associated with different GBS variants, the mere existence of the antibodies found from blood tests was not only *not* mandated for a diagnosis, but that the confirmation of the existence of antibodies was not "reliably predictive of a different clinical course or response to treatment." *Id.* at 11 (*quoting* P. Bourque et al., *Autoimmune Peripheral Neuropathies,* 449 Clinica Chimica Acta 37 (2015), filed as Ex. A Tab 16 (ECF No. 22-17) ("Bourque"), at 41 (discussing CIDP variants). Indeed, with respect to AIDP, Bourque stated that "for the prototypical AIDP syndrome, the search for reliable antigenic biomarkers has not yielded consistent associations with high titers *and thus antibody screening is not part of accepted management strategies or diagnostic criteria.*" Bourque at 39 (emphasis added).

Much of Dr. Jamieson's report addressed aspects of Petitioner's medical history. For example, she disputed there was any significance in the fact that Petitioner's GBS onset began during late pregnancy, a timeframe Dr. Tornatore suggested was characterized by immune suppression (making it an unlikely backdrop for an uncontrolled autoimmune response—and, in Dr. Tornatore's view, increasing the likelihood that vaccination was the "x factor"). First Jamieson Rep. at 8–9. Dr. Jamieson noted that immune-mediated diseases often could begin in this timeframe, and cited one (neuromyelitis optica disorders) known to worsen in connection with pregnancy. Y. Mao-Draayer et al., *Neuromyelitis Optica Spectrum Disorders and Pregnancy: Therapeutic Considerations,* 16 Nature Reviews – Neurol. 154 (2020), filed as Ex. A Tab 1 (ECF No. 22-2), at 155, 166.

In addition, GBS's incidence was the same for pregnant women when contrasted with the general population. *See, e.g*., Cheng at 603; L. Chan et al., *Guillain-Barré Syndrome in Pregnancy,* 83 Acta Obstet. Gynecol. Scand. 83 (2004), filed as Ex. A Tab 3 (ECF No. 22-4). Pregnancy itself has been deemed a possible GBS trigger, and Dr. Jamieson contended that "the most likely time that GBS occurs during pregnancy is later, *especially* in the third trimester." First Jamieson Rep. at 9 (emphasis added); K. Sheikh, *Guillain-Barré Syndrome,* Am. Acad. Neurol. 1184 (Oct. 2020), filed as Ex. A Tab 9 (ECF No. 22-10) ("Sheikh"), at 1193.

Dr. Jamieson also noted that Petitioner's late-August 2019 neurologic symptoms were preceded by evidence of reported fevers plus sweating or dizziness. Although Dr. Tornatore proposed that these could be GBS-associated (as evidence of autonomic nervous system dysfunction), Dr. Jamieson contended such symptoms would in fact occur only "in the setting of severe disease," not before it. These initial symptoms (which were first reported on August 19, 2019—more than three weeks after vaccination) could thus not be persuasively linked to Petitioner's subsequent actual GBS onset a few days later. First Jamieson Rep. at 2–3, 7, 9.

Dr. Jamieson took issue with Dr. Tornatore's emphasis on evidence of abnormal lymphocyte levels, as found from blood testing performed at the end of August 2019. First Jamieson Rep. at 9–10. Dr. Tornatore maintained this could occur both in the context of a viral infection or post-vaccination, but Dr. Jamieson deemed it a nonspecific feature, and more likely attributable to a viral infection (especially given Petitioner's report of fever and sweats a week before). *Id.* And a treater note recording the *fact* of GBS occurring post-vaccination was not akin to a confirmation of a causal relationship. *Id.* at 10. GBS frequently was understood to reflect a post-infectious illness, even when a specific viral/bacterial trigger that caused the originating illness could not be identified. *Id* at 10–11*;* P. Donofrio, *Guillain-Barré Syndrome,* 23 Continuum 1295, 1295 (2017) filed as Ex. A Tab 11 (ECF No. 22-12) ("Donofrio").

Dr. Jamieson further disputed the reliability and quality of evidence offered to link the Tdap vaccine to GBS. She noted at the outset that Dr. Tornatore had offered a number of articles, like Schonberger, that did not involve the Tdap vaccine at all. First Jamieson Rep. at 8. Otherwise, he referenced case reports, but Dr. Jamieson did not deem them particularly deserving of evidentiary weight. Only one involved the Tdap vaccine itself, as opposed to earlier versions containing whole-cell pertussis. *See, e.g.*, Ammar. And case reports were generally known to be weak evidence of causation. First Jamieson Rep. at 12.

Dr. Tornatore had also cited some retrospective studies relying on passive surveillance reporting, such as VAERS data. First Jamieson Rep. at 12; Souayah at 1–2. But Souayah acknowledged that it did not observe a higher incidence of post-Tdap vaccination GBS when compared to the general population (and otherwise relied on what Dr. Jamieson deemed the "specious assumption" that a casual association with an environmental trigger and GBS could be assumed if the two occurred within a six-week timeframe). First Jamieson Rep. at 12. By contrast, some other studies (also utilizing surveillance data) did not observe a statistically significant incidence of Tdap-associated GBS. First Jamieson Rep. at 12–13; Tuttle at 3.

Second Report

After it had been determined that this matter would be resolved without hearing, Dr. Jamieson prepared a final written report—like Dr. Pasetti, mostly responding to the contentions in Dr. Tornatore's final report.

Dr. Jamieson began by addressing the purported governmental-related endorsements of Tdap causation that Dr. Tornatore had invoked. *See generally* Second Jamieson Rep. at 1–2, 3, 5–6. She accepted that the 1994 IOM Report had confirmed *some* association between tetanus toxoid-containing vaccines and GBS, but that it had not deemed the risk significant—or even ascertainable in magnitude. Second Jamieson Rep. at 2; 1994 IOM Report at 71 ("the conclusions are not based on controlled studies, no estimate of incidence or relative risk is available. It would seem to be low"). The subsequent, 2012 IOM Report had deemed several existing epidemiologic studies[12] weak evidence of causation, either because they relied on "data from passive surveillance systems," "lacked unvaccinated comparison populations," or "lacked an unvaccinated comparison population for the GBS analysis." 2012 IOM Report at 557. But it still did not conclude that sufficient evidence existed to make an affirmative causation finding.

Other statements from governmental medical/scientific committees about Tdap vaccine causation were also less probative, in Dr. Jamieson's estimation, than Dr. Tornatore contended.

[12] One of those rejected studies was Tuttle—relied upon by Dr. Jamieson, even in this second report. Second Jamieson Report at 2. Dr. Jamieson seemed to still find Tuttle useful, given its findings. But her individual acceptance of it does not adequately explain why it merits evidentiary weight herein, even though the IOM seems to have discarded it. I will accordingly not give Tuttle weight in my analysis.

Second Jamieson Rep. at 8–9. Ezeanolue, for example, relied on what appeared to be outdated sources for its precautions against the Tdap vaccine for those who previously experienced GBS after a prior dose. *Id.* at 8; Ezeanolue at 52. The ACIP recommendation (consistent with Ezeanolue) was outdated and said nothing about the first-line risk of incurring GBS due to receipt of the Tdap vaccine. Second Jamieson Rep. at 9.

