In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: December 30, 2025

DONNA FAYE MCKENNEY,	*	
	*	No. 19-1799V
Petitioner,	*	
v.	*	Special Master Gowen
	*	
SECRETARY OF HEALTH	*	
AND HUMAN SERVICES,	*	Prevnar-13; Guillain-Barre
	*	Syndrome.
Respondent.	*	

Amber D. Wilson, Wilson Science Law, Washington, D.C., for petitioner.
Jennifer L. Reynaud, U.S. Department of Justice, Washington, D.C., for respondent.

RULING ON ENTITLEMENT[1]

On November 22, 2019, Donna Faye McKenney ("petitioner") filed her claim in the National Vaccine Injury Compensation Program.[2] Petition (ECF No. 1). Petitioner alleged that the Prevnar-13[3] vaccine she received on August 13, 2018, was the cause-in-fact of her developing Guillain-Barre Syndrome ("GBS"). *Id.* at Preamble. After a review of the record, including petitioner's medical records, expert reports, and the medical literature filed in this matter, I find that petitioner is entitled to compensation.

[1] Pursuant to the E-Government Act of 2002, *see* 44 U.S.C. § 3501 note (2012), because this decision contains a reasoned explanation for the action in this case, I am required to post it on the website of the United States Court of Federal Claims. The court's website is at http://www.uscfc.uscourts.gov/aggregator/sources/7. **This means the decision will be available to anyone with access to the Internet.** Before the decision is posted on the court's website, each party has 14 days to file a motion requesting 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). "An objecting party must provide the court with a proposed redacted version of the decision." *Id.* **If neither party files a motion for redaction within 14 days, the decision will be posted on the court's website without any changes.** *Id.*

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to 34 (2012) (hereinafter "Vaccine Act" or "the Act"). Hereinafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

[3] Petitioner also received a Shingrix vaccine the same day, which is a non-covered vaccine and thus not considered in this case.

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

I. Procedural History

Petitioner filed her claim on November 22, 2019 and she filed accompanying medical records to support her allegations. *See* Petitioner's Exhibits ("Pet'r Exs") 1-14 (ECF No. 41).

On August 20, 2020, respondent filed the Rule 4(c) report recommending against compensation, stating that petitioner had not provided preponderant evidence that the Prevnar-13 vaccine caused her GBS. Respondent ("Resp't") ("Rept.") (ECF No. 18). In response, petitioner filed an expert report from Dr. David Axelrod[4] on February 19, 2021. Pet'r Ex. 15 (ECF No. 23). On August 11, 2021, respondent filed a responsive expert report from Dr. Harold Moses, Jr.[5], a neurologist. Resp't Ex. A (ECF No. 30). Petitioner filed a responsive expert report from Dr. Axelrod on August 25, 2021. Pet'r Ex. 16 (ECF No. 32). Then respondent filed a supplemental expert report from Dr. Moses. Resp't Ex. C (ECF No. 36).

After an entitlement hearing was cancelled, petitioner moved for a Ruling on the Record on November 29, 2023. Pet'r Motion ("Mot") (ECF No. 49). Respondent filed a response on February 8, 2024. Resp't Response (ECF No. 52). Petitioner filed a reply to the response on April 15, 2024. Pet'r Reply (ECF No. 54). On May 7, 2024, the parties filed a Joint Status Report stating that they agree that the record is now complete. Joint Status Rept. (ECF No. 56). This case was transferred to the undersigned's docket on August 29, 2024.

Accordingly, this case is now ripe for adjudication.

II. Legal Standard for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The

[4] Dr. David Axelrod is a clinical immunologist who is now currently retired from patient healthcare. Pet'r Ex. 15 at 1. He received his medical degree from University of Michigan Medical School in 1974 and was a resident at the University of Toronto School of Medicine and at William Beaumont Hospital. *Id.* at 15. He is board certified in Rheumatology, Internal Medicine, and Allergy and Immunology. *Id.* at 1. Dr. Axelrod was an Associate Professor of Medicine in the Division of Adult Rheumatology at the Medical College of Ohio and he was also an Associate Profess of Medicine, Division of Allergy, Immunology and Rheumatology at the UMDNJ. *Id.* at 16. Dr. Axelrod is currently a visiting professor at the Penn State Hershey Medical Center. *Id.* He is licensed to practice medicine in the states of Michigan, Pennsylvania, and Maryland. *Id.* Further, he has authored or co-authored numerous articles in the field of immunology and rheumatology. *Id.* at 17-18. Therefore, the undersigned finds Dr. Axelrod an expert in the field of Rheumatology and Immunology.

[5] Dr. Harold Moses is an Associate Professor of Neurology in the Division of Neuroimmunology and Multiple Sclerosis at Vanderbilt Medical Center. Resp't Ex. A at 1. Dr. Moses received his medical degree from the University of North Carolina School of Medicine in 1993. Resp't Ex. B at 1. He was a neurology resident at the Mayo Clinic in Rochester, Minnesota and was a Neurology Instructor/Fellow at the Vanderbilt University Medical Center. *Id.* He is licensed to practice medicine in the state of Minnesota, Arizona and Tennessee. *Id.* at 2. Dr. Moses is board certified in psychiatry and neurology. *Id.* Dr. Moses is a journal reviewer for the Journal of Neuroimmunology, Clinical Neurology and Neurosurgery, and American Academy of Neurology. *Id.* at 3. He treats patients with neurological diseases at several hospitals in Tennessee. *Id.* at 3. Dr. Moses has authored or co-authored numerous articles in the field of neurology and neuroimmunology. Therefore, the undersigned finds that Dr. Moses is an expert in neurology and neuroimmunology.

Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" *Rooks v. Sec'y of Health & Hum. Servs.,* 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). 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 or she] may find in favor of the party who has the burden to persuade the judge of the fact's existence. *Moberly v. Sec'y of Health & Hum. Servs.,* 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010).

To receive compensation through the Program, petitioner must prove either (1) that [he] suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); *Capizzano v. Sec'y of Health & Hum. Servs.,* 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Because petitioner does not allege that he suffered a Table Injury, he must prove that a vaccine he received caused his injury. To do so, he must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and his injury ("*Althen* Prong One"); (2) a logical sequence of cause and effect showing that the vaccine was the reason for her injury ("*Althen* Prong Two"); and (3) a showing of a proximate temporal relationship between the vaccine and her injury ("*Althen* Prong Three"). § 13(a)(1); *Althen*, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Hum. Servs*., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Recently, in *Kottenstette,* the Federal Circuit reiterated that proof of causation does not "require identification and proof of specific biological mechanisms[.]" *Kottenstette v. Sec'y of Health & Hum. Servs.,* --Fed.Appx.—(Fed. Cir. June 15, 2021) (citing *Knudsen v. Sec'y of Health & Hum. Servs.,* 35 F.3d 543, 549 (Fed. Cir. 1994). Causation "can be found in vaccine cases….without detailed medical and scientific exposition of the biological mechanisms." *Knudsen,* 35 F.3d 543, 548-49 (Fed. Cir. 1994). It is not necessary for a petitioner to point to conclusive evidence in the medical literature linking a vaccine to the petitioner's injury, as long as the petitioner can show by a preponderance of evidence that there is a causal relationship between the vaccine and the injury, whatever the details of the mechanism may be. *Moberly v. Sec'y of Health & Hum. Servs.,* 592 F.3d 1315, 1325 (Fed. Cir. 2010).

Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in petitioner's favor when the evidence weighs in his favor. *See Moberly*, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the

persons presenting that evidence."); *Althen*, 418 F.3d at 1280 (noting that "close calls" are resolved in petitioner's favor).

In Vaccine Act cases, expert testimony may be 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 also Cedillo*, 617 F.3d at 1339 (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). In Vaccine Program cases, the *Daubert* analysis has been used in the weighing of the scientific evidence actually proffered and heard rather than as a tool for the pre-trial exclusion of expert testimony. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (Fed. Cl. 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"), *aff'd*, 420 F. App'x 923 (Fed. Cir. 2011). The flexible use of the *Daubert* factors to determine the persuasiveness and/or reliability of expert testimony in Vaccine Program cases has routinely been upheld. *See, e.g., Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 742–45 (2009). 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").

Close calls regarding causation must be resolved in favor of the petitioner. *Althen*, 418 F.3d at 1280 (holding that Congress created a system in which "close calls regarding causation are resolved in favor of injured claimants"); *Knudsen*, 35 F.3d at 551 ("If the evidence (on alternative cause) is seen in equipoise, then the government has failed in its burden of persuasion and compensation must be awarded.").

III. Summary of Evidence Submitted

a. Petitioner's Medical Records

Prior to petitioner's August 13, 2018 vaccination, petitioner, a 68 year old woman, had been treated for a left corneal ulcer and abrasion and appeared to receive annual physical exams. *See* Pet'r Ex. 4 at 7, 37. At her annual health check on August 13, 2018, it was noted that petitioner "needs update for hypothyroidism," but her physical exam was unremarkable. Pet'r Ex. 4 at 55. Petitioner received the Prevnar-13 vaccine at this appointment. *Id.* at 57.

On August 17, 2018, a message was sent to petitioner noting that she needed a lower dose of thyroid medication and requested petitioner to come back for repeat blood work in six weeks. Pet'r Ex. 4 at 59.

On August 26, 2018, petitioner went to the Kaiser Permanent Emergency Department for epigastric abdominal pain for two days, with no associated vomiting or nausea. Pet'r Ex. 5 at 2. Petitioner took her husband's pain medication but got "minimal relief." *Id.* She also reported "tingling of fingers and toes" which started one day ago. *Id.*

The plan was to administer an enema and admit her to the hospital for observation. One day, later August 27, 2018, a nursing note indicated that petitioner was having low urine output and that she "can no longer 'hold anything, because I cannot feel my hands.'" Pet'r Ex. 5 at 9. Petitioner had an initial consult with Dr. Amelia Lee on August 27, 2018, when it was noted that petitioner had "bilateral upper and lower extremity" polyneuropathy and petitioner was having difficulty holding objects, but her strength "appeared normal." *Id.* at 9-10. Petitioner reported to Dr. Lee that "prior to the onset of her abdominal pain, she started developing numbness in her hands and feet," and that her symptoms started on August 23rd. *Id.* at 11. Petitioner reported her hands "felt weak" and that she tried to walk with physical therapy, but "felt too weak and collapsed to the floor." *Id.* Her physical examination was positive for decreased sensation to touch on both sides of her hands extending to mid-forearm and both sides of her feet extending to mid-shin. *Id.* at 12. Dr. Lee's opinion was that petitioner's polyneuropathy "may be related to critical illness or possibly GBS variant" and that it was a "possible autoimmune process." *Id.* Further, Dr. Lee stated that if petitioner's diagnosis was not clear or she was not improving, a neurology consult should be considered. *Id.* at 10.

The following day, on August 28, 2018, petitioner's polyneuropathy had an acute worsening overnight. Pet'r Ex. 5 at 16. Petitioner reported that her numbness was worse in both legs and arms. *Id.* at 17. Her physical examination was remarkable for decreased muscle strength in her lower extremities and "worse than prior exam." *Id.* It was noted that petitioner had a slight left facial droop. Petitioner had a consult with neurologist, Dr. Hema Subbaratnam, who ordered an MRI of petitioner's spine to assess for a possible lesion. *Id.* at 21. With petitioner's MRI being negative for any lesions that could explain her symptoms, Dr. Subbaratnam recommended a lumbar puncture "to check for signs of inflammation and changes consistent with GBS." *Id.* at 23. She received one dose of IVIG on August 28, 2018 due to concern for GBS. *Id.* at 30.

Petitioner was transferred to the ICU on August 29, 2018 due to respiratory failure. Pet'r Ex. 5 at 27. When she was examined by neurologist, Dr. Subbaratnam in the ICU, she had absent deep tendon reflexes in the knees and ankles, and no ability to flex her ankles and could "barely" flex her knees. *Id.* at 34. Petitioner's CSF protein was slightly elevated at 67 and she was negative for Enterovirus. *Id.* Her diagnosis was "GBS-Patient with significant respiratory deterioration along with increased weakness motor system. Has pattern of asymmetric motor-sensory neuropathy, acute. Unable to get NCS to check if predominantly demyelinating or axonal at this time." *Id.* On August 30, 2018, Dr. Subbaratnam noted that even with two IVIG doses, petitioner was still deteriorating, and that petitioner would need the full course of IVIG. *Id.* at 43.

Petitioner remained hospitalized until September 26, 2018, when she was transferred to a Skilled Nursing Facility. During her hospitalization, petitioner had a consult with Dr. Stefan Law on August 13, 2018, who indicated that petitioner's GBS had an "unclear etiology" and wrote: "? Related to prior history of Graves disease, *also consider recent VZV/pneumonia vaccinations*." Pet'r Ex. 5 at 268. Dr. Law also wrote that petitioner had no evidence of CMV, West Nile Virus, Lyme or typical viral/bacterial meningitis on [lumbar puncture]." *Id.* On September 25, 2018, prior to petitioner's discharge, Dr. Marie Nera, assessed petitioner with

GBS and wrote, "unclear what caused GBS, maybe related to prior history of Graves disease, also consider recent VZV/pneumonia vaccinations or possible viral illness prior to admission. No evidence of [cytomegalovirus], [West Nile Virus], Lyme or typical viral/bacterial meningitis on LP." *Id.* at 440.

Petitioner made slow improvements in her functionality while hospitalized. *See e.g.* Pet'r Ex. 5 at 197 (Pt now has 4/5 biceps strength, 3/5 triceps and shoulder flexion, and 3+/5 grip); Pet'r Ex. 5 at 274 ("Pt with improved fine motor coordination during grooming tasks but would benefit from non-skid coban material to increase success with holding containers."); Pet'r Ex. 5 at 440 ("Clinically, she continues to make steady improvement."). She was discharged to a Skilled Nursing Facility on September 26, 2018 with a primary diagnosis of Guillain-Barré Syndrome. Pet'r Ex. 5 at 480.

