# In the United States Court of Federal Claims
## OFFICE OF SPECIAL MASTERS
## No. 23-12V

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| STACEY WYBLE, | * | Chief Special Master Corcoran |
| Petitioner, | * | Filed: February 27, 2026 |
| v. | * | |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |

*David J. Carney*, Green & Schafle, LLC, Philadelphia, PA, for Petitioner.

*Tyler King*, U.S. Department of Justice, Washington, DC, Respondent.

**ENTITLEMENT DECISION**[1]

On January 4, 2023, Stacey Wyble filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program).[2] Petitioner alleges that she suffered chronic inflammatory demyelinating polyradiculopathy ("CIDP") due to receipt of a tetanus-diphtheria-acellular-pertussis ("Tdap") vaccine on November 15, 2020. Petition (ECF No. 1) at 1. I determined that this matter could be fairly resolved via ruling on the record, and both sides filed briefs in support of their positions. *See* Petitioner's Motion for Ruling on the Record, dated Feb. 25, 2025 (ECF No. 37) ("Mot."); Respondent's Response, dated May 12, 2025 (ECF No. 40) ("Opp."); Petitioner's Reply, dated June 4, 2025 (ECF No. 41) ("Reply").

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

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

The matter is now ripe for resolution. For the reasons set forth in more detail below, I hereby deny entitlement. Petitioner has not preponderantly established that the Tdap vaccine can cause CIDP, or likely did so to her.

## I. Factual Background

*Preexisting Health & Treatment*

Petitioner (born on July 5, 1977) was 43 years old at the time of the relevant vaccination. Ex. 1 at 1. She had a fairly-lengthy medical history featuring a wide variety of preexisting issues, some of which arguably bear on the injury alleged in this case (such as arthropathy, chronic gluteal pain, fibromyalgia, hyperthyroidism, hypothyroid, polyarticular arthritis, right leg swelling, spinal disc herniations, and disc degeneration). Ex. 5 at 61 (history list).

Also relevant are some treatment instances that occurred before Petitioner's November 2020 vaccination. On March 17, 2020, for example, Ms. Wyble saw orthopedist Ninad Sthalekar, M.D., and reported lumbar pain. Ex. 7 at 6–7. It was noted at this time that Petitioner had experienced "herniated discs in 2007 and a car accident that exacerbated symptoms in 2012," and that she had since been receiving treatments for her lumbar and cervical spine. *Id*. at 6. Dr. Sthalekar assessed Petitioner with lumbar radiculopathy and degenerative disc disease, and recommended a lumbar MRI prior to any additional treatment. *Id*. at 6.

Later that same March, Petitioner had a telehealth visit with neurologist Daniel Skubick, M.D., for pain management. Ex. 11 at 27–28. Dr. Skubick noted that Ms. Wyble was potentially suffering from a "generalized polyarthropathy," and that she might be properly at some point classified as disabled due to her significant comorbidities. *Id*. at 27. Dr. Skubick assessed Petitioner with fibromyalgia among other things, "noting the widespread nature of her pain" and "myofascial pain" as a "substrate" of her fibromyalgia, and proposed she continue to utilize medical marijuana for treatment of her pain. *Id*. at 28.

*Vaccination and Subsequent Symptoms*

On November 15, 2020, Petitioner received the Tdap vaccine at issue in her left deltoid at a CVS Pharmacy. Ex. 1 at 2. The next day, she saw her primary care provider ("PCP"), Donald Brislin, D.O., at St. Luke's Dublin Internal Medicine, for a follow up visit relating to her hemoptysis.[3] Ex. 5 at 74–77. Later that month, an x-ray was performed due to these hemoptysis

[3] "Hemoptysis" is defined as "the expectoration of blood or of blood-stained sputum." *Hemoptysis*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=22096&searchterm=hemoptysis (last visited Feb. 27, 2026).

concerns, but it yielded normal results. Ex. 9 at 445–46. But she also underwent in late-November an upper gastrointestinal double contrast exam that was positive for severe gastroesophageal reflux disease. *Id.* at 447–48. Petitioner reported no neurologic symptoms in this two-week timeframe that might bear on her later CIDP diagnosis, and there is no medical record evidence from November of any vaccine reaction.

On December 3, 2020, Petitioner saw pulmonologist, Kathy Tran, D.O., for treatment of an ongoing productive cough. Ex. 3 at 7–9. A physical examination noted no abnormalities, although Petitioner did display wheezing. *Id*. After some evaluation, Dr. Tran assessed Petitioner's condition as "consistent with asthma[]," and started her on an inhaled corticosteroid. *Id*. at 7. Approximately two weeks later, Petitioner had a telehealth visit with her PCP, Dr. Brislin, and she now reported that she had for two days been experiencing chills, fatigue, and diarrhea but no fever, and that some of her relatives had recently been diagnosed with COVID. Ex. 5 at 59–63. Dr. Brislin ordered COVID testing, diagnosed Petitioner with a viral illness, and directed her to return for care if her symptoms did not improve. *Id*. at 62. Thus, although by early December there was no evidence of a vaccine reaction, Petitioner was reporting symptoms that could reasonably be associated with a *viral infection*.

*Initial Manifestation of Neurologic Symptoms*

By the second half of December—now approximately one-month post-vaccination—Petitioner began to experience symptoms that appeared more neurologic in nature. On December 18, 2020, she had an in-person visit with Dr. Brislin, and she now complained of all-body tingling sensation, plus "[c]old [t]ingling fingers," cold lips, pain with movement, and chest pain that would "come and go with breathing," beginning December 6th. Ex. 5 at 53; *see also* 51–58. A review of systems and physical examination noted no concerns, however, and a brain MRI performed later that month and was intended to rule out the possibility of a transient ischemic attack resulted in normal findings. *Id*. at 52, 53–54, 56–57

Petitioner went back to see Dr. Skubick a few days later (on December 22, 2020). Ex. 11 at 29–30. She reported "the development of new symptoms consisting of a sense of burning and numbness affecting all 4 extremities to include her face and her lips." *Id*. at 29. Dr. Skubick opined that the "exact etiology of [Petitioner's] complaint [was] not clear," but noted that exam revealed no focal motor weakness, and no true weakness with full exertion. *Id*. However, Ms. Wyble did display diminished vibration sensation in the feet, with symmetric reflexes in the upper extremities but absent at the knees and ankles. *Id*.

Based on the foregoing, Dr. Skubick opined that Petitioner continued to "clearly qualif[y] for the diagnosis of fibromyalgia," but also appeared to be experiencing myofascial pain, likely caused by her numerous conditions. Ex. 11 at 29–30. But he noted the need to "[r]ule out

polyneuropathy," given Petitioner's observed lower extremity areflexia. *Id*. at 30. He ordered an EMG[4] to further assess her condition. *Id*.

At the end of December 2020, Petitioner went to the Doylestown Hospital Emergency Department and reported numbness that she reported had begun a few weeks prior, beginning bilaterally in her hands, and also featured weakness and difficulty in walking. Ex. 9 at 357. A physical examination revealed "[u]pper extremity strength intact, subjective numbness bilaterally, [and] she [was] able to lift her legs off the bed, ankle reflexes [were] present[.]" *Id*. at 358–59.

Later that same day, Petitioner saw a different neurologist, Jeffrey Gould, M.D. Ex. 9 at 374–77. She now identified December 13, 2020 (four weeks post-vaccination), as the date of "sudden onset of awareness of bilateral hand sensation of cold and numb and tingling, worse with extension 'like falling asleep[]'" and added that she had experienced slurred speech and sensory changes in her tongue (but which had resolved). *Id*. at 374–75. Dr. Gould completed a full examination and only observed mildly-pressured speech, and a mildly-reduced cold sensation, along with no other evidence of altered sensation, coordination, or weakness. *Id*. at 376–77. Dr. Gould's impression was that Petitioner had experienced an "[a]brupt change in gait dysfunction," and his differential diagnosis was a "[s]omatization disorder; no evidence of a progressive neuromuscular disorder currently[.]" *Id.* at 374. He prescribed neurologic pain medication (gabapentin), and directed Ms. Wyble to undergo EMG testing within two weeks. *Id*.

*Hospitalization in Early 2021*

On January 4, 2021, Ms. Wyble saw neurologist Divisha Raheja, M.D., for the planned EMG testing. Ex. 11 at 38–40. Petitioner's reports of progressive weakness and difficulty walking were again memorialized, and an exam revealed poor activation in distal feet muscles and absent reflexes in the lower extremities. *Id.* at 38. But in Dr. Raheja's estimation, the EMG testing results did not "provide any electrophysiologic evidence to support a generalized neurogenic or myopathic process affecting the peripheral nervous system, and there was "no evidence to support a radiculopathy in the left upper or lower extremities." *Id*. at 38. She also noted that certain conduction velocity findings were not consistent with the existence of demyelination. *Id.* at 40.