Some independent literature, however, did undermine causation in Dr. Jamieson's estimation, despite Dr. Tornatore's efforts to show otherwise. A table from Yih, for example (published before the 2012 IOM Report—but not referenced therein (Second Jamieson Rep. at 2)), was cited by Dr. Tornatore to show an increased risk of GBS post-receipt of the Tdap vaccine. Yet in Dr. Jamieson's reading, the chart "clearly shows that the Tdap vaccine *did not increase* the risk of GBS. A log-likelihood ratio[13] of 0.38 for GBS is not significant when compared to a critical value of 3.12, as 0.38 is a much lower number than 3.12." (Second Jamieson Rep. at 2 (emphasis added) (referencing Yih Table 3). In fact, Yih's authors had expressly stated (in interpreting their own findings) that "[f]or all outcomes [including GBS], the expected number of events reached the upper limits without the log-likelihood ratio exceeding the critical value, providing no evidence that Tdap raises the risk of GBS above the background incidence or that it increases the risk of the other studied outcomes relative to Td." Yih at 6.

Dr. Jamieson did not accept evidence offered by Dr. Tornatore about the risks of CIDP flares after vaccination. In invoking those pieces of evidence, she maintained that Dr. Tornatore "conflates two distinct and different neurological diseases"—GBS and CIDP. Second Jamieson Rep. at 5. In her view, "[d]ata from . . . relapses of CIDP cannot be extrapolated to support an argument relevant to an initial bout of a monophasic disease." *Id*. at 6. Dr. Jamieson similarly disputed the value of evidence involving relapse of existing GBS, noting that Petitioner had not experienced GBS before the vaccination in question. *Id.* Articles like Pritchard, therefore, were of limited utility, since they were focused on relapse (while also relying on questionnaire data from responding patients, rather than confirmed instances of medically-diagnosed relapse). Pritchard at 1.

Reliance on case series was also an unpersuasive basis for a finding of causation, Dr. Jamieson contended. Top, for example, not only concluded risk of GBS due to infection was rare, but in the cases observed post-receipt of the Tdap vaccine, confounding factors (evidence of a prior infection) made it difficult to conclude vaccine causation—and Top's own authors expressly stated that the relationship observed in a few cases was most likely coincidental. Top at 13. Kongbukkiat featured individuals who had developed GBS in a much longer timeframe than relevant to this case, and did not reliably confirm the diagnoses either. Kongbukkiat at 2. And Chang relied on

[13] A log-likelihood ratio refers to the statistical measure that compares the likelihood of two models. *See* M. Kulldorff et al., *A Maximized Sequential Probability Ratio Test for Drug and Vaccine Safety Surveillance*, 30 Sequential Analysis 58, p. 3.1 (2011), https://www.tandfonline.com/doi/full/10.1080/07474946.2011.539924 (last visited Aug. 25, 2025).

passive surveillance data, concluding that causation should not be inferred from its findings as a result. Chang at 2.

Individual case reports were equally unreliable, according to Dr. Jamieson. Some could be called into question because the GBS diagnosis was unconfirmed or evidentiarily shaky. Second Jamieson Rep. at 5; Others specifically noted the uncommon nature of their finding, thus highlighting the likelihood that they were outliers rather than predictive of a real causal possibility. Bakshi & Graves at 2; Ammar at 2. Pollard & Selby was also criticized by Dr. Jamieson, both due to its outdated quality (more than 40 years old, and thus implicating formulations for the vaccine no longer administered), and because it said more about relapse risk after multiple doses of tetanus-containing vaccines than the initial risk of GBS after vaccine exposure. Second Jamieson Rep. at 6.

Dr. Jamieson ended her final report by rejecting Dr. Tornatore's argument that a wild bacterial tetanus infection could itself be associated with GBS. Second Jamieson Rep. at 9–10. Given that many different kinds of infections—viral and bacterial—were associated with GBS, Dr. Tornatore's contention was "not inconceivable," but he had failed to corroborate it with reliable independent evidence. Sonnabend, for example, involved a botulism infection caused by a distinguishable bacterium—not GBS due to a bacterial infection of any kind.[14] Sonnabend at 1. Lee involved a subset of individuals with diagnosed tetanus infections, some of whom may have developed it from "secondary hospital-acquired infections." Second Jamieson Rep. at 9; Lee at 2. (Lee in fact notes the importance of distinguishing GBS associated with tetanus toxoid and GBS associated with a tetanus infection). Of that smaller group (eight out of thirteen), two received GBS diagnoses that (on the basis of the evidence set forth in Lee) Dr. Jamieson deemed "thinly described"—and there were too many alternative explanations for their GBS (for example, the fact that the two patients were on a course of neuromuscular blockade medications). Second Jamieson Rep. at 10.

III. Procedural History

The Petition was filed in January of 2021. After that, Petitioner and Respondent filed the above-discussed expert reports. During that time, Respondent filed a joint 4(c) Report and Pre-hearing Brief challenging Petitioner's claim. The final expert report was filed in August of 2024, thereby concluding the process. I determined that this matter could be fairly resolved via ruling on the record and set a briefing schedule for resolution of the matter. The parties completed and filed their respective briefs between November of 2024 and February of 2025, making his matter ripe for resolution.

[14] Tetanus is attributed to a specific bacterium, *Clostridium tetani*. *Clostridium tetani,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=65648&searchterm=Clostridium+tetani (last visited Aug. 26, 2025).

IV. Parties' Arguments

Petitioner

Petitioner maintains that Dr. Tornatore has provided preponderant evidence of a biologically credible medical theory causally connecting the tetanus vaccine to GBS. Dr. Tornatore has opined that exposure to viral or bacterial antigens caused the development of GBS through the mechanism of molecular mimicry. Br. at 20. Molecular mimicry requires that these antigens share homology with host antigens that lead to an autoimmune response, which, as Dr. Tornatore points out, is seen in swine flu and tetanus vaccines. *Id.* at 20–21. The Petitioner parallels the swine flu and tetanus vaccines and their documented autoimmune responses, arguing that what is shown in one context reasonably applies elsewhere. *Id.* Dr. Tornatore also more specifically notes that peptides found in the flu vaccine and tetanus toxin are a molecular mimic with P1L/MBP epitopes and P2 epitopes respectfully. *Id.* at 22–23.

Further support for causation is found in a number of studies, articles, case reports, and governmental practice guidelines regarding vaccination. Br. at 23–24. The 1994 IOM report, for example, concluded that the Tdap vaccine could cause GBS through molecular mimicry or other biologic mechanisms. *Id.* at 24 (citing to 1994 IOM Rep. at 89). The 2012 IOM Committee, however, backed off from this likely because it employed a higher standard for finding causation than the 1994 IOM report. *Id.* at 25–26. And in any event, the 2012 IOM Report neither accepted nor rejected a causal relationship between the two, and does not otherwise replace the IOM's earlier determinations. *Id.* at 26–29 (citing to *Harris v. Sec'y of Health & Hum. Servs.,* No. 18-944V, 2023 WL 2583393, at *22 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (special master determining that the 2012 IOM report did not invalidate the 1994 IOM report)).

In addition, Dr. Tornatore offered other articles and case reports linking Tdap to GBS such as Yih, which found a causal relationship between the Tdap vaccine and developing GBS in "about 1 excess case" out of every 100,000 doses of the Tdap vaccine. Br. at 30 (discussing Yih). This exceedingly low number does not mean that there is no causal relationship between Tdap and GBS, but, rather, that the risk of this outcome is low. *Id.* And Petitioner accepted that case reports have historically been given less evidentiary weight in the Program, but asserts they have value when coupled with other evidence, and therefore cannot be reflexively disregarded. *Id*. at 31–32. The Petitioner also offered evidence from clinical studies showing that GBS has been associated with natural tetanus infections. *Id*. at 32–33. Specifically, in the Sonnabend and Lee articles, it was observed that patients with tetanus infections also had features or diagnoses of GBS. *Id.* (citing Sonnabend; Lee).

