# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

Filed: March 2, 2026

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| CAROLANN SINCLAIR, | * | |
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| Petitioner, | * | No. 22-140V |
| | * | |
| v. | * | Special Master Gowen |
| | * | |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| | * | |
| Respondent. | * | |

*Matthew N. Menzer,* Menzer Law Firm PLLC, Seattle, WA, for petitioner.
*Parisa Tabassian,* U.S. Dept. of Justice, Washington, D.C., for respondent.

## RULING ON ENTITLEMENT[1]

On February 10, 2022, Carolaan Sinclair ("petitioner") filed her claim in the National Vaccine Injury Compensation Program.[2] Petition (ECF No. 1). Petitioner alleged that as a result of receiving the Prevnar-13 vaccine on February 12, 2019, she suffered from Guillain-Barré Syndrome ("GBS") and subsequent PRES. *Id.* at Preamble. After reviewing petitioner's medical records, expert reports submitted by both parties, and the medical literature, I find petitioner is entitled to compensation.

### I. Procedural History

Petitioner timely filed her petition for compensation and medical records to support her claim on February 10, 2022. Petitioner's ("Pet'r.") Exhibits ("Exs.") 2-8 (ECF No. 1). On

[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.

December 22, 2022, respondent filed the Rule 4(c) report recommending against compensation, stating that petitioner had not submitted an expert report in support of her claim. Respondent ("Resp't.") Report ("Rept.") (ECF No. 21). Petitioner filed an expert report from Dr. John Hixson[3] on March 22, 2023. Pet'r. Ex.12 (ECF No. 22). Respondent filed responsive expert reports from Dr. Dara Jamieson[4] and Dr. Robert Fujinami, PhD.[5] Resp't. Exs. A & C (ECF Nos. 26, 27).

On October 23, 2023, the undersigned held a Rule 5 status conference ordering the parties to file supplemental expert reports. Scheduling Order (ECF No. 29). Petitioner filed additional expert reports from Dr. Lawrence Steinman. *See* Pet'r. Exs. 34, 78 (ECF Nos. 32, 39). Respondent also filed supplemental expert reports from Drs. Jamieson and Dr. Fujinami. *See* Resp't. Exs. E, F (ECF Nos. 35, 36).

On September 24, 2024, the parties filed a joint status report stating that the parties wanted to resolve entitlement through a ruling on the record. Joint Status Rept. (ECF No. 41). On January 14, 2025, petitioner filed her motion for a ruling on the record. Pet'r. Brief ("Br.") (ECF No. 44). Respondent filed a response to petitioner's motion on March 12, 2025. Accordingly, this matter is now ripe for adjudication.

## II. Evidence Submitted

### a. Summary of Petitioner's Medical Records

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[3] Dr. John Hixson is a board-certified neurologist and Professor of Neurology at the San Francisco, VA Medical Center and the University of California San Francisco in California. Pet'r. Ex. 12 at 1. He received his medical degree from Johns Hopkins School of Medicine, followed by a neurology residency at the Hospital of the University of Pennsylvania. *Id.* In 2006, Dr. Hixson began a faculty position at the University of California San Francisco in and is currently a member of the Epilepsy Division of the Department of Neurology. *Id.* He diagnoses and treats patients with acute inflammatory demyelinating polyneuropathy ("AIDP") and provides general neurology services. *Id.* For the purposes of this case, Dr. Hixson is accepted in the field of neurology.

[4] Dr. Dara Jamieson is a board-certified neurologist and Clinical Associate Professor of Neurology at Weill Cornell Medicine. Resp't. Ex. A at 1. She received her medical degree from the University of Pennsylvania and completed a residency in neurology at the Hospital of the University of Pennsylvania. *Id.* She previously taught at Temple University and Pennsylvania Hospital (Thomas Jefferson University, University of Pennsylvania). *Id.* Dr. Jamieson became an Associate Professor of Clinical Neurology at Weill Cornell Medicine and was on staff at the New York Presbyterian Hospital. *Id.* She has editorship positions for Neurology Alert, Current Treatment Opinions in Neurology, and the Journal of Neuroimaging. *Id.* Dr. Jamieson has diagnosed and treated patients in the clinical setting with AIDP and AMAN, as well as other neurological diseases. *Id.* at 1-2. Dr. Jamieson is accepted as an expert in the field of neurology.

[5] Dr. Robert Fujinami, PhD, is currently the Assistant Vice President for Academic Affairs for University of Utah Health. Resp't. Ex. C at 1. He completed his Ph.D at Northwestern University and received post-doctoral training and Assistant Professorship at The Scripps Research Institute. *Id.* Dr. Fujinami has received the Harry M. Weaver Neuroscience Scholar of the National Multiple Sclerosis Society. *Id.* Dr. Fujinami, with Dr. Oldstone, first introduced the concept of molecular mimicry involving viruses and autoimmunity. *Id.* Dr. Fujinami has served as an expert for the Institute of Medicine of the National Academic of Sciences pertaining to the influenza vaccine and GBS. *Id.* at 2. Dr. Fujinami has provided his opinion regarding vaccine causation in previous Vaccine Program cases. He is accepted as an expert in the field of immunology.

Petitioner was 65 years old when she received the Prevnar-13 vaccine on February 19, 2019. Pet'r. Ex. 2 at 6. On the date of vaccination, petitioner reporting taking Celexa and Wellbutrin, but had no other health concerns. *Id.* Her physical exam was normal, and she was referred for a mammogram screening and OB/GYN consultation. *Id.* at 7.

On March 2, 2019, approximately 18 days post-vaccination, petitioner went to emergency department of Island Hospital complaining of tingling in her fingers bilaterally, and a headache for four days. Pet'r. Ex. 3 at 81. Petitioner reported developing back pain over "the past several hours" and additional tingling in her left arm. *Id.* Petitioner's examination was normal and she was given Toradol. *Id.* at 86. The emergency room physician, Dr. Michael Lanker, recommended petitioner follow-up with her primary care physician and suspected her symptoms were musculoskeletal in nature. *Id.* Dr. Lanker wrote a musculoskeletal cause would explain her back, neck, and headache. *Id.*

The following day, petitioner returned to Island Hospital emergency department with "midthoracic paraspinal back pain" that was "sharp and stabbing." Pet'r. Ex. 3 at 68. Additionally, petitioner's tingling in her hands and fingers continued. *Id.* An MRI of her cervical and thoracic spine revealed an older cervical fusion at C5-C7. At C2-C3 and T1-2 soft tissue inflammation and enhancement involving the paraspinal soft tissue was seen. *Id.* at 93-94. Petitioner also underwent a lumbar puncture demonstrating elevated protein in the CSF of 64. *Id.* at 65.

Petitioner was transferred to Swedish Hospital in Seattle for continuing care on March 4, 2019. Pet'r. Ex. 4 at 701. Petitioner reported numbness in her toes now, along with the numbness in her hands. *Id.* She also indicated that now she had lost strength in her hands symmetrically. *Id.* Petitioner's neurological exam was notable for reduced grip strength and she endorsed numbness in her fingers bilaterally, although her deep tendon reflexes were recorded as present and normal. *Id.* at 704. Dr. Brett Hiroto wrote that petitioner's clinical exam was not consistent with a musculoskeletal spinal issue, and opined that there was a concern for GBS, although she demonstrated normal deep tendon reflexes. *Id.*

During her hospital admission from March 4-6, 2019, petitioner continued to experience numbness and tingling in her hands and toes. Pet'r. Ex. 4 at 711. She also developed facial numbness and reported it on March 5, 2019. *Id.* Petitioner was examined by neurologist, Dr. William Berg on March 5, 2019 and he wrote that petitioner's physical exam showed "some distal arm weakness, primarily within the median and ulnar nerve distribution," but that petitioner had "excellent strength through the legs," and his concern for GBS "was low." *Id.* at 714. Dr. Berg ruled out HSV myelitis, and a central cord process. *Id.* He opined that petitioner's "patchy numbness" was related to "pre-existing compressive neuropathies." *Id.* Petitioner was discharged on March 6, 2019, with a recommendation to follow-up with a neurologist for an EMG. *Id.* at 707.