The next day, Petitioner again sought ER care for numbness, and she was seen a second time by Dr. Gould after being hospitalized. Ex. 9 at 6, 10, 33. A physical and neurologic exam revealed normal strength, trace reflexes, and normal sensation. *Id.* at 36. Given this (plus the prior

[4] "Electromyography" is defined as "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation, performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals." *Electromyography*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography (last visited Feb. 27, 2026).

negative/normal MRIs and EMG testing results), Dr. Gould assessed Petitioner with "[a]brupt onset in gait dysfunction," but also included in the differential "[s]omatization disorder; no symptoms consistent with spinal cord damage[.]" *Id*. at 33. He recommended that Petitioner undergo neuropsychological testing as an outpatient, but that she also should receive medication specific for nerve pain. *Id.*

Treaters who saw Petitioner around the time of her hospital admission struggled to identify the proper diagnostic classification for her symptoms. For example, in early January 2021, Ms. Wyble was evaluated by physical medicine and rehabilitation specialist David James Van Why, M.D. Ex. 9 at 25. The sensation testing Dr. Van Why performed did reveal an "electrical sensation . . . from her face down her entire body down to her left foot," but light sensation issues were only evident in a descending pattern down her lower extremities, with areflexia also evident only in the knees and ankles. *Id.* at 28. It was also difficult to assess Petitioner's strength. *Id.* Ultimately, Dr. Van Why allowed that Petitioner's overall symptoms were consistent with a peripheral neuropathy, even though her presentation was variable, but he also highlighted her anxiety as possibly impacting testing results. *Id.* at 30. By contrast, Petitioner was also around this same time also evaluated by orthopedist Guy Lee, M.D. *Id.* at 20. Dr. Lee proposed that Ms. Wyble was experiencing a herniated cervical disk, observing "no signs of myelopathy on physical exam," and no explanation for her ambulation issues, with normal strength findings when in bed. *Id.*

While admitted, Petitioner underwent additional MRIs (of the cervical, thoracic, and lumbar spine), but no imaging findings were deemed to shed light on the etiology for her presentation. Ex. 9 at 81–87; *see also* at 11. Some blood testing revealed elevated inflammation biomarkers, but testing for rheumatologic or infectious etiologies was negative. Ex. 11 at 74–77. Petitioner was at discharge assessed with progressive lower extremity weakness with ambulatory dysfunction, hypothyroidism, chronic back and cervical pain, and fibromyalgia. *Id.* at 93. At most, it was noted that if Petitioner's neurologic work-up continued to result in normal findings, a neuropsychiatric etiology should be considered. *Id.* at 94. Petitioner was thereafter transferred to an inpatient therapy center due to her mobility issues, and she remained in rehab until January 15, 2021. Ex. 9 at 95; Ex. 11 at 66–68.

On February 4, 2021, Ms. Wyble had a telehealth appointment with yet another neurologist (Marianos Dalakas, M.D.) and complained of "diffuse burning pain." Ex. 6 at 3–4. Based on review of Petitioner's history and reported symptoms, Dr. Dalakas opined that Petitioner had "[n]europathic symptoms probably small fiber sensory neuropathy," but also noted a [p]ossible cervical discogenic process[.]" *Id*. at 4. He added, however, that if another EMG produced normal results, "the only possible diagnosis is small fiber sensory neuropathy." *Id*. To that end, Dr. Dalakas proposed further EMG testing, spine imaging, and an increase in Petitioner's gabapentin (300 mg daily). *Id.*

*Embrace of CIDP Diagnosis*

On March 2, 2021, Petitioner had a telehealth visit with another neurologist, Sami Khella, M.D., at Neurology Penn Medicine University City. Ex. 12 at 73. After taking note of Petitioner's reported symptoms, Dr. Khella ordered additional evaluative testing, most of which was normal or not of diagnostic value. *Id*. at 51–53, 54–60. Toward the end of March, Petitioner saw Dr. Khella in person and received a physical/neurologic exam. *Id.* at 47–70. Dr. Khella conducted a physical examination of Petitioner noting in part "[m]ild graded sensory loss to pin in legs," plus variable strength testing, an ataxic gait, and absent reflexes *Id.* at 49. Dr. Khella also completed an EMG, and it was interpreted to reveal "mild chronic partial denervation distally but not in proximal muscles." *Id*. at 63–66. Based on these findings, Dr. Khella opined that Ms. Wyble had possibly experienced "[a]cquired demyelinating polyneuropathy." *Id*. at 66.[5]

Petitioner had a follow-up telemedicine appointment with Dr. Dalakas on April 5, 2021. Ex. 6 at 41–48. Relying on EMG results and a visual exam of Petitioner's ambulation efforts, Dr. Dalakas assessed her with "[a]cute onset demyelinating neuropathy with conduction blocks, consistent with CIDP." *Id*. at 43. But he deemed it "unclear" whether the November 2020 Tdap vaccine could have triggered Petitioner's December symptoms. *Id.* Dr. Dalakas ordered more lab testing and prescribed a three-month course of once-a-month IVIG infusions, and recommended as well that Petitioner continue with her gabapentin. *Id*.

A whole-body CT scan performed on April 20, 2021, provided no evidence of skeletal fractures suggestive of myeloma, nor did a cranial MRI suggest any etiologic explanations. Ex. 14 at 60, 119. Three days later, on April 23, 2021, Petitioner presented to Adam Waxman, M.D., at the Division of Hematology and Oncology at Penn Medicine. *Id.* at 57–62. Following a physical examination, there was serious consideration of the possibility that Petitioner was experiencing "POEMS syndrome,"[6] with a differential diagnosis of "CIDP + MGUS [Monoclonal gammopathy of undetermined significance] vs paraprotein mediated disease such as POEMS syndrome." *Id.* at 62. Although Petitioner tested positive for certain biomarkers for POEMS syndrome, she was advised otherwise to continue IVIG treatment for CIDP. Ex. 14 at 45–47, 53, 57–62, 86, 104. Those treatments continued through June. *Id.* at 28; Ex. 17.

[5] A second EMG study was performed on March. 31, 2021. Ex. 6 at 21. This testing also revealed a "suggestion of an acquired demyelinating neuropathy, with evidence of prolonged distal latencies, conduction block, and slow conduction velocity." *Id.*

[6] "POEMS" is a rare, complex plasma cell disorder, and stands for "Polyneuropathy, Organomegaly, Endocrinopathy, Monoclonal gammopathy, and Skin changes," *R.S. v. Sec'y of Health & Hum. Servs.*, No. 15-1207V, 2019 WL 7631017, at *2 (Fed. Cl. Spec. Mstr. Dec. 19, 2019) (defining POEMS syndrome and discussing its common features and clinical presentation). It causes debilitating nerve damage (neuropathy), among other things, and it is associated with evidence of vascular endothelial growth factor, or "VEGF." *R.S,* 2019 WL 7631017, at *2.

Ms. Wyble saw Dr. Khella again (for a telehealth visit) on July 6, 2021. Ex. at 12 at 42–46. Dr. Khella now more firmly embraced CIDP as the proper diagnosis, noting that earlier MRIs did not in his estimation provide a viable alternative characterization for her overall symptoms. *Id*. at 42. He deemed the IVIG treatments to be appropriate as well, especially since Petitioner seemed to be responding favorably to them. *Id*. at 43. Later that month, other treaters took note of Petitioner's improvement after IVIG (although she remained bed-ridden), finding her responsiveness to such treatments made CIDP a more likely diagnostic characterization than POEMS. Ex. 14 at 27–31.

Dr. Khella reiterated his view that CIDP was an appropriate diagnosis in October 2021, noting further her ongoing improvement (and continued prescriptions of IVIG). Ex. 12 at 32–41. Petitioner's improvement continued into 2022, despite some lingering gait/leg movement issues. *Id*. at 21–32. The CIDP diagnosis was also accepted by Dr. Skubick in January 2022, although he deemed some of her continued pain issues to relate to preexisting comorbidities. Ex. 11 at 72–73. Other records pertaining to treatment events thereafter are consistent with the CIDP diagnosis but do not otherwise shed much light on the causation issues to be resolved in this case.

## II. Expert Reports

### A. *Petitioner's Experts*

1. Dr. Joseph Jeret — Dr. Jeret, a neurologist, prepared a single written report on Petitioner's behalf. Report, dated July 18, 2023, filed as Ex. 20 (ECF No. 16-1) ("Jeret Rep.").

Dr. Jeret received his undergraduate degree from CUNY Brooklyn College, New York in 1984, and his medical degree from SUNY Health Science Center at Brooklyn in 1988. *See* Curriculum Vitae, filed as Ex. 21 (ECF No. 16-2) ("Jeret CV") at 1. Thereafter, he completed a one-year general internal medicine preliminary year at Maimonides Medical Center, followed by a three-year residency in Neurology and a one-year fellowship in Clinical Neurophysiology at SUNY Downstate. *Id.* He is board certified in Neurology by the American Board of Psychiatry and Neurology and is currently employed by Optum Health Care as an active neurologist and is on staff at two community hospitals—Mount Sinai South Nassau Hospital and Mercy Medical Center. *Id.*; Jeret Rep. at 1. Dr. Jeret has published numerous articles in areas related to neurology, reflecting his broad general practice. *Id.* at 2–7; Jeret Rep. at 2.

Dr. Jeret's report included a review of Petitioner's medical history. Jeret Rep. at 5–15. Based upon her clinical symptoms, course, and EMG/NCS testing (which he described in great detail), he concluded that CIDP was the appropriate diagnosis. *Id.* at 3–4, 16. Dr. Jeret defined CIDP as falling within the context of other, related peripheral neuropathies, like GBS. He deemed CIDP the "chronic counterpart" to GBS, highlighting the fact that both are immune-mediated, driven in part by damaging inflammation, involve demyelination (damage to a nerve's myelin sheath), and impact more than one nerve (hence "polyneuropathy"). Jeret Rep. at 2–3.

Vaccines generally, Dr. Jeret maintained, are associated with CIDP. Jeret Rep. at 4; K. Gorson & A. Ropper, *Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP): A Review of Clinical Symptoms and Treatment Approaches in Clinical Practice*, 4 J. Clin Neuromusc. Dis. 174, 177 (2003), filed as Ex. 24(f) (ECF No. 16-8) ("Gorson & Ropper"). And Dr. Jeret deemed molecular mimicry (an autoimmune mechanism in which foreign antigens with similarity to a self-structure/tissue result in the production of antibodies against the antigen that mistakenly then attack self) an "immune error" by which demyelinating neuropathies like GBS or CIDP could occur. Jeret Rep. at 16. As additional support for a causal link, Dr. Jeret observed that an organization for those struck by GBS/CIDP specifically noted the dangers of vaccination a second time if the disease followed an earlier vaccination. GBS|CIDP Foundation International, https://www.gbscidp.org/faq/should-i-get-the-flu-vaccine-if-i-have-gbs-or-have-cidp/ (last visited Feb. 27, 226). Dr. Jeret offered no other more granular explanation for how the Tdap vaccine could cause CIDP, however.

