In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 21-867V

* * *

Petitioner has not preponderantly established that the Tdap vaccine can cause GBS, or did so to him.

I. Factual Background

In the same month as the vaccination at issue, Petitioner (who was in his early 60s at the time) experienced an illness bearing on his claim. Specifically, on May 13, 2019, Petitioner sought urgent care treatment for sinus/upper respiratory infection ("URI") symptoms that he reported had begun at the end of April. Ex. 13 at 24–25. He was prescribed antibiotics and a corticosteroid nasal spray. *Id.* at 25–26. Two days later (May 15, 2019), Petitioner contacted his primary care provider's ("PCP") office reporting worsening headaches, light sensitivity, nausea, and weakness upon standing. Ex. 3 at 111.

On May 24, 2019, Petitioner visited his PCP after being bitten by a new puppy. Ex. 2 at 11. For that reason, Petitioner was administered an intramuscular Tdap vaccine. Ex. 1 at 2; Ex. 2 at 11–13. There is no record evidence of any immediate vaccine reaction.

Over three weeks later, on June 17, 2019, Petitioner called his PCP reporting numbness in his toes and hands for the previous four to five days (meaning beginning on June 12–13, 2019—almost three weeks after vaccination, and five weeks after his URI). Ex. 2 at 14. He also reported pain in his knees and hips, muscle cramps and malaise. *Id*. An in-person appointment was scheduled for June 20, 2019. *Id.* (Prior to that appointment, Petitioner saw his nephrologist for ongoing care, and he was informed that his symptoms could be consistent with a peripheral neuropathy best treated by a neurologist. Ex. 8 at 16).

At the scheduled PCP appointment, Petitioner reported his symptoms—as well as the fact that he recalled being bitten by a tick about two weeks prior (or on June 6, 2019). Ex. 2 at 15. Petitioner's neurological exam was normal, however, and the PCP ordered Lyme disease testing, which later came back negative. *Id*. at 8, 17. The next day (June 21st), Petitioner went to urgent care for treatment of acute abdominal pain, but did not report any neurologic symptoms. Ex. 13 at 27–31. He also failed to mention such symptoms at a June 24th blood pressure check with his PCP. Ex. 2 at 17–20.

On July 4, 2019 (now almost six weeks from the date of vaccination), Petitioner went to a hospital emergency room complaining of progressive bilateral upper and lower extremity weakness, dyspnea upon exertion, shuffling gait, muscle cramping, and some numbness in his mouth. Ex. 9 at 402. He informed treaters that his lower bilateral numbness began a month before, and that after two weeks he began to experience similar symptoms in his upper extremities. *Id.* Petitioner was subsequently admitted to the hospital. *Id.*

Petitioner was hospitalized from July 4–7, 2019. Ex. 4 at 402. A neurology exam revealed decrease strength in all extremities, decreased sensation, slightly diminished reflexes, and a slow unsteady gait. Ex. 9 at 409–11. He underwent a lumbar puncture, and his cerebral spinal fluid ("CSF") white blood cell count was 0 mm^3 (reference range 0 mm^3 to 5 mm^3)—meaning on the low end of normal—while his protein level was 37 mg/dL (reference range 12 mg/dL to 60 mg/dL)—also in the normal range. Ex. 8 at 68; Ex. 9 at 353. A spinal MRI produced no significant findings other than evidence of some degenerative changes. Ex. 9 at 534. And Mr. Bickel was evaluated by an occupational therapist, who noted deficits in strength, endurance, sensation, standing balance, and functional mobility, although a physical therapist deemed him capable of independent movement. *Id.* at 437, 442.

On July 7, 2019, petitioner was discharged to his home. Ex. 9 at 324. At this time, he displayed 4/5 strength or better in his extremities and a relatively stable gait, but decreased sensation and reflexes. *Id*. at 327. A consulting neurologist diagnosed Petitioner with an acute, possibly-axonal polyneuropathy likely with nerve pathology (based in part on the physical exam), but expressed doubt that Petitioner had GBS because of his normal CSF protein findings. *Id*. at 325. The neurologist referred Mr. Bickel to physical or occupational therapy, and recommended an outpatient electromyography ("EMG") and nerve conduction study ("NCS"). *Id*.

On July 11, 2019, the EMG and NCS testing occurred, and they revealed a motor and sensory polyneuropathy with axonal and demyelinating features. Ex. 9 at 853. In addition, Petitioner began outpatient physical therapy mid-July 2019. Ex. 12 at 7.[3] On initial evaluation, he displayed deficits in strength, balance, proprioception, navigating stairs, and standing for more than thirty minutes. *Id*. at 52. Toward the end of that month, Petitioner reported to his PCP at a blood pressure check visit that he was experiencing a loss of sensation in his hands and feet. Ex. 2 at 22.

Several months later (in December 2019), Mr. Bickel was evaluated by a neurologist. Ex. 8 at 148. He now reported improvements in strength and balance from physical therapy, but complained of continued cramping in his legs, and mild tingling and occasional burning in hands and feet, and hand tremors since that October. *Id*. He was now assessed with a toxic or metabolic subacute peripheral neuropathy or axonal GBS that was not progressing at the time of hospitalization. *Id*. at 150. This was based on Petitioner's MRI findings that showed no evidence of nerve root enhancement, and EMG findings that showed demyelinating features *Id.*

[3] There is also medical record evidence of Petitioner's initial occupational therapist evaluation from August 2019. Ex. 15 at 3. The occupational therapist noted that Petitioner had decreased balance, strength, sensation, and independence with activities of daily living. *Id.* at 4. But although Petitioner was scheduled for additional sessions, no record evidence was filed corroborating their occurrence.

Subsequent treatment encounters reflect Petitioner's recovery, but are not particularly helpful in establishing causation. On January 3, 2020, Mr. Bickel requested to be discharged from physical therapy (having completed 25 sessions since mid-July) because of his work schedule, but also reported that he felt better, displaying improved strength and balance despite some continued problems with the latter. Ex. 12 at 7.

By the fall of 2022 (now over three years after the vaccination at issue—and also after this case had been filed), Petitioner reported to a neurologist that he still experienced tremors and felt some fingertip and sole numbness. Ex. 30 at 9, 11. He was at this point assessed with having more likely experienced an extremely mild instance of GBS rather than a toxic or metabolic subacute peripheral neuropathy, given his clinical course. *Id.* Petitioner reported comparable symptoms in 2023 and 2025, despite overall improvement. Ex. 31 at 3, 6 (March 2023 neurology visit); Ex. 129 at 11–13 (April 2025 neurology visit).

II. Expert Reports

A. *Petitioner's Experts*

1. Dr. Norman Latov – Dr. Latov is a neurologist, and he prepared two written reports on behalf of Petitioner. Report, dated May 9, 2023, filed as Ex. 32 (ECF No. 37-1) ("First Latov Rep."); Report, dated Feb. 28, 2024, filed as Ex. 59 (ECF No. 43-1) ("Second Latov Rep.").

Dr. Latov is board-certified by the American Board of Psychiatry and Neurology, and is licensed to practice in New York state. Curriculum Vitae, filed as Ex. 33 (ECF No. 37-2) ("Latov CV") at 2. He attended the University of Pennsylvania to complete both his medical and doctorate degree, before completing his residency in neurology and immunology at Columbia University. *Id.* at 2–3. Dr. Latov currently serves as a Professor of Neurology and Neuroscience at the Weill Medical College at Cornell University, and as an Attending Neurologist at New York Presbyterian Hospital. *Id.* at 3. Dr. Latov has devoted much of his practice to the evaluation, diagnosis, and treatment of patients with peripheral neuropathies like GBS. *See* First Latov Rep. at 2. He directs a peripheral neuropathy center where his research has been credited with the discovery of antibodies and assays currently used for testing patients with suspected autoimmune diseases. *Id.* In addition, Dr. Latov has published over 200 pieces of medical literature pertaining to peripheral neuropathies. Latov CV at 4–17.

First Report

Dr. Latov opined that that vaccines can induce autoimmune disease such as GBS through two possible mechanisms :molecular mimicry or bystander activation. First Latov Rep. at 5–6. Both molecular mimicry and bystander activation have been accepted by the Institute of Medicine ("IOM") as reliable explanations for adverse post-vaccination events. *Id.* at 6 (citing *Adverse*

Effects of Vaccines: Evidence and Causality, Institute of Medicine (K. Stratton et al., eds. 2012), filed as Ex. 44 (ECF No. 37-13) ("2012 IOM Report")).