Two days later, petitioner went to the University of Washington emergency department with complaints of dizziness and presyncope. Pet'r. Ex. 5 at 48. She reported that the night before she fell, and she had nausea and vomiting. *Id.* She was having difficulty walking and was unable to open jars due to reduced grip strength. *Id.* at 36. Additionally, she reported

numbness and tingling in her fingertips, along with her toes. *Id.* Her neurological exam showed that her eyes were "symmetric and equal but with sluggish reactivity to light," and her motor strength in her right upper extremity was decreased compared to her left. *Id.* Petitioner was observed in the emergency department overnight and then discharged the following day. *Id.* at 36-37.

On March 11, 2019, petitioner went to Skagit Regional Health emergency department complaining of a tightening in her throat and shortness of breath. Pet'r. Ex. 6 at 21-22. Aside from her respiratory issues her physical exam appeared to be "normal" and petitioner was now able to walk with the use of a walker. *Id.* at 23. Petitioner's EKG and X-ray were normal, and she was discharged home with instructions to follow-up with her primary care physician. *Id.* at 25.

Two days later, on March 13, 2019, petitioner went to Swedish Cherry Hill emergency department with tingling in her bilateral feet and hands. Pet'r. Ex. 4 at 691. Petitioner had subjective decrease in sensation to light touch throughout and she was unable to walk. *Id.* Her physical exam was notable for absent reflexes in her knees and ankles. *Id.* at 694. Petitioner was admitted for a neurology consult. *Id.* at 695. Petitioner was seen by neurologist, Dr. Eric Price, who observed that petitioner had bilateral eye closure weakness and a persistent lower facial droop. *Id.* at 574. A lumbar puncture and MRI of her brain and cervical cord were ordered. *Id.* The brain MRI was "highly suspicious for PRES," and protein in her CSF had now risen to 107. *Id.* at 400, 428.

Dr. Price was able to obtain an arterial blood gas which was significantly acidotic. Her ph was 7.22 (normal range 7.35-7.45), PaCO2 66.5 (normal <45), Pa O2, 78 (Normal 80 to 100%) HCO3 27 (Normal 22-p26), but her petitioner's blood pressure was 233/118. *Id.* at 402. On March 15, 2019, petitioner was intubated due to respiratory failure. *Id.* at 399, 402. Given her elevated protein in her CSF and symptoms, she was treated with IVIG for five days. *Id.* at 385. On April 2, 2019, petitioner was discharged to an acute care facility with her principal diagnosis being "PRES with autonomic instability 2/2 Guillain-Barre." *Id.* at 384.

Petitioner was discharged on April 11, 2019 from the Swedish Medical Center Rehabilitation, with a final principal diagnosis of Guillain-Barré Syndrome. Pet'r. Ex. 4 at 280. Her discharge summary notes that the etiology of petitioner's condition was "possible vaccine provoking GBS provoking autonomic dysregulation causing PRES and seizures." *Id.* at 284. Petitioner expressed having neuropathic pain in her fingers and toes and was given a prescription for Gabapentin. She had moderate cognitive communication deficits, but she was improving overall, and she had absent deep tendon reflexes throughout, except a weak right biceps reflex at discharge from the rehab facility on April 11, 2019. Pet'r. Ex. 4 at 285.

Petitioner had a follow-up rehabilitation appointment on August 27, 2019, where she reported needing additional help, as she did not want to go to a skilled nursing facility. Pet'r. Ex. 4 at 258. Petitioner still had neuropathic pain, decreased peripheral proprioceptive sensation, and paresthesia. *Id.* at 260. Further, she was still experiencing visual deficits due to the PRES. *Id.*

On September 25, 2019, petitioner had a follow-up appointment with Dr. Keung, a neuro-ophthalmologist. Pet'r. Ex. 4 at 249. Petitioner's sister who attended this appointment with petitioner explained that petitioner's vision had improved and she could perceive the curb better, although reading was still problematic. *Id.* at 250. Petitioner reported having difficulty reading her phone numbers and dialing. *Id.* Dr. Keung noted that petitioner's vision had made significant improvement, but petitioner still had cortical visual impairment. *Id.* Driving was not recommended and Dr. Keung also stressed that petitioner should undergo neuropsychological testing. *Id.* at 253.

On December 16, 2019, petitioner had an appointment at the Everett Clinic to "discuss vaccine reaction." Pet'r. Ex. 2 at 3. Petitioner questioned whether she had received a flu vaccine in February, prior to the onset of her GBS, but it was noted that there was no documentation for the flu vaccine. *Id.* She had only received Prevnar at that time. Petitioner also reported "mostly recovered from her illness" although she was having difficulty reading and writing. *Id.* Dr. Young diagnosed petitioner with GBS and wrote, "I am not certain if the GBS that she had is related to the Prevnar-13 vaccination. She could have coincidental viral or bacterial infection." *Id.* at 4.

At petitioner's annual wellness exam on January 8, 2020, it was noted that petitioner was still recovering from her GBS but still had some visual and cognitive impairment. Pet'r. Ex. 4 at 226. She was going to resume physical and occupational therapy as well. *Id.*

On June 6, 2020, petitioner went to the Island Hospital emergency room with right sided facial droop and "word salad and not making sense." Pet'r. Ex. 3 at 4. While petitioner was in the emergency department, she had a seizure and given Ativan. *Id.* at 9. Petitioner was transferred to Swedish Medical Center due to her condition and respiratory failure. *See* Pet'r. Ex. 4 at 46.

On June 9, 2020, petitioner underwent a video-EEG due to her "PRES with consequent ischemic strokes with acute encephalopathy likely due to seizures." Pet'r. Ex. 4 at 162. The result was an abnormal EEG study suggestive of, "(1) diffuse encephalopathy; (2) focal structural/functional abnormalities with active epileptogenic focus in the right temporal lobe." *Id.* at 164.

An MRI on July 24, 2020 demonstrated petitioner had "chronic appearing bilateral occipital encephalomalacia, right greater than left, with chronic encephalomalacia extending into the posterior parietal region on the right. Pet'r. Ex. 3 at 3-4.