Dr. Jeret also mentioned other aspects of Petitioner's course relevant to her entitlement burden, maintaining that the medical record supported the conclusion that the Tdap vaccine likely "did cause" Petitioner's injury. Jeret Rep. at 16–17. Petitioner had undergone extensive testing for an explanation for her symptoms, including bloodwork, a bone marrow biopsy, and a variety of imaging, but nothing was identified other than the vaccine. *Id.* at 16. Under such circumstances, causality assessment criteria established by the World Health Organization suggested it was "very likely" the vaccine was causal. J.-P Collet et al., *Monitoring Signals for Vaccine Safety: The Assessment of Individual Adverse Event Reports by an Expert Advisory Committee*, 78 Bulletin of the World Health Organization 178, 181 (2000), filed as Ex. 24(d) (ECF No. 16-6).

Regarding onset, Dr. Jeret maintained Petitioner's CIDP symptoms most likely began 28 days after the November 15, 2020 vaccination (December 13, 2020). Jeret Rep. at 16. By that time, she was experiencing "chills and loose stools" for which she sought treatment December 15th. *Id.* at 7; Ex. 5 at 59–62. Later that month, and after her neurologic symptoms had become more pronounced, she informed neurology specialists like Dr. Gould that she had first felt "sudden onset of bilateral hand coldness, numbness, and tingling" on December 13th. Ex. 9 at 374–77. Such an onset date was consistent with vaccine causality, and in support Dr. Jeret invoked the 3-42-day timeframe required to prove a Table flu vaccine-GBS claim. Jeret Rep. at 15.

2. Dr. Noga Or-Geva — Dr. Or-Geva is a neuroimmunology research scientist, and she prepared two written reports opining that the Tdap vaccine could cause a peripheral neuropathy like CIDP. Report, dated Aug. 14, 2023, filed as Ex. 22 (ECF No. 18-1) ("First Or-Geva Rep."); Report, dated June 7, 2024, filed as Ex. 29 (ECF No. 32-1) ("Second Or-Geva Rep.").

Dr. Or-Geva received her bachelor's degree from Tel Aviv University, and her master's degree and Ph.D. in Immunology and Regenerative Medicine at the Weizmann Institute of Science. *See* Curriculum Vitae, filed as Ex. 23 (ECF No. 18-2) ("Or-Geva CV") at 1. She is currently a

research scientist in Neuroimmunology and Transplantation Medicine at Stanford University, where she conducts research on post-infectious neuroimmune and psychiatric disease aimed at the discovery of novel mechanisms and the identification of biomarkers to improve patient treatment. *Id.* She has also published numerous articles in areas related to neuroimmunology. *Id.* at 2–3.

*First Report*

Dr. Or-Geva's first report included a summary of Petitioner's medical history (First Or-Geva Rep. at 3–5), and she accepted the CIDP diagnosis (although her credentials and expertise do not extend to diagnosing neurologic conditions). She then provided an overview of autoimmune disease processes, and how vaccines might arguably relate to them (through the functioning of the immune system). *Id.* at 6–7.

CIDP, Dr. Or-Geva contended, is known as an acquired autoimmune condition affecting the peripheral nervous system in approximately up to 8 per 100,000 individuals. First Or-Geva Rep. at 16 (citing H. Koller et al., *Chronic Inflammatory Demyelinating Polyneuropathy*, 352 N Engl J Med 1343 (2005), filed as Ex. 26 (ECF No. 21-1); B. Kieseier et al., *Immune-Mediated Neuropathies*, 4 Nature Rev. 1 (2018), filed as Ex. 26(b) (ECF No. 20-10)). Its exact cause remains unidentified, but studies suggest that both cell-mediated and antibody-mediated immune responses are likely contributors. First Or-Geva Rep. at 17. Vallat et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Diagnostic and Therapeutic Challenges for a Treatable Condition*, 9 Lancet neurol 402 (2010), filed as Ex. 27(q) (ECF 25-1). Unlike GBS, where approximately 70% of cases are preceded by infections, such as *Campylobacter jejuni*, only a small fraction of CIDP cases follow an infectious or post-vaccination event, and thus, suggesting a pathogenesis possibly involving molecular mimicry. First Or-Geva Rep. at 17.

Certain immune response stages or components are relevant to CIDP's pathogenesis. A type of T-helper cell (T cells that assist B cells in the production of antibodies) has "been found to be elevated in CIDP," and hence reflect "a skewing towards an inflammatory autoimmune chronic response." First Or-Geva Rep. at 7. At the same time, the Tdap vaccine (a refined version of the whole-cell pertussis form that was utilized more than 30 years ago) is also associated with encouragement of a T-helper cell response. *Id.* at 8; R. da Silva Antunes et al., *Th1/Th17 Polarization Persists following Whole-Cell Pertussis Vaccination despite Repeated Acellular Boosters*, 128 J. Clin Invest 3853 (2018), filed as Ex. 25 (ECF No. 19-6) ("da Silva Antunes"). Receipt of a vaccine can "provoke[] an inflammatory reaction mediated by innate immune cells, triggering cytokine secretion and antigen presentation." First Or-Geva Rep. at 8. B cells, according to Dr. Or-Geva, play a vital role in generating and discharging antibodies that bind to foreign antigens. *Id.* And because the immune system has evolved to allow for the creation of a vast array of antibodies from various B-cell components (i.e., polyclonal B cell response), "[t]his strategy…boosts the chances of an effective response against pathogens but also raises the potential

risk of autoimmune disease due to unintended immune reactions against the body's own molecules." *Id.* Similarly, the involvement of various inflammatory cells and immune mediators (i.e., cytokines and chemokines) in CIDP's pathogenesis, further emphasizes its complex immune pathology. *Id.* at 18–20.

Animal models (specifically, Experimental Allergic Neuritis ("EAN") and Spontaneous Autoimmune Peripheral Polyneuropathy ("SAPP")), according to Dr. Or-Geva, have been instrumental in helping medical science understand CIDP, by providing further information about the condition's likely pathologic features. First Or-Geva Rep. at 22; K. Hagen & S. Ousman, *The Immune Response and Aging in Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, 18 J. Neuroinflammation 1, 12 (2018), filed as Ex. 25(v) (ECF No. 20-4). CIDP is likely a multifactorial autoimmune disease that involves innate immunity, T-cell responses, and autoantibody attacks, therefore challenging the notion that CIDP is solely mediated by autoantibodies, as once previously thought. First Or-Geva Rep. at 22. Dr. Or-Geva added that "the spectrum of autoantibodies found in CIDP patients is large, with at least some of these patients exhibiting identical auto-ganglioside antibodies to those seen in patients with GBS." *Id.*

When discussing vaccine-induced autoimmunity, Dr. Or-Geva noted that vaccines are often developed without an exhaustive understanding of their immune activation mechanisms—although it is known they trigger inflammatory responses that promote cytokine secretion and antigen presentation to T and B cells. First Or-Geva Rep. at 23. While a causal connection between the flu vaccine and GBS has been identified, other associations between vaccination and other autoimmune disease remain unclear. Nevertheless, causality can be inferred through several factors, including event timelines, consistency of adverse event report, biological plausibility, as well as genetic and environmental triggers. *Id.*; N. Agmon-Levin et al., *Vaccines and Autoimmunity*, 5 Nat. Rev. Rheumatol. 648 (2009), filed as Ex. 25(c) (ECF No. 18-5). And though the primary goal of vaccination is to stimulate immunity, there are instances in which it can lead to a more severe reaction, especially in the context of combination vaccines such as Tdap. First Or-Geva Rep. at 23. Dr. Or-Geva also invoked the concept of a "two-hit" hypothesis for autoimmunity, in which a disease process results from two separate triggers (i.e., a genetic predisposition and then an external factor, such as an infection or vaccine). *Id.* Despite significant peptide similarity between viral and human proteins, it is well understood that not all individuals go on to develop an autoimmune disease—but susceptible individuals do. *Id.* at 24.

It is true that combined vaccines, such as Tdap, are designed to improve immune protection against multiple diseases by reducing the number of injections, as they can trigger a more robust immune response due to the activation of several antigens simultaneously. *Id.* at 25. While such polyclonal activation can strengthen immunity, it can also diminish the overall immune response and lead to an increased risk of autoimmune reactions. *Id.* Dr. Or-Geva notes that the vaccine's components, including the tetanus toxoid and pertussis antigens, have been found to cross-react

with self-antigens like cardiolipin, which can lead to potential neurological conditions. *Id.*; *see also* T.R. Poulsen, et al., *Kinetic, Affinity, and Diversity Limits of Human Polyclonal Antibody Responses against Tetanus Toxoid*, 179 J. Immunology 3841 (2007), filed as Ex. 27(a) (ECF No. 23-5); V. Waters & S. Halperin, *Bordetella Pertussis, in Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases* 2619, (2015), filed as Ex. 27(w) (ECF No. 25-7).

Similarly, the aluminum adjuvant in the Tdap vaccine and the cytokine profiles observed in CIDP indicate notable overlaps, specifically in the stimulation of pro-inflammatory cytokines like IL-1β, IL-6, and TNT-α. First Or-Geva Rep. at 28. As a result, there is a suggested linkage between aluminum-induced immune responses and CIDP's inflammatory processes, according to Dr. Or-Geva. *Id.* Thus, the observed similarities between CIDP's cytokine alterations and the effects of aluminum in Tdap underscore the importance of understanding the immune system and dysregulation of it, as it can ultimately lead to heightened inflammation and possible adverse reactions. *Id.*

Dr. Or-Geva discussed several post-vaccination studies that show a connection between Tdap and a new onset, recurrence or worsening of CIDP, with some patients demonstrating a rapid symptoms onset following vaccination. First Or-Geva Rep. at 25, 26; P.E. Doneddu, et al., *Risk Factors for Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP): Antecedent Events, Lifestyle and Dietary Habits. Date from the Italian CIDP Database*, 27 Eur. J. Neurology 136, 138 (2020), filed as Ex. 25(p) (ECF No. 19-8) (finding that an antecedent event, such as vaccination, occurred within one to forty-two days before CIDP onset in 1.5% of the studied cohort); *see also* J. Pritchard, et al., *Risk of Relapse of Guillain-Barré syndrome of Chronic Inflammatory Demyelinating Polyradiculoneuropathy following Immunization*, 73, J Neurol Neurosurg. Psychiatry 348, 348 (2002), filed as Ex. 27(b) (ECF No. 23-6) (concluding that "[o]f the patients with CIDP who experienced a relapse after immune[z]ation, two relapses occurred among 23 patients who received the tetanus vaccine, giving a risk of relapse of 8.7%."). Moreover, the Pollard & Selby case study discussed an individual who experienced relapses of an inflammatory demyelinating polyneuropathy after receiving three doses of the tetanus toxoid vaccine—suggesting that "[t]here is little doubt that the three clinical episodes of demyelinating neuropathy resulted from the administration of tetanus toxoid. J.D. Pollard & G. Selby, *Relapsing Neuropathy due to Tetanus Toxoid*, 37 J. Neurological Sciences 113, 117 (1978), filed as Ex. 26(z) (ECF No. 23-4) ("Pollard & Selby"). Dr. Or-Geva thus maintained that Pollard & Selby, "serves as a potent indicator that the tetanus toxoid may not only exacerbate existing CIDP but also potentially act as a trigger for its onset in predisposed individuals." First Or-Geva Rep. at 26.