Molecular mimicry, Dr. Latov explained, "occurs when there is a structural homology, in sequence or conformation, between an exogenous agent, such as a vaccine or infection, and a self or autoantigen that is subsequently targeted by the immune response. Induction of immune reactivity against the foreign agent results in cross reactivity with the self-antigen, with subsequent tissue damage and autoimmune disease." First Latov Rep. at 6. An example of such a process is seen in those who develop the Acute Motor Axonal Neuropathy ("AMAN") variant of GBS after an infection with *Campylobacter jejuni* bacterium. *Id.* (citing R. Stein, *Campylobacter Jejuni and Postinfectious Autoimmune Diseases: A Proof of Concept in Glycobiology*, 8 ACS Infect. Dis. 1981 (2022), filed as Ex. 56 (ECF No. 37-25) (discussing the contribution of molecular mimicry to the pathogenesis of postinfectious GBS)). Molecular mimicry has also been observed to be capable of causing experimental allergic neuritis (a research model applicable to GBS) in animals after vaccination. *Id.* (citing C. Caporale et al., *Experimental Axonopathy Induced by Immunization with Campylobacter Jejuni Lipopolysaccharide from a Patient with Guillain-Barre Syndrome*, 174 J. Neuroimmunol. 12, (2006), filed as Ex. 38 (ECF No. 37-38) (successfully recreating axonal neuropathy in rabbits after immunization with *C. jejuni*); B. Soliven, *Animal Models of Autoimmune Neuropathy*, 54 ILAR J. 282, (2014), filed as Ex. 53 (ECF No.37-22) (summarizing studies of autoimmune neuropathies based on animal models); H. Willison, *Glycoconjugates and Neuroimmunological Diseases*, 9 Adv. Neurobiology 543, (2014), filed as Ex. 58 (ECF No. 37-27).

Bystander activation, on the other hand, occurs when infections or immunizations stimulate the immune system in such a way as to overcome immune tolerance and lead to autoimmune disease. First Latov Rep. at 6. This is done by the spurring and activation of existing autoreactive cells. *Id.* Examples of this "stimulation" is seen in animals that develop demyelinating autoimmune disorders after introduction of bacteria. *Id.* (citing A. Nogai et al., *Lipopolysaccharide Injection Induces Relapses of Experimental Autoimmune Encephalomyelitis in Nontransgenic Mice via Bystander Activation of Autoreactive CD4+ Cells*, 175 J. lmmunol. 959, (2005), filed as Ex. 48 (ECF No. 37-17) ("Nogai") (finding that previously healthy mice developed experimental autoimmune encephalomyelitis ("EAE") via bystander activation after being introduced to bacteria); P. Soulas et al., *Autoantigen, Innate Immunity, and T Cells Cooperate to Break B Cell Tolerance During Bacterial Infection*, 115 J. Clin. Invest. 2257, (2005), filed as Ex. 55 (ECF No. 37-24) ("Soulas") (observing bystander activation of rheumatoid factor antibodies from B-cell tolerance breakdown from bacteria); J. Goverman et al., *Transgenic Mice that Express a Myelin Basic Protein-Specific T Cell Receptor Develop Spontaneous Autoimmunity*, 72 Cell 551, (1993) ("Goverman") (finding that pertussis stimulated the immune system in mice, resulting in the development of EAE.)). Immune stimulation can also lead to the release of cytokines capable of inducing GBS episodes in vulnerable individuals. *Id.* at 7 (citing X. Lim et al, *Pseudo-*

Anaphylactic Reactions to Pfizer BNT162b2 Vaccine: Report of 3 Cases of Anaphylaxis Post Pfizer BNT162b2 Vaccination, 9 Vaccines (Basel) 974, (Aug 31, 2021), filed as Ex. 46 (ECF No. 37-15); I. Solmaz et al, *Recurrent Demyelinating Episodes as Sole Manifestation of Inherited CD59 Deficiency*, 51 Neuropediatrics 206, (2020), filed as Ex. 54 (ECF No. 37-23)).

The components of the Tdap vaccine (such as pertussis toxoids and aluminum adjuvant), Dr. Latov contended, are stimulants for the immune system. First Latov Rep. at 7. Relying on case reports and VAERS[4] data, Dr. Latov stated that the introduction of the vaccine or its stimuli in both animals and humans have resulted in demyelinating disorders, and he referenced case reports in support. *Id.* (discussing Goverman at 552–53 (observing mice develop EAE after being exposed to pertussis); H. Ammar et al., *Guillain-Barre Syndrome After Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccine: a Case Report*, 5 J. Med. Case Reports 1, (2011), filed as Ex. 35 (ECF No. 37-4) ("Ammar") (case report of a forty year old man developing Tdap; Boostrix vaccine and was later diagnosed with GBS within days); R Bakshi & M. Graves, *Guillain-Barre Syndrome After combined Tetanus-Diphtheria Toxoid Vaccination*, 147 J. Neurological Scis. 201, (1997), filed as Ex. 37 (ECF No. 37-6) ("Bakshi & Graves"); N. Newton & A. Janati, *Guillain-Barre Syndrome After Vaccination with Purified Tetanus Toxoid*, 80 South Med. J. 1053, filed as Ex. 47 (ECF No. 37-16) ("Newton & Janati") (case report documenting GBS after injection of pure tetanus toxoid in forty-seven year old man); P. Holliday & R. Bauer, *Polyradiculoneuritis Secondary to Immunization with Tetanus and Diphtheria Toxoids*, 40 Arch. Neural. 56, (1983), filed as Ex. 43 (ECF No. 37-12) ("Holliday & Bauer") (case report of a twenty-two-year-old man developing polyradiculoneuritis after receiving an immunization with tetanus and diphtheria toxoids)).

Moreover, another government health organization—the 1996 "Advisory Committee on Immunization Practices" ("ACIP")—acknowledged that tetanus toxoid can trigger a GBS onset. First Latov Rep. at 7 (citing Center for Disease Control, *Vaccine Side Effects, Adverse Reactions, Contraindications, and Precautions: Recommendations of the Advisory Committee on Immunization Practices* (ACIP), MMWR 1996; 60 [No. RR-12]: 1–35 (Sep. 6, 1996), filed as Ex. 39 (ECF No. 37-8) ("1996 ACIP Report"). That same ACIP committee later published a 2019 "best practices" update directing treaters to take care in administering tetanus-containing vaccines to individuals who had previously experienced GBS within six weeks of receipt of a tetanus-containing vaccine. *Id.* (citing E. Ezeanolue et al., "Best Practices Guidance of the Advisory Committee on Immunization Practices (ACIP)," *General Best Practice Guidelines for*

[4] VAERS is the Vaccine Adverse Event Reporting System, a database maintained by the Centers for Disease Control. VAERS collects information about adverse events that occur after the administration of licensed vaccines in the U.S. *See* About VAERS, Vaccine Adverse Event Reporting System (VAERS), https://vaers.hhs.gov/about/index (last visited January 21, 2026).

Immunization, https://www.cdc.gov/vaccines/hcp/acip-recs/general-recs/downloads/general-recs.pdf (last visited Jan. 11, 2026), filed as Ex. 41 (ECF No. 37-10) ("2019 ACIP Report").[5]

After arguing in favor of general causation, Dr. Latov opined that the three-week timeframe between Petitioner's vaccination and GBS onset was medically appropriate—and establishes a logical sequence of cause and effect between the two. First Latov Rep. at 7. Review of GBS occurrence following the National Influenza Immunization program in the 1970s resulted in the observation that GBS can develop in individuals as long as nine to ten weeks after receiving a vaccine. *Id.* (citing L. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977,* 110 Am. J. Epid. 2:105–23 (1979), filed as Ex. 51 (ECF No. 37-20) ("Schonberger")). Petitioner developed his first GBS symptoms within three weeks of vaccination, leading Dr. Latov to conclude that it was medically appropriate and logical for the vaccine to have triggered the onset of Petitioner's injury. *Id.*