Petitioner was admitted to Marquis Mt. Tabor Skilled Nursing Facility from September 26, 2018 through December 5, 2018. *See generally* Pet'r Ex. 7. During this time, petitioner had a follow-up appointment with neurologist, Dr. Branaven Mahadeva on October 18, 2018, who noted that petitioner "did receive vaccinations a few weeks prior and it is unclear how much the latter played a role in this." Pet'r Ex. 5 at 615. Petitioner's reflexes were recorded as "areflexic throughout" and she still had diminished sensation to pinprick and light touch in her arms and legs. *Id.* at 618. Dr. Branaven also reviewed petitioner's EMG/NCS which he stated showed, "demyelination with axonal loss, the latter predominantly in the lower extremities." *Id.* at 615; *see also* Pet'r Ex. 7 at 1697 ("There is evidence for an underlying demyelinating neuropathy as can be seen in Guillain-Barre syndrome; There is evidence for axonal features in the lower extremity with denervation and absent motor responses which could indicate a more prolonged recovery in the lower extremities; There are predominantly demyelinating features in the upper extremity with the slowed conduction velocities, prolonged motor distal latencies and absent F responses which is more indicative of segmental demyelination which could indicate a better recovery in the upper extremities.").

Despite petitioner's extended time in the skilled nursing facility, additional therapy was recommended in order for her to become independent and she was transferred to the Good Samaritan Rehabilitation Institute from December 5, 2018 through January 4, 2019. *See generally* Pet'r Ex. 8. When she was discharged on January 4, 2019, her remaining functional issues were "lower and upper extremity weakness," and "anxiety." Pet'r Ex. 8 at 28. After discharge from Good Samaritan Rehabilitation Institute, petitioner engaged in extensive out-patient physical therapy. *See* Pet'r Ex. 9 at 14. At a follow-up appointment on January 23, 2019, petitioner was using a power wheelchair and reported neuropathic pain in her extremities, but she declined additional medication. *Id.* at 27. Petitioner completed out-patient in home physical therapy as well.

On February 11, 2019, she was assessed by physical therapist, Ms. Ann Defazio, who wrote that petitioner upper extremity strength was approximately 4/5 in most movements and her lower extremities were 2/5 with most movements. Pet'r Ex. 9 at 39. Petitioner was using a motorized wheelchair still. *Id.* at 38. Petitioner required assistance to get from sitting to standing, getting her lower extremities into the bed, and she uses a chair in the shower that is moved up a ramp to get her into the shower. *Id.* at 39. By February 26, 2019, petitioner was

using a walker in her home to move independently but still used the motorized wheelchair when she left the house. *Id.* at 52. Unfortunately, due to scheduling, her next appointment was not until March 28, 2019. *Id.* at 76. By her next appointment, on April 12, 2019, petitioner was walking and only using the wheelchair approximately 30% of the time. *Id.* at 79. At her May 20, 2019 appointment, petitioner was able to walk without assisted devices, but was still experiencing lower extremity weakness. *Id.* at 88-89. Petitioner's lower extremity strength was still recorded as 3/5 to 4/5. *Id.* at 89. She was continuing physical therapy, but also had to be treated for lower leg swelling in 2019. *Id.* at 102. No additional records have been filed past June 2019.

b. Kevin Jamison M.D.

Petitioner submitted a letter from Dr. Kevin Jamison, a board-certified neurologist. Pet'r Ex. 10. In the letter, Dr. Jamison states that petitioner was hospitalized for GBS, two weeks after receiving the Prevnar-13 vaccine. *Id.*

Dr. Jamison explained that GBS "occurs when the immune response cross-reacts with peripheral nerve components," and that the "immune response can follow infection, immunization, surgery, or even trauma." *Id.*

He wrote that he reviewed her medical records, and it was his opinion that "she did not suffer any of the other common triggering events for [GBS], such as an infection, surgery, or trauma," prior to her hospitalization for GBS. *Id.* It was his opinion that petitioner's "immune activation [was] caused by the vaccinations," which were the cause of her GBS. *Id.*

c. Petitioner's Expert's Opinion: Dr. David Axelrod

Petitioner submitted two expert reports from neurologist immunologist/rheumatologist, Dr. David Axelrod. Pet'r Exs. 15 & 16. Dr. Axelrod agreed with the diagnosis of GBS and stated that the onset of petitioner's symptoms, twelve days post-vaccination, was consistent with a primary adaptive immune response to her vaccinations. Pet'r Ex. 15 at 13. Dr. Axelrod proposed a theory of molecular mimicry to explain how the Prevnar-13 vaccine can cause GBS. *Id.* at 5.

In his first report, Dr. Axelrod explained that given that the vast majority of individuals do not develop autoimmune conditions, it is likely that certain individuals are predisposed to developing autoimmune diseases following exposure to an infectious agent or vaccine. Pet'r Ex. 15 at 3. He explained, "molecular mimicry is postulated to be the most likely mechanism of post-infectious autoimmunity," and that "vaccines are developed to mimic the infectious agents without causing infection." *Id.* He stated that, "If there are amino acid sequences in the vaccine components that are similar or homologous structures to self-antigens, then the immune response to the vaccine structures can also result in immune responses to self-antigens, and cause dysfunction and/or damage, autoimmune disease." *Id.* at 4. Dr. Axelrod opined that molecular mimicry was the likely mechanism that caused petitioner to develop GBS after receiving the Prevnar-13 vaccine. *Id.* at 3-5.

Referring to the chapter on Autoimmune Peripheral Neuropathies in the Clinical Immunology and Principles and Practice book, Dr. Axelrod stated that "antibodies and T-cell responses to a number of sialic acid gangliosides can be found in subjects with GBS." Pet'r Ex. 15 at 4. The chapter explains, "GBS is an inflammatory demyelinating polyneuropathy in which the peripheral myelin, the axon, the nodes of Ranvier, or the Schwann cells are putative target antigens of an immune attack, possibly triggered by various antecedent events." Pet'r Ex. 17 at 5. Further, the chapter provides that gangliosides are especially present in the nervous system, and different gangliosides are involved in different GBS subtypes. *Id.* However, the authors note that IgG antibodies that react with GM1, GD1a, GalNAc-GD1a, and GM1b are found in 80% of cases with the motor axonal form of GBS, but in the most common GBS subtype, AIDP, ganglioside-specific antibodies are uncommon. *Id.* at 6. While recognizing that *c.jejuni* is associated with GBS, the authors wrote "molecular mimicry may not be limited to *C.jejuni* because GM1 and GQib epitopes are also found in the bacteria wall of *hemophilus influenzae,* which is also a triggering factor in GBS….Another potential for molecular mimicry is *M.pneumoniae*, which precedes GBS in 5% of cases and is known to stimulate antibodies against human carbohydrate antigens, including galactocerebroside, the main glycolipid antigen in peripheral nerves." *Id.* at 6.