To further bulwark Petitioner's proposed theory, Dr. Or-Geva conducted BLAST searches to analyze potential structural similarities between the Tdap vaccine and CIDP. *Id.* at 29. Based on the results, Dr. Or-Geva maintained that the structural and sequence similarities between components of the Tdap vaccine and proteins associated with CIDP, strongly suggest molecular

mimicry at play. *Id.* at 30. Moreover, such similarities could instigate autoimmune responses, leading to the production of autoantibodies against CIDP-related proteins, especially in individuals who develop CIDP post-vaccination. *Id.* Dr. Or-Geva further emphasized the individuals who experience this adverse reaction might also be predisposed to underlying sensitivities influenced by genetic and environmental factors. *Id.*

In conclusion, Dr. Or-Geva briefly discussed the three *Althen* prongs—arguing that Petitioner satisfied each one. Specifically, Dr. Or-Geva maintained that the evidence relied upon herein strongly suggests that the Tdap's components are likely to trigger molecular mimicry and potentially lead to an autoimmune response that results in the development of CIDP. First Or-Geva Rep. at 31. In addition, Dr. Or-Geva noted that the primary cause of Petitioner's remains "nebulous"; however, the absence of other alternative triggers—after extensive investigation and testing—strongly indicates the proposition that Petitioner's receipt of the Tdap vaccine was the likely instigator for her subsequent development of CIDP. *Id.* at 33. Lastly, Petitioner's symptoms onset began on December 13, 2020, twenty-eight days post-vaccination, which is consistent with an accelerated onset often seen in patients with acute-onset CIDP. *Id.* at 33.

*Second Report*

Dr. Or-Geva spent the entirety of her second report addressing comments made by Drs. Sriram and He. In response to Dr. Sriram's opinion that Petitioner's CIDP was more likely than not caused by monoclonal gammopathy of undetermined significance ("MGUS"), Dr. Or-Geva argued that MGUS should not be considered as a usual cause for CIDP given that "[e]pidemiological studies show that MGUS is prevalent in the general population, especially among older adults, [but] only a small fraction of these patients develop neuropathy." Second Or-Geva Rep. at 2; H. Chaudhry, et al., *Monoclonal Gammopathy—Associated Peripheral Neuropathy: Diagnosis and Management*, 92 Mayo Clin Proc. 838, 840 (2017), filed as Ex. 30(f) (ECF No.32-7) (noting that MGUS is prevalent in approximately three to four percent of the normal adult population over 50 year of age). Indeed, Dr. Or-Geva noted that several studies suggest that the prevalence of neuropathy among MGUS patients ranges between five to seventeen percent—further indicating that neuropathy is not a common presentation in MGUS. Second Or-Geva Rep. at 2. More importantly, Dr. Or-Geva argued that MGUS-associated neuropathy typically presents as a "chronic, slowly progressive large-fiber sensory-motor polyneuropathy, initially affecting the lower extremities" whereas CIDP oftentimes presents more acutely and accompanied by a relapsing-remitting nature. *Id.* Additionally, alternative causes were ruled out (i.e., diabetes, chronic alcohol consumption, and POEMS) in Petitioner's case, and the temporal association between vaccination and onset was acknowledged by Dr. Sriram. *Id.* at 3.

The remainder of Dr. Or-Geva's second report addressed Dr. He's comments. *See generally* Second Or-Geva Rep. at 5–20. Dr. Or-Geva disagreed with Dr. He's assertion that there is no

evidence at all to suggest that Tdap can cause a strong immune response capable of dysregulating peripheral tolerance. *Id.* at 6. Indeed, she referenced several articles that show otherwise. *Id.*; L. Guimaraes, et al., *Vaccines, Adjuvants and Autoimmunity*, 100 Pharmacological Research 190 (2015), filed as Ex. 25(t) (ECF No. 20-2) (supporting the notion that adjuvants like aluminum slats used in Tdap, are designed to enhance the immune response by activating antigen-presenting cells and promoting the release of pro-inflammatory cytokines); O. Hen, et al., *Dysautonomia following Tetanus, Diphtheria, and Pertussis Vaccine (Tdap): The First Case of Extreme Cachexia caused by Autoimmune/Inflammatory Syndrome Induced by Adjuvants (ASIA Syndrome) in a Human*, 57 Medicina 1 (2021), filed as Ex 25(w) (ECF No. 20-5) (reporting a case of an 18-year old female who presented with extreme cachexia due to severe dysautonomia caused by the ASIA syndrome induced by the tetanus, diphtheria, and pertussis vaccine). Although rare, Dr. Or-Geva maintained that these, among other documented cases showing a temporal and mechanistic link to the onset of autoimmune diseases, "challenge the assertion that vaccines cannot induce strong immune responses like those observed in natural infections." Second Or-Geva Rep. at 6. Indeed, Dr. Or-Geva noted that "[r]esearch indicates that adjuvants in vaccines like TDAP can specifically enhance Th1 and Th17 responses, potentially exceeding typical activation levels and leading to a pro-inflammatory state conducive to autoimmunity." *Id.*; L. Guimaraes, et al., *Vaccines, Adjuvants and Autoimmunity*, 100 Pharmacological Research 190 (2015), filed as Ex. 25(t) (ECF No. 20-2).

Trained immunity, explained Dr. Or-Geva, is the process by which innate immune cells are reprogrammed via metabolic or epigenetic changes that cause an enhanced or nonspecific immune response following subsequent exposure to various antigens. Second Or-Geva Rep. at 7. Studies have demonstrated that vaccines can induce a trained immunity response, allowing for the potential to develop autoimmune conditions, according to Dr. Or-Geva. *Id.*; A. Beignon, et al., *Trained Immunity as a Possible Newcomer in Autoinflammatory and Autoimmune Diseases Pathophysiology*, 9 Front. Med. 1085339 (2023), filed as Ex. 30(b) (ECF No. 32-3) (stating that the increased responsiveness of innate and inflammatory responses may contribute to the development and/or maintenance of chronic or recurrent inflammation and tissue destruction). Notably, the Tdap vaccine is given repeatedly, therefore, the concept of innate immunity is highly relevant. Second Or-Geva Rep. at 7. Here, Dr. Or-Geva emphasized the timing and sequence of Petitioner's CIDP symptoms post-vaccination and noted that they "provide a plausible narrative that trained immunity might have contributed to her condition" having triggered a heightened immune response. *Id.*

Dr. Or-Geva maintained that her proposed theory of molecular mimicry is a well-documented mechanism and is "crucial in the development of autoimmune diseases and is not limited to infections but is also triggered by vaccines." Second Or-Geva Rep. at 9. She further acknowledged that sequence homology is not a conclusive factor for determining its role in any disease, however, it remains an important factor, nonetheless. *Id.* And the context and specifics of an individual's immunological history (i.e., genetic background and environmental exposures) by which the homologies interact with the immune system are critical to understanding the role of

molecular mimicry as a general instigator of autoimmune diseases and vaccine-induced autoimmune diseases. *Id.* at 10.

Dr. Or-Geva then briefly addressed Dr. He's assertion that Petitioner's pre-existing conditions (i.e., Hashimoto's thyroiditis, arthritis, and fibromyalgia) and their established association with CIDP presents a more probable cause of her CIDP than her receipt of the Tdap vaccine. Second Or-Geva Rep. at 15. In response, Dr. Or-Geva argued that Petitioner's medical history simply provided a "backdrop" for her overall heightened autoimmune reactivity, and that the timing of her CIDP onset and the absence of any new autoimmune symptoms or triggers prior to the Tdap vaccine "argue strongly for a vaccine-related onset." *Id.* Petitioner's medical records also do not indicate a significant exacerbation or changes in her pre-existing autoimmune conditions that would typically been seen with a clinical presentation of CIDP. *Id.* Moreover, "[t]he sharp demarcation between the vaccination date and the onset of symptoms strongly supports the vaccine as the trigger rather than a gradual development expected from existing conditions like fibromyalgia." *Id.*

To conclude, Dr. Or-Geva reiterated her prior arguments regarding the *Althen* prongs and noted that she has provided sound and reliable evidence to support the Tdap vaccine as the more likely cause of her subsequent development of CIDP. Second Or-Geva Rep. at 17–21.

B. *Respondent's Experts*

1. Dr. Subramaniam Sriram — Dr. Sriram, a clinical and academic neurologist, offered a written report for Respondent. Report, dated Feb. 14, 2024, filed as Ex. B (ECF No. 30-2) ("Sriram Rep."). Although Dr. Sriram agreed that CIDP was a proper diagnosis for Petitioner's symptoms, he proposed it was more likely associated with "Monoclonal Gammopathy of Unknown Significance" ("MGUS") than attributable to receipt of a Tdap vaccine. Sriram Rep. at 10.