Petitioner underwent a neuropsychological test on March 23, 2022 and petitioner reported issues relating to attention, memory deficits, and visuospatial skills. Pet'r. Ex. 11 at 89. The results of the neuropsychological testing demonstrated that petitioner had "nearly global impairments in cognition," and petitioner exhibited "significant visuospatial dysfunction, being unable to complete even a test of basic visual perception/judgment." *Id.* Further, the test noted that petitioner was unable to locate the signature line on the consent form even with assistance. *Id.* The test confirmed a "major neurocognitive disorder," and noted that petitioner's "current findings, with difficulties note primarily in attention, executive functioning, memory (seemingly

in the form of a retrieval deficit), and expressed language, as well as prominent visuospatial disturbance and features of Gerstmann syndrome, are consistent with her history of occipitoparietal strokes." *Id.*

**b. Petitioner's Experts' Opinions**

**1. Dr. John Hixson**

Petitioner submitted an expert report by Dr. John Hixson, neurologist, who opined that petitioner had Guillain-Barré Syndrome ("GBS") with an "atypical presentation and course." Pet'r. Ex. 12 at 5. Dr. Hixson also stated that petitioner developed PRES due to her GBS, which then "led to the development of bilateral parieto-occipital strokes, causing epileptic seizures and permanent neurologic deficits." *Id.*

Dr. Hixson stated that the cause of petitioner's GBS and development of PRES was the Prevnar-13 vaccine she received on February 12, 2019. *Id.* at 5. He stated that it was "well-established that GBS is triggered by an autoimmune response to an antecedent infection or other immune stimulant that provokes an immune reaction." *Id.* at 6. The Chandrashekhar article Dr. Hixson referred described the pathogenesis and clinical features of GBS, stated that "GBS is triggered by a cross-reaction of an immune response to an antecedent infection or other event with shared epitopes on peripheral nerve (molecular mimicry). Autoantibodies that react with epitopes on peripheral nerve appear to be the immune trigger after an acute infection in many cases of GBS." Pet'r. Ex. 13.[6] The article notes that there was an increased risk of GBS following vaccination to the swine flu and the 2009 H1N1 flu vaccine, but then other studies have failed to "identify a risk of GBS associated with influenza vaccination." *Id.* at 7.

Dr. Hixson opined that the Prevnar-13 vaccine caused petitioner's GBS through the mechanism of molecular mimicry. Pet'r. Ex. 12 at 6. He referred to Chapter 21 in Autoimmunity, which gave examples of molecular mimicry between antigens and self-epitopes as the inducer of autoimmune diseases, including GBS, MS, and rheumatic fever. Pet'r. Ex. 16.[7] The authors noted that GBS, through molecular mimicry, has been associated with a *campylobacter jejuni* infection, viral infection such as EBV, cytomegalovirus, or *mycoplasma pneumoniae.* *Id.* at 2. Dr. Hixson described molecular mimicry as a "well-known phenomenon for many medical conditions." Pet'r. Ex. 12 at 6.

Dr. Hixson stated that the temporal sequence in petitioner's case demonstrates a "classic temporal sequence for [petitioner's] development of neurologic signs and symptoms. Pet'r. Ex. 12 at 7. He observed that she received the Prevnar-13 vaccine on February 12, 2019 and then by early March 2019, she was experiencing neurological symptoms. *Id.* Further, Dr. Hixson noted that prior to the vaccination, petitioner had no documented neurological complaints or findings, and she also did not report any intercurrent illness during the period prior to vaccination. *Id.* at 6. Dr. Hixson also stated that petitioner's development of PRES was a result of her GBS. He

[6] Chandrashekhar, S. et al, *Guillain-Barre Syndrome in Adults: Pathogenesis, Clinical Features, and Diagnosis,*

[7] Blank, M. et al., *Chapter 21: Molecular Mimicry in Autoimmunity and Vaccinations* 344-350. [Pet'r. Ex. 16].

stated, "There is a body of literature that supports this causal linkage between AIDP, dysautonomia, and PRES," and referred to an article which identified case reports of GBS patients developing PRES. *Id.* at 1; *see* Pet'r. Ex. 23.[8] Garrido et al., which is a letter to the editor, discussed a case report describing the onset of PRES in a patient with GBS. Pet'r. Ex. 23. Garrido identified 30 published cases describing the occurrence of PRES in patients with GBS and explained that the most common characteristic among the case reports is acute elevation of blood pressure or blood pressure fluctuations followed by severity of GBS defined by the inability to walk unassisted at any point during the GBS, need of intensive care or any life-threatening complication. *Id.* The authors wrote that "co-occurrence of PRES with GBS is most probably associated with the presence of dysautonomia, and being more precise, of acute phase elevated blood pressure or fluctuating blood pressure." *Id.* Garrido further explained that "[d]ysautonomia in GBS is a manifestation of more severe involvement of the peripheral nervous system and is associated with increased mortality and poor functional outcome." *Id.*

The Storti et al. article also described the case of a 64-year-old patient who developed right conjugate eye deviation and left hemiparesis, who was then found to have severe subacute demyelinating polyneuropathy. Pet'r. Ex. 27.[9] The authors observed that PRES in GBS "seems to be a health issue highly prevalent in women, with only one described case of a man with PRES, GBS, and a concomitant head injury." *Id.* at 3. The authors noted that PRES typically occurs prior to the onset of GBS symptoms, stating that "in 5 out of 9, overt sensory or motor GBS symptoms presented more than 48 hours after PRES onset." *Id.*

Finally, Dr. Hixson opined that the timeframe between when petitioner received the Prevnar-13 vaccine and the onset of her neurological symptoms established a clear temporal relationship and that there were no intervening illnesses that could have been associated with the development of petitioner's GBS. Pet'r. Ex. 12 at 7.

**2. Dr. Lawrence Steinman**

Petitioner submitted two expert reports from Dr. Lawrence Steinman in support of her claim that the Prevnar-13 vaccine caused her to develop GBS. Pet'r. Exs. 34 & 78. Dr. Steinman agreed with Dr. Hixson that GBS and secondary PRES are the appropriate diagnoses for petitioner. Pet'r. Ex. 34 at 2.

Dr. Steinman opined that the Prevnar-13 vaccine petitioner received on February 12, 2019 caused her to develop GBS through the mechanism of molecular mimicry. Pet'r. Ex. 34 at 9. Hi s theory is based on molecular mimicry between two different components of the Prevnar-13 vaccine and the peripheral nervous system.

First, he explained that the Prevnar-13 vaccine's components include a phosphoglycerol link to the capsular polysaccharide antigens contained in the vaccine. Pet'r. Ex. 34 at 19. He

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[8] Garrido, D. et al., *Triggers of Posterior Reversible Encephalopathy in Guillain-Barre Syndrome,* 198 Clin. Neurol. & Neurosurg. https://doi.org/10.1016/j.clineuro.2020.106250 (2020). [Pet'r. Ex. 23].

[99] Storti, B. et al., *Posterior Reversible Encephalopathy and Guillain-Barre Syndrome: Which came first, the chicken or the egg? A Review of Literature,* 41 Neurol. Sci. 3663-3666 (2020). [Pet'r. Ex. 27].

referred to the Prevnar-13 patent which describes the importance of the phosphoglycerol component in the vaccine in order to preserve the immunogenicity of the vaccine. The patent explains that "the retention of potentially sensitive non-saccharide substituent functional groups of the individual components, such as 0-Acyl, phosphate or glycerol phosphate side chains that may form part of the saccharide epitope." Pet'r. Ex. 34 at 24. Dr. Steinman referenced the Chang et al. article, which examined the structure of the 18C serotype, and stated that phosphate head group on the glycerophasphate is critical for the immunogenicity and effectiveness of the 18C components of Prevnar 13. Pet'r. Ex. 34 at 19; *see also* Pet'r. Ex. 50.[10] Dr. Steinman also references Bryson et al. to demonstrate that antibodies generated to the 23F serotype, which is included in the Prevnar-13 vaccine, bind to the glycerophosphate component of the 23F saccharide. Pet'r. Ex. 34 at 28; Pet'r. Ex. 53 at 6-7.[11] Dr. Steinman stated, "The data from the Bryson article demonstrates *unequivocally* that the immune response to the serotype 23F component of Pneumovax 23 targets the phosphoglycerol in serotype 23F." Pet'r. Ex. 34 at 29 (original emphasis).