In addition to the antibodies to gangliosides identified in GBS patients, Dr. Axelrod stated that there are other autoantibodies that have been found to attack other parts of the peripheral nervous system that are affected in GBS, such as the Nodes of Ranvier. Pet'r Ex. 15 at 5. He referred to the article by Kira et al., which identified other autoantibodies in GBS patients. *Id.* Kira et al. stated that in GBS, "antibodies against nodal proteins such as NF186, gliomedin, and contactin, were detected in a minority of patients," and that "anti-NF186 and anti-NF155 antibodies have been found in both [central nervous system] and [peripheral nervous system] demyelinating disorders. Pet'r Ex. 42 at 1.[6] Dr. Axelrod noted that the Davies et al. study, which examined the serum of patients with inflammatory neuropathies, also found antibodies to NF155, NF186, pan-neurofascin, contactin-1, and contactin associated protein (Caspr-1) or CNTN1/Caspr1-complex. Pet'r Ex. 15 at 5; *see also* Pet'r Ex. 24 at 4.[7]

Dr. Axelrod wrote that the "Prevnar-13 [vaccine] is a pneumococcal 13 valent polysaccharide conjugate vaccine with Diphtheria toxin CRM-197 (inactivated Diphtheria toxin by substitution of an amino acid at position 197)," and he wrote that the diphtheria toxin protein shares the same or conserved similar amino acids to various self-antigens. Pet'r Ex. 15 at 6-7. In agreement with the article by Frankild,[8] he stated that "amino acid similarity, not identity, is a predictive measure of cross-reactivity….as a result of molecular mimicry." *Id.* at 4. Dr. Axelrod stated, "amino acid sequences found within the Prevnar-13 vaccine have homologous and/or conserved similar amino acid sequences to normal peripheral nerve components," and it was his opinion that petitioner's immune system reacted to the components in the vaccine, mainly the

[6] Kira, J. et al., *Anti-Neurofascin Autoantibody and Demyelination,* 130 Neurochem. Int. https://doi.org/10.1016/j.neuint.2018.12.011 (2018). [Pet'r Ex. 42].

[7] Davies, A.J. et al., *Immunoadsorption and Plasma Exchange in Seropositive and Seronegative Immune-mediated Neuropathies,* 9 J Clin. Med. (2020). [Pet'r Ex. 24].

[8] Frankild et al, *Amino Acid Similarity Accounts for T Cell Cross Reactivity and for "Holes" in the T Cell Repertoire,* 3 Plos One e1831 (2008). [Pet'r Ex. 21].

diphtheria component, and cross-reacted with self-antigens in the peripheral nervous system, resulting in GBS.

In his supplemental report, responding to respondent's expert, Dr. Moses' criticism of the theory of molecular mimicry to the diphtheria component of the Prevnar-13 vaccine, Dr. Axelrod noted that the diphtheria toxoid component is a common component administered with other vaccines, such as the DTaP and meningococcal conjugate. Pet'r Ex. 16 at 3. He stated that the diphtheria toxin CRM-197 protein is identical to the inactivated diphtheria toxin found in Tdap, DtaP, TDP, and TD vaccines. Pet'r Ex. 15 at 12.

Dr. Axelrod cited the Delp et al. article, which described five cases of patients developing GBS after diphtheria infections, to support his opinion that the diphtheria infection can be associated with post-infectious GBS. Pet'r Ex. 16 at 4. Diphtheria is a bacterium that can be treated with antibiotics, such as penicillin.[9] In Delp, one of the patients developed difficulty swallowing and mild ocular palsy, along with palatal paralysis bilaterally ten days into his diphtheria infection and after the patient had been treated with antibiotics. Pet'r Ex. 34 at 3.[10] Another case described in Delp indicated that a patient developed weakness in his legs, mild shortness of breath, and difficulty closing his right eye approximately 10-12 days after the onset of a diphtheria infection in his throat and a lesion on his skin. *Id.* at 2. This patient also demonstrated weak deep tendon reflexes in his upper extremities and ankles, and had absent knee reflexes. *Id.*

He then referenced several articles that describe patients developing GBS following the administration of vaccines that contained the diphtheria toxoid CRM-197. *Id.* at 5. For example, the Myers et al. article reported on adverse events following administration of the Menactra vaccine (meningococcal diphtheria toxoid conjugate vaccine) and identified 32 cases of GBS following vaccination. Pet'r Ex. 38 at 7.[11] The authors wrote:

> In October 2005, reports to the Vaccine Adverse Event Reporting System (VAERS) indicated a possible safety signal for Guillain-Barre syndrome (GBS) following vaccination with MenACWY-D, and three publications subsequently described 17 reports to VAERS of GBS after MenACW-D, occurring between June 2005 and September 2006. These reports described onset of symptoms of GBS ranging from 2 to 33 days after vaccination in persons 11-43 years of age. While the available data suggested *a*

[9] According to the Mayo Clinic, diphtheria is caused by the bacterium *Corynebacterium diptheriae.* The bacterium usually multiples on or near the surface of the throat or skin. Diphtheria signs and symptoms usually begin 2 to 5 days after a person becomes infected. Signs and symptoms include: a thick, gray membrane covering the throat and tonsils, a sore throat and hoarseness, swollen glands in the neck, difficulty breathing or rapid breathing, nasal discharge, or fatigue. Complications of untreated diphtheria can lead to breathing problems, heart damage or nerve damage. https://www.mayoclinic.org/diseases-conditions/diphtheria/symptoms-causes/syc-20351897

[10] Delp, M. et al., *Post-Diphtheritic Polyneuritis: A Report of Five Cases with Albuminocytologic Dissociation Simulating Guillain-Barre Syndrome,* 24 Ann. Intern Med. 618-28 (1946). [Pet'r Ex. 34].

[11] Myers, T. et al., *Adverse Events Following Quadrivalent Meningococcal Diphtheria Toxoid Conjugate Vaccine (Menactra) Reported to the Vaccine Adverse Event Reporting System (VAERS),* 38 Vaccine, 6291-6298 (2020). [Pet'r Ex. 38].

> *small increased risk of GBS after MenACWY-D vaccination,* the authors concluded that uncertainty regarding background incidence rates for GBS and the inherent limitations of the data source warranted additional evaluation in a more robust data source such as the Vaccine Safety Datalink. *After a review of the data, ACIP determined that protection provided by MenACWY-D against meningococcal disease outweighed a possible small increased risk of GBS.*

Id. at 2 (emphasis added). The Bakshi article is the first case report of a 22-year-old patient who developed GBS four days after receiving a combined tetanus-diphtheria vaccination. Pet'r Ex. 36 at 1.[12] The authors noted that two other cases of GBS were reported after the "pure tetanus toxoid" vaccine, and they "suspected that the tetanus portion of the vaccination produced the GBS," but they also wrote that they were "unable to exclude that the GBS was secondary to the diphtheria portion of the vaccination." *Id.* at 2.