Equally important, Petitioner contends, is the evidence of public warnings concerning the risks of administering the Tdap vaccine to patients who have previously experienced post-

vaccination GBS. Br. at 33–34. These public warnings are especially significant because they have played a role in assisting with establishing prong one showings (under the causation standard set for causation claims by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005)) in previous vaccine cases before the Court. *See id.* (citing *Mohamad v. Sec's Health & Hum. Servs*., 2022 WL 711604 (Fed. Cl. Spec. Mstr. Jan. 27, 2022)). It is the combination of this evidence that Petitioner asserts has shown Dr. Tornatore's causal theory is shown by a preponderance of the evidence and has satisfied her *Althen* prong one.

Petitioner then contends that she has established that the Tdap vaccine "did cause" her injuries, and in a medically-acceptable timeframe (as required by *Althen* prongs two and three). *See* Br. at 37–41. Petitioner largely relies on temporal proximity to satisfy her burden for these two requirements. *See id.* For the second prong specifically, Dr. Tornatore argues that there was a logical sequence of cause and effect between Petitioner's GBS and Tdap vaccine because development of symptoms occurred after the vaccine—and when the Petitioner was in an immunocompromised state (due to her pregnancy)—and with no other possible explanation for her GBS in her medical records. *Id.* at 38–39.

In Dr. Tornatore's view, Petitioner's GBS was vaccine-related, and not post- or para-infectious, because her body was in a state that was primed for an abnormal immunologic event. Br. at 38. According to Dr. Tornatore, the Petitioner's immune system was suppressed due to her third trimester pregnancy. *Id*. In addition, receiving two Tdap vaccines roughly a year apart would create a stimulating effect that would lead to a high antibody count. *Id.* at 38–39. The compounding of these two factors left the Petitioner in an immunocompromised state, prime for a rare autoimmune reaction following a vaccination. *Id.*

Dr. Tornatore also asserts that there was no other logical cause for Petitioner's GBS identified on the record. Br. at 39. Even though Petitioner did report experiencing some symptoms of sweats and dizziness five to six days prior to developing the first neurologic symptoms of GBS, these could all be associated with GBS because there is no other recorded etiology for these symptoms listed in Petitioner's medical records. *Id.* Beyond that, Petitioner's treating providers took down that she was in a "usual state of health" before her vaccine. *Id.* at 40. Petitioner claims that this circumstantial evidence is sufficient to satisfy *Althen* prong two.

A similar argument is used to satisfy *Althen* prong three. *See* Br. at 40–41. Petitioner claims she has shown a medically acceptable temporal relationship between her Tdap vaccine and onset of her GBS symptoms. Petitioner's GBS symptoms began on August 22, 2019 when she experienced pain and tingling in her extremities. *Id.* at 41. This occurred twenty-eight days after receiving the Tdap vaccine. *Id.* at 40.

Petitioner also relies on both Dr. Tornatore's and Dr. Pasetti's statements to establish a medically appropriate time frame for onset. Both of these experts opine that onset of symptoms within twenty-eight days is appropriate. *See* Br. at 41 (quoting Dr. Pasetti believing the development of GBS within 3.5 weeks of vaccination is indicative of a temporal relationship between the two, and citing to Dr. Tornatore's expert report where he relayed that onset could be up to ten weeks after vaccination). Petitioner also analogizes GBS onset from the flu vaccine to the Tdap vaccine to support a medically appropriate time frame. Because, as Petitioner points out, the Secretary of Health and Human Services presumes causation of GBS from the flu vaccine if onset occurs within three to forty-two days. Br. at 41. Therefore, in Petitioner's view, her twenty-eight day GBS onset is also medically appropriate following a Tdap vaccine. *Id.*

On reply, Petitioner stresses that the relevant standard for her to prevail on *Althen* is not medical certainty, but instead only requires her to show enough evidence to show onset, causation, exacerbation, and temporal relationship by a preponderance of the evidence. Reply at 5. As a result, Dr. Tornatore's lack of evidence confirming the specific biologic mechanism of molecular mimicry between tetanus vaccine and GBS is not fatal to Petitioner's case, and is consistent with the outcome in *Mohamad*. *Id.* at 5, 9–11.

Petitioner further defends Dr. Tornatore's offered articles as sound scientific evidence that supports the biological, causal effect between the Tdap vaccine and GBS via molecular mimicry, and refutes that Respondent's offered evidence undermines her own. Reply at 3–9. While the Petitioner acknowledges that science today has not confirmed a definite link between tetanus and GBS, she reiterates the legal importance of circumstantial evidence. *Id.* at 9–15. Lastly, the Petitioner reasserts that her fever and symptoms before neurologic symptom onset and disease timeline are indicative of a vaccine-based etiology, as opined by her treating provider. *Id.* Treating provider statements, she stresses, carry great weight and cannot be rejected so easily by the Respondent. *Id.* at 16–17.

Respondent

Respondent's opposition brief argues that the Petitioner has failed her requisite showing on all three *Althen* prongs. Opp. at 2.

Respondent begins by arguing that Dr. Tornatore's molecular mimicry theory is inadequate. Respondent maintains Dr. Tornatore has engaged in hand waiving and chanting of the phrase "molecular mimicry," but without specifically showing a reliable and persuasive link between the Tdap vaccine and GBS. Opp. at 10–11. As the authors in Yuki noted, molecular mimicry is only a persuasively-established mechanistic explanation for a specific autoimmune disease process if four criteria are met. *Id.* at 13 (quoting Yuki at 1–2). But Dr. Tornatore has failed to substantiate them all. In particular, he has not offered reliable, scientific evidence of a true molecular mimic. *Id.* at 11–12. Even Dr. Tornatore's BLAST search results are themselves

insufficient, as Dr. Pasetti only observed a few isolated amino acids and large gaps in sequence alignment—nothing that would indicate a partial sequence homology. *See id.* at 12.

Next, Respondent contends that the available epidemiologic evidence demonstrates that the Tdap vaccine is not likely associated with GBS. Opp. at 13. Studies like Baxter II and Daley looked at the correlation between a number of vaccines (including tetanus toxoid-containing vaccines) and GBS, but none of them were able to find an associated risk. *Id.* at 14 (*citing* Baxter II at 200–02). Even the Yih and Souayah studies that Dr. Tornatore relies upon could not conclusively establish a link between the Tdap vaccine and GBS. *Id*. at 15–16. Respondent concedes that Souayah *suggests* an association between Tdap and GBS, but argues that the study cannot be given much evidentiary weight because it relies on VAERS data. *Id.* at 15 (citing Souayah at 5). The overwhelming and most reliable epidemiologic evidence argues against general causation. *Id*. at 16.

Respondent then attacks Petitioner's case reports and medical literature offered in this case, arguing that they do not advance Dr. Tornatore's causation theory. Opp. at 19. Respondent first takes aim at the Petitioner's use of case reports—which have historically been given less evidentiary weight in the Program, since they rely largely on timing to establish a cause-and-effect relationship. *Id.* (citing First Jamieson Rep. at 12). Also, the circumstances of the case report subjects are distinguishable from Petitioner's own history. *Id*. at 19–20 (discussing Top (which involved infections and other conditions that contradict an association between vaccination and GBS); Kongbunkiat (assessing Tdap vaccines formulated and manufactured differently than the Boostrix Tdap vaccine the petitioner received); Chang (finding three of the four instances of Tdap/GBS cases had a preceding infection); Newton & Janati (assessing a male patient with an unclear neurological disorder; Ammar (the patient fell from a second story roof before receiving the Tdap vaccination)).