Dr. Sriram received a Bachelor of Medicine and a Bachelor of Surgery from the University of Madras in Madras, India. *See* Curriculum Vitae, filed as Ex. D (ECF No. 34-8) ("Sriram CV") at 1. He then served as an intern and resident at Wayne State University and completed a residency in Neurology at Stanford University, where he also served as chief resident and eventually completed a fellowship in Neuroimmunology. *Id.* Dr. Sriram is board-certified in both Neurology and Internal Medicine. *Id.* In addition, he holds academic positions as a Professor of Experimental Neurology and Therapeutics, as well as a joint appointment Professor of Pathology, Microbiology, and Immunology at Vanderbilt University Medical Center. *Id.* at 1–2. Dr. Sriram is heavily involved in clinical work as he directs the Multiple Sclerosis Clinic at Vanderbilt University Medical Center where he sees approximately 1,450 patients a year (including both inpatient and outpatient). Sriram Rep. at 1. He has also published numerous articles on various aspects of clinical and immune mediated disease of the nervous system. Sriram CV at 9–21.

Dr. Sriram, like his neurologic counterpart expert Dr. Jeret, provided a summary of Petitioner's medical history. Sriram Rep. at 2–7. He also embraced CIPD as the proper diagnosis, given Petitioner's clinical presentation and the results of electrodiagnostic testing. *Id.* at 7–8. Dr. Sriram did, however, identify from Petitioner's history an alternative etiologic explanation for her CIDP: that it was an "MGUS associated form of peripheral neuropathy." *Id.* at 8.

In support of this contention, Dr. Sriram noted the existence of medical literature observing that there is a "well documented" association between peripheral neuropathies and monoclonal gammopathy (meaning a blood disorder in which abnormal plasma cells overproduce a single type of protein, called an M protein). Sriram Rep. at 9; J. Vallat et al., *The Wide Spectrum of Pathophysiologic Mechanisms of Paraproteinemic Neuropathy*, 96 Neurology 214 (2021), filed as Ex. B Tab 4 (ECF No. 34-4) (emphasizing the well-documented association between peripheral neuropathy and monoclonal gammopathy, and noting that such association has also been recognized in cases of MGUS); E. Nobile-Orazio et al., *Peripheral Neuropathy in Monoclonal Gammopathy of Undetermined Significance: Prevalence and Immunopathogenic Studies*, 85 Acta Neurol Scand 383, 388 (1992), filed as Ex. B Tab 2 (ECF No. 34-2) ("Nobile-Orazio") (demonstrating that peripheral neuropathies can be observed in any type of MGUS or malignant monoclonal gammopathy and that monoclonal gammopathy can be detected in three to five percent of all peripheral neuropathies). MGUS can often be benign, but it also features "a risk of progression to malignant disorders," and has been associated with peripheral neuropathies in a meaningful minority of MGUS patients. Sriram Rep. at 9; Nobile-Orazio at 388. In such cases, the neuropathy would "present as a chronic, slowly progressive large-fiber sensory-motor polyneuropathy, usually affecting initially the lower extremities." Sriram Rep. at 9. Notably, Dr. Sriram contended, pain, dysesthesia, and cramps—some of which Petitioner reported—were features of peripheral neuropathy in the context of MGUS. Y. Rajabally et al., *Prevalence, Correlates and Impact of Pain and Cramps in anti-MAG Neuropathy: A Multicentre European Study*, 25 Euro. J. Neurology 135 (2017), filed as Ex. B Tab 7 (ECF No. 34-7) at 137 Table 1.

By contrast, Dr. Sriram contended, it was far less likely that the Tdap vaccine explained Petitioner's CIDP. He denied that medical science associated the vaccine with CIDP, contrasting present circumstances with the evidence linking GBS to vaccines due to support for "molecular mimicry with bacterial or viral antigens triggers." Sriram Rep. at 10. In addition, Petitioner's neurologic treaters never proposed a vaccine cause, with one (Dr. Dalakas) downplaying the likelihood of a relationship. *Id.*; *see also* Ex. 6 at 43.

Dr. Jeret's causation theory also, in Dr. Sriram's view, over-relied on the temporal association with vaccination. Sriram Rep. at 10. And Dr. Jeret had cited a case report involving an acute polyneuropathy like GBS. P. McCombe, et al., *Chronic Inflammatory Demyelinating Polyradiculoneuropathy: A Clinical and Electrophysiological Study of 92 Cases*, 110 Brain 1617 (1987), filed as Ex. 24(b) (ECF No.16-10). But Dr. Sriram distinguished CIDP from GBS, noting

that the two conditions were ultimately "unlike one another," with "no candidate antigens identified for CIDP"—meaning that molecular mimicry as a pathogenic mechanism could not simply be assumed relevant—and no other infectious or causal factors known for CIDP otherwise. Sriram Rep. at 10.

2. Dr. You-Wen He — Dr. He, a medical doctor and academic immunologist, prepared one report for Respondent. *See* Report, dated Feb. 12, 2024, filed as Ex. A (ECF No. 30-1) ("He Rep.").

Dr. He is a Professor of Integrative Immunobiology in the Department of Integrative Immunobiology at Duke University School of Medicine. *See* Curriculum Vitae, dated Nov. 14, 2023, filed as Ex. C (ECF No. 33-43) ("He CV") at 1. He received his medical degree from the Fourth Military Medical University in China and received his Ph.D. from the Miami School of Medicine. *Id.* Dr. He went on to complete a senior fellowship in the Department of Immunology at the University of Washington and completed his residency at Qindu Hospital in China. *Id.* Dr. He has been conducting research in immunology since he graduated from medical school in 1986. He Rep. at 1. Over the past 27 years, he has been invited to lecture nationally and internationally on the topic of host immune responses to microbial infections and tumors. *Id.* Dr. He has also served as a co-Principal Investigator for four clinical trials focusing on cancer immunotherapy using personalized cancer vaccines. *Id.* In addition, he has been published extensively and has served as an ad hoc reviewer for more than 30 scientific journals. *Id.*; He CV at 7–18.

Dr. He defined CIDP as "an acquired, immune-mediated neuropathy affecting peripheral nerves and nerve roots, characterized by a relapsing-remitting or progressive course, glucocorticoid responsiveness, and electrodiagnostic or pathologic features of demyelination." He Rep. at 4; R. Lewis et al., *Chronic Inflammatory Demyelinating Polyneuropathy: Etiology, Clinical Features, and Diagnosis*, UpToDate (2023), filed as Ex. A Tab 1 (ECF No. 33-1). While specific predisposing risk factors or an underlying cause for CIDP is currently unknown, there is evidence to support the notion that it can have multiple triggers. He Rep. at 5. There is not, however, in Dr. He's view any evidence causally linking the Tdap vaccine to the development of CIDP. *Id.* To bulwark this assertion, Dr. He cited to several systemic studies that had failed to identify any causal link between the Tdap vaccine and CIDP. *Id.* at 5, 6; *see also Adverse Effects of Vaccines: Evidence and Causality* (K. Stratton et al., eds., 2012), filed as Ex. A Tab 2 (ECF No. 33-2) (the "2012 IOM Report") (concluding the evidence as inadequate to accept or reject a causal relationship between diphtheria toxoid-, tetanus toxoid-, or acellular pertussis-containing vaccine and CIDP); M.Z. Dudley et al., *The State of Vaccine Safety Science: Systematic Reviews of the Evidence*, 20 Lancet Infect Dis e80, e84 (2020), filed as Ex. A Tab 3 (ECF No. 33-3) (finding that vaccines have not been shown to cause CIDP). Taken together, and in light of the fact that millions of Tdap vaccine are administered annually, Dr. He maintained that there is simply no reliable evidence to associate the Tdap vaccine and CIDP. He Rep. at 6.

Dr. He then briefly responded to comments by Drs. Jeret and Or-Geva. Dr. He first agreed with Dr. Jeret that a temporal relationship between Petitioner's receipt of the Tdap vaccine and her development of CIDP did exist—but added that there was no additional clinical evidence to further support a causal link. He Rep. at 6. Moreover, Dr. He emphasized that Petitioner was diagnosed with "multiple diseases" prior to her vaccination, which *also* had a temporal relationship. *Id.* (emphasis added).

With respect to molecular mimicry as the relevant pathologic mechanism, Dr. He argued that the "theory has been strongly challenged by scientific evidence from large sequencing of proteomes of microbial pathogens that demonstrate massive peptide sharing between viral and human proteomes." He Rep. at 7, 8, 9; *see also* B. Trost et al., *Bacterial Peptides are Intensively Present throughout the Human Proteome*, 1 Self/Nonself 71, 72 (2010), filed as Ex. A Tab 8 (ECF No. 33-8) (demonstrating an overlap between bacterial proteomes and the human proteome at the nonamer level (i.e., 9 amino acids) for *Corynebacterium diphtheriae*, *Clostridium tetani*, and *Bordetella pertussis* as 2608, 2594, and 5612, respectively); D. Kanduc et al., *Massive Peptide Sharing between Viral and Human Proteomes*, 29 Peptides 1755, 1757 (2008), filed as Ex. A Tab 6 (ECF No. 33-6) (demonstrating that 90% of the viral 5-mer peptides (i.e., a consecutive stretch of 5 amino acids) are widely and repeatedly scattered throughout the human proteome and a total of 2,907,096 matches were found between viral and human proteome).Were autoimmunity likely based solely on amino acid sequence similarity, Dr. He maintained, the above-referenced sequence analogies "would result in a 100% autoimmune disease rate in the general population after either infection or vaccination, according to the molecular mimicry theory." He Rep. at 9. Thus, he opined that mere sequence homology is insufficient to conclude a causal relationship between vaccination and the development of autoimmune diseases. *Id.*

The remaining portion of Dr. He's report responded to Dr. Or-Geva's opinion. *See generally* He Rep. at 9–25. He first criticized Dr. Or-Geva's attempt to equate infection-induced immune activation with *immunization-induced* activation—noting that "[i]nfections are fundamentally different from vaccination in their capability to induce host immune responses." *Id.* at 10. For example, the microbial pathogens of wild type bacterium or viruses contain many components that allow for the stimulation of a much broader immune response than that of their corresponding vaccines. *Id.*; U. Koppe et al., *Recognition of Streptococcus pneumoniae by the Innate Immune System*, 4 Cellular Microbiology 460 (2012), filed as Ex. A Tab 23 (ECF No. 33-23); Y.S. Kang et al., *The C-type Lectin SIGN-R1 Mediates Uptake of the Capsular Polysaccharide of Streptococcus pneumoniae in the Marginal Zone of Mouse Spleen*, 101 PNAS 215 (2004), filed as Ex. A Tab 22 (ECF No. 33-22). In addition, wild type bacterium and viruses have natural, uncontrolled infection pathways, oftentimes penetrating the many layers of peripheral regulation to lead to autoimmune diseases such as CIDP. He Rep. at 11; *see also* W. Land, *Role of DAMPs in Respiratory Virus-Induced Acute Respiratory Distress Syndrome—with a Preliminary Reference to SARS-CoV-2 pneumonia*, 2 Genes & Immunity 141 (2021), filed as Ex. A Tab 27 (ECF No. 33-27) (finding that

during the infection process, a large number of non-immune cells (i.e., epithelial cells) undergo cell death and subsequently release large quantities of immune stimuli known as danger-associated molecular patterns ("DAMPs"), and thus further exacerbate the host immune response). On the other hand, vaccinations are administered intramuscularly and can thus be highly controlled. He Rep. at 11.