The case report article from *Morbidity and Mortality Weekly Report* describes five cases of GBS following administration of the Meningococcal Polysaccharide Diphtheria Toxoid vaccine. Pet'r Ex. 37.[13] The article explains that onset of GBS symptoms began between 14-31 days post-vaccination. *Id.* at 2-3. The same article describes the case of woman who received the Mentactra vaccine and then fourteen days later developed heaviness in her legs when walking up stairs. *Id.* at 3. The patient eventually became unable to walk, and her neurological examination revealed bilateral acute flaccid weakness and decreased deep tendon reflexes. *Id.* The patient's viral and bacterial cultures were negative and had no history of respiratory or gastrointestinal illnesses. *Id.* She was treated with plasmapheresis and IVIG, but developed difficulty breathing and had to be intubated 53-days after onset. *Id.* The patient was discharged to a rehabilitation facility, and 53 days later was able to walk, feed herself, sit and stand again. *Id.*

Dr. Axelrod wrote that although these case reports include exposure to vaccines with other antigens, such as tetanus or meningitis, they all contained the diphtheria toxoid, including CRM-197, "suggesting a possible role of the diphtheria toxoid in the development of GBS." Pet'r Ex. 16 at 5. More specific to the Prevnar-13 vaccine, Dr. Axelrod referenced the Tseng et al. article which discussed the safety of the pneumococcal conjugate vaccine in comparison to the PPSV23 in older adults, but the study identified four cases of GBS post-Prevnar-13 vaccine. Pet'r Ex. 33 at 2.[14] Tseng noted that there was only one case of GBS in the clinical trials that was considered "possibly related to PCV13," and that 3 reports of GBS were in VAERS data. *Id.* at 2. The article found that the adverse events were "no more common than those following the PPSV23 in elderly populations," and that there was "no significantly elevated risk of….Guillain-Barré syndrome," compared with PPSV23. *Id.* at 7. Dr. Axelrod acknowledged

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[12] Bakshi, R. & Graves, M.C., *Guillain-Barre Syndrome After Combined Tetanus-Diphtheria Toxoid Vaccination,* 147 J. Neurol. Sci. 201-02 (1997). [Pet'r Ex. 36].

[13] Centers for Disease Control & Prevention, *Guillain-Barre Syndrome Among Recipients of Menactra Meningococcal Conjugate Vaccine-United Sates, June-July 2005,* 54 MMWR Morb. Mortal Wkly. Rep. 1023-25 (2005). [Pet'r Ex. 37].

[14] Tseng, H.F. et al., *Pneumococcal Conjugate Vaccine Safety In Elderly Adults,* 5(6) Open Forum Infect. Dis. 1-8 (2018). [Pet'r Ex. 33].

this finding by Tseng and stated "[t]his does not mean that these authors found no risk of GBS following Prevnar-13 vaccination." Pet'r Ex. 16 at 2

Dr. Axelrod opined that petitioner developed her GBS symptoms approximately 12 days after the Prevnar-13 vaccination, which was an acceptable timeframe for her to develop an immune response to the vaccine through molecular mimicry, resulting in damage to her peripheral nervous system. Pet'r Ex. 15 at 11. He noted that petitioner had previously received Tdap vaccine on April 3, 2013, and that vaccine also contained the same CRM-197 diphtheria toxin. Thus, the CRM197 in the Prevnar vaccine could induce a stronger and more rapid immune response. Pet'r Ex. 15 at 13. The Abbas article Dr. Axelrod cites, explains that "exposure of the immune system to a foreign antigen enhances its ability to respond to that antigen again. Responses to second and subsequent exposures to the same antigen, called secondary immune responses, are usually more rapid, greater in magnitude, and often qualitatively different from the first, or primary immune response to that antigen." Pet'r Ex. 19 at 9.[15] Further, the authors explain that the secondary response is typically "stronger" than the primary immune response. *Id.*

The Stone et al. article describes different types of immune-mediated adverse reactions to vaccines, and states that "Immunologically mediated neurological complications such as Guillain-Barre syndrome and other demyelinating neuropathies…are a known reported adverse event related to immunization, but such events are exceedingly rare." Pet'r Ex. 32 at 5.[16] The article explains that GBS has been found to occur after the flu, oral polio, rabies, or tetanus toxoid containing vaccines and that "onset of symptoms is considered as possibly linked to vaccination if it occurs within six weeks" of vaccination. *Id.* at 6.

Finally, Dr. Axelrod stated that there was nothing in the medical records to suggest an alternative cause. Pet'r Ex. 15 at 10. Petitioner was tested for various viral infections, all which were negative. He explained that through the mechanism of molecular mimicry, approximately 12 days after she received the Prevnar-13 vaccine, she developed an immune response to both antigens in the vaccine, but also to her peripheral nervous system that resulted in damage to her myelin, manifesting in clinical symptoms. *Id.* at 9.

Dr. Axelrod concluded that it was his opinion that the Prevnar-13 vaccine caused her to develop GBS through the mechanism of molecular mimicry, there was no alternative cause identified in the medical records that could have caused her GBS, and the onset of petitioner's symptoms twelve days after the vaccination is consistent with the mechanism of molecular mimicry inducing post-vaccination GBS. Pet'r Ex. 15 at 13; Pet'r Ex. 16 at 6.

[15] Abbas, A.K. et al., *Cellular and Molecular Immunology*, 9th Ed. Elsevier, (2008). [Pet'r Ex. 19].

[16] Stone, C. Jr. et al., *Immune-mediated Adverse Reactions to Vaccines,* 85 Br. J. Clin. Pharmacol. 2694-2706 (2019). [Pet'r Ex. 32].

d. Respondent's Expert's Opinion: Dr. Harold Moses, Jr.

Respondent submitted two expert reports from Dr. Harold Moses, Jr., a neurologist. *See* Resp't Ex. A & C. Dr. Moses agreed with the diagnosis of GBS but argued that the Prevnar-13 vaccine petitioner received was not the cause of her GBS. Resp't Ex. A at 5.

Dr. Moses acknowledged that the onset of petitioner's symptoms of GBS after vaccination "falls within an acceptable timeframe for an association." Resp't Ex. A at 4; Resp't Ex. C at 2. However, he argued that the temporal association between the vaccination and onset of symptoms is the *only* evidence that supports vaccine causation. Resp't Ex. C at 2.

Dr. Moses' expert reports primarily focused on studies that have not shown an increased risk of GBS following the Prevnar-13 vaccine and that the incidences of neurological complications post-vaccination is rare and impliedly unlikely. *See* Resp't Ex A at 4; Resp't Ex. C at 2.

With respect to the theory of molecular mimicry as the causal mechanism for the Prevnar-13 vaccine causing GBS, Dr. Moses y stated that the homology between the diphtheria toxin protein to various proteins within the peripheral nervous system as detailed by Dr. Axelrod, was "comprehensive and accurate," but argued that it "does not demonstrate that Prevnar…resulted in the petitioner's GBS." Resp't Ex. A at 4. Dr. Moses noted that the Israeli et al. paper referenced by Dr. Axelrod lists 15 different vaccines as "implicated as possibly being associated with GBS," but the Prevnar-13 vaccine is not listed as one. Resp't Ex. A at 4; *see also* Resp't Ex. D at 5. However, this article was published online in 2010, and the FDA did not approve Prevnar-13 for older adults until December 30, 2011, so there would have been no history of Prevnar immunizations of adults at the time of publication. More importantly, this paper acknowledged that molecular mimicry is a mechanism for triggering an autoimmune condition such as GBS. The authors wrote, "A common explanation for how infectious agents stimulate autoimmunity in an antigen-specific way is via molecular mimicry." Pet'r Ex. 39 at 6.