Then he explained that phospholipids are components of the cellular membrane of myelin sheath in humans and the phosphoglycerol headgroup attached to the lipids are also targets of antibodies in neuroinflammatory diseases. Pet'r. Ex. 34 at 11. He refers to the Ho et al. article, which examined the immune targets in multiple sclerosis, and it found that autoantibodies in MS "target a phosphate group in phosphatidylserine and oxidized phophatidylcholine derivatives." *Id.*; *see also* Pet'r. Ex. 41 at 1.[12] Dr. Steinman also referenced the Nakos et al. article, which studied antibodies found in GBS patients, to support his theory that the immune response to the Prevnar-13 vaccine can generate antibodies to phospholipids. Pet'r. Ex. 34 at 13; *see also* Pet'r. Ex. 44.[13] Nakos explained that anti-ganglioside antibodies are associated with GBS and associated with *c.jejuni* through the mechanism of molecular mimicry, however, only 15-50% of GBS patients have anti-ganglioside antibodies. Pet'r. Ex. 44 at 2. The authors examined antibodies to different phospholipids in GBS patients and found that all nine GBS patients developed anti-phospholipid antibodies against one lipid during the course of the disease. *Id.* at 5. The authors explained that none of the GBS patients they studied had any other autoimmune condition that may have been responsible for generating the anti-phospholipid antibodies. *Id.*

Additionally, Dr. Steinman referenced Al-Temeemi et al., to again demonstrate that antibodies to phospholipids are pathogenetic in GBS. Pet'r. Ex. 34 at 16; *see also* Pet'r. Ex.

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[10] Chang, J. et al., *Relevance of O-acetyl and Phosphoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysaccharide,* 30 Vaccine 7090-7096 (2012). [Pet'r. Ex. 50].

[11] Bryson, S. et al., *Structures of Preferred Human IgV Genes Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation,* 196 J. Immunol. 4723-4730 (2016). [Pet'r. Ex. 53].

[12] Ho, P. et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath that Resolve Neuroinflammation,* 4(137) ScienceTranslationMedicine.org (2012). [Pet'r. Ex. 41].

[13] Nakos, G. et al., *Anti-phospholipid Antibodies in Serum from Patients with GBS,* 31 Intensive Care Med. 1401-1408 (2005). [Pet'r. Ex. 44].

48.[14] Al-Temeemi explained that "the immune target is still unknown in patients with acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the most frequent variant of GBS," despite extensive research. Pet'r. Ex. 48 at 1. The study sought to measure anti-phospholipid antibodies in patients with GBS and found that elevated IgM anti-phospholipid antibody titers were "statistically significant" during the first week of illness in GBS patients and that IgG was more prevalent in the second week. *Id.* at 8.

Dr. Steinman stated that the information above "provides compelling support for the proposition that the immune response to serotypes 18C and 23F in Prevnar-13 targets the phosphoglycerol moiety in those serotypes and through molecular mimicry, may cross-react with the phospholipids on the myelin, resulting in damage and GBS. Pet'r. Ex. 34 at 30-31.

The second area of molecular mimicry Dr. Steinman proposed is between the CRM-197 protein (diphtheria conjugate) and components of the peripheral nervous system, specifically contactin-1 and Caspr2. Pet'r. Ex. 34 at 32, 49. Contactin-1, which is present in the paranodal subdomains adjacent to the nodes of Ranvier in peripheral nerves. It is also an immune target in GBS. *Id.* at 13, 32. As explained in the Rasband article, "Axons conduct electrical signals…among neurons in circuit in response to sensory input, and between motor neurons and muscles. In mammals and other vertebrates, many axons are myelinated. Myelin, made by Schwann cells in the peripheral nerves and oligodendrocytes in the central nervous system, respectively, is a multilamellar sheet of glial membrane that wraps around axons to increase transmembrane resistance and decrease membrane capacitance." Pet'r. Ex. 49. Myelinated axons are divided into distinct domains, including nodes of Ranvier, paranodal axoglial junctions (PNJ), juxtaparnodes (JXP), and internodes. *Id.* at 1-2. The Devaux et al. article, referenced by Dr. Steinman, explained that the paranodal axoglial junctions are composed of contactin-associated protein (Caspr)/contactin/neurofascin-155 that form a barrier to the lateral diffusion of nodal channels. Pet'r. Ex. 58 at 2.[15] Devaux found that antibodies to the nodes of Ranvier and paranodes were immune targets in GBS and CIDP. *Id.* at 66. They found "58 patients with GBS or CIDP showed IgG deposition at nodes of Ranvier and paranodes," and concluded that NF-186, gliomedin, and contactin are novel target antigens in patients with GBS. *Id.* at 8. Devaux also noted that a higher percentage of GBS patients with antibodies that targeted nodal adhesion molecules presented with respiratory disturbances. *Id.*

Dr. Steinman performed a BLAST search between the CRM-197 diphtheria conjugate to identify homology between contactin-1 and Caspr2. Pet'r. Ex. 34 at 33. He stated that based on the Gautam paper, which Dr. Steinman co-authored, "a viral peptide with homology at just 5 amino acids with a self-peptide can induce clinical signs of EAE in mice." *Id.* at 34. Dr. Steinman that the major study of molecular mimicry in MS reported in the Lanz et al. paper to which he contributed, demonstrated antibodies to the Epstein-Barr virus also recognize the protein GlialCAM, a component of glial cells in the brain that may contribute to MS. The Lanz

[14] Al-Temeemi, T. et al., *Antiphospholipid Antibody in Serum of Guillain-Barre Syndrome Patients,* 10(2) Iraqi J. Med. Sci. 191-199 (2012). [Pet'r. Ex. 48].

[15] Devaux, J. et al., *Nodal Proteins are Target Antigens in Guillain-Barre Syndrome,* 17 J. of the Periph. Nerv. Sys. (2012). [Pet'r. Ex. 58].

study was able to demonstrate mimicry with Epstein Barr through a very large study with very specific testing of CSF of people with MS, but such scientific certainty is not available in this matter. *Id.* at 40. Through his BLAST search between CRM-197 and contactin-1, he found two sequences that had five out of ten identical amino acids, which would be capable of inducing neuroinflammation. Pet'r. Ex. 34 at 43-46. He then searched the Immune Epitope Database (IEDB) and found that humans have been shown to develop T-cell immune responses to both sequences of the diphtheria molecule. *Id.* at 46-47.

In his supplemental report, Dr. Steinman responded to Dr. Jamieson's criticism of his theory that autoantibodies to the nodal or paranodal regions of myelin play a role in GBS in some patients, by referencing the Appelshauser et al. article, which found autoantibodies against Caspr-1 and contactin-1 in patients with GBS. Pet'r. Ex. 78 at 1-2; *see also* Pet'r. Ex. 80 at 1.[16] The authors also examined the class of autoantibodies through the course of GBS and CIDP patients and found that antiparanodal autoantibodies IgG2/3 classes were in monophasic GBS, but IgG4 was found in CIDP, suggesting a subclass switch in chronic diseases. *Id.* at 8. The authors wrote, "our data support pathogenicity of autoantibodies and suggest that autoantibodies and titer are valid indicators for disease activity, as proposed in previous studies on paranodopathy and further IgG4-related neurologic disease." *Id.* at 8.