Next, Respondent questions the value of Dr. Tornatore's reliance on literature pertaining to vaccines and diseases *other* than Tdap and GBS. Opp. at 20–22. Respondent notes that Dr. Tornatore uses the flu vaccine and CIDP, for example, as parallels for the Tdap vaccine and GBS. *Id.* at 21–22. And the flu and Tdap vaccines formulation and manufacturing differences are too many to deem them comparable. *Id.* at 21 (citing First Pasetti Rep. at 6; Second Pasetti Rep. at 1). Similarly, as Dr. Jamieson established, GBS and CIDP are not interchangeable diseases, and, therefore, studies using data from one "cannot be extrapolated to support an argument relevant" to the other. *Id.* at 21 (citing at Second Jamieson Rep. at 5–6).

Further, Respondent argues that the 1994 IOM Report was unpersuasive. Not only did it rely upon the Pollard & Selby study for its findings (which involved CIDP), but it was superseded by the 2012 IOM Report, and additional reliable epidemiologic evidence has been made available since the reports that only underscore the absence of a causal association. Opp. at 22–24. Lastly,

Respondent notes that GBS has some recognized causes, but it is mostly idiopathic, and likely was idiopathic in this case. *Id.* at 25. Even if a trigger for GBS was necessary, Dr. Tornatore has not offered sufficient evidence in support of his contention that a wild tetanus infection is associated with GBS. *Id*. at 25–26.

Respondent further maintains that Petitioner has fallen short of satisfying her *Althen* prong two showing, because it relies solely on *post hoc ergo propter hoc* logic while not grappling with evidence of other potential GBS causes evident in the record. *See* Opp. at 26. In particular, Respondent experienced a fever in the days leading up to the first neurologic symptom of her GBS, and tested positive for atypical lymphocytes—evidence of an infectious process. *Id.* at 27. And Dr. Jamieson noted association of GM2 IgG-IgM positivity with cytomegalovirus infection, which could explain Petitioner's GBS as well. *Id.* And the Petitioner received two Tdap vaccines before the one at issue, but experienced no reaction. *Id.* at 13.

Similarly, Respondent discounted the significance of Petitioner's pregnancy as impacting her GBS. He argues that pregnancy-specific studies do not show a different incidence of pregnant women developing GBS relative to the general population, thus undermining Dr. Tornatore's assertion that the Petitioner was in a state of immunosuppression and was susceptible to an adverse autoimmune reaction from the Tdap vaccine via molecular mimicry. Opp. at 17–19 (citing Cheng; Tseng; Sukumaran).

Otherwise, Petitioner relied mainly on the temporal association between Petitioner's Tdap vaccine and GBS onset, that in turn relies mainly on a treating provider's conclusory statements. Opp. at 26–27. Such statements, Respondent contends, lack reason and evidence beyond temporality, which is insufficient to satisfy *Althen* prong two. *Id.* at 27.

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 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 ex rel. 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).[15] There is no Table claim for GBS caused by a tetanus-containing vaccine.

[15] 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

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 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*, 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 even 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

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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).

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 and remanded*, 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. 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*, 993 F.2d at 1528.

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary

evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

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

B. *Legal Standards Governing Factual Determinations*

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

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical

records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

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

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

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the

accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

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

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

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

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

determine whether expert testimony offered is reliable and/or persuasive.

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

D. *Consideration of Medical Literature*

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

E. *Determination to Resolve Case without a Hearing*

I have opted to decide entitlement in this case based on written submissions and evidentiary filings, including the expert reports filed by each side. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers rather than via evidentiary hearing, where (in the exercise of their discretion) they conclude that the former means of adjudication will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d).

The choice to do so has been affirmed on appeal. *See D'Toile v. Sec'y of Health & Human Servs.*, No. 15-85V, 2018 WL 1750619, at *2 (Fed. Cir. Apr. 12, 2018); *see also Hooker v. Sec'y of Health & Human 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 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. *See Hovey v. Sec'y of Health & Human Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417.

ANALYSIS

I. Overview of GBS and its Treatment in Prior Program Cases

GBS has been defined as an acute, monophasic peripheral neuropathy involving rapidly-progressive and ascending weakness and paralysis, and which is thought to have an autoimmune mechanism. Leonhard at 1–2; Sheikh at 4.

GBS is usually believed to present with "pain, numbness, paraesthesia, or weakness in the limbs." Hughes & Cornblath at 1661; J. Griffin & K. Sheikh, "The Guillain-Barré Syndrome," in *2 Peripheral Neuropathy* 2197 (P. Dyck & P. Thomas, eds., 4th ed. 2005) ("Dyck & Thomas"), at 2199–2200. Fever, by contrast, is *not* considered a presenting feature. Leonhard at 676 Box 1; Donofrio at 1299 Table 4-2 ("Diagnostic Criteria for [GBS]"); Dyk at 2199–2200 (fever not listed as a clinical manifestation). Thus, while preexisting infections (which can later result in GBS) may present with fever or other evidence of "malaise," GBS *itself* presents with different clinical features, like weakness or numbness. Autonomic involvement is also "common" in GBS, and can involve evidence of "urine retention, . . . sinus tachycardia, hypertension, cardiac arrhythmia, and postural hypotension." Hughes & Cornblath at 1661. But dysautonomia (like fever) is also not considered a presenting feature, arising instead secondarily to symptoms directly reflecting the consequences of demyelination. Dyck & Thomas at 2199–2200.

GBS can be vaccine-caused, specifically by the flu vaccine (although the risk from wild flu *infection* is much greater). Sheikh at 1193. Consistent with this, a large body of reasoned Program decisions[16] recognize an association between the flu vaccine and GBS (as well as other related peripheral neuropathies). Indeed, there is a Table claim for GBS due to receipt of a flu vaccine. 42 C.F.R. § 100.3.14. This means the Government accepts that sufficiently-probative and reliable science on the topic exists to justify conceding causation, at least for Program purposes.

[16] Although prior decisions from different cases do not control the outcome herein, special masters may reasonably take into account, for guidance, the logic of such reasoned determinations. In fact, it is wise to do so, given how often similar causation theories or fact patterns arise in Vaccine Program cases.

Haskins v. Secretary of Health & Hum. Servs., No. 18-1776V, WL 2020 1870279 (Fed. Cl. Spec. Mstr. Mar. 13, 2019). Even in cases where a Table element for such a claim cannot be met (for example, when onset is too short or too long to fit within the timeframe of 3–42 days set for the claim), any subsequent causation-in-fact analysis performed by the special masters rarely requires the claimant to offer proof in support of the first *Althen* prong, "can cause" element; instead, it is reasonably assumed to be satisfied already. *See Welch v. Sec'y of Health & Hum. Servs*. No. 18-494V, 2019 WL 349360 (Fed. Cl. Spec. Mstr. July 2, 2019).

Other vaccines have also been found causal of GBS, although there is disagreement among the special masters as to the preponderant strength of these proposed associations. *See, e.g.*, *Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075, 2022 WL 9669651, at *36–37 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (finding the pneumococcal vaccine caused GBS); *but see Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at *30 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review den'd*, 167 Fed. Cl. 127 (2023) (holding that the pneumococcal vaccine was not shown to cause GBS); *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *3 (Fed. Cl. Spec. Mstr. Dec. 9, 2022) (same). It thus cannot be said that the Program has developed a consistent view as to what the science preponderantly "says" about causation of GBS when the flu vaccine is not involved. Instead, it appears that the outcome in such cases is mostly a function of the evidence before the special master (along with the special master's individual view about the applicability of causation theories to different vaccines), with no clear trend one way or the other.