Dr. Latov ruled out any differential diagnosis or potential cause of Petitioner's injury. First Latov Rep. at 8. While differential diagnoses included myelopathy and myopathy, Dr. Latov claimed that the MRI and EMG results ruled out both of these diagnoses. *Id.* Raised concerns about low CSF protein concentrations did not rule out a GBS diagnosis, as an elevated concentration is only seen in roughly 70% of GBS patients. *Id.* (referencing H. Al-Hakem et al., *Cerebrospinal Fluid Findings in Relation to Clinical Characteristics, Subtype, and Disease Course in Patients with Guillain-Barre Syndrome*, 101 Neurology 592, (2023)).[6] Dr. Latov also considered other potential causes of hypothyroidism and renal disease unlikely, as Petitioner's medical records and progression do not support a finding of either. *Id.*

Second Report

Dr. Latov's second report explored his previously-explained causation theory in greater detail. Vaccines, like infections, activate both the acquired and innate immune system, creating a cascading effect that can result in the desired immune response, or a self-targeting, autoimmune response. Second Latov Rep. at 1. The mechanisms he previously discussed (molecular mimicry or bystander activation) were both possible ways an aberrant reaction could lead to GBS. *Id.* at 3. As additional support, Dr. Latov cited to studies that have observed central nervous system demyelinating events following vaccinations—specifically ADEM after receipt of a Tdap vaccine. *Id.* (citing R. Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis*, 63 Clin. Infect. Dis. 1456, (2016), filed as Ex. 60 (ECF No. 43-2) (finding no association between transverse myelitis and prior vaccines, and a "possible association of ADEM with the

[5] Petitioner failed to file this piece of literature in this case, despite filing a coversheet for this exhibit. *See* ECF No. 37-10.

[6] Petitioner failed to file this piece of literature in this case despite filing a coversheet for this exhibit. *See* ECF No. 37-3.

Tdap vaccine" at the risk of 1.16 cases per million vaccines administered). Dr. Latov conceded that there have been no studies to identify the specific and most likely cross reactive antigens, but opined that those cross reactivities *do* probably occur. *Id.*

Dr. Latov also once again relied on VAERS database search results and case reports mentioned in his first report to further support a causal link between the Tdap vaccine and GBS. Second Latov Rep. at 3–4. The volume of reports of GBS after the Tdap vaccination or one of its components points to an association. *Id.*

2. Dr. Sohail Ahmed – Dr. Ahmed is a physician and academic with board certifications in both internal medicine and rheumatology. Curriculum Vitae, dated June 18, 2024, filed as Ex. 80 (ECF No. 47-2) ("Ahmed CV") at 5. He prepared two reports regarding immunological causation on behalf of the Petitioner. Report, dated June 18, 2024, filed as Ex. 79 (ECF No. 47-1) ("First Ahmed Rep."); Report, dated Mar. 12, 2025, filed as Ex. 100 (ECF No. 55-1) ("Second Ahmed Rep.").

Dr. Ahmed received his B.A. from the Johns Hopkins University and his medical degree from the University of Texas at Houston. Ahmed CV at 3. Afterwards, he pursued a residency in internal medicine and a fellowship in rheumatology at the University of Texas Medical School. *Id.* Dr. Ahmed is licensed to practice medicine in both the United States and Italy. *Id.* at 5. Dr. Ahmed has over ten years of experience as a clinical practitioner, and over twenty years as an academic investigator. *See id.* at 1–5. He currently works as a Medical and Scientific Consultant, where he assists companies accelerate research and development efforts. *Id.* at 2.

First Report

Dr. Ahmed focused his first expert report on establishing how the Tdap vaccine can cause GBS, and (consistent with many other prior experts opining in cases involving GBS) invoked molecular mimicry as the likely mechanism at issue. First Ahmed Rep. at 5. To substantiate its relevance herein, Dr. Ahmed performed a BLAST[7] search to look for amino acid sequence similarities between peptides in the vaccine's antigens (tetanus toxoid, diphtheria toxoid, and acellular pertussis antigens) and five neuronal membrane proteins likely to be targets for autoimmune, cross-reactive attack. *Id.* at 6–7. The peptide sequence lengths used in his BLAST search ranged from 12 to 16 amino acids, as this is a suggested length sufficient for T cells to

[7] Basic Local Alignment Search Tool ("BLAST") is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The software database compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Jan. 6, 2026). A BLAST search thus involves review of an online database to "compare[ ] nucleotide and protein sequences, to search for a homology between the … vaccine and [the body's myelin basic protein]." *Montgomery v. Sec'y of Health & Hum. Servs*., No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019).

identify surface antigens and trigger an immune response. *Id.* at 6 (citing B. Hemmer et al., *Minimal Peptide Length Requirements for CD4$^+$ T Cell Clones—Implications for Molecular Mimicry and T Cell Survival,* 12 International Immunology 375, (2000), filed as Ex. 87 (ECF No. 47-9) (concluding that T lymphocytes usually recognize and mount an immune response to foreign peptides made up of 12–16 amino acids)). Of the studied vaccine peptides, only one sequence had a 54% "identical match," while multiple sequences returned "similar matches" ranging from 53% similar to 83% similar. *Id.* at 7.

Reliable medical and scientific studies also identified six of the same tetanus and diphtheria toxoid peptide sequences Dr. Ahmed's BLAST search returned as implicated in cross-reactions. First Ahmed Rep. at 8 (citing R. da Silva Antunes et al., *Definition of Human Epitopes Recognized in Tetanus Toxoid and Development of an Assay Strategy to Detect Ex Vivo Tetanus CD4+ T Cell Responses*, 12 PLoS One 1, (Jan. 12, 2017), filed as Ex. 88 (ECF No. 47-10); B. Diethelm-Okita et al., *Universal Epitopes for Human CD4+ Cells on Tetanus and Diphtheria Toxins*, 181 J. Infect. Dis. 1001, 1002 (2000), filed as Ex. 89 (ECF No. 47-11) (finding peptide sequences on tetanus and diphtheria toxoids similar to human proteins)). Dr. Ahmed deemed his results to be statistically significant, especially when coupled with other research purporting six of the same peptide strands were comparable to human proteins. *Id.* at 8–9. These findings were so significant, Dr. Ahmed opined, that it was likely that any of these six mimics could trigger post-vaccination antibodies that trigger the development of GBS. *Id.*

Dr. Ahmed deemed his homology determinations consistent with more general scientific findings about the pathogenesis of GBS. Wild viruses and infections have been linked to GBS, and vaccines are designed to mimic natural infection processes. First Ahmed Rep. at 28–29. However, Dr. Ahmed claimed, the tetanus and diphtheria toxins have been concentrated in vaccines to create the desired immunity. *Id.* at 29. Dr. Ahmed stated that vaccinologists are aware of the risk these concentrations create and the potent immune response adjuvanted vaccines mimic in individuals. *Id.* Therefore, vaccines, like wild viral or bacterial infectious counterparts, can likely instigate an adverse effect. *Id.*

Turning from general causation, Dr. Ahmed opined that there was a sufficient cause-and-effect relationship linking Petitioner's receipt of the Tdap vaccine to the development of his GBS, with onset occurring within a medically acceptable timeframe. First Ahmed Rep. at 31. Dr. Ahmed did not expand on these conclusions, except to note he relied on Dr. Latov's expert reports and his own review of Petitioner's medical record to reach them. *Id.*

Dr. Ahmed then spent the remainder of his initial report addressing claims made by Respondent's experts. He maintained, for example, that there are a number of reputable case studies and VAERS-based items of literature to support his theory. First Ahmed Rep. at 10–12, 14 (citing Newton & Janati; Ammar; Bakshi & Graves; J. Pollard & G. Selby, *Relapsing Neuropathy due to Tetanus Toxoid. Report of a Case*, 37 J. Neural Sci. 113, (1978), filed as Ex. 50 (ECF No.

37-19) ("Pollard & Selby") (documenting a case of a forty-two-year-old who developed demyelinating neuropathy three separate times after receiving three separate tetanus toxoid injections over the course of fourteen years)). Going further, Dr. Ahmed argued that these sources are reliable due to the direct involvement of those who reporting or overseeing those events. *Id.* Similarly, studies and articles that assess tetanus toxoid—not the Tdap vaccine specifically—and GBS should be treated as equally relevant to sources that study Tdap-GBS, because tetanus toxoid shares the same mimics as the Tdap vaccine and human proteins. *Id.*

Further, Dr. Ahmed denied that any of Petitioner's preexisting health issues could have caused his GBS. First Ahmed Rep. at 14–15. Respondent's contention that Petitioner experienced a pre-vaccination URI was predicated on the assumption that Petitioner's URI was viral. *Id.* at 14. However, Dr. Ahmed opined that Petitioner's URI was more likely bacterial, based on the course of antibiotics (which fight bacteria) Petitioner received in treatment. *Id.* Therefore, Dr. Ahmed held that Petitioner's URI could not have been a viral explanation for his GBS. *Id.* at 15. (Of course, this argument ignores the fact that the wild infectious agent best associated with GBS—*C. jejuni*—is *itself* bacterial).