Dr. Steinman also expanded his mechanism of molecular mimicry between the Prevnar-13 vaccine and other components of myelin in his supplemental report. Pet'r. Ex. 78 at 4. He examined whether there was molecular mimicry between CRM-197 to myelin components P2, P0, and PMP-22. *Id.* He did this in part because of a new article published in 2024 by Sukenikova et al. that examined which myelin proteins are targeted by T cells in demyelinating GBS. *Id.* at 3; *see also* Pet'r. Ex. 81.[17] Sukenikova explained that GBS pathogenesis "is likely to be a consequence of an aberrant immune response triggered by environmental factors, and so far no consistent associations with certain human leukocyte antigen (HLA) class I or II alleles have been described." Pet'r. Ex. 81 at 1. Further, anti-ganglioside antibodies are absent in most patients with GBS, although associated with *c.jejuni* infection through molecular mimicry. *Id.* Sukenikova examined autoreactive T-cells in patients with GBS and found CD4 and CD8 T-cells targeting P0, P2 and PMP22 myelin antigens in the serum, CSF, and nerve tissues of patients with demyelinating AIDP. *Id.* at 8.

Dr. Steinman performed two BLAST searches comparing CRM-197 and P0 and PMP-22, which were referenced targets of autoreactive T-cells in the Sukenikova article, and found sequence homology sufficient to trigger neuroinflammation. Pet'r. Ex. 78 at 7-9. His search between CRM-197 and P0 identified two sequences which share six out of nine identical amino acids and his search between CRM-197 and PMP-2 resulted in two sequences that had five out of ten identical amino acids. Dr. Steinman then explained that the Raju et al. article showed T-cell

[16] Appeltshauser, L. et al., *Antiparanodal Antibodies and IgG Subclasses in Acute Autoimmune Neuropathy,* 7 Neurol. Neuroimmunol. Neuroinflamm. doi:10.1212/NXI.0000000000000817 (2020). [Pet'r. Ex. 80].

[17] Sukenikova, L, et al., *Autoreactive T-cells Target Peripheral Nerves in Guillain Barre Syndrome,* 626 Nature 160-167 (2024). [Pet'r. Ex. 81].

recognition to the sequences of the diphtheria toxin and both P0 and PMP-22. Pet'r. Ex. 78 at 9-10; *see also* Pet'r. Ex. 69.[18]

With respect to the timeframe for petitioner's GBS, Dr. Steinman opined that petitioner's GBS began approximately twenty days post-vaccination, which is an appropriate timeframe for an autoimmune condition to develop after vaccination through the mechanism of molecular mimicry. Pet'r. Ex. 34 at 56. He referred to the Schonberger study GBS developing after administration of the swine flu vaccine, Dr. Steinman stated that "20 days post-immunization is well in the range that was above the estimated background rate of GBS." *Id.* at 56-57. Dr. Steinman stated that there was no epidemiologic study specific to the Prevnar-13 vaccine and GBS that he could reference, so he used Schonberger as an analogy to determine an appropriate temporal relationship between vaccine and onset of GBS. *Id.* at 57. In his supplemental report, Dr. Steinman noted that the 2016 Haber et al. paper, referenced by Dr. Fujinami, identified eleven cases of GBS after reviewing cases reported to VAERS but concluded that those cases did not signal attributable increased risk of GBS after the Prevnar-13 vaccine. Resp't. Ex. F, Tab 11.[19] The median onset interval of GBS symptoms was 9 nine days post-vaccination. *Id.* at 4. Dr. Steinman noted thatthe authors of Haber explained their study had limitations, stating, "It is important to note the limitations of VAERS, which may include underreporting, varying quality of reports….and the lack of an unvaccinated comparison group….Because of these limitations, it is extremely difficult to determine causal associations between vaccines and AEs. However, VAERS may be effective for promptly detecting rare AEs and potential vaccine safety problems." Resp't. Ex. F, Tab 11 at 5.

**c. Respondent's Experts' Opinions Regarding Vaccine Causation**

**1. Dr. Dara Jamieson**

Respondent submitted two expert reports from Dr. Jamieson, a neurologist. Resp't. Exs. A & E. Dr. Jamieson reviewed petitioner's medical history and agreed that petitioner developed GBS and resultant PRES. Resp't. Ex. A at 14; *see also* Resp't. Ex. E at 1. She agreed with Dr. Hixson that petitioner's development of GBS was atypical, stating, "In my opinion, [petitioner's] neurological presentation with acutely elevated blood pressure prior to developing severe weakness and areflexia, while likely due to GBS, was atypical." Resp't. Ex. E at 1. She also wrote, "[petitioner's] dysautonomia appears to be related to the onset of acute inflammatory demyelinating neuropathy (AIDP), a subtype of GBS." *Id.* Without providing a possible alternative cause, Dr. Jamieson opined that petitioner's development of GBS and PRES, were unrelated to the Prevnar-13 vaccine. Resp't. Ex. A at 14; Resp't. Ex. E at 2.

In her first report, Dr. Jamieson focused on medical literature that did not find a "causative correlation between pneumococcal vaccination and GBS." Resp't. Ex. A at 17.

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[18] Raju, R. et al., *Epitopes for Human CD4+ Cells on Diphtheria Toxin: Structural Features of Sequence Segments Forming Epitopes Recognized by Most Subjects,* 25 Eur. J. Immunol. 3207-3214 (1995). [Pet'r. Ex. 69].

[19] Haber, P. et al., *Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged ≥ 19 years old in the United States, Vaccine Adverse Events Reporting System (VAERS), June 1, 2012-December 31, 2015,* 24 Vaccine 6330-6334 (2016). [Resp't. Ex. F, Tab 11].

She first referenced the epidemiological study by Baxter et al., which examined GBS after vaccination and found "no increased risk of GBS following any vaccination," within a six-week period after vaccination. Resp't. Ex. A, Tab 9.[20] The authors stated that their "study did not find any association between influenza vaccine or any other vaccine and development of GBS within 6 weeks following vaccination," but also acknowledged that they were "unable to exclude *any* possible association between vaccines and GBS," and that they had " limited power to fully assess the risk of GBS following vaccination *due to the rarity of the outcome*." *Id.* at 8 (emphasis added). Further, the authors also wrote that their study should provide "reassurance that the risk of GBS following any vaccine, including influenza vaccines, is extremely low." *Id.*

Dr. Jamieson also referenced the Souayah et al. article, which examined VAERS reports of GBS following different types of vaccination, including after the pneumococcal polyvalent vaccine and found 1 case. Resp't. Ex. A, Tab 11 at 2.[21] Dr. Jamieson also referenced the Haber article, to support her opinion that the pneumococcal vaccine is not associated with GBS. She acknowledged that Haber identified 11 cases of GBS following administration of the Prevnar-13 vaccine, which included 10 cases listing the PCV-13 as the only vaccine administered. Resp't. Ex. A at 17. Finally, Dr. Jamieson stated that the Tseng et al. article did not find a "significantly elevated risk of GBS with vaccination with PCV-13 as compared to PPSV23." *Id.* Tseng examined adverse events in adults who received the Prevnar-13 vaccine and identified 4 cases of GBS following administration of the Prevnar-13 vaccine, but 8 cases following administration of the PPSV23. Resp't. Ex. A, Tab 13 at 6.[22] The authors concluded that there was "no significantly elevated risk of cardiovascular events, Bell's palsy, Guillain-Barré syndrome, syncope, erythema multiforme, thrombocytopenia, cellulitis and infection, or allergic reaction compared with PPSV23." *Id.* at 7.