In response to Dr. Or-Geva's discussion of the immune response to vaccination and the role of the innate and adaptive immune systems as well as the T-helper cells; Dr. He emphasized that T-helper cells provide "general processes of the host immune reaction and [are] involved in many different physiological and pathological processes, including maintaining host immune homeostasis, mounting immune responses to immunizations and infections, fighting against tumor cells, and participating in various autoimmune diseases[, such as CIDP]." He. Rep. at 12; J.M. Carter et al., *A History and Atlas of the Human CD4(+) T Helper Cell*, 11 Biomedicines (2023), filed as Ex. A Tab 31 (ECF No. 33-31). Thus, the involvement of T-helper cells in immunization and autoimmune disease does not mean they instigate a disease process. He Rep. at 12. Similarly, Dr. Or-Geva's suggestion that the general concept of trained immunity is at play herein in provoking an autoimmune response leading to Petitioner's development of CIDP was merely an unsubstantiated hypothesis. *Id.* at 13. Instead, Dr. He maintained that "[i]mmune responses are subjected to counter-balance controls by negative regulators, including regulatory T cells, immune checkpoint molecules/receptors, regulatory B lymphocytes, tolerogenic dendritic cells, and other intrinsic pathways." *Id.* at 14.

Regarding the theory of molecular mimicry and its application herein, Dr. He argued that there are known scientific facts that strongly argue against the theory. First, and as mentioned earlier, large sequencing of proteomes of microbial pathogens have unequivocally demonstrated that microbial pathogens and human proteomes have massive sequence sharing, but do not result in commonplace disease. He Rep. at 14; *see also* A. Kusalik, et al., *Widespread and Ample Peptide Overlapping between HCV and Homo sapiens Proteomes*, 28 Peptides 1260 (2007), filed as Ex. A Tab 7 (ECF No. 33-7); D. Kanduc, et al., *Massive Peptide Sharing between Viral and Human Proteomes*, 29 Peptides 1755 (2008), filed as Ex. A Tab 6 (ECF No. 33-6). Second, T-cell receptors are degenerative and have the capability to recognize different epitopes for polyspecificity, making it unlikely that mimics will inerrantly cause autoimmune cross-reactions. Lastly, cross-reactive T lymphocytes and antibodies to self-proteins and foreign antigens are widely detectable in humans, but do not necessarily result in disease simply due to their presence. He Rep. at 15, 19; *see also* 2012 IOM Rep. at 70–71. Thus, not only did the IOM state that "[f]inding a tissue-specific antibody response following exposure to an exogenous agent is also, by itself, not proof of molecular mimicry as the pathologic mechanism of disease" but that "in some circumstances, infection with viruses that express antigens having immunologic cross-reactivity with self-proteins can actually protect against autoimmune disease in certain animal models." He Rep. at 15; 2012 IOM Rep. at 70–71.

Ultimately, the critical determinant of autoimmune disease development upon antigen stimulation, according to Dr. He, is the strength of the immune activation induced by the overall immunological encounters (i.e., infection or immunization), as opposed to the mere existence of sequence homology between foreign antigens and self-proteins. He Rep. at 15–16. And here, Petitioner had multiple pre-existing autoimmune conditions, including Hashimoto's Thyroiditis and Fibromyalgia, that were more likely to have created the context for disease. Dr. He stated that it is well understood that "individuals diagnosed with one autoimmune disorder often exhibit a heightened risk of contracting others, a condition termed 'polyautoimmunity.'" *Id.* at 16 (citing Rojas et al., 2018). However, absent any evidence supporting an association between receipt of the Tdap vaccine and the development of CIDP in genetically susceptible individuals, Dr. He maintained that Petitioner's genetic vulnerability elevated her to a heightened risk of contracting CIDP, regardless of her receipt of the Tdap vaccine. He. Rep. at 16.

## III. Procedural History

As noted, the claim was initiated approximately three years ago, and after its activation from "pre-assignment review" the Petition was assigned to my own docket. In August 2023, Respondent filed his Rule 4(c) Report objecting to entitlement, and the parties subsequently went about obtaining expert reports, with that process being completed by June 2024. I thereafter set a schedule for resolution of the claim via ruling on the record, and the briefing process was completed in June 2025.

## IV. Parties' Arguments

*Petitioner*

Petitioner contends that her CIDP was likely caused by her receipt of the Tdap vaccine on November 15, 2020. Reply at 1. In her briefing, Petitioner address all three prongs of the test set by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005) for causation claims.

She first maintains that she has met her burden pursuant to *Althen* prong one by demonstrating preponderant evidence of a "biologically plausible (and reliable) medical theory." Br. at 52. Although the exact etiology of CIDP remains unclear, it is understood that both cell and antibody-mediated immune responses are implicated. *Id.* at 64; First Or-Geva Rep. at 17–18. The CIDP disease process, Petitioner maintains, is characterized by the infiltration of inflammatory cells, such as T cells and macrophages, as well as various immune mediators like cytokines and chemokines, thus highlighting its multifaceted pathology. Br. at 64. Because some CIDP cases are known to follow an infectious or post-vaccination event, the disease's pathogenesis likely involves molecular mimicry (which is already associated with infectious triggers). *Id.* To bulwark this argument, Dr. Or-Geva relied upon several animal models (EAN and SAPP), which purportedly

demonstrate molecular mimicry in CIDP's pathogenesis. *Id.* at 76; First Or-Geva Rep. at 21–22. In addition, Petitioner had offered proof of structural and sequence similarities between components of the Tdap vaccine and various proteins associated with CIDP. Br. at 70.

Petitioner further emphasizes that the Tdap vaccine (a combination vaccine containing tetanus and diphtheria toxoids and acellular pertussis antigens) has "a heightened ability to activate a robust immune response," and can thus "trigger broad immune responses due to the activation of multiple antigens simultaneously." Br. at 69. While such polyclonal activation can enhance immunity, it can also cause an increased risk of autoimmune reactions. *Id.* Studies show that (1) there is an association between the Tdap vaccine and cases of autoimmune conditions, such as transverse myelitis, and (2) the tetanus toxoid and pertussis antigens have been found to trigger immune responses that cross-react with self-antigens, and lead to potential neurologic issues. *Id.*; *see also* First Or-Geva Rep. at 24. Indeed, Petitioner emphasized that some post-vaccination studies have demonstrated a connection between the Tdap vaccine and the onset, recurrence, or exacerbation of CIDP. *Id.*

Thus, based on identified homology between the components of the Tdap vaccine and CIDP-related proteins, epidemiological studies, and Petitioner's clinical presentation (i.e., rapid symptom onset), Petitioner argues that it is more likely than not that her CIDP was triggered by the Tdap vaccine via molecular mimicry, "scientifically plausible medical theory." Br. at 71, 72.

As for the second, "did cause" *Althen* prong, Petitioner maintains that she has preponderantly established a clear connection between her receipt of the Tdap vaccine in November 2020 and her subsequent development of CIDP. Br. at 76. Petitioner's medical records not only demonstrate that she exhibited no symptoms of CIDP prior to vaccination, but that "within days [of vaccination], she was diagnosed with acute onset demyelinating neuropathy." *Id.* at 77. Petitioner further emphasizes that her treating physician, Dr. Dalakas, recognized a concern for a potential link to the Tdap vaccine (although he did not conclusively confirm this association). *Id.* And because viral and bacterial illnesses were ruled out, that left only the vaccine as a possible antecedent trigger. *Id.*

Finally, Petitioner argued that the onset of her CIDP occurred within a medically acceptable timeframe following vaccination. Br. at 78. Twenty-eight days post-vaccination, Petitioner began to experience numbness and tingling in her arms and hands. *Id.* at 83. Such symptom manifestation prompted further investigation for demyelinating neuropathies, and eventually led to Petitioner's CIDP diagnosis in July 2021. *Id.* at 83. Both Drs. Jeret and Or-Geva acknowledged that an immune response beginning 3 to 42 days post-vaccination is consistent with a casual relationship based on the theory of molecular mimicry. *Id.* In fact, both of Respondent's experts concurred as to the timing of Petitioner's onset of CIDP symptom. *Id.* at 78; s*ee also* He Rep. at 6 ("[a] temporal relationship between [Petitioner's receipt of the TDaP vaccine and her CIDP onset exists"); Sriram Rep. at 10

("I agree with the diagnosis and the temporal relationship between the receipt of TdAP and the development of neurological symptoms of CIDP").

In her reply, Petitioner emphasizes that her medical records and expert opinions clearly establish that she suffers from CIDP, as her treating neurologists at Jefferson and the University of Pennsylvania "repeatedly documented the condition based on their diagnosis on clinical findings and EMG results." Reply at 2. She further maintains that both Drs. Jeret and Or-Geva provided a detailed and science-based theory supported by medical literature and prior Program cases detailing how the Tdap vaccine could lead to CIDP. *Id.* at 2, 3. Regarding *Althen* prongs Two and Three, Petitioner did not begin experiencing sensory symptoms until approximately four weeks post-vaccination, no intervening even better explains her onset of CIDP, and the purported timeframe is medically appropriate and consisted with the autoimmune nature of CIDP. *Id.* at 3. Moreover, Petitioner contends that Respondent's alternative cause argument is based merely on speculation. Noting that none of Petitioner's prior conditions are known to cause CIDP, and her treating physicians consistently ruled them out. *Id.* In short, Petitioner argues that she has satisfied her burden under *Althen* through the testimony of her experts, consistent clinical documentation, and the clear absence of a more likely alternative cause. Reply at 45.