Dr. Ahmed then discussed the Respondent's experts' questions about literature filed in this case, and/or items offered by Respondent in rebuttal. First Ahmed Rep. at 16–29. For example, Dr. Ahmed challenged Respondent's interpretation of the 2012 IOM Report. *Id.* at 16–18. Dr. Ahmed read it to conclude that there was insufficient data to identify or rebut a connection between the vaccine and GBS (hence leaving open the issue). *Id.*. Dr. Ahmed then claimed that Respondent's immunologic expert, Dr. You-Wen He, relied on studies that were not relevant to his analysis, and/or molecular mimicry's utility as a possible mechanism, since they showed "massive sharing" in nature of peptides composed of five to nine amino acids, whereas Dr. Ahmed's results looked at longer chains of twelve to fifteen. *Id.* at 20–23 (discussing B. Trost et al., *Bacterial Peptides are Intensively Present Throughout the Human Proteome*, 1 Self/Nonself 71, (2010), filed as Ex. C Tab 11 (ECF No. 45-12) ("Trost") (finding thousands of perfect mimics between humans and bacteria); D. Kanduc et al., *Massive Peptide Sharing Between Viral and Human Proteomes*, 29 Peptides 1755, (2008), filed as Ex. 65 (ECF No. 43-7) ("Kanduc") (calling into question the possibility of direct causal association between virus and hosts due to the large number of overlapping peptide sequences between the two seen in nature)).

Second Report

Despite its length, Dr. Ahmed's second report mostly echoed the opinions set forth in his first. He defended his use of case reports and VAERS data, reiterated that the 2012 IOM Report was ambivalent on the evidence of a link between the Tdap vaccine and GBS, and maintained that his BLAST search results and offered literature established general causation by a preponderance of the evidence. Dr. Ahmed's criticisms of Respondent's expert reports were largely the same as

well. He criticized Dr. He for cherry-picking portions of medical literature, and claimed Respondent's experts were misinterpreting findings of studies. *See id.* at 6–7, 25–45.

Dr. Ahmed did, however, elaborate on his prior argument that there were no likely preexisting causes for Petitioner's GBS. Second Ahmed Rep. at 7. Regarding Petitioner's alleged URI, Dr. Ahmed pointed out that Petitioner's medical records revealed he had been diagnosed with sinusitis. *Id.* at 8. Sinusitis, Dr. Ahmed noted, is not associated with GBS. *Id.* There is also no evidence from Petitioner's medical record indicating that Petitioner was experiencing a virus. *Id.* Petitioner showed no signs of fever, cough, sore throat, trouble swallowing, rhinitis, myalgias, ear issues, headache, or loss of appetite—classic viral symptoms. *Id.* This, coupled with the fact Petitioner was being treated with antibiotics, was sufficient for Dr. Ahmed to conclude that Petitioner's preexisting illness could not have caused his GBS. *Id.*

B. *Respondent's Experts*

1. Dr. Brian Callaghan – Dr. Callaghan is a neurologist who prepared two reports on behalf of Respondent. Report, dated Sep. 18, 2023, filed as Ex. A (ECF No. 40-1) ("First Callaghan Rep."); Report, dated Sep. 30, 2024, filed as Ex. E (ECF No. 49) ("Second Callaghan Rep.").

Dr. Callaghan is Associate Professor of Neurology and an Associate Program Director for Research at the University of Michigan. Curriculum Vitae, filed as Ex. B (ECF No. 40-8) at 2. Dr. Callaghan both received his medical degree and completed his residency in Neurology at the University of Pennsylvania Medical Center. *Id.* at 1. Afterwards, he earned his master's degree and completed two postdoctoral fellowships at the University of Michigan. *Id.* Dr. Callaghan is board certified in both Neurology and Electrodiagnostic Medicine. *Id.* Throughout his practice, Dr. Callaghan has served as the Director of the Amyotrophic Lateral Sclerosis Clinic at the University of Michigan Health System, treated many patients with neuropathies and neurologic disorders, and has published over one-hundred peer reviewed articles. *Id.* at 2, 19–27.

First Report

Dr. Callaghan disagreed with Dr. Latov's general causation theory. First Callaghan Rep. at 5. He conceded that there is published data to support an association of certain forms of influenza vaccines with an increased chance of contracting GBS, but disputed the existence of comparable reliable evidence for the Tdap vaccine. *Id.* (citing J. Marks & T. Haplin, *Guillain-Barre Syndrome in Recipients of A New Jersey Influenza Vaccine,* 243 JAMA 2490, (1980), filed as Ex. A Tab 1 (ECF No. 40-2); C. Grave et al., *Seasonal Influenza vaccine and Guillain-Barre Syndrome: A Self-Controlled Case Series Study*, 94 Neurology 2168, (2020), filed as Ex. A Tab 3 (ECF No. 40-4) ("Grave") (finding no association between the seasonal flu vaccine and GBS within forty-two days after vaccination, but observing a higher instance of GBS following an acute respiratory tract

infection or gastrointestinal infection)). There is also no established similarity between the two vaccines, as one targets a singular virus and the other targets *three* kinds of bacteria. *Id.* at 5–6. Dr. Callaghan also criticized Dr. Latov's reliance on case reports and VAERS data to demonstrate an association between the Tdap vaccination and GBS. *Id.* at 5. The problem with case reports, he argued, was that they only demonstrate a temporal relationship, and with respect to a single or small group of subjects as well. *Id.* Similarly, VAERS data is methodologically untrustworthy; anyone can submit a VAERS report without confirmation of the existence of the alleged adverse reaction, and there are no control or comparison groups to determine if there is a significant difference between the vaccinated and non-vaccinated populations. *Id.*

Other evidence referenced by Dr. Latov did not support a link between the Tdap vaccine and GBS, in Dr. Callaghan's view. First Callaghan Rep. at 6. The 1996 ACIP Report, for instance, based its conclusion that a tetanus-containing vaccine could potentially cause GBS on vaccines other than the Tdap vaccine, plus a single case report (Pollard & Selby) of an instance of post tetanus-toxoid vaccine GBS. *Id.* (discussing 1996 ACIP Report). Similarly, he pointed out that the 2012 IOM Report agreed with his assessment that there was inadequate evidence to establish a causal relationship between Tdap and GBS (although this report does not close the door on the possibility either). 2012 IOM Report at 587.

Further, Dr. Callaghan claimed there was no reliable evidence connecting molecular mimicry, bystander activation, or cytokine release to Tdap and GBS. First Callaghan Rep. at 6. While Dr. Latov gave the example of *C. jejuni* as a potential cause of AMAN, this known association did not make it more likely the Tdap vaccine components are *also* associated with GBS. *Id.* Dr. Callaghan further observed an absence of studies establishing that Tdap (or any vaccine for that matter) likely causes GBS via molecular mimicry. *Id.* Regarding the alternative mechanism of bystander activation, Dr. Callaghan questioned whether studies involving EAE as an experimental model were helpful in this context. *Id.* Lastly, Dr. Callaghan claimed that Dr. Latov failed to make a connection between cytokine release and the Tdap vaccine. *Id.*

Dr. Callaghan ultimately opined that the most likely cause of Petitioner's GBS was not his Tdap vaccine but rather the URI Petitioner appears to have developed forty-four days prior to symptom onset. First Callaghan Rep. at 7. There is strong epidemiologic support for a URI-GBS association that outweighs the proposed association between Tdap and GBS, making Petitioner's URI a more likely trigger for his injury. *Id.* (citing S. Greene et al., *Guillain-Barre´ Syndrome, Influenza Vaccination, and Antecedent Respiratory and Gastrointestinal Infections: A Case-Centered Analysis in the Vaccine Safety Datalink, 2009–2011*, 8 Plos One 1, (2013), filed as Ex. A Tab 6 (ECF No. 40-7) (finding no evidence of an elevated risk of GBS following influenza vaccines, but a heightened risk of developing GBS after an infection); F. Galeotti et al., *Risk of Guillain-Barre Syndrome After 2010–2011 Influenza Vaccination*, 28 Eur. J. Epi. 433, (2013),

filed as Ex. A Tab 5 (ECF No. 40-6) (finding that the association of GBS after experiencing gastrointestinal infections was far greater than after receiving a flu vaccination); Grave at 2168)).