Dr. Jamieson concluded her first report stating that petitioner developed symptoms of AIDP approximately 2 weeks after receiving the Prevnar-13 vaccine, and following the onset of AIDP symptoms, petitioner developed elevated blood pressure and developed clinical and imaging findings consistent with PRES. Resp't. Ex. A at 19. Dr. Jamieson stated that "no reproducible or reliable epidemiological studies have postulated any causal association, and isolated case reports of GBS occurring after vaccination do not establish a causal link between [petitioner's] vaccination with Prevnar-13 and her subsequent neurological conditions." *Id.*

In her supplemental report, Dr. Jamieson responded to Dr. Steinman's report and his theory of molecular mimicry between components of the Prevnar-13 vaccine and the peripheral nervous system. Resp't. Ex. F. Specifically, Dr. Jamieson argues that autoantibodies directed to contactin-1 and/or Caspr2 are related to a new category of peripheral neuropathies that have clinical manifestations more consistent with CIDP than GBS. *Id.* at 3. She criticized Dr.

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[20] Baxter, R. et al., *Lack of Association of Guillain-Barre Syndrome with Vaccinations,* 57(2) Clin. Infect. Dis. 197-204 (2013). [Resp't. Ex. A, Tab 9].

[21] Souayah, N. et al., *Guillain-Barre Syndrome After Vaccination in United States: A Report from the CDC/FDA Vaccine Adverse Event Reporting System,* 25 Vaccine 5253-5255 (2007). [Resp't. Ex. A, Tab 11].

[22] Tseng, H. et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults,* Open Forum Infec. Dis. (2017). [Resp't. Ex. A, Tab 13].

Steinman's reference to Devaux to demonstrate that autoantibodies to nodes and paranodes exist in patients with GBS, stating, "Since the publication of Devaux et al. in 2012, the diseases associated with multiple specific autoantibodies that target proteins in the peripheral nervous system, including in the nodal/paranodal regions of nerves, have been more specifically delineated as distinctly different, with unique clinical presentations and pathophysiological mechanisms." *Id.* at 3. Dr. Jamieson stated, "Contactin as a disease target applies to these recently identified CIDP-like autoimmune nodopathies, not to an acute demyelinating subtype of GBS, which is the type of peripheral neuropathy that caused [petitioner's] symptoms." *Id.* Dr. Jamieson stated that "[c]ontactin plays a crucial role in the organization and maintenance of the nodal/paranodal regions, not the myelin sheath, in myelinated axons." Resp't. Ex. E at 4. She stated that in GBS, the immune system produces antibodies that target gangliosides or peripheral nerve myelin sheath, not contactin. *Id.*

Dr. Jamieson concluded her second report reiterating her opinion that petitioner's Prevnar-13 vaccine was not the cause of her developing GBS and PRES, based on the lack of epidemiological studies and a lack of causal connection between the vaccine and GBS. Resp't. Ex. E at 5.

**2. Dr. Robert S. Fujinami's Opinion**

Respondent also submitted two expert reports from Dr. Robert Fujinami, PhD. Resp't. Ex. C & F. Dr. Fujinami deferred to Dr. Jamieson's opinion on petitioner's diagnosis and focused his two reports responding to petitioner's experts, Dr. Hixson and Dr. Steinman.

In his first report, Dr. Fujinami opined that petitioner's GBS was not caused by the Prevnar-13 vaccine because, in general, "vaccines, including the Prevnar-13 vaccine, do not increase the risk of GBS." Resp't. Ex. C at 3. Dr. Fujinami, referring to the National Institute of Neurological Disorders and Stroke Fact Sheet on GBS, wrote, "The exact cause of GBS is not known," and that most cases of GBS "usually start a few days or weeks following a respiratory or gastrointestinal viral infection." *Id.* at 4. Referring to the Sejvar et al. article, which is a paper that explained the use of the Brighton Collaboration GBS Working Group's standardized case definition for GBS, Dr. Fujinami asserts that GBS cases following vaccines was a temporal association only and that the mechanism of molecular mimicry between the flu vaccine and GBS has yet to be proven. *Id.* at 4. Dr. Fujinami wrote, "There is no scientific information demonstrating that influenza vaccine caused GBS during the vaccination program in 1976 through the mechanism of molecular mimicry." *Id.* Dr. Fujinami referenced his own work between the hepatitis B antigen and autoimmune conditions but distinguishes his work from Dr. Hixson's proposed theory between the Prevnar-13 vaccine and GBS. *Id.* at 7.

Dr. Fujinami largely criticizes Dr. Hixson's opinion regarding the mechanism of molecular mimicry between the Prevnar-13 vaccine and GBS because Dr. Hixson "does not provide any information about what it is in the Prevnar-13 vaccine that has molecular mimicry with any component in the [peripheral nerve tissue]." Resp't. Ex. C at 5, 7. In his criticism of Dr. Hixson's theory, Dr. Fujinami explains that the Yih et al. article, which Dr. Hixson referenced, is not applicable because the article is examining the safety of the Tdap vaccine and

adverse events. *See* Pet'r. Ex. 19.[23] Yih which examined adverse events following the Tdap vaccine, including GBS, from information in the Vaccine Safety Datalink, identified 4 cases of GBS following administration of the Tdap vaccine. Pet'r. Ex. 19 at 4. Despite Dr. Fujinami's assertion that "rationale for the 42-day observation window is not provided," the authors stated that they chose the six-week timeframe for GBS after vaccination because that "is considered the period of elevated risk after administration of other vaccines." *Id.* at 1. Discussing their finding for GBS after vaccination, the authors wrote, "….we can be relatively confident that the vaccine does not increase the risk of GBS 4-5 times above the background risk….We can be relatively confident that the vaccine does not confer a risk greater than about 1 excess case per 100,000 of Tdap." *Id.* The authors concluded that their study "provides some reassurance that the Tdap vaccine has a similar safety profile to that of Td vaccine." *Id.* at 5. Further, while Dr. Fujinami asserts that the Yih article is irrelevant because it compares the Td vaccine and the Tdap vaccine, he fails to consider the fact that the Prevnar-13 vaccine contains the same diphtheria molecule that is included in the Tdap vaccine. Dr. Fujinami also referred to the Haber article, which is discussed above, as evidence that there is no association between the Prevnar-13 vaccine and GBS. Resp't. Ex. C at 5.

Dr. Fujinami also opined that the appropriate timeframe for the development of an "autoimmunologic response" is approximately 10 days post-vaccination, not two-weeks, as asserted by Dr. Hixson. Resp't. Ex. C at 7. Dr. Fujinami explained that the onset of an immunologic response would be faster because the Prevnar-13 vaccine includes an adjuvant, which would result in a faster response than two to three weeks. *Id.* Dr. Fujinami closes his first report opining that the development of petitioner's GBS was coincidental to her receipt of the Prevnar-13 vaccine and that the molecular mimicry provided by Dr. Hixson is non-specific.

In his second report, Dr. Fujinami opined that Dr. Steinman's theory of molecular mimicry between two different components of the Prevnar-13 vaccine to the peripheral nervous system is flawed and unreliable. Resp't. Ex. F at 1, 11.