*Respondent*

Respondent accepts Petitioner's CIDP diagnosis, but disputes that Petitioner has preponderantly proven that CIDP *can* be caused by the Tdap vaccine. Opp. at 24 n.4. Petitioner has not provided reliable scientific evidence demonstrating that tetanus toxoid-containing vaccines, such as Tdap, can cause CIDP, or that the vaccine itself or its components likely trigger molecular mimicry leading to CIDP. *Id.* at 25. Respondent notes that Dr. Or-Geva simply relies on the mere mention of molecular mimicry to support her theory, but without additional evidence tying the mechanism to the specific injury and/or vaccine at issue. *Id.* Dr. Or-Geva's reliance on demonstrations of sequential homology is not only misplaced, according to Respondent, but "[the results] are entirely expected because of the known massive sequence homologies between microbial pathogens and human proteins." *Id.* at 26 (citing First Or-Geva Rep. at 24–25). Moreover, the animal studies relied upon by Petitioner argue against the idea of molecular mimicry in CIDP's pathogenesis, in Dr. He's opinion. Rather, their results demonstrate that self-antigens alone are incapable of causing autoimmune diseases, absent the introduction of potent immune adjuvants used only experimentally (and thus in order to provoke reactions that can be studied). *Id.* at 27; *see also* First Or-Geva Rep. at 20. Notably, Respondent points out that the 2012 IOM Report specifically stated that "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. Many such homologies exist, and the vast majority of these are not associated with biologically relevant autoimmune phenomena or actual human disease." *Id.*; First Or-Geva Rep. at 11.

Respondent further argues that the second, "did cause" *Althen* prong has not been met. He notes that Petitioner suggests a "two hit" hypothesis for how the Tdap vaccine caused her CIDP – but even assuming Petitioner possessed a threshold susceptibility, there is no evidence herein that Petitioner's receipt of the Tdap vaccine likely acted as the "hit" necessary to cause a reaction. Opp. at 28. Instead (and as Dr. He maintained), Petitioner's pre-existing conditions (i.e., Hashimoto's thyroiditis, arthritis, and fibromyalgia) were far more likely causal, as all are closely associated with CIDP, consistent with polyautoimmunity. *Id.* at 29. In addition, the medical records lack any substantive causal statements that reliably connect Petitioner's CIDP to her receipt of the Tdap vaccine—with instead multiple records documenting treater uncertainty as to the most likely etiology for Petitioner's injury. *Id.*, *citing* Ex. 11 at 29 (12/20/2020 visit noting that the "exact etiology of [Petitioner's complaint [was] not clear"), Ex. 6 at 41 (4/5/2021 visit with Dr. Dalakas stating that "[w]hether this was triggered by the tetanus vaccine [Petitioner] received 3 week prior is unclear").

Lastly, Respondent briefly argues that Petitioner has failed to produce reliable evidence of a temporal relationship between the Tdap vaccine and her CIDP—noting that she solely relied on the literal temporal proximity. Opp. at 30.

## 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).[7] There is no Table claim for CIDP caused by any covered 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,

[7] 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).

486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y Health & Hum. Servs*., 165 F.3d 1344, 1352–53 (Fed.Cir.1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

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

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

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or even a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed.Cir.2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant 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. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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

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

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

B. *Legal Standards Governing Factual Determinations*

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

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL

1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

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

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

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

other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

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

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

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

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

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S.

146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

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

E. *Determination to Resolve Case without a Hearing*

I have opted to decide entitlement in this case based on written submissions and evidentiary filings, including the expert reports filed by each side. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers rather than via evidentiary hearing, where (in the exercise of their discretion) they conclude that the former means of adjudication will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The choice to do so has been affirmed on appeal. *See D'Toile v. Sec'y of Health & Human Servs.*, No. 15-85V, 2018 WL 1750619, at *2 (Fed. Cir. Apr. 12, 2018); *see also Hooker v. Sec'y of Health & Human Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *See Hovey v. Sec'y of Health & Human Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417.

## ANALYSIS

### I. Overview of CIDP and its Treatment in Program Cases

Petitioner's CIDP diagnosis is not disputed, but some discussion of the condition is still warranted. CIDP is defined as "a slowly progressive, autoimmune type of demyelinating polyneuropathy characterized by progressive weakness and impaired sensory function in the limbs and enlargement of the peripheral nerves." *CIDP*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=99346&searchterm=chronic+inflammatory+demyelinating+polyneuropathy (last visited Feb. 27, 2026). CIDP often requires persistent maintenance treatment with IVIG or corticosteroids. H. Koller, et al., *Chronic Inflammatory Demyelinating Polyneuropathy*, 352 N Engl J Med 1343, 1351 (2005), filed as Ex. 26(d) (ECF No. 21-2). GBS, by contrast, is an inflammatory polyneuropathy characterized by an *acute* onset, rapid progression, symmetric muscle weakness and hyporeflexia or areflexia. Vallat at 402. GBS is monophasic and unresponsive to steroid treatment (unlike CIDP). *Nieves v. Sec'y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148, at *35 (Fed. Cl. Spec. Mstr. May 22, 2023), *mot. for review den'd*, 167 Fed. Cl. 422 (2023). And GBS and CIDP are not understood to have the same self-targets for cross-reactive attack, with CIDP thought to involve damage occurring at the "nodes of Ranvier" running along a nerve, rather than on the general surface of the myelin. *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022).

Despite their differences, it is not uncommon for CIDP to present with GBS-like, sudden clinical symptoms, given the overlap between both conditions—and for treaters to correspondingly assume at first that they are "seeing" GBS. Because of this overlap, Program petitioners often treat CIDP as merely "long GBS." *See*, *e.g.*, *Nieves*, 2023 WL 3580148, at *35. The temptation to avoid distinguishing between the two diseases likely stems from the fact that GBS has been credibly linked to certain vaccinations (namely the influenza vaccine),[8] while the evidence supporting a causal link between CIDP and vaccines is less robust. *See Nieves*, 2023 WL 3850148, at *35 ("evidence that strongly supports a GBS-flu vaccine causal relationship rings weaker when applied to CIDP").

But it remains the case that CIDP and GBS are separate, immune-mediated polyneuropathies, even if they share many features. *See*, *e.g., Houston v. Sec'y of Health & Hum. Servs.*, No. 18-420V, 2021 WL 4259012, at *17 (Fed. Cl. Spec. Mstr. Aug. 19, 2021) (noting CIDP vs. GBS distinctions). It thus is improper when deciding a Vaccine Act claim to treat CIDP and

[8] Indeed, this causal evidence is the basis for a Table claim. 42 C.F.R. § 100.3.14 This means the Government has agreed that sufficiently-probative and reliable science on the topic exists to justify (in effect) conceding causation, at least for Program purposes. *Haskins v. Secretary of Health & Hum. Servs.*, No. 18-1776, 2020 WL 1870279 (Fed. Cl. Spec. Mstr. Mar. 13, 2020).

GBS as interchangeable.[9] *See Nieves*, 2023 WL 3580148 at *36; *Howard v. Sec'y of Health & Hum. Servs*., No. 16-1592V, 2022 WL 4869354, at *22 (Fed. Cl. Spec. Mstr. Aug. 31, 2022) ("for purposes of Program determinations, it is improper to think of GBS and CIDP as 'two sides of the same coin,' despite their overlap…Petitioners cannot just 'borrow' what is known about GBS and vaccination generally as a template for proving causation in the context of a CIDP injury"), *mot. for review den'd,* 2023 WL 4117370, (Fed. Cl. May 18, 2023), *aff'd,* 2024 WL 2873301 (Fed. Cir. June 7, 2024). I will therefore not blindly apply the evidence supporting a causal link between GBS and other vaccines to this case. While *some* aspects of what is understood about GBS and its relationship to vaccines may well be relevant to my analysis, Petitioner's success ultimately turns on whether she has preponderantly linked *CIDP* to the Tdap vaccine.

Nevertheless, many Program decisions have assumed that the medical and scientific evidence supporting a GBS-flu vaccine link applies with the same force to CIDP. *See*, *e.g.*, *Jastisan v. Sec'y of Health & Hum. Servs.*, No. 13-937V, 2016 WL 4761950 (Fed. Cl. Spec. Mstr. Aug. 10, 2016). Special masters particularly tend to lump GBS and CIDP together when the flu vaccine is at issue. *See*, *e.g.*, *Tomsky v. Sec'y of Health & Hum. Servs.*, No. 17-1132V, 2020 WL 5587365, at *5 (Fed. Cl. Spec. Mstr. Aug. 24, 2020) ("[F]or purposes of this decision I merely assume but do not decide that petitioner has established a medical theory causally linking the flu vaccine to CIDP"). I myself have done the same (albeit *only* in the context of the flu vaccine), motivated by the desire to adhere to past Program resolutions of such claims for the sake of judicial consistency. *See Strong v. Sec'y of Health & Hum. Servs.*, No. 15-1108V, 2018 WL 1125666, at *20 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (finding that the flu vaccine could cause CIDP).

But there remain very few *reasoned* decisions linking the flu vaccine to CIDP—and fewer still involve *tetanus-containing vaccines*. In fact, the most well-reasoned and persuasive decisions do not find an association at all.[10] *See, e.g.*, *DeVaughn v. Sec'y of Health & Hum. Servs.*, No. 22-832V, 2025 WL 758128, at *18–21 (Fed. Cl. Spec. Mstr. Feb. 10, 2025) (Td vaccine not shown to likely cause CIDP); *Howard*, 2022 WL 4869354 (same); *Sanchez v. Sec'y of Health & Hum. Servs.*,

[9] By contrast, other kinds of Program claims involving conditions with multiple titles sometimes reference the same thing. Brachial neuritis, for example, is also referred to as Parsonage-Turner syndrome, or neuralgic amyotrophy—and therefore it makes no difference what term is used to characterize the diagnosis. *See Marshall v. Sec'y of Health & Hum. Servs.*, No. 21-1445V, 2024 WL 2059813, at *1 n.3 (Fed. Cl. Spec. Mstr. Apr. 12, 2024).