Second Report

Dr. Callaghan's second report responded to the theories and conclusions set forth in Dr. Ahmed's first report. Second Callaghan Rep. at 1. Dr. Callaghan questioned Dr. Ahmed's reliance on case reports, noting that they merely establish a proximate temporal relationship between vaccination and an injury. *Id.* Similarly, Dr. Callaghan criticizes Dr. Ahmed's reliance on VAERS data, since it has many limitations: it relies on self-reporting, does not need confirmation of diagnosis, and does not use control or comparison groups. *Id.* Results established in VAERS reports cannot help determine the existence of a causal relationship between a vaccine and disease. *Id.*

Dr. Callaghan also cited to the 2012 IOM Report as more supportive of his views than Dr. Ahmed's theory. Second Callaghan Rep. at 2 (discussing 2012 IOM Report). As noted by both Drs. Callaghan and Ahmed, the 2012 IOM Report concluded that there was insufficient evidence to support a causal link between the Tdap vaccine and GBS. *Id.* (discussing 2012 IOM Report). However, Dr. Callaghan pointed out that there have been no studies *since* the 2012 IOM Report that would alter its findings, and, therefore the evidence remains insufficient to support a causal association between the Tdap vaccine and GBS. *Id.*

In response to Dr. Ahmed's claims that the Petitioner's injury could not have been initiated by an antecedent infection, Dr. Callaghan asserted that URIs can be bacterial or viral, and the fact that Petitioner was prescribed antibiotics does not confirm the infection's bacterial nature. *Id.* In addition, Petitioner was never tested to identify the nature of this infection. *Id.* Regardless, Petitioner's URI could still be a potential cause of his injury—not the vaccine. *Id.*

2. Dr. You-Wen He – Dr. He is a medical doctor and immunologist who authored two written reports commenting on the primary causation questions raised by Petitioner's claim. Report, dated Mar. 30, 2024, filed as Ex. C (ECF No. 45-1) ("First He Rep."); Report, dated Nov. 26, 2024, filed as Ex. F (ECF No. 52-1) ("Second He Rep.").

Dr. He received his M.D. from the Fourth Military Medical University in Xian, China, and his Ph.D. in Microbiology and Immunology from the University of Miami School of Medicine in Miami, Florida. Curriculum Vitae, Mar. 30, 2024, filed as Ex. D (ECF No. 45-32). Dr. He is currently a Professor of Immunology at the Department of Immunology at Duke University Medical Center. *Id.* During the course of his career, Dr. He has reviewed National Institutes of Health studies, serves on editorial boards, and has authored or co-authored numerous publications. *Id*. at 2–17. Dr. He's research focuses on His research areas include "innate and adaptive immunity

against viral and bacterial infections[,] as well as tumors." First He Rep. at 1. He has conducted research on human immune responses to viral infections and is currently a Co-Principal Investigator for clinical trials focusing on cancer immunotherapy. *Id*.

First Report

Dr. He did not offer an opinion with respect to diagnostic or medical facts, but rather focused his report on responding to Dr. Latov's theories. Dr. He denied that Petitioner's GBS was caused by a Tdap vaccine, maintaining a lack of sufficient evidence existed to support a causal relationship between the two. First He Rep. at 15.

Dr. He described GBS as a postinfectious disorder that can be triggered by a number of identified infections, bacterial and viral (*C. jejuni*, cytomegalovirus, Epstein-Barr Virus, Mycoplasma pneumonia, Hemophilus influenza, influenza A virus, enterovirus, hepatitis E virus, varicella-zoster virus, Zika virus, etc.). First He Rep. at 4 (citing B. van den Berg et al., *Guillain-Barre Syndrome: Pathogenesis, Diagnosis, Treatment and Prognosis,* 10 Nat. Rev. Neurol. 469, (2014), filed as Ex. C Tab 1 (ECF No. 45-2) ("van den Berg"); P. Donofrio, *Guillain-Barre Syndrome,* 23 Continuum 1295, (2017), filed as Ex. C Tab 3 (ECF No. 45-4) ("Donofrio"); B. Islam et al., *Guillain-Barre Syndrome Following Varicella-Zoster Virus Infection,* 37 Eur. J. Clin. Microbiol. Infect. Dis. 511, (2018), filed as Ex. C Tab 5 (ECF No. 45-6) ("Islam").

But Dr. He denied there was similar evidence supporting such a causal relationship between the Tdap vaccine and GBS. First He Rep. at 4. In fact, the possibility of a relationship has been systematically studied, but never confirmed by any reliable findings. *Id.* The 2012 IOM Report specifically assessed multiple studies and case reports involving the vaccine (and its components) and GBS, but found mechanistic evidence to establish an association "lacking." *Id.* (discussing 2012 IOM Report at 557–58). Similarly, medical studies and reviews conducted thereafter have failed to establish a causal association. *Id.* at 4–5 (discussing M. Dudley et al., *The State of Vaccine Safety Science: Systematic Reviews of the Evidence,* 20 Lancet Infect. Dis 80, (2020), filed as Ex. C Tab 7 (ECF No. 45-8) ("Dudley") (vaccine safety systematic review intended to update possible causal associations of Adverse Events Following Immunization compiled in the 2012 IOM Report, and finding no causal relationship between the Tdap vaccine and development of GBS)).

Based on current research, Dr. He opined, molecular mimicry is not the likely pathogenic mechanism implicated in the pathogenesis of GBS. First He Rep. at 5. Dr. He explained the understanding of sequence homology (the core principle of molecular mimicry) has evolved over time, with medical science now better aware of the multitudes of shared homologies between foreign antigens and human proteins. Indeed, peptides of five to eight amino acids found in viruses are widely and repeatedly found throughout the human body—yet with no resultant cross-

reactivity leading to disease. *Id.* at 5–6 (discussing Kanduc at 1757–62). Such findings undermine the past belief that a mere showing of sequence homology is sufficient to establish that an autoimmune disease is likely due to molecular mimicry. *Id.* at 6 (discussing Trost; Kanduc; *see also* A. Kusalik et al., *Widespread and Ample Peptide Overlapping Between HCV and Homo Sapiens Proteomes*, 28 Peptides 1260, (2007), filed as Ex. C Tab 12 (ECF No. 45-13) (finding the HCV polyprotein has high level of similarity to over nineteen thousand human proteins); 2012 IOM Report at 70–71 ("linear amino acid sequence homology or even similar conformational structure between an exogenous agent and a self-antigen alone are not sufficient to prove that molecular mimicry is the pathogenic mechanism for a disease").

Dr. He also argued that bystander activation would not likely be the pathologic mechanism for GBS. First He Rep. at 6. Bystander activation, Dr. He noted, "has never been shown to be a *cause* of autoimmune diseases." *Id.* (emphasis added). Rather, the determining factor for autoimmunity beginning due to antigen stimulation is the strength of the initial immune activation. *Id.* at 8. This is seen with infections (which can be highly damaging) but not vaccines. *Id.* at 6–10 (citing van den Berg; Donofrio; Islam). Wild-type pathogens have different processes by which they induce immune responses, and thus bystander activation was more likely to play some secondary role in an autoimmune process due to infection than after immunization. *Id.* at 10. And the animal studies offered by Petitioner showing development of autoimmune diseases after immunization involved the use of powerful, experimental adjuvants not used in human vaccines but instead reserved for studies only. *Id.* at 10–11 (discussing Nogai; Soulas; Goverman).

Dr. He then criticized Petitioner's use of case reports and VAERS data to support his claim. First He Rep. at 12. Case reports only show a temporal relationship between the injury and vaccine. *Id.* In addition, the 2012 IOM Report reviewed many of the offered case reports and found that they were insufficient to show a causal relationship between the Tdap vaccine and GBS. *Id.* (citing 2012 IOM Report at 557–58). Similarly, VAERS data cannot be used as epidemiologic evidence due to its many shortcomings, including the fact that such reports can be filed by anyone, and do not require confirmation of the purposed adverse event. *Id.*

Turning to timing and specific causation, Dr. He conceded that there is in this case a medically-appropriate temporal relationship between Petitioner's Tdap vaccine and his symptom onset. First He Rep. at 13. However, there are two other factors that could have contributed to, or caused, his injury based on the medical record evidence. *Id*. The Petitioner, for example, was obese, and had also experienced a URI before and during his vaccination. There is strong support for a causal association between GBS and infections—more so than with the Tdap vaccine. *Id.* (citing van den Berg at 470; Donofrio at 1295).