With respect to Dr. Steinman's theory regarding antibodies to phospholipids generated by the Prevnar-13 vaccine cross-react with phospholipid structures in the peripheral nervous system, Dr. Fujinami asserts that Ho et al. simply demonstrates that the phospholipids that were identified can help reduce neuroinflammation and maybe useful in identifying new therapies for neuroinflammatory demyelinating diseases. Resp't. Ex. F at 4. However, he somewhat conflates the discussion of the role of the myelin phospholipids that was discussed in the article and how autoantibodies affect the myelin phospholipids. Dr. Fujinami states that "the lipids were found to "treat" or ameliorate neuroinflammatory demyelinating disease," which is consistent with Ho, however, he does not explain that Ho found that these phospholipids are targeted by autoantibodies in MS, reducing their neuroinflammatory role. *See* Resp't. Ex. F at 4. Ho states:

> We show that autoantibody-targeted phospholipids ameliorate disease in a mouse model of MS by inhibiting the auto-aggressive T cell response that underpins autoimmune demyelination….Autoantibody targeting may thus reduce the lipids' anti-inflammatory

[23] Yih, W. K. et al., *An Assessment of the Safety of Adolescent and Adult Tetanus-Diphtheria-Acellular Pertussis (TdaP) Vaccine, Using Active Surveillance for Adverse Events in the Vaccine Safety Datalink,* 27 Vaccine 4257-4262 (2009). [Pet'r. Ex. 19].

> effect by enhancing their clearance or inhibiting their activity-and thereby compromise the lipid-mediated protection against neuroinflammation.

*Id.* Further, Ho et al. states clearly, "The destruction of myelin involves anti-lipid autoantibodies, which can induce demyelination and prevent remyelination in mouse models of MS." *Id.* The article continues:

> Through antigen array screening, we found that the CSF of MS patients contains antibodies to oxidized phosphatidylcholine derivatives, and antibodies to oxidized phosphatidylcholine have been detected in MS brain lesions, supporting the idea that the antibodies to oxidized phosphatidylcholine derivatives that we detect in CSF can bind their lipid targets in MS brain. Moreover, studies in EAE indicate that antibodies to oxidized phosphatidylcholine are generated as part of the pathological process of autoimmune demyelination. *Apart from the general destruction of the myelin sheath, such autoantibody targeting of oxidized phosphatidylcholine derivatives could conceivably contribute to MS pathogenesis by reducing the levels or blocking the immunoregulatory activity of these protective lipids.*

*Id.* (emphasis added).

Dr. Fujinami argues that the anti-phospholipid antibodies that Dr. Steinman is proposing were created by the immune response to the Prevnar-13 vaccine do not actually initiate the demyelination, but that the autoantibodies to the phospholipids are generated because of the demyelination through epitope spreading. Resp't. Ex. F at 5-6. Referring to Gilburd et al., Dr. Fujinami states that the article found that the autoantibodies reactive to phospholipids were produced as a result of the myelin damage rather than the cause of the demyelination. *Id.* at 5; *see also* Resp't. Ex. F, Tab 10.[24] However, it should be noted that Gilburd was a 2012 paper published before Ho, Nakos, Bryson and others and that the authors merely speculated that the autoantibodies were produced by the disease and not a cause of it. He also cites to Kander et al., which Dr. Steinman co-authored, to support his opinion that anti-phospholipids are not inciting inflammation, but instead, are a result of epitope spreading because of the neuroinflammation. Resp't. Ex. F at 6-7. Kander et al. found that autoreactive B-cells in EAE "undergo intermolecular epitope spreading to target lipid components of the myelin sheath," but that autoreactive antibodies to myelin lipids also contribute to the pathogenesis of autoimmune demyelinating disease. Resp't. Ex. F, Tab 13 at 4-5.[25]

Dr. Fujinami also argues that Dr. Steinman's CRM-197 molecular mimicry theory is also unreliable because most individuals that get immunized against diphtheria do not develop GBS. Resp't. Ex. F at 8. He referred to the Chen et al. article, which examined adverse events after vaccination in three Chinese cities and found that there was no increased risk of GBS following

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[24] Gilburd, B. et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with Guillain-Barre Syndrome: Cross-Reactive or Pathogenic,* 16(1) Autoimmun. 23-27 (1993). [Resp't. Ex. F, Tab 10.]

[25] Kanter, J. et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation,* 12 Nature Medicine, doi:10.1038/nm1344 (2005). [Resp't. Ex. F, Tab 13].

any vaccination among any age group. *Id.*; *see also* Resp't. Ex. F, Tab 2.[26] Chen states, "Although the underlying etiology and pathophysiology of GBS has not been clearly elucidated, it is thought to be an autoimmune process triggered by antigenic stimulation, resulting in demyelination and destruction of peripheral nerves." Resp't. Ex. F, Tab 2 at 1. While Chen did not study cases of GBS following the Prevnar-13 vaccine or PPSV-23, the study 7 cases of GBS following the Tdap vaccine occurring 4-42 days post-vaccination. *Id.* at 5. The authors concluded that they could not find any evidence to demonstrate "an association of vaccines with an increased risk of GBS and its recurrence among either pediatric or adult individuals," however, they study also noted that "it is biologically plausible that immunizations may lead to subsequent GBS in rare cases." *Id.* at 6. To Dr. Fujinami, Dr. Steinman's mechanism of molecular mimicry between the CRM-197 and components of the peripheral nerves fails to take into account the fact that there is no increased risk of GBS following any vaccination that includes the diphtheria toxoid. Resp't. Ex. F at 8.

Finally, Dr. Fujinami also argues that Dr. Steinman's opinion regarding the timing of GBS onset and vaccination (which is similar to Dr. Hixson's opinion), is unreliable because it is based on the Schonberg article which examines GBS cases following the 1976-77 swine flu vaccine and unrelated to the vaccine at issue in this case. Resp't. Ex. F at 11. Dr. Fujinami refers back to the Haber article, which he reiterated found "no evidence that the Prevnar-13 vaccination increased the risk of GBS." *Id.*; *see also* Resp't. Ex. C, Tab 3. But like Gilburd, Haber merely concluded based on early and limited data that Prevnar did not increase the risk of GBS it did not attempt to do more than report the cases reported to VAERS and draw a conclusion based on those reports alone. Dr. Fujinami also referenced the Chen article, discussed above, and the Baxter study, to support his opinion that there is no increased risk of GBS following a pneumococcal vaccination. Resp't. Ex. F at 11. He ultimately concluded his supplemental report the same way as his first, opining that petitioner's development of GBS and subsequent PRES was coincidental to her receiving the Prevnar-13 vaccine. *Id.*

## III. 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 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).

[26] Chen, Y. et al., *Vaccines and the Risk of Guillain-Barre Syndrome,* 35 Euro. J. of Epidem. 363-370 (2020). [Resp't. Ex. F, Tab 2].

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.").

**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 I find that petitioner has demonstrated a sound and reliable theory to explain how the Prevnar-13 vaccine can cause GBS, thus satisfying *Althen* prong one.