This is definitely true for claims that the Tdap vaccine can cause GBS. Several cases decided in the past ten years (some of which I authored) found *no causal association* between the two.[17] *See, e.g.*, *Dennington v. Sec'y of Health & Hum. Servs.*, No. 18-1303V, 2023 WL 2965239 (Fed. Cl. Spec. Mstr. Apr. 17, 2023), *mot. for review den'd*, 167 Fed. Cl. 640 (2023), *appeal dismissed,* No. 2024-1214, 2024 WL 1255318 (Fed. Cir. Mar. 25, 2024); *Montgomery v. Sec'y of Health & Hum. Servs.*, No. 15-1037V, 2019 WL 2511352 (Fed. Cl. Spec. Mstr. May 21, 2019); *Tompkins v. Sec'y of Health & Hum. Servs.*, No. 10-261V, 2013 WL 3498652 (Fed. Cl. Spec. Mstr. June 21, 2013), *mot. for review den'd*, 117 Fed. Cl. 713 (2014); *Isaac*, 2012 WL 3609993 at 19.[18]

[17] I have also decided a few cases in which I determined that the petitioner failed to establish a causal association between the Tdap vaccine and CIDP—a different injury from GBS, although also still a peripheral neuropathy (and Program claimants frequently rely on GBS-specific evidence in arguing that a vaccine can cause CIDP). *See, e.g.*, *DeVaughn v. Sec'y of Health & Hum. Servs.*, No. 22-832V, 2025 WL 758128 (Fed. Cl. Spec. Mstr. Feb. 10, 2025); *Howard*, 2022 WL 4869354 at *26; *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 18-1012V, 2022 WL 1013264, at *1 (Fed. Cl. Spec. Mstr. Mar. 11, 2022).

[18] I recently decided another case involving the contention that the Tdap vaccine can cause GBS, but resolution turned on the third *Althen* prong, rendering that decision less valuable for guiding the present outcome. *Langert v. Sec'y of Health & Hum. Servs.,* No. 22-809V, 2025 WL 1892418 (Fed. Cl. Spec. Mstr. June 13, 2025).

Prior Tdap-GBS cases have often involved causation theories comparable to what is offered here. In *Isaac*, for example, a petitioner proposed molecular mimicry as the causal mechanism. *Issac*, 2012 WL 3609993, at *6. But the special master determined that the petitioner's expert had over-relied on a single case report[19] to prove causation, without adequately substantiating the mechanism. *Id.* at *20–21. This determination was affirmed on appeal at the Court of Federal Claims and Federal Circuit. In *Tompkins*, the special master denied entitlement in a case alleging that a number of vaccines received at the same time (including Tdap) caused a petitioner's GBS, but the causal theory put forward attempted to assert that the vaccines could also individually trigger the disease. *Tompkins*, 2013 WL 3498652, at *15. The petitioner's expert, however, relied heavily on VAERS passive surveillance data, and otherwise invoked a number of theories (molecular mimicry, or endotoxin in tetanus-containing vaccines) that were only cursorily substantiated. *Id.* at *19–23.

Admittedly, some special masters have deemed causation demonstrated in Tdap vaccine-GBS cases. *See Harris*, 2023 WL 2583393; *Mohamad*, 2022 WL 711604, at *18. In *Mohamad*, a special master ruled in petitioner's favor in a Tdap-GBS case, but almost wholly based on the determination that the Government had effectively conceded the first *Althen* Prong. In particular, the special master observed that (a) in 2011, the IOM had noted a precaution to receipt of the Tdap vaccine in the future if an immunized individual had developed GBS within six weeks of a prior dose,[20] and (b) this precaution note (along with an acknowledgment of the possibility of encephalopathy in a seven-day timeframe) had been maintained in subsequent ACIP reports, despite interim findings that the tetanus-GBS link was not as well-established as previously thought. *Mohamad,* 2022 WL 711604, at *13–15. From this (and also on the basis of credibility determinations specific to the experts who had testified in that case), the special master concluded that the first *Althen* prong was satisfied. *Id.* at *7, 15–18.

I make one final point. As stated above, the only Table claim for GBS relates to the flu vaccine. And yet too often the Program has featured cases in which the framework for this Table claim—including the timeframe *specific* to onset after receipt of the flu vaccine—is applied in blanket form to claims involving other vaccines, as if all are interchangeable for purposes of causing peripheral neuropathies involving demyelination. In effect, as if there exists an "off the menu" quasi-Table claim for all other covered vaccines as potentially causal of GBS that the Government has simply not gotten around to adding to the Table.

[19] The case report mentioned in *Issac* was Pollard & Selby—cited herein by Dr. Tornatore as well.

[20] Also of note is the fact that this specified circumstance (advising against receipt of the Tdap vaccine if an individual previously developed GBS after a prior Tdap dose) is facially distinguishable from the claim that a single dose can cause GBS *for the first time*. And it gives weight to the development of GBS after a second dose, but ignores the implications of receipt of prior doses that did not also result in GBS.

This kind of reasoning has obvious legal/evidentiary deficiencies. Nor should special masters be in the business of duplicating Table claims and then applying them broadly to other vaccines, based upon the reasoning that *any* vaccine is capable of causing an autoimmune injury, merely because vaccines impact the human immune system. In fact, just as the viruses and bacteria that vaccines protect against are distinguishable in their effects on the human body, so too are vaccines variable in their potential aberrant outcomes. Thus, the most that can be determined from the Program's prior treatment of Tdap vaccine-GBS claims is that they are never categorically ruled out—but that their underlying causal reasoning can properly be questioned.[21]

II. Petitioner Has not Carried Her Burden of Proof

It is well-accepted in the Vaccine Program that (because claimants must preponderantly establish all three *Althen* prongs to receive damages) special masters need only evaluate those causation elements relevant to a denial of entitlement. *Dobrydnev v. Sec'y of Health & Hum. Servs*., 566 Fed. Appx. 976, 980 (Fed. Cir. 2014). Here, I find the first and second *Althen* prongs are not satisfied (and I address them in order of their significance).

A. *Althen* Prong Two

As noted above (and below as well), the association between the Tdap vaccine and GBS is reasonably disputed—allowing for the possibility that the vaccine *could* cause this injury (although it is my conclusion in this case that this element has not been preponderantly established). But even if I assumed "can cause" causation *was* established, I could not find on the basis of the present record that Petitioner's receipt of the vaccine likely "did cause" her GBS—and any finding to the contrary would be engaging in the kind of *post hoc ergo propter hoc* reasoning that the Program rightly rejects.