In his supplemental report, Dr. Moses repeats the same general arguments, stating that, "Dr. Axelrod has a theory that I believe is not substantiated," without providing any additional support and seemingly focused on the lack of epidemiological evidence between the Prevnar vaccine and GBS. Resp't Ex. C at 2. He argued that "there is no persuasive medical or scientific evidence supporting causal relationship between the vaccines [petitioner] received on August 13, 2018 and the development of GBS." *Id.* Dr. Moses also criticized the case reports Dr. Axelrod cited as "not regarded as substantive evidence" and asserted that the Tseng paper "did not find a relationship between Prevnar-13 vaccination and GBS." *Id.* However, Dr. Axelrod correctly observed that the Tseng paper's conclusion was that there was not found *increased* risk of an adverse event following the Prevnar-13 vaccine when compared to the PPSV23 vaccine, not that no adverse events occurred. *See* Pet'r Ex. 33 at 7.

Finally, without identifying an alternative cause for the initiation of petitioner's GBS, Dr. Moses opined that petitioner's previous Grave's disease, suggests that she was susceptible to developing GBS. Resp't Ex. C at 1. He likens her development of GBS after vaccination to a coincidence, stating, "I think it's apparent that by chance some people will develop GBS within

6 weeks after any vaccination, including Prevnar-13." Resp't Ex. C at 2. He concluded both reports stating that the Prevnar-13 vaccine could not have cause petitioner's GBS. Resp't Ex. A at 5; Resp't Ex. C at 2.

IV. Analysis

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006). Such theory must only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 548-49. Petitioner may satisfy the first *Althen* prong without resorting to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *See 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). However, a "petitioner must provide a 'reputable medical or scientific explanation' for [petitioner's] theory." *Boatmon v. Sec'y of Health and Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Moberly*, 592 F.3d at 1322). While the theory need not be medically or scientifically certain, "it must still be 'sound and reliable.'" *Id.* (quoting *Knudsen*, 35 F.3d at 548-49). The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 548-49. Causation "can be found in vaccine cases…without detailed medical and scientific exposition of the biological mechanisms." *Id.*

For the reasons set forth below, in this matter, petitioner has demonstrated by preponderant evidence a sound and reliable theory explaining how the Prevnar-13 vaccine can cause GBS, thus satisfying *Althen* prong one.

Dr. Axelrod proposed a theory of molecular mimicry to explain how the Prevnar-13 vaccine can cause GBS. Pet'r Ex. 15 at 3-7. As he explained in his report, molecular mimicry occurs when there are amino acid sequences or structures in the vaccine that are similar or homologous to self-antigens, then the immune response to the vaccine structures can also result in immune response to the self-antigens causing dysfunction and/or damage in the form of autoimmune disease. *Id.* at 4. Molecular mimicry is referenced in articles as the cause of GBS that both Drs. Axelrod and Moses cite. *See* Resp't Ex. D at 2 ("A common explanation for how infectious agents stimulate autoimmunity in an antigen-specific way is via molecular mimicry."); Pet'r Ex. 25 at 3[17] (noting that molecular mimicry between *Haemophilus, cytomegalovirus, C.jejuni* and myelin structures have been identified); Pet'r Ex. 22 at 322 ("Most of the antiglycolipid antibodies are generated by immunoreaction against glycoconjugates in pathogens causing antecedent infections, the mechanism of which is called "molecular mimicry."). The Stone article also endorsed molecular mimicry as the mechanism for inducing GBS post-vaccination, stating, "Postvaccination Guillain-Barre syndrome, similar to that occurring after an acute infection, is thought to be a mixed, delayed immune-mediated reaction, which probably represents a T-cell response where CD4+ and CD8+T cells cross-recognize specific virotopes

[17] Winer J.B, *An Update in Guillain-Barre Syndrome*, 2014 Autoimmune Dis. (2014). [Pet'r Ex. 24].

and similar self-antigens in the nervous system, leading to either an axonal or demyelinating clinical subtype." Pet'r Ex. 32 at 6.

Additionally, molecular mimicry has been generally accepted as a sound and reliable theory for causing GBS in many cases in the Vaccine Program. *See Conte v. Sec'y of Health & Hum. Servs.*, No. 17-403V, 2020 WL 5743696, at *23 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program."); *Maloney v. Sec'y of Health & Hum. Servs.,* No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); *Osso v. Sec'y of Health & Hum. Servs.,* No. 18-575V, 2023 WL 5016473, at *21 (Fed. Cl. Spec. Mstr. July 13, 2023); *Whitener v. Sec'y of Health & Hum. Servs.*, No. 06-477V, 2009 WL 3007380 (Fed. Cl. Spec. Mstr. Sept. 2, 2009) (finding that meningococcal vaccine can cause GBS); *Mohamad v. Sec'y of Health & Hum. Servs.*, No. 16-1075V, 2022 WL 711604, *9-18 (Fed. Cl. Spec. Mstr. Jan. 27, 2022) (finding that Tdap can cause GBS through molecular mimicry); *Peirson v. Sec'y of Health & Hum. Servs.,* No. 17-1136V, 2022 WL 322836, at *31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022). (finding that Prevnar through molecular mimicry can cause GBS) Further, molecular mimicry has been accepted as a theory of causation for GBS even in the absence of any demonstration of homology and cross reaction. *See Salmins v. Sec'y of Health & Hum. Servs.,* No. 11-140V, 2014 WL 1569478, at *14 (Fed. Cl. Spec. Mstr. Mar. 31, 2014) (finding that HPV vaccine can cause GBS).

More specific to the Prevnar-13 vaccine, Dr. Axelrod proposed a theory of molecular mimicry between the diphtheria component of the vaccine and multiple potential components of the peripheral nervous system, including contactin-1, contactin-associated protein-1 (CASPR 1), human myelin protein, myelin basic protein, ganglioside GA1, and human myelin associated glycoprotein. Pet'r Ex. 15 at 6-7. Molecular mimicry between the diphtheria toxin, CRM-197 and self-antigens, including contactin-1, has been found as sound and reliable in other Prevnar-13 cases. *See Maloney*, 2022 WL 1074087; *Anderson v. Sec'y of Health & Hum. Servs.,* No. 18-484V, 2024 WL 557052, at *32 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); and *Simeneta v. Sec'y of Health & Hum. Servs.*, No. 18-859V, 2024 WL 4881411, at *32 (Fed. Cl. Spec. Mstr. Oct. 31, 2024). I have also accepted the theory of molecular mimicry between the CRM-197 (diphtheria toxin) and contactin-1 in other cases involving GBS and the Prevnar-13 vaccine. *See e.g. Musick v. Sec'y of Health & Hum. Servs.,* No. 18-451V, 2025 WL 2452232 (Fed. Cl. Spec. Mstr. July 8, 2025); *Byrd v. Sec'y of Health & Hum. Servs.,* No. 20-1476, 2024 WL 4003061, at *21-26 (Fed. Cl. Spec. Mstr. July 8, 2024).[18]