Petitioner's experts, Drs. Hixson and Steinman propose a theory of molecular mimicry to explain how the Prevnar-13 vaccine can cause GBS. Pet'r. Exs. 12, 34, & 78. Dr. Hixson does not specifically identify molecular mimics between the components of the Prevnar-13 vaccine and the peripheral nervous system, however, Dr. Steinman identified two possible molecular mimics between the Prevnar-13 vaccine and self-antigens in the peripheral nervous system. *See* Pet'r. Ex. 34 at 25-44; Pet'r. Ex. 78 at 4-12. While many of the articles filed explain that the

exact pathogenesis of GBS is not well understood, molecular mimicry is a possible mechanism is described in articles cited by both parties. *See* Pet'r. Ex. 13 at 2 ("The acute polyneuropathy of GBS is often triggered when an immune response to an antecedent infection or other event cross-reacts with shared epitopes on peripheral nerve (molecular mimicry.)); Resp't. Ex. A, Tab 5 at 6[27] ("Some evidence supports the presence of molecular mimicry between gangliosides and antecedent infectious agents in patients with Guillain-Barré syndrome and those with Miller Fisher Syndrome."); Resp't. Ex. A, Tab 3 at 8[28] ("Postinfectious molecular mimicry is the predominant pathophysiologic mechanism in GBS, particularly in Miller Fisher syndrome and axonal variants….Although the pathogenesis of AIDP is largely undetermined, evidence of molecular mimicry exists in a small minority of patients with AIDP. A number of patients develop GBS after *M. pneumoniae* infection. *M. pneumoniae* expresses antigens that cross-react with galactocerebroside (GalC), a glycolipid that is enriched in the myelin sheath of Schwann cells in peripheral nerves."). Additionally, the literature also endorses as GBS occurring post-vaccination, albeit a rare event. *See* Resp't. Ex. A, Tab 3 at 10 ("Noninfectious events, including trauma, vaccinations, immunosuppression, and pregnancy, may rarely trigger GBS.").

Further, molecular mimicry has been generally accepted as a sound and reliable theory to explain vaccine causation of 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).

But more specific to the vaccine at issue in this case, Prevnar-13, I and other special masters have accepted molecular mimicry as a sound and reliable theory for vaccine causation of GBS. *See McKenney v. Sec'y of Health & Hum. Servs.,* No, 19-1799V, 2025 WL 4229212 (Fed. Cl. Spec. Mstr. Dec. 30, 2025); *Diponziano v. Sec'y of Health & Hum. Servs.,* No. 17-1130V, 2025 WL 942744, at *19-24 (Fed. Cl. Spec. Mstr. Feb. 11, 2025); *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. 20-1476V, 2024 WL 4003061, at *21-26 (Fed. Cl. Spec. Mstr. July 8, 2024); *Koller v. Sec'y of Health & Hum. Servs.,* No. 16-439V, 2021 WL 5027947, at *10-20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021); *see also 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)

[27] Yuki, N. et al., *Guillain-Barre Syndrome,* 266 N. Eng. J. Med. 2294-304 (2012). [Resp't. Ex. A, Tab 5].
[28] Sheikh, Karen, *Guillain-Barré Syndrome,* 23(5) Continuum (Minneap Minn.) 1295-1309 (2017). [Resp't. Ex. A, Tab 3].

(Horner); *Datte v. Sec'y of Health & Hum. Servs.,* No. 18-2V, 2025 (Fed. Cl. Spec. Mstr. May 9, 2025) (Horner); *Parker v. Sec'y of Health & Hum. Servs.,* No. 20-411V, 2023 WL 9261248 (Fed. Cl. Spec. Mstr. Dec. 20, 2023) (Dorsey); *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).

Acceptance of molecular mimicry in Prevnar-13-GBS cases has not been uniform among special masters. *See Bialek v. Sec'y of Health & Hum. Servs.,* No. 18-761V, 2023 WL 35509, at *33-37 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (Corocran); *Trollinger v. Sec'y of Health & Hum. Servs.,* No. 16-473V, 2023 WL 2521912, at *25 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for rev. den'd,* 2023 WL 5249583 (Fed. Cl. 2023) (Corcoran); *Morrison v. Sec'y of Health & Hum. Servs.,* No. 18-386V, 2024 WL 3738934, at *17-23 (Fed. Cl. Spec. Mstr. July 18, 2024) (Oler). Most recently, in *Gamboa-Avila,* the Federal Circuit upheld the special master's decision which found that Dr. Steinman's theory of molecular mimicry between the Prevnar-13 vaccine and components of the peripheral nervous system was unreliable. But the Circuit observed the inconsistent results in Vaccine Program cases based on identical facts. *Gamboa-Avila v. Sec'y of Health & Hum. Servs.,* ----F.4th--- 2026 WL 375758 (Fed. Cir. 2026). Regardless of those decisions, they are not binding on me, but I have considered the reasoning of those decisions and come to a different conclusion. *See Boatmon v. Sec'y of Health & Hum. Servs.,* 941 F.3d at 1358-59.

Dr. Steinman presented his theory of molecular mimicry between the phospholipids contained in the Prevnar-13 vaccine and phospholipid components of myelin in previous cases I have considered, as he has done in this one. *See e.g. Koller,* 2021 WL 5027947, at *10-20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). He presented several pieces of literature, including the patent application for the vaccine itself, to demonstrate that the Prevnar-13 vaccine contains a phosphoglycerol component, and that the phosphoglycerol must be retained in order to achieve a sufficient immune response to the vaccine. He presented his theory, as set forth in this case, that antibodies to the phosphoglycerol cross react with the vaccine and the phosphoglycerol headgroup attached to phospholipids in the cellular membrane of myelin to demonstrate that molecular mimicry with components of the vaccine can cause patients to develop anti-phospholipid antibodies, to the phospholipids in myelin tissue giving rise to GBS. *See* Pet'r. Ex. 34 at 25-28; Pet'r. Ex. 50 at 7; Pet'r. Ex. 44 at 5-6.

As Dr. Steinman has done in other cases involving the Prevnar-13 vaccine and GBS, he has gone beyond simply invoking the phrase molecular mimicry, to demonstrate that an immune response to the vaccine, which it is intended to do, could recognize similar structures that are found on peripheral nerves, and cause demyelination. The Chang et al. article explains how the immune system reacts to the phosphoglycerol component of serotypes 18C, which is included in the vaccine and that this component is necessary to confer immunogenicity. Pet'r. Ex. 50 at 7. Then Nakos identified antiphospholipid antibodies in patients with GBS, and opined that their patients did not have any other underlying autoimmune condition to which these autoantibodies

to the phospholipids could have been generated. Pet'r. Ex. 44 at 6. Nakos was clear that "it is not well understood whether these anti-phospholipid antibodies play a role in the pathogenesis of the polyneuropathy or represent a part of a more extensive immunoreaction that takes place in GBS," but that the presence of these antibodies "suggest that in GBS there is a more extensive immune reaction beyond the well-known anti-ganglioside production." *Id.*

As many of the articles explain, the exact cause of pathogenesis of the AIDP variant of GBS is not well understood, even as anti-ganglioside antibodies have been identified in the AMAN variant of GBS. *See e.g.* Resp't. Ex. A, Tab 4 at 10 ("Despite our understanding of the pathology and electrodiagnosis of AIDP and its much greater prevalence than AMAN or AMSAN, the immunologic sequence causing AIDP is less understood. Since a wide range of viruses and bacterial agents can incite an antibody in AIDP, it has been difficult to find a common antigenic stimulus for the illness."); *see also* Resp't. Ex. A, Tab 5 at 5 ("The pathogenesis of the demyelinating Guillain-Barre syndrome has yet to be clarified, despite the documentation of characteristic histologic changes."). Willison and van Doorn articulate this exact point, stating:

> By contrast with acute motor axonal neuropathy (form of GBS), the immunological cascade involved in acute inflammatory demyelinating polyneuropathy is less well understood for various reasons. First, a wider range of immune stimulants cause acute inflammatory demyelinating polyneuropathy compared with acute motor axonal neuropathy, which includes bacterial and viral infections, and vaccines. Second, specific antibody biomarkers have yet to be characterized, despite widespread screening efforts to identify the putative nerve antigens. *At present, a wider range of anti-nerve autoantibodies directed