The record in this case establishes several points that immediately cut against a finding that the Tdap vaccine was likely associated with Petitioner's injury. For starters, Petitioner never reported any immediate reaction—not strong evidence against the "did cause" prong, but something that undermines the likelihood that the vaccine initiated some kind of reaction. Next, Petitioner's first medical encounter after vaccination (almost four weeks later) involved complaints far more reflective of an intercurrent infection, including a fairly high fever (which as noted above *would not constitute evidence of GBS onset*) rather than neurological deficits. And no testing at

[21] I also do not give weight to instances in which Respondent has opted to settle claims that allege GBS or a comparable demyelinating neuropathy was caused by a Tdap vaccine. *Howard,* 2022 WL 4869354, at *22–23. No matter how many times Respondent may have resolved cases involving the same theory, the choice by a litigant to settle a case does not stand as evidence that the causal theory underlying such claims is preponderant, or has been accepted as such by Respondent (and of course the decision to settle a case is not evidence of the strength of one side's position in any event). Only *reasoned decisions* by other special masters (which do exist—and which admittedly in some instances are favorable to Petitioner herein) deserve any consideration as guidance.

this time suggested GBS was then occurring. Ex. 3 at 475, 478–81. Dr. Tornatore unconvincingly attempted to recharacterize these initial, pre-neurologic symptoms as proof of GBS-related dysautonomia, but it is far more preponderantly likely (as reflected in numerous treatises and articles discussing GBS) that such concerns would arise *after* onset of the kind of neurologic symptoms classically associated with GBS. And Petitioner only started to display such GBS-like symptoms (pain and limb tingling sensations) on August 22, 2019—now nearly a month post-vaccination, and far closer in time to the intervening complaints she reported a few days before. Ex. 7 at 411. Dr. Tornatore's highlighting of abnormal lymphocytes as associated with vaccination was persuasively rebutted by Dr. Jamieson.

Dr. Tornatore gave special emphasis to the fact that Petitioner was in her third trimester at the time of vaccination, suggesting that natural immunosuppression occasioned by pregnancy made an autoimmune disease like GBS unlikely (and therefore put a spotlight on vaccination as a possible triggering event). But the authority offered for the impact of pregnancy on the development of autoimmune diseases was far more equivocal than he proposed. As Respondent's experts noted, pregnancy-specific studies do not show a different incidence of pregnant women developing GBS relative to the general population. *See* First Pasetti Rep. at 3; *see also* Tseng; Sukumaran. And while pregnancy *could* have been a confounding factor, the record in this case (which again revealed no closer-in-time reaction to receipt of the vaccine) is inconsistent with a finding that the Tdap vaccine caused an aberrant immune response.

Otherwise, treater support for a vaccine association in this case is largely lacking. And no other clinical or testing results support the conclusion that Petitioner's vaccination constituted a special factor in her development of GBS. All that is left is the fact that within about a month of receipt of the Tdap vaccine, Petitioner developed GBS. While that might be sufficient to satisfy the Table claim for GBS after receipt of the *flu vaccine*, it is not enough for a distinguishable vaccine like Tdap.

B. *Althen* Prong One

There are a number of deficiencies in Petitioner's theory that prevent me from finding it has been preponderantly established. Given that a Tdap-GBS association has been found in other cases, I will regiment my analysis more formally, so the bases for my findings are clear.

1. *Evidence Associating the Tdap Vaccine's Antigenic Components with GBS is Lacking*

In many cases, claimants build their causation theory upon the proposition that a vaccine's underlying wild viral or bacterial component is associated with a particular disease or illness. Since vaccines provide a controlled immune response that somewhat replicates what a wild infection

accomplishes (albeit without the same degree of damage to health), a showing that a wild infection can cause "disease X" opens the door to the possibility that a vaccine designed to thwart that infection, and which includes some antigenic components of it, could also in rare instances cause the same illness.

Here, however, there is insufficient evidence bulwarking the view that any of the vaccine's components might be associated with GBS (other than case reports, which are addressed below). Dr. Tornatore's arguments on this front were particularly weak and unpersuasive. The bacterium causal of tetanus has not been shown related to GBS—unlike other bacteria such as *Campylobacter jejuni.* Dyck & Thomas at 2199. Just because one bacterium is associated with GBS does not mean all are. And the support he offered for this point, like Sonnabend, involved an instance in which an individual actually suffering from a *different* bacterial infection developed symptoms that mimicked GBS, and/or were secondary to the bacterial infection. Sonnabend at 360. Lee was no better. Of 13 individuals being treated for preceding tetanus infections, only two individuals went on to develop GBS that was not deemed treatment-associated, and its authors did not propose that the GBS was likely *caused* by the prior infection. Lee at 21.

Dr. Tornatore further attempted to establish amino acid sequence homology between the tetanus component of the vaccine and a self protein associated with nerve MBP. But he did so indirectly and by inference. Thus, he purports that if an antigenic target involved in some experimental forms of CNS or peripheral autoimmune neuropathies has cross-reactive homology with peptide sequences used to stimulate the disease into existence, then homology between a component of the vaccine *in question* and that same target means molecular mimicry "works" here as well. *See generally* Third Tornatore Rep.

Of course, Dr. Tornatore only identified antigenic similarity between tetanus *toxin* and the target—and the toxin is not itself a vaccine component. And Dr. Pasetti accurately noted that the process of inactivation of the toxin could well modify the sequence such that the homology he observed would disappear. Second Pasetti Rep. at 2. But even if that is ignored, Dr. Tornatore's mimicry showing is too many steps removed from "reality" to be considered reliable, to the extent it relies on an indirect comparison between sequences in tetanus with the P2 protein. For it assumes that *any* protein containing the same homologic sequence could also stimulate GBS into existence. Yet it is well understood by medical science that homology is widespread in nature—and so the fact that autoimmune diseases are not rampant or widespread alone undercuts the significance of bare homology. *J.C. v. Sec'y of Health & Hum. Servs.*, No. 17-69V, 2024 WL 3412625, at *19 (Fed. Cl. May 16, 2024) (experts acknowledged that "molecular mimics are 'widespread' even among healthy individuals."). Homology would need to be shown more specifically to target antigens relevant to GBS directly—and even then is not enough to carry the day in showing it likely the Tdap vaccine causes GBS *at all*.

In addition, there is limited evidentiary value to homology showings as a general matter. Rather, as Dr. Pasetti observed, there are additional criteria that medical science looks for in such contexts. First Pasetti Rep. at 4; Yuki at 691–92.[22] More must be done if molecular mimicry is a reasonable mechanistic explanation for an autoimmune process. I have on too many prior occasions noted that a showing of homology alone does not suffice to prove that molecular mimicry is a likely mechanism for a vaccine-caused injury—even if the *concept* of molecular mimicry has reliable scientific support (which in fact it does). *DeVaughn v. Sec'y of Health & Hum. Servs*., No. 22-832V, 2025 WL 758128, at *19 (Fed. Cl. Spec. Mstr. Feb. 10, 2025) (tetanus-containing vaccine not causal of CIDP). This is true even when, as here, the injury is believed to be mediated by this mechanism—for that fact does not also lead to the conclusion that GBS occurring after administration of a vaccine other than the flu vaccine likely caused it. Rather, more must be done to fit the mechanism to a case involving a different vaccine, like Tdap—and Dr. Tornatore's thin and unsubstantiated homology showing is insufficient.

2. *The Government Has Not Conceded a GBS-Tdap Vaccine Association*

The different publications offered in this case to suggest that the Government has (at least implicitly) embraced the possibility of a GBS-Tdap association do not in fact stand all that well for that proposition. The 1994 IOM Report can reasonably be understood not as equal and independent proof, but to have been *supplanted* by the 2012 IOM Report—and the earlier view that a formulation of the vaccine no longer administered could be associated with GBS was not carried forward. *See* 2012 IOM Rep. at 94.

Petitioner correctly notes that other special masters have given weight to the IOM's serial views on the topic (and in some cases have gone so far as deeming the matter conceded). *See Mohamad,* 2022 WL 711604, at *18; *Harris*, 2023 WL 2583393, at *27. Of course, the determinations of other special masters do not bind me—and I have previously noted that *Mohamad's* reasoning is not wholly persuasive in treating the issue of causation as conceded in any form by the Government. *K.A*., 2022 WL 20213037, at *25. In fact, the logic employed in *Harris* and *Mohamad* was called into question years before their respective publication, in *Tompkins*. There, former Chief Special Master Vowell was tasked with determining whether a number of disparate vaccines could cause GBS. That decision includes a lengthy and specific discussion of each relevant vaccine—including the tetanus vaccine. *Tompkins*, 2013 WL 3498652, at *23-30.