Second Report

Dr. He's second report focused again on Petitioner's general causation theories, but also directly responded to and countered certain contentions made by Dr. Ahmed. Second He Rep. at 1. Dr. Ahmed's BLAST search returning positive results for mimics was entirely expected, Dr. He proposed, but not good evidence that molecular mimicry likely drove the disease process in question. *Id.* at 2–4. Dr. He further undermined Dr. Ahmed's results by claiming that his "rigorous approach" fell below the stringent criteria set by the Trost and Kanduc studies. *Id.* at 2. These studies required a consecutive stretch of eight or nine amino acids, rather than Dr. Ahmed's stretch of five, to show a likely mimic. *Id.* Without longer chain of matching amino acids, Dr. Ahmed's results could not be associated with biologically relevant autoimmune diseases. *Id.* at 2–4. Thus, although Dr. He allowed that molecular mimicry could explain how *some* autoimmune diseases would mechanistically occur, he maintained that more was needed to invoke it as the likely mechanism for Tdap vaccine-caused GBS. Second He Rep. at 9–10. Dr. He continued to opine instead that Petitioner's URI was far more likely to be the cause of his GBS, since infections stimulate the immune system differently than vaccines, and infection is a well-known trigger for GBS. *Id.* at 6, 10–12.

Dr. He otherwise reiterated his opinion that the evidence put forward by Petitioner did not further his claim. Case results and VAERS data only show a temporal association between a vaccine and injury. First He Rep. at 12. The CDC Precautions and Recommendations by the AICP did not establish any causal relationship either. *Id.* at 13. And the 2012 IOM Report (which at best disclaimed the ability to confirm or deny a vaccine-GBS link) analyzed more recent data than the 1994 IOM Report, making it more reliable overall. *Id.* at 4 (discussing 2012 IOM Report; 1994 IOM Report).

III. Procedural History

The Petition was filed in February 2021. Respondent filed an informal response that served as their Rule 4(c) Report challenging Petitioner's claim. Status Report, dated Jan. 31, 2023, (ECF No. 33). The expert reports discussed above were subsequently obtained and exchanged, with the final expert report filed in March 2025. I determined that this matter could be fairly resolved via ruling on the record and set a briefing schedule. This matter is now ripe for resolution.

IV. Parties' Arguments

Petitioner

Petitioner argues that he has provided sufficient evidence to preponderantly demonstrate that the Tdap vaccine can cause GBS, meeting all three of the prongs set by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005). Br. at 21. Relying

on Drs. Latov's and Ahmed's expert reports, Petitioner asserts that molecular mimicry, bystander activation, or the two processes working in tandem could have facilitated the development of GBS after receipt of a Tdap vaccine. *See id.* at 21–30 (citing First Latov Rep. at 4–5; Second Latov Rep. at 3; First Ahmed rep at 4–7). Both mechanisms are known to mediate demyelinating, immune disorders. *Id.* at 21–22. Dr. Latov offered examples of rare forms of GBS that have been observed developing after immunization with different peripheral nerve or bacterial antigens. *Id.* (discussing Nogai; Goverman). Bystander activation has also been shown to induce autoimmune diseases after the introduction of bacteria in animal studies. *Id.* at 22 (discussing Soulas; Nogai). The components of the Tdap vaccine (tetanus toxoid, diphtheria toxoid, pertussis bacteria, and aluminum adjuvant) are known to stimulate the immune system, and case reports and VAERS data have recorded instances of GBS after receipt of this vaccine. *Id.* at 22–23 (*see, e.g.,* Bakshi & Graves; Holliday & Bauer; Pollard & Selby). While either of these processes could have induced an immune disorder on their own, Dr. Latov noted they could also interact, triggering in tandem demyelinating diseases in individuals. *Id.* at 23–24 (quoting Second Latov Rep. at 3).

Petitioner also relies on Dr. Ahmed to supplement Dr. Latov's reports and give further weight to his general causation theory. Br. at 24. Dr. Ahmed's BLAST results confirmed the possibility of mimicry between components of the Tdap vaccine and self-antigens. *Id.* at 24–27. Dr. Ahmed's search results, cross referenced with medical literature, returned six mimic peptides between Tdap vaccine components and human proteins that he concluded would, "more likely than not," trigger antibodies in a devastating effect that leads to the development of GBS in susceptible individuals. *Id.* at 25 (discussing First Ahmed Rep. at 7, 30).

The association between tetanus and demyelinating neuropathies is otherwise well documented. Br. at 26–27. Petitioner and his experts specifically referenced seven case reports in which individuals developed demyelinating peripheral neuropathies after receiving a tetanus-containing vaccine (or experimental immunization). *Id.* (citing to Pollard & Selby; Bakshi; Ammar; Newton & Janati; Holliday & Bauer). Petitioner's experts themselves considered there to be a scientific basis for such a connection. *Id.* at 27. And the ability of wild tetanus infections to cause GBS (albeit reflected in case reports) lends itself to the causation theory that a vaccine used strain of tetanus could also cause GBS. *Id.*

Responding to arguments from Respondent and his experts, Petitioner maintained that there is a contradiction in Dr. He's contentions. Br. at 27. Dr. He stated that molecular mimicry as a theory to explain autoimmune disorders has recently been called into question. *Id.* However, Petitioner points out that some of Dr. He's own cited literature *confirm* instances in which the pathogenesis of GBS likely occurs via molecular mimicry. *Id.* (citing to van den Berg). Further, Petitioner disagrees with Dr. He's conclusion that the 2012 IOM Report is a better representation of science's evolving understanding of epidemiological and mechanistic properties of GBS. *Id.* at 28 (referencing Second He Rep. at 4). Petitioner points to prior Program caselaw that found value

in earlier iterations of the IOM Reports, including the 1994 version. *Id.* (discussing *Harris v. Sec'y of Health & Hum. Servs.*, No. 18-944V, 2023 WL 2583393, at *23 (Fed. Cl. Spec. Mstr. Feb. 21, (2023) ("The 1994 IOM Report Provides Some Evidence to Support Petitioner's Claim and Is Not Automatically Outweighed by the 2012 Report")). Otherwise, Petitioner emphasized that that the Program does not require a Petitioner to provide epidemiological studies to prevail on his claim. *Id.* at 29.

Petitioner also contends that he has shown a logical cause and effect between his receipt of the Tdap vaccine and development of his GBS. To do so, he relies upon temporal proximity to onset, plus the medical records. *See* Br. at 31–32. Those records show that he was generally healthy, with no signs of neuropathy prior to, and at the time of, his vaccination. *Id.* at 32 (referencing Ex. 3). In addition, Petitioner's onset occurred within the peak medically appropriate timeframe for GBS onset observed in Schonberger, and his experts have put forward a medically sound theory for general causation. *Id.* Lastly, Petitioner claims that there are no alternative causes for his GBS identified by either his treating physicians or Respondent's experts. *Id.* Therefore, Petitioner claims the totality of the evidence provided has satisfied a logical sequence of cause and effect and has satisfied his *Althen* prong two showing.

A similar argument is used to satisfy *Althen* prong three. Petitioner claims he has shown a medically acceptable temporal relationship between his Tdap vaccine and his GBS symptom onset. *See* Br. at 33–34. After four to five days of experiencing numbness in his hands and toes, Petitioner called his PCP to report the neuropathy. *Id.* at 33 (citing Ex. 2 at 13). From this record, Petitioner puts his onset at roughly nineteen to twenty days after receiving his Tdap vaccine. *Id.* Dr. Latov specifically opined that an onset of symptoms occurring within three weeks of vaccination is medically appropriate. Br. at 33. And Schonberger observed an elevated risk for GBS in individuals mostly within five weeks of vaccination. *Id.* (discussing Schonberger). (Petitioner adds that Respondent has not challenged his success in establishing this element of the claim. Br. at 34).

Respondent

Respondent's opposition brief argues that the Petitioner has failed his requisite showing on all three *Althen* prongs. Opp. at 2. To begin with, Respondent purports that Petitioner has failed to put forward a reliable medical theory that preponderantly shows that the Tdap vaccine *can* cause GBS, maintaining that medical literature does not find such a relationship, and in particular that Petitioner has not offered sufficient evidence to support his molecular mimicry mechanism. Br. at 11–16.