[18] In addition to these cases, there have been numerous cases in the Vaccine Program finding that the Prevnar-13 vaccine was the cause of GBS. *See Davison ex rel. Davison v. Sec'y of Health & Hum. Servs.*, No. 19-1404V, 2025 WL 2692664, at *11-15 (Fed. Cl. Spec. Mstr. Aug. 19, 2025) (Horner); *Datte v. Sec'y of Health & Hum. Servs.*, No. 18-2V, 2025 (Fed. Cl. Spec. Mstr. May 9, 2025) (Horner); *Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022) (Horner); *Bartoszek v. Sec'y of Health & Hum. Servs.*, No. 17-1254V, 2024 WL 4263604, at *17-22 (Fed. Cl. Spec. Mstr. Aug. 27, 2024) (Horner); *Maloney v. Sec'y of Health & Hum. Servs.*, No. 19-1713V, 2022 WL 1074087, at *30-31 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (Dorsey); *Sprenger v. Sec'y of Health & Hum. Servs.*, No. 18-279V, 2023 WL 8543435, at * 18-19 (Fed. Cl. Spec. Mstr. Nov. 14, 2023) (Dorsey). While there is not uniformity among all the special masters regarding the Prevnar-13 vaccine and GBS, the similarity of the facts between these cases is striking, with many petitioners suffering onset of symptoms between four- and 22-days post-vaccination, and many petitioners suffering acute diseases, requiring ICU interventions. *See e.g. Davison ex. rel Davison*, 2025 WL 2692664, at *4-6 (petitioner requiring ICU care and intubation); *Datte*, 2025 WL 1565894, at *4-6 (petitioner requiring ICU intervention, intubation, and despite numerous interventions, continued to deteriorate).

Additionally, molecular mimicry as the mechanism by which the diphtheria toxin found in other vaccines, such as Tdap or Menactra, can induce autoantibodies to the peripheral nervous system, has been found persuasive in other Vaccine cases. *See Giannetta v. Sec'y of Health & Hum. Servs.*, No. 13-215V, 2017 WL 4249946 (Fed. Cl. Spec. Mstr. Sept. 2017) (finding the theory of molecular mimicry with the diphtheria toxin in Menactra to be persuasive); *Harris v. Sec'y of Health & Hum. Servs.,* No. 18-994, 2023 WL 2583393 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (finding a study that demonstrated "some homology between diphtheria and myelin associated proteins" persuasive for demonstrating molecular mimicry with the diphtheria toxin included in the Tdap vaccine).

Moreover, the evidence submitted in this case supports Dr. Axelrod's theory of molecular mimicry between the diphtheria toxin and structures of the peripheral nervous system. Dr. Axelrod presented an extensive list of potential peripheral nervous system targets of similar antigens in the vaccine as identified through Uniprot searches. The target antigens are found not only in myelin but in the nodes of Ranvier as well as in the paranodal and juxtanodal parts of the peripheral nerves.

The Kira and Winer articles Dr. Axelrod references explain that autoantibodies to different proteins in the peripheral nervous system have been identified in GBS patients. *See* Pet'r Ex. 43 at 1; Pet'r Ex. 25 at 2. Winer explains, "the evidence in support of antiganglioside antibodies as a cause of [Miller Fisher Syndrome] and AMAN was strong, the most common form of GBS [in] Western countries (AIDP) was only rarely associated with ganglioside antibodies," and that "although antiganglioside antibodies are the most commonly reported antibody in GBS, there are other reports of antibodies that might be pathogenic in a smaller number of patients. Antibodies against a protein in the node of Ranvier "neurofascin" have received recent attention with serum of 4% of patients with AIDP." Pet'r Ex. 25 at 3. The Kira study stated that antibodies against nodal and paranodal proteins, such as NF186, gliomedin and contactin were detected in GBS patients. Pet'r Ex. 43 at 1. Wang further explains that antibodies to myelin sheath associated proteins, such as P2, P0, PMP22 and connexin 32 were also detected in GBS patients. Pet'r Ex. 26 at 6. Taken together, these articles suggest that in GBS there are numerous self-antigens in the peripheral nervous system that can be the target of autoantibodies—and Dr. Axelrod identified a number of these possible targets in his report as sharing similar or conserved amino acids with the diphtheria toxin (CRM-197).

Further, Dr. Moses does little to rebut Dr. Axelrod's proposed mechanism of molecular mimicry between the Prevnar-13 vaccine and GBS. Dr. Moses reliance on the Israeli et al. paper as evidence that the Prevnar-13 vaccine is not implicated in association with GBS is misplaced, given that the paper was published in 2010 and the Prevnar-13 vaccine had only been made available for children in February 2010 and was not approved by the FDA for adults over the age of 50 until December30, 2011.[19] Additionally, Dr. Moses's conclusion that the Tseng paper "did not find a relationship between the Prevnar-13 vaccination and GBS" mischaracterizes the conclusion of that paper. Tseng identified four cases of GBS after administration of the Prevnar-13 vaccine and concluded that "there is no significantly elevated risk of cardiovascular events,

[19] Licensure of 13-Valent Pneumococcal Conjugate Vaccine for Adults Aged 50 Years and Older, https://www.cdc.gov/mmwr/preview/mmwrhtml/mm6121a3.htm (last visited on December 18, 2025).

Bell's palsy, Guillain-Barre syndrome, syncope, erythema multiforme, thrombocytopenia, cellulitis and infection or allergic reaction *compared with* the risk associated with *PPSV23*." Pet'r Ex. 33 at 7 (emphasis added). The paper simply notes that the Prevnar-13 vaccine does not pose an *increased* risk of certain adverse events compared to the pneumococcal-23 vaccine, not that there is no relationship between the vaccine and adverse events. The concept of increased risk to the general population of vaccine recipients as a measure of causation is also not identical to determining whether there is preponderant evidence that the vaccine caused the harm to the specific petitioner in the case before the court.

The theory presented by Dr. Axelrod supported by reference to multiple similarities in peripheral nerve epitopes and peptides in the Prevnar vaccine more directly addressed the question before the court then the failure of various studies to identify rare events in statistically significant numbers. Ultimately, petitioner does not need to establish a theory of vaccine causation by scientific certainty. *See Gross v. Sec'y of Health & Hum. Servs.,* No. 17-1075V, 2022 WL 9669651, at *36 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (finding that the criteria, including supportive epidemiology, identification of antibodies directed against human antigens, identification of the mimics of the target antigen, and reproduction in an animal model, is tantamount to "require[ing] scientific certainty, which is a bar too high.").

Likewise, is Dr. Moses' insistence upon a testable hypothesis which he indicates is not possible with rare events suggesting that epidemiological proof is required. While I appreciate his view that medicine has not been able to definitively determine the cause of many autoimmune diseases, Congress created the Vaccine Program in order to address the rare adverse events that occur secondary to vaccines even when that causal relationship is not fully understood. As the Federal Circuit held in *Althen:* While this case involves the possible link between a tetanus toxoid-containing vaccine and a CNS injury, "*a sequence hitherto unproven in medicine,"* the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field "*bereft of complete and direct proof*" of how vaccines affect the human body. *Althen*, 418 F.3d at 1280 (emphasis added).