With respect to tetanus, Special Master Vowell noted that the 1994 IOM report seemed to allow for a tetanus-GBS association, but had been supplanted by a later version "concluding that

[22] The elements set forth in articles like Yuki need not be met in rote form by Program petitioners (who of course are not required to prove causation with certainty—and are never required to offer a mechanism in the first place). But these criteria do provide some guidance value in suggesting the kinds of factors relevant to the explanatory value of molecular mimicry as a mechanism for an autoimmune disease.

epidemiologic evidence was insufficient to demonstrate a causal association between tetanus toxoid and GBS." *Tompkins*. 2013 WL 3498652, at *26. In fact, a specific reason for this apparent about-face was the fact that the IOM report's authors noted that Pollard & Selby was a basis for the earlier view—and yet it involved *CIDP* and not GBS. 2012 IOM Rep. at 95–96. (This not only underscores the unreliable quality of Pollard & Selby, but also the lack of mechanistic evidence it provides herein. *See id.*)

Perhaps the most some governmental publication evidence stands for is recognition of a potentially-increased risk of a demyelinating polyneuropathy *relapse* after receipt of a tetanus-containing vaccine (as reflected in the "precaution" statements from entities like the ACIP), But this is not equivalent to a finding that the vaccine would likely be capable of triggering GBS *ab initio*. *K.A.*, 2022 WL 20213037, at *24. Thus, *some* articles filed in this case, like Hughes, do in fact expressly warn against a second administration of the Tdap vaccine, *if* the vaccinee previously developed GBS within some period post-administration. Hughes at 2. But Petitioner herself *never had GBS before*, nor did she experience any Tdap vaccine-associated relationship, diminishing the relevance of such precautions to this case.

Regardless, the existence of the precaution alone is not particularly probative of causation. Evidence a vaccine could promote a symptomatic flare does not also mean it can be *causal* of the original injury. *See*, *e.g.*, *Porch v. Sec'y of Health & Hum. Servs.*, No. 17-802V, 2023 WL 21875, at *13 n.39 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (vaccinations may cause MS flares, but this is not the same as proof of direct causation). It is thus unreasonable to read this evidence collectively to mean that government entities have adopted a position on Tdap-GBS causation supportive of Petitioner's causation theory. At best, the theory has not been directly rejected.

3. *Petitioner's Reliance on Case Reports is Misplaced—and Some Are Facially Unreliable or Irrelevant*

It is almost axiomatic in the Program at this point in its existence that case reports provide tepid support for causation. *See Campbell v. Sec'y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) ("Case reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value compared particularly to formal epidemiological studies"). They may stand as evidence of a "signal" that should encourage studies or methodologically-confirmable experimentation, and provide the kind of "smoke" that would suggest the presence of a fire. But otherwise, case reports (or even collections of a few patient subject experiences into a case series report) ultimately only establish a *temporal relationship* between vaccination and some adverse events.[23]

[23] I have also observed in other cases the logical inconsistency of emphasizing a handful of case reports (involving small numbers of individuals who experienced an adverse event) as durable proof of causation, while at the same time disparaging large-scale, methodologically-sound epidemiologic studies involving *thousands* of vaccine recipients. *Kelly v. Sec'y of Health & Hum. Servs.*, No. 16-878V, 2021 WL 5276373, at *26 n.24 (Fed. Cl. Spec. Mstr. Oct. 18,

Dr. Tornatore nevertheless relies on a number of such case reports, although they are often distinguishable factually—or stand out for their overall paucity, given how intermittent they are over many years in which the Tdap vaccine has been administered. Thus, some of the case series noted either that confounding factors (like an intercurrent/preceding infection) prevented reliable determinations that there was a causal association, involved different vaccine formulations, relied on passive reports of GBS, or longer timeframes for onset than what is relevant here. *See,* e.g., Top at 13; Kongbukkiat at 2; Chang at 2. Individual case reports had comparable deficiencies, and sometimes predated the time in which the form of vaccine administered today was in use. Second Jamieson Rep. at 5; Bakshi & Graves at 2; Ammar at 2.

The individual case reports offered in this case have other deficiencies specific to their circumstances. For example, I have in relevant prior decisions observed issues with the reliability of Pollard & Selby. It was, in fact, the determination that Pollard & Selby better described the risk of relapse of *CIDP* after receipt of tetanus-containing vaccines that lead the IOM to change its view about Tdap vaccine's GBS association. 2012 IOM Report at 95–96. And as I noted in *DeVaughn*, 2025 WL 758128, at *20:

> In this quite-old case study, a patient's acute idiopathic polyneuropathy relapsed on three occasions, each purportedly following a tetanus vaccination. Pollard & Selby at 113. But its authors did not consider alternative explanations for these spontaneous relapses, nor did they provide evidence beyond a temporal association. . . The authors also failed to explain *how*, or by *what* mechanism, a tetanus toxoid antigen could stimulate CIDP, even if some association had been demonstrated in this single patient. . . . And Pollard & Selby's findings remain uncorroborated, over 45 years later, by subsequent (and more reliable) studies that might confirm what it suggests is possible. It cannot stand as persuasive evidence for causation. *Tompkins*, 2013 WL 3498652, at *26 (observing that an absence of evidence in the years after publication of a case report or series corroborating its suggestions about a vaccine-injury association undermines the initial report's causal significance, and suggests its findings reflect only chance).

4. *Other Independent Studies Purporting to Associate Tdap Vaccine With GBS are Unreliable or Rebutted by Better Studies*

Petitioners are never obligated to marshal epidemiologic evidence in support of causation. But the evidence they *do* offer is properly subject to scrutiny—and to being weighed against the evidence offered by Respondent (who in many cases can identify epidemiologic studies of his own that undermine a finding of causation).

2021), *mot. for review den'd,* 160 Fed .Cl. 316 (2022). Dr. Tornatore makes just such arguments in this case. Second Tornatore Rep. at 14.

In this case, Petitioner has offered some studies that are based on VAERS data rather than confirmed instances of GBS. *See* Souayah; Chang. But they do not record the total number of vaccinated subjects in the general population for each vaccine, and were (given the reliance on passive surveillance information) subject to a number of methodologic deficiencies, including underreporting, bias, and variability in quality and completeness. *See, e.g.*, Souayah at 5. Yih is also unsupportive of Petitioner's causation argument. And Dr. Tornatore's contention that he was able to glean an admission from within it about a vaccine association that presumably its authors missed, or ignored outright, was shown by Dr. Jamieson to be specious. *See, e.g.*, Second Jamieson Rep. at 2–3. By contrast, Respondent's experts offered several large-scale epidemiologic studies (or articles commenting on such studies) that undermine any GBS-Tdap association. *See, e.g.*, Nordin; Baxter II at 199, 200, 203.

Dr. Tornatore's more general response to some of these studies was particularly unpersuasive, and merits comment. In effect, he contends that because an epidemiologic study can *never* "rule out a rare event" (Second Tornatore Rep. at 14) they are meaningless as evidence in the context of a Vaccine Act claim.