Available epidemiologic evidence, Respondent argues, does not support a causal relationship between the Tdap vaccine and GBS, much less that molecular mimicry explains how it would occur. *See id.* at 11–16. The 2012 IOM Report reviewed epidemiologic evidence on the

relationship between GBS and the Tdap vaccine, but found "[t]he evidence is inadequate to accept or reject a causal relationship between diphtheria toxoid–, tetanus toxoid–, or acellular pertussis–containing vaccines and GBS." *Id.* at 11–12 (*quoting* 2012 IOM Report at 587). There has been no medical/scientific evidence generated since then that would support a causal relationship. *Id.* at 12. Further, the Dudley article, which was intended to update the 2012 IOM Report, was unable to identify reliable evidence to link Tdap to GBS since. *Id.* (citing Dudley at 4).

Similarly, Respondent attacks Petitioner's reliance on case reports, claiming they are unreliable and do not advance a causal relationship between the Tdap vaccine and GBS. Opp. at 13. Case reports have historically been given less evidentiary weight in the Program, since they only establish temporal relationships between a vaccination and a subsequent adverse event. *Id*. (citing *Pearson v. Sec'y of Health & Hum. Servs*., No. 17-489V, 2019 WL 1150044, at *11 (Fed. Cl. Spec. Mstr. Feb. 7, 2019) (case reports receive only limited evidentiary weight, and cannot cure *Althen* prong one deficiencies); *Harris v. Sec'y of Health & Hum. Servs*., No. 10-322V, 2014 WL 3159377, at *18 (Fed. Cl. Spec. Mstr. June 10, 2014) (noting that "case reports are generally not a valuable form of evidence")). Dr. Callaghan explained that case reports of GBS after Tdap vaccines only establish a proximate temporal association. *Id.* (citing First Callaghan Rep. at 7). Dr. He's report also added that the 2012 IOM Report included case reports in its analysis, yet still determined there was insufficient evidence to establish a link between GBS and the Tdap vaccine. *Id.* (citing First He Rep. at 12).

Respondent then takes aim at Petitioner's molecular mimicry theory, asserting that it does not demonstrate a likely pathogenic mechanism relevant to this context. Opp. at 13. Instead, Petitioner merely relied on the concept's general reliability, invoking it in a "magic words" manner but without sufficient corroboration. *Id.* at 13–15. Past Program cases have rejected this theory when advanced without evidence more specifically substantiating an association with the vaccine and injury at issue. *Id.* (citing *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352 (Fed. Cir. 2013); *Loyd v. Sec'y of Health & Hum. Servs*., No. 16-811V, 2021 WL 2708941, at *1 (Fed. Cl. Spec. Mstr. May 20, 2021), *mot. for review den'd,* (Fed. Cl. Nov. 12, 2021)*, aff'd*, 2023 WL 1878572 (Fed. Cir. Feb. 10, 2023) ("though molecular mimicry is a generally accepted scientific concept, and is frequently invoked in Program cases, the mere mention of it does not constitute satisfaction of the preponderant evidentiary standard . . . . Rather, it must be shown that the mechanism likely does link the vaccine in question to the relevant injury.")). Respondent highlights other Program cases that have reached similar conclusions regarding molecular mimicry in Tdap-GBS cases. *Id.* at 14; *K.A. v. Sec'y of Health & Hum. Servs*, No. 16-989V, 2022 WL 20213037 (Fed. Cl. Spec. Mstr. April 18, 2022) ("it is not enough for a claimant to invoke the concept of molecular mimicry along with *some* identified homology between an amino acid sequence and a target antigen in order to carry her burden"), *mot. for review den'd,* 146 Fed. Cl. 98 (2022), *aff'd,* 2024 WL 2012526 (Fed. Cir. May 7, 2024).

Petitioner's evidence for such a process at play was also based on Dr. Ahmed's BLAST search results showing "short sequences" of identical amino acid peptides shared by the vaccine's components and human proteins. Opp. at 15. These short sequences, Dr. Ahmed claims, were sufficient homology to establish an autoimmune response. *Id.* But as Dr. He explained, the frequence of homology between foreign bodies and human proteins is far higher than previously believed. *Id.* And prior decisions recognize that petitioners must offer more than BLAST search results showing some sequence homology between a vaccine and self target relevant to an injury to prevail. *Id*. (*citing Schultz v. Sec'y of Health & Hum. Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) ("[m]ere demonstration of theoretical homology alone, based on computer driven searches involving databases of amino acid sequences, does not carry the day")). Thus, the general reliability of molecular mimicry as an explanation for the pathogenesis of *some* autoimmune processes does not mean it has been reliably substantiated in this case. *Id.* at 15–16.

Respondent next maintains that Petitioner has failed to show that the Tdap vaccine specifically caused his GBS. Opp. at 16–17. Respondent points out that there must first be "'a reputable medical or scientific explanation [that] must support this logical sequence'" which Respondent argues does not exist, as explained above. *Id.* (quoting *Hodges v. Sec'y of Health & Hum. Servs,* 9 F.3d 958, 962 n.4 (Fed. Cir. 1993)). Respondent also argues that there are other viable, alternate causes for Petitioner's injury, such as his pre-vaccination URI that likely started around April 29, 2019—well before his vaccination. *Id.* at 2, 16. Both Drs. He and Callaghan provided strong epidemiologic support for a connection between URIs and GBS that undermines Petitioner's specific causation argument. *Id.* at 16.

Finally, Respondent argued that Petitioner has failed to satisfy the third *Althen* prong (albeit largely for the reason that he has allegedly failed to show that the Tdap vaccine can cause GBS). Opp. at 17.

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).[8] There is no Table claim for GBS caused by any tetanus-containing vaccine.

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

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

empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs*, 121 Fed. Cl. 230, 245 (2015), *vacated 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 Cerrone v. Sec'y of Health & Hum. Servs.*, 146 F.4th 1113, 1122 (Fed. Cir. 2025); *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 Demore v. Sec'y of Health & Hum. Servs.*, No. 20-1265V, 2024 WL 4542934 (Fed. Cl. Spec. Mstr. Sept. 26, 2024), *aff'd*, No. 20-1265V, 2025 WL 868902, at *4 (Fed. Cl. Mar. 20, 2025) (rejecting the argument that a petitioner's burden is to prove that a causation theory is *plausible* and instead requiring petitioner to prove the theory by a preponderance of the evidence) (emphasis added). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C.*, 704 F.3d at 1356; *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y*

of Health & Hum. Servs., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review 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 & Hum. Servs.*, No. 15-85V, 2018 WL 1750619, at *2 (Fed. Cir. Apr. 12, 2018); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided 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 & Hum. 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

The parties agree on Petitioner's GBS diagnosis (*see, e.g.*, Br. at 20; Opp. at 11), but some discussion of the injury is nevertheless warranted. 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. van den Berg at 469–70. It is usually believed to present with pain, numbness, paresthesia, or weakness in the limbs. *Id.* at 472–73. Autonomic involvement is also "common" in GBS, and can involve evidence of urine retention, tachycardia, hypertension, cardiac arrhythmia. Donofrio at 1305; van den Berg at 478.

GBS can be vaccine-caused, specifically by the flu vaccine (although the risk from a wild flu *infection* is much greater). *See, e.g.,* Schonberger; Grave. Consistent with this, a large body of reasoned Program decisions[9] recognizes 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. *Haskins v. Secretary of Health & Hum. Servs.*, No. 18-1776V, WL 2020 1870279 (Fed.

[9] 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.

Cl. Spec. Mstr. Mar. 13, 2019). Even in cases where a Table element for such a claim cannot be met (for example, when onset occurs outside 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). The Program has not 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 views 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.[10] *See, e.g.*, *Kaczerowski v. Sec'y of Health & Hum. Servs*., No. 21-758V, 2025 WL 2798865, at *1 (Fed. Cl. Spec. Mstr. Aug. 28, 2025); *Hiatt v. Sec'y of Health & Hum. Servs.,* No. 19-1363V, 2025 WL 3230494, at *18 (Fed. Cl. Oct. 24, 2025), *mot. for review filed,* No. 19-1363 (Fed. Cl. Dec. 22, 2025); *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.[11]

[10] 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 v. Sec'y of Health & Hum. Servs.*, No. 16-1592, 2022 WL 4869354, at *26 (Fed. Cl. Spec. Mstr. Aug. 31, 2022); *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 18-1012V, 2022 WL 1013264, at *1 (Fed. Cl. Spec. Mstr. Mar. 11, 2022).