For the reasons set forth above, the undersigned finds that petitioner has provided preponderant evidence of a sound and reliable theory of causation, demonstrating that the Prevnar-13 vaccine can cause GBS, satisfying *Althen* prong one.

b. *Althen* prong three

Under the third *Althen* prong, a petitioner must demonstrate a "proximate temporal relationship" between the subject vaccination and the alleged injury. *Althen,* 418 F.3d at 1278. To do this, petitioner must provide "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *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 also coincide with the theory of how the relevant vaccine can cause an injury. *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 Fed. App'x 952 (Fed. Cir. 2013).

In this case, both Drs. Axelrod and Moses agree that the onset of petitioner's symptoms of GBS began 12-days after she received the Prevnar-13 vaccine. *See* Pet'r Ex. 15 at 10-13; Resp't Ex. A at 4. Dr. Moses wrote, "The 12-day period between vaccination and development of symptoms falls within an acceptable timeframe for an association." Resp't Ex. A at 4.

The Israeli article, which discusses GBS induced by vaccination, explained that the increased risk of GBS following administration of the swine flu vaccine was a 5-to-9-week period, with the mean interval between vaccination and onset of neurological symptoms was 3.9 weeks. Pet'r Ex. 15 at 10; Resp't Ex. D at 4. The Stone article also endorses the onset of GBS post-vaccination within a 6-week period post-vaccination. *See* Pet'r Ex. 32 at 6. Additionally, the case reports described in the *Morbidity and Mortality Weekly Report*, found the onset of GBS symptoms after administration of the meningococcal-diphtheria toxin containing vaccine (MCV4), ranging from 14 to 25 days post-vaccination. Pet'r Ex. 37 at 3. The Myers article, which identified 42 GBS cases after administration of the meningococcal diphtheria-toxoid conjugate vaccine, found a median time interval of 15 days between vaccination and symptom onset. Pet'r Ex. 38 at 5.

The time interval between vaccination and the onset of petitioner's symptoms is appropriate given the theory of molecular mimicry proposed by Dr. Axelrod, as described in the medical literature. Further, this timeframe has been found to be appropriate in other Prevnar-13 cases in which molecular mimicry has been proffered as the causal mechanism by myself and other special masters. *See Musick*, 2025 WL 2452232, at *40 (finding onset of nine days after administration of the Prevnar-13 vaccine to be medically acceptable to infer vaccine causation); *Diponziano*, 2025 WL 942744, at *27-28 (finding onset of symptoms 11 days post-vaccination to be consistent with molecular mimicry); *Gross*, 2022 WL 966951, at *38-39 (finding a GBS onset of 13 days after the Prevnar-13 vaccination to be appropriate); *Koller*, 2021 WL 5027947, at *23 (finding a GBS onset of 12 days after Prevnar-13 vaccination to be "within the medically accepted timeframe consistent with petitioner's theory of molecular mimicry).

Petitioner received her Prevnar-13 vaccine on August 13, 2018, and she went the emergency department on August 26, 2018 complaining of abdominal pain, along with tingling in her fingers and toes that began on August 25, 2018. Pet'r Ex. 5 at 2-4. Petitioner was ultimately hospitalized and treated for GBS, as described in her medical records. The experts do not dispute the onset of petitioner's symptoms, nor her diagnosis of GBS. Accordingly, the undersigned finds that petitioner has demonstrated by preponderant evidence a medically acceptable temporal relationship between the Prevnar-13 vaccine and the onset of her neurological symptoms, satisfying *Althen* prong three.

c. *Althen* prong two

Under *Althen* prong two, petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Capizzano*, 440 F.3d at 1324 (quoting *Althen*, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm…or in other words, that the vaccine was the 'reason for the injury.' " *Pafford*, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. *Andreu,* 569 F3d at 1367; *Capizzano,* 440 F.3d at 1326 ("[M]edical 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 trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. *Cucuras,* 993 F. 2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court.").

Petitioner has demonstrated by preponderant evidence a logical sequence of cause and effect, establishing that the Prevnar-13 vaccine she received on August 13, 2018 caused her to develop GBS twelve days later.

There is no disagreement between the parties that petitioner suffered from GBS following her vaccination. Pet'r Ex. 15 at 3; Resp't Ex. A at 3. As the undersigned explained above, petitioner proffered a sound and reliable mechanism of vaccine causation under prong one and the onset of petitioner's symptoms was approximately 12-days post-vaccination, an appropriate time frame in which cross reactions secondary to molecular mimicry could cause GBS. The twelve-day post vaccination symptom onset was squarely in the middle of the time period in which the previously decided Prevnar /GBS cases occurred. "Evidence demonstrating petitioner's injury occurred within a medically acceptable timeframe bolsters a link between the injury alleged and the vaccination at issue under the "but-for" prong of the causation analysis." *Capizzano,* 440 F. 3d at 1326 (finding medical opinions that explain how a vaccine can cause the injury alleged coupled with evidence demonstrating a close temporal relationship "are quite probative" in proving actual causation.") *Pafford,* 451 F.3d at 1358; *see also Contreras,* 107 Fed. Cl. at 295 (finding that there is a "logical overlap between three *Althen* prongs, and that evidence that goes to one prong may also be probative for another prong"). However, it is not only the finding of *Althen* prongs one and three for which the undersigned finds preponderant evidence to support *Althen* prong two.

Petitioner's medical records demonstrate some consideration by her treating physicians for the role of the Prevnar-13 vaccine as the cause of her GBS. *See* Pet'r Ex. 5 at 440 ("unclear what caused GBS, maybe related to prior history of Graves disease, also consider recent VZV/pneumonia vaccinations"). Further, petitioner was tested for other possible infections that could have caused GBS, but her panel was negative. Dr. Moses also concedes that petitioner's medical records do not support the finding of an antecedent infection prior to her developing GBS. Resp't Ex. A at 4. Finally, petitioner's clinical history and course are consistent with several other GBS cases following the Prevnar-13 vaccination. For example, in *Datte,* the petitioner developed tingling in her hands and feet ten days post-vaccination, became so weak she was unable to walk, was treated with IVIG, eventually intubated due to her condition deteriorating, and once discharged spent extensive time in skill nursing facilities and rehabilitation facilities. *Datte,* 2025 WL 1565894, at *4-5. In *Diponziano,* the petitioner received the Prevnar-13 vaccine and developed tingling in her hands and feet 11 days later, she was ultimately hospitalized, and even though she began treatment with IVIG, had to be

transferred to the ICU and intubated, and when discharged spent considerable time in long-term care facilities to regain her function. *Diponziano*, 2025 WL 942744, at *6-7. The clinical courses of the petitioners in *Datte, Diponziano,* and in this case, all describe a similar timeframe of symptom onset, the need for higher level care in the hospital, and extended rehabilitation.

Thus, in accordance with the above, petitioner has preponderantly demonstrated a logical sequence of cause and effect establishing that the Prevnar-13 vaccine did cause her GBS.

V. Conclusion

For the reasons discussed above, the undersigned finds that petitioner has established by preponderant evidence that the Prevnar-13 vaccine she received on August 13, 2018, caused her to develop GBS with severe symptoms that lasted more than six months. Therefore, petitioner is entitled to compensation. A separate damages order will be issued.

IT IS SO ORDERED.

s/Thomas L. Gowen
Thomas L. Gowen
Special Master