[10] Similar reasoning has even lead special masters to reject contentions of a causal link between the Tdap vaccine and GBS itself. *Kaczerowski v. Sec'y of Health & Hum. Servs.*, No. 21-758V, 2025 WL 2798865 (Fed. Cl. Spec. Mstr. Aug. 28, 2025); *Winkler v. Sec'y of Health & Hum. Servs.*, No. 18-203V, 2021 WL 6276203 (Fed. Cl. Spec. Mstr. Dec. 10, 2021), *mot. for review den'd*, No. 18-203V, 2022 WL 1528779 (Fed. Cl. Spec. Mstr. May 13, 2022), *aff'd*, 88 F.4th 958 (Fed. Cir. 2023); *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); *see also Isaac v. Sec'y of Health & Hum. Servs.*, 108 Fed Cl. 743 (2013) (affirming special master denial of claim alleging tetanus vaccine was causal of GBS), *mot. for review den'd*, 540 Fed. App'x 999 (Fed. Cir. 2013).

No. 18-1012V, 2022 WL 1013264 (Fed. Cl. Spec. Mstr. Mar. 11, 2022) (same); *Houston*, 2021 WL 4259012 (same).

These decisions often arise from the fact that the claimant is simply applying in blanket form medical and scientific theories mainly applicable in the context of the flu vaccine and GBS, while relying on generalizations about these theories and their relationship to the immune process. As I noted in *DeVaughn,* for example, arguments about molecular mimicry between components of Tdap and self/nerve amino acid sequences over-emphasized the possible cross-reactive effect, but without enough preponderant evidence to show an autoimmune outcome was likely *in the specific context* of the Tdap vaccine's administration. *DeVaughn,* 2025 WL 758128, at *19. The *DeVaughn* claimant also offered too many case reports (a kind of evidence understood not to deserve significant weight), unreliable studies, purported Government concessions that stood for no such thing, or otherwise stale medical literature—rather than evidence more specific to the pathogenesis of CIDP, showing why the immune stimulation caused by a vaccine was *likely* to spark an autoimmune process causing nerve demyelination. *Id.* at *20–21.

Such decisions are not *binding* on the outcome herein. And I readily acknowledge that different facts from a person's medical record could produce a different outcome; it is conceivable, for example, that the medical record of a susceptible person might establish a reason that the mere stimulation provided by a vaccine could result in CIDP. But I properly take into account my experience resolving similar cases when considering the theory offered herein. *Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 339 (2007). These prior, relevant decisions may be considered as persuasive guidance supporting the view that a Tdap vaccine-CIDP association is not all that well-established—and absent new scientific or medical evidence on the subject not previously considered, these prior determinations support a denial of entitlement in future cases.

In summary, then, there is ample Program caselaw suggesting that CIDP is not likely preponderantly associated with the Tdap vaccine. The fact that molecular mimicry is often embraced to explain how autoimmune pathologic processes might unfold is of no help to Petitioner in this case. Nor could Petitioner simply rely on the scientific and medical support for a flu vaccine-GBS association—which *has* been preponderantly demonstrated—and in turn apply it wholesale to the context of distinguishable demyelinating peripheral neuropathies, or substituted where non-flu vaccines are at issue. Despite the overlap between GBS and CIDP, far less is known about CIDP and its overall pathogenesis. And this case does not in any event involve the flu vaccine—the vaccine best associated, from a scientific/medical perspective, with GBS or (arguably) other peripheral neuropathies involving demyelination. While other vaccines, like Tdap, have has also been found causal of GBS, such an association weakens significantly in the context of CIDP. Moreover, I have identified no recent *reasoned* decisions in which a special master explained how or why the Tdap vaccine was likely causal of the claimant's CIDP. I have therefore considered in

prior cases the specific theory offered herein associating the Tdap vaccine to CIDP, but have not found it to be reliably established with sufficient preponderant evidence.

## II. Petitioner Has Not Carried Her Burden of Proof

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

### A. *Althen* Prong One

As noted above, 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 CIDP. *See generally DeVaughn*, 2025 WL 758128, at *18-21; *Howard*, 2022 WL 4869354, at *23–26; *Sanchez*, 2022 WL 1013264, at *21–22; *Houston*, 2021 WL 4259012, at *17–18.

In so ruling, I have considered expert testimony from qualified and experienced neurologists, and reviewed in detail the literature and studies offered on the topic. I have also taken into account the oft-repeated argument that the Government has "conceded" entitlement (based on somewhat-old publications discussing possible vaccine adverse events), as well as the logical contention that because these kinds of claims are frequently settled, there is likely some basis for a favorable causation finding. I have been presented numerous case reports in which individuals developed CIDP after receipt of a tetanus-containing vaccine. I have reviewed expert opinions from neurologists and immunologists well qualified to offer their views. And I have heard the kinds of arguments about the possibility of autoantibody-mediated cross-attacks against nerve myelin, with the antibodies generated due to mimicry between the amino acids or other subunits of a vaccine component and self nerve components.

Yet I have not been able to determine that this mix of evidence is enough to tip the preponderant scale in a claimant's favor. And my determinations have been tested on appeal, and found to reflect reasonable analysis in accordance with the evidentiary weighing I perform as a special master. My decisions on the lack of a preponderantly-supported CIDP-Tdap vaccine association cannot simply be wished away, or diminished in weight as turning on some fact specific to the relevant claimant.

This case involves a slightly different group of experts, and some items of literature not previously reviewed (although nothing particularly new either). But the outcome is—

appropriately—the same. Dr. Or-Geva's reports contain articulately-presented opinions, and are thorough in scope, but overall they largely repeat the kinds of contentions I have encountered before—repeatedly. There is nothing novel set forth in those reports that would suggest revisiting this causation theory a fifth time.

Petitioner also (having briefed this matter prior to the Federal Circuit's decision in *Cerrone*) repeatedly emphasizes her contention that she only needed to establish a *plausible* theory to prevail. Br. at 71, 72. And indeed, Dr. Or-Geva's report outlines the many plausible biologic processes that are likely involved in CIDP's pathogenesis, and how vaccination could theoretically impact those processes in a way that would result in disease. But the relevant standard is *preponderance*–not plausibility. *Cerrone,* 146 F.4th at 1122. Petitioner's theory is no better preponderantly established than near-identical theories I have considered in so many prior matters. There is simply insufficient evidence (other than in the form of case reports observing instances of CIDP after receipt of a Tdap vaccine—a kind of *post hoc ergo proper hoc* evidence reasonably given low weight in Program cases) that CIDP is likely due to administration of this specific vaccine.

I note in closing that my negative evaluation of this theory is not the product of some kind of personal bias, or works an unfairness against claimants that could be avoided if only a different neutral, unfamiliar with the claim or more "open minded" about the possibility of causation, decided the matter. The Vaccine Program's special masters are expected and *intended,* over the course of their terms in service to the Court, to develop expertise in resolving Vaccine Act claims – the sole kind of claim they are directed to resolve. *Doe*, 76 Fed. Cl. at 339. This "on the job" training teaches them not only much about what specific fact issues to home in upon, but also educates them about the kinds of causation theories medical science has generated for how a vaccine could result in an adverse effect. The special masters thus possess expertise in differentiating between claims that raise novel or legitimately-disputed questions of medical science from those that reflect tired reasoning usually if not always resulting in dismissal. I reject the causation theory herein based upon my exposure to versions of it numerous times in the past—and because *it has been found repeatedly to be insufficient*.

B. *Althen* Prong Two

The record in this case establishes several points that immediately cut against a finding that the Tdap vaccine was likely associated with Petitioner's CIDP. There is, for example, no record evidence of any unusual inflammatory response to the vaccine. In addition, treater support for a vaccine association in this case is largely lacking, or was deemed a question they could not answer pro or con. Ex. 6 at 43. No other clinical or testing results support the conclusion that Petitioner's vaccination constituted a special factor in her development of

CIDP. Petitioner's constellation of preexisting comorbidities also greatly complicates the picture; while I certainly do not purport to find one or a combination of them was likely causal, Petitioner's overall health picture makes it difficult to single out the vaccine as the triggering factor. And the record reveals Petitioner was experiencing symptoms of an infection in early December 2020, before manifestation of her neurologic symptoms later that month. Ex. 5 at 59–63. This also complicates an argument that a vaccine received two weeks prior to these symptoms, and a month before neurologic symptoms onset, was likely causal of her neuropathy.

Dr. Sriram posed an intriguing counter-explanation for Petitioner's CIDP, based on testing performed on Petitioner at the time treaters were considering POEMS syndrome. In so doing, Dr. Sriram did establish that MGUS *can* be associated with CIDP. However, the evidence in this record is insufficiently robust for me to make a finding that Petitioner's CIDP was *likely* caused by MGUS, or even that the possibility of this greatly diminishes the vaccine as having been causal. And the evidence of a pre-onset infection is itself somewhat ambiguous.

I thus cannot find on this record that there is preponderantly some other explanation for Petitioner's injury. But in the end, what is left is the fact that within about a month of receipt of the Tdap vaccine, Petitioner first manifested neurologic symptoms that in retrospect can be said to reflect burgeoning CIDP—with no evidence of an initial reaction to the vaccine, inconsistent and insufficient treater support for an association, and nothing else (other than the fact of the diagnosis itself) to link the vaccine to the injury. While that might be sufficient to satisfy the Table claim for *GBS* after receipt of the *flu vaccine* (especially since onset occurred within the Table timeframe) it is not enough for a distinguishable vaccine like Tdap—or for a separate, chronic demyelinating disease like CIDP.

## 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.[11]

**IT IS SO ORDERED**.

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

[11] 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.