[11] I also 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[12] to prove causation, without adequately substantiating the mechanism as likely. *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 v. Sec'y of Health & Hum. Servs.*, No. 16-1075, 2022 WL 711604, at *18 (Fed. Cl. Spec. Mstr. Jan. 27, 2022). 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,[13] 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 involves the flu vaccine. And yet too often the framework for this specific Table claim—including the timeframe *specific* to onset of GBS after receipt of the flu vaccine—has been applied in blanket form to claims involving *other* vaccines, as if all are interchangeable for purposes of causing peripheral, demyelinating neuropathies. In effect, petitioners act as if there exists an "off the menu" quasi-

[12] The case report mentioned in *Issac* was Pollard & Selby—cited herein as well.

[13] 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 as proof of causality, while disregarding the implications of receipt of prior doses that *did not* also result in GBS.

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.

This kind of reasoning has obvious legal/evidentiary deficiencies. Nor should special masters be in the business of creating their own Table claim, and then applying its elements broadly to other vaccines, based upon the reasoning that *any* vaccine is likely 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.[14]

II. Petitioner Has not Carried His 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 *Althen* prong is not satisfied—and this alone is a basis for denying entitlement.[15]

I have now several times determined that existing medical science does not support the contention that the Tdap vaccine can *likely* (i.e., be preponderantly shown to) cause GBS. *See, e.g., Kaczerowski*, 2025 WL 2798865 at *29–38; *Hiatt,* 2025 WL 3230494, at *18; *Dennington*, 2023 WL 2965239 at *19. I have noted a large number of deficiencies in the theory, including but not limited to the following:

[14] 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 the claim 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.

[15] Although I need not also analyze Petitioner's success in meeting the other *Althen* prongs, I note that the second, "did cause" prong is also not likely met, on the basis of the evidence in this case. The record clearly establishes that in mid-May 2019, Petitioner sought treatment for some form of illness that began at the end of April. Ex. 13 at 24–25. He had a second encounter not long after, once his symptoms worsened. Ex. 3 at 111. While the experts disagreed as to whether the illness was viral or bacterial (and the importance of that fact), it matters not for present analysis, since in either case the antecedent infection could *more likely* have triggered an autoimmune process leading to GBS—precisely in the manner proposed by Petitioner, moreover—than a vaccine. Infections *are far more associated with GBS than vaccines*. First He Rep. at 4. This record thus makes it unlikely the vaccine was causal, even if the first *Althen* prong were met.

- The underlying wild tetanus bacterial infection is at best *weakly* associated with GBS, and only by case reports (a form of evidence not deemed worthy of much weight in Program cases)—hence this case does not present circumstances in which the wild viral/bacterial analog to the relevant vaccine is itself known to be associated with the alleged injury (a factor that can bulwark a prong one finding);[16]

- It is improper to apply in blanket fashion scientific evidence pertaining to the flu virus/vaccine association with GBS to other, distinguishable, vaccines;

- Homology alone between components of the Tdap vaccine and purported self-antigens on nerve myelin where cross-reactivity leading to GBS is thought to occur is insufficient grounds for finding the vaccine *likely* causal of GBS;

- The Government has not conceded a Tdap vaccine-GBS association, and older publications that suggest as much should not be given substantial weight;

- Settling cases, and Respondent's willingness to settle those cases, that allege GBS after Tdap vaccine does not stand as evidence that the causal theory underlying such claims is correct, preponderantly-established, or reflects reasoning the Respondent shares;

- Publications that caution against the use of the Tdap vaccine after contracting GBS are only relevant to instances where a petitioner *previously* developed GBS after vaccination—and hence *before* the vaccination event at issue. Even then, this is not particularly reliable evidence of a causal relationship;

- Passive surveillance data or case reports are weak proof of causation, and only demonstrate a naked temporal association with vaccination; and

- Existing epidemiologic evidence better supports the conclusion that the Tdap vaccine is not likely associated with GBS.

See generally Hiatt, 2025 WL 3230494, at *18–19; *Kaczerowski,* 2025 WL 2798856, at *33–38; *Dennington,* 2023 WL 2965239 at *19.

[16] In making this observation, I am not requiring Petitioner to establish that any of the Tdap vaccine's bacterial antigenic components can cause, or are associated with, GBS. I am simply observing the fact that Petitioner's claim cannot take advantage of such a known, preexisting relationship. Claimants routinely contend that vaccination is comparable to an infection—so if the wild viral/bacterial components are not linked to a given injury, that absence of association will have to be overcome by more robust evidence of an association from other sources of proof.

These factors lead me to reach the same conclusion in this case—and which special masters before me adopted, after evaluation of comparable evidence. And in so ruling before, I have considered expert testimony from qualified and experienced neurologists, and reviewed in detail the literature and studies offered on the topic. The mix of reasoning and evidentiary showings are not enough to tip the preponderant scale in a claimant's favor.

I acknowledge that this case involves a slightly different set of experts. But the outcome is—appropriately—the same. This record reveals nothing that would make it more likely than not that the Tdap vaccine Petitioner received had initiated some kind of autoimmune process that would spark GBS symptoms within a few weeks. The medical literature provided in this case does not wholly disprove a causal association between the Tdap vaccine and GBS, but it certainly does not support one either. Petitioner has not filed new scientific or medical reports/studies that would better support causation than in prior matters. Rather, Petitioner offers multiple case reports that only show a temporal association between his vaccination and GBS. That alone does not make it more likely the vaccination is the explanation for what came after.

Petitioner's experts have also reiterated the same molecular mimicry theory as in previous cases, relying on proof of homology as the cornerstone of their case. However, there is limited evidentiary value to homology showings as a general matter; more must be provided to show molecular mimicry is a reasonable mechanistic explanation for an autoimmune process. I have expressed this sentiment on multiple, previous occasions. *See, e.g., Kaczerowski,* 2025 WL 2798865 at *45; *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). Therefore, Dr. Ahmed's BLAST search results returning sequence homologies is not sufficient evidence to support the conclusion that the Tdap vaccine likely causes GBS in this manner.

The literature cited by Petitioner's experts is also common to many prior cases, but is not supplemented with new findings that would justify another look at this oft-rejected theory. Petitioner's experts offer the same reports and studies offered in previous cases and make the same arguments as to their conclusions. Experts in both *Hiatt* and *Kaczerowski* cite to Pollard & Selby, Ammar, Bakshi & Graves, Newton & Janati, and other case reports as causal evidence, when I have repeatedly explained that case reports offer nothing more than evidence of a temporal association. *Kaczerowski,* 2025 WL 2798856, at *9, 35 (citing to Bakshi & Graves; Ammar; Pollard & Selby; Newton & Janati); *Hiatt,* 2025 WL 3230494, at *5 (discussing Pollard & Selby). The same is true for the various governmental reports on vaccine adverse events. *Hiatt,* 2025 WL 3230494, at *3 (discussing 2019 ACIP Report); *Kaczerowski*, 2025 WL 2798865, at *4, 8 (discussing 2019 ACIP Report). And this is not the first time I have rejected the argument that cautioning patients who previously experienced GBS after receipt of a Tdap vaccine to avoid the vaccine in the future is evidence of causation. *Hiatt*, 2025 WL 3230494, at *18 (citing *Kaczerowski,* 2025 WL 2798865, at *34).

It is certainly not *impossible* that additional scientific discoveries will better support a Tdap vaccine-GBS association. But they have not been offered in this case. I also allow that the core basis of Petitioner's theory—that a Tdap vaccine could stimulate GBS in the same way the flu vaccine (or other wild infections) are thought to do so—has a veneer of plausibility. But as noted above, *plausibility is not the evidentiary standard applied to the first Althen prong. Cerrone*, 146 F.4th 1113, 1122. In rejecting the theory advanced by Petitioner, I am drawing on my repeated prior exposure to its components in comparable cases, and finding—again—that it lacks preponderant support. Although Petitioner's GBS *followed* receipt of the Tdap vaccine, it is not likely the vaccine caused it.

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.[17]

IT IS SO ORDERED.

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

[17] 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.