This supposition is self-evidently wrong as a matter of law. Special masters should, of course, never deem one item of literature fully dispositive on a causation claim—especially epidemiologic evidence, which is always reasonably subject to criticism about its scope, methodology, or statistical significance. But Dr. Tornatore's view is still misguided, because reliable scientific studies unquestionably cast light on the fundamental question presented in Vaccine Act cases: *can a particular vaccine likely cause a given injury?* That kind of evidence goes to the heart of the *Althen* prong one analysis—and where it undermines the contention that a vaccine can act aberrantly, it makes a petitioner less likely to succeed. It is not to be disregarded simply because vaccine-related adverse events are rare, or hard to prove. *Crutchfield v. Sec'y of Health & Hum. Servs.*, No. 09-0039V, 2014 WL 1665227, at *15 (Fed. Cl. Spec. Mstr. Apr. 7, 2014), *mot. for review den'd,* 125 Fed. Cl. 251 (2014).[24]

Indeed, the uncommon nature of a vaccine-related injury is not an all-purpose rebuttal to evidence that helps defeat a claimant's showing. The Program "prices in" this fact in many other ways (by embracing the preponderant evidentiary standard, for example, or allowing Petitioners

[24] As specifically noted in *Crutchfield,* 2014 WL 1665227, at *15:

> It is, in fact, *always* true that epidemiological studies can *never* prove definitively that Factor A *never* causes Condition B. Even when large studies fail to identify an association between Factor A and Condition B, it is *always theoretically possible* that Factor A causes Condition B in a very small number of cases, an effect too rare for the study to detect. But it is not the Respondent's burden in this case to prove that it is *impossible* that [a vaccine] can cause [an injury]. It is, rather, the *Petitioner's burden* to show not only that the . . . vaccine *can* cause [the relevant injury] . . . , but also that her own [vaccination] *did* cause [the injury]. And, therefore, the epidemiological studies cited in this case, . . . clearly do *not* help Petitioner carry her burden..

to offer any and all evidence they wish, without regard for federal evidentiary rules). And of course studies offered against a causation theory can be challenged, and found to merit less weight than their formal conclusions may suggest. But epidemiology that exists on a given causation theory should not only be evaluated, but can and does assist in determinations that the "can cause" prong was not preponderantly met. Dr. Tornatore's sweeping pronouncement to the contrary is incorrect.

5. *Petitioner Improperly Relies on Analogizing the Tdap Vaccine to The Flu Vaccine*

Another flaw in Petitioner's reasoning is the extent she has attempted to transpose thinking about the flu vaccine's association with GBS to the context of a Tdap vaccine recipient. Numerous items of literature filed in this case discuss only the impacts of the flu vaccine. *See, e.g.*, Schonberger; Nachamkin. But there is considerably more scientific and medical evidence linking the flu vaccine than the Tdap vaccine to GBS. And axiomatically, claimants cannot analogize what is preponderant for one vaccine to all other covered vaccines. I thus do not give much weight to arguments that rely on medical literature specific to the flu vaccine.

6. *Other Arguments Advanced by Dr. Tornatore Were Speculative*

While Dr. Tornatore is an accomplished physician and highly competent treater of neurologic illnesses, in this case he often engaged in speculative arguments to advance his opinion. I have already noted the extent to which he overemphasized Petitioner's pregnant status, despite equivocal medical and scientific evidence regarding the actual risk of development of autoimmune disease while pregnant. But he also relied repeatedly on other poorly-substantiated contentions that seem more reflective of a "tit for tat" desire to rebut a valid point raised by Respondent's experts—even when the evidence he relied upon was exceedingly thin.

For example, Dr. Tornatore attempted to explain away why Petitioner had not previously developed GBS after receipt of a Tdap vaccine booster—*and* when also pregnant the year before. To do so, he invoked the "kinetics" of antibodies to tetanus toxoid, maintaining that the "unusual" administration of two Tdap boosters within the span of a little over a year made an aberrant response more likely, given what is known about how long tetanus antibodies last. Fourth Tornatore Rep. at 9–10.

But this argument is unsupported by other independent evidence establishing that receipt of too many Tdap booster doses close in time (and over the span of a year is not appreciably that close) is medically contraindicated or even dangerous. Hammarlund does suggest that repeated Tdap boosters may be medically *unnecessary* due to the long-lasting impact of one dose—but it mainly proposes that cost savings would be achieved if subsequent boosters were not administered. Hammarlund at 1116–17. To the extent it considers the possibility of any adverse event risks from

repeated receipt of boosters, it mentions only anaphylaxis or brachial neuritis—*not* GBS. *Id.* at 1116. Dr. Pasetti more persuasively noted that Dr. Tornatore's arguments about antibody kinetics was undercut by Petitioner's actual medical history. Because Petitioner's 2018 booster was received nine years after a prior booster, the 2018 booster (which was also administered while Petitioner was pregnant, as in 2019) should have been more robust in its immunologic stimulation than the next booster in 2019. Third Pasetti Rep. at 6. And Sukumaran seems to directly rebut this whole line of reasoning in any event. Sukumaran at 1585. Thus, this point is largely if not wholly speculative, and not a basis for finding the vaccine can cause GBS.[25]

7. *Dr. Tornatore's Opinion Did not Gain Heft Simply Because of His Say So, and was Otherwise Persuasively Rebutted*

A final, somewhat-secondary factor that leads me to find causation not preponderantly established is my assessment of the overall reliability/persuasiveness of the opinions the experts have offered. I found the opinions offered by Drs. Pasetti and Jamieson to be reasoned, measured, and to engage in fair readings of the evidence in this case. Petitioner, by contrast, relies on only one expert, Dr. Tornatore—and although Respondent does not gain the upper hand on the question of causation simply because he hired more experts, Dr. Tornatore's opinion was not persuasive or reliable, even after four iterations.

As a competent neurologist with considerable expertise in the study of CNS demyelinating diseases like MS, Dr. Tornatore was qualified to offer an opinion on a neurologic injury like GBS and its immune etiology. The opinions he offered cogently set forth his reasoning (even if I did not find them well-substantiated after close analysis), and he did not hide his views behind a thicket of dense medical jargon (an approach often attempted by Program experts). But peripheral neuropathies are not his specialty[26]—nor has it been shown that he has gained insight regarding their nature as a result of any specific research he has performed. In addition, he lacks the same deep background in immunology possessed by Dr. Pasetti, leading me to give her opinions on those matters greater weight (although I certainly acknowledge he possessed the expertise to opine on causation issues relating to immunology).

At bottom, I discount the opinion offered on Petitioner's behalf more than I find fault in the personal qualifications of the expert espousing it. What Dr. Tornatore has done is offer a learned

[25] I do acknowledge, of course, that evidence a person previously received a particular vaccine but did not become ill is not strong evidence *against* causation either. There is no reverse form of "challenge-rechallenge," and it is always possible that a combination of factors can cause a vaccine reaction when prior exposures did not. But this particular argument by Dr. Tornatore is nevertheless too insubstantial to find persuasive.

[26] In many other cases, Dr. Tornatore has willingly *exceeded* his expertise to an even greater extent, offering opinions on a wide variety of alleged vaccine injuries even though they only loosely involved neurologic issues. *See Kelly*, 2021 WL 5276373, at *4–8 (opinion with respect to sensorineural hearing loss).

yet *advocating* opinion in which he has marshaled numerous items of evidence that collectively do not make it more likely that the Tdap vaccine can cause GBS. Too much of his theory attempted to rely upon the flu vaccine-GBS framework, but without evidence specific to Tdap; invoked out-of-date evidence and case reports; and did not preponderantly link the Tdap vaccine to GBS.

CONCLUSION

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

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[27]

IT IS SO ORDERED.

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

[27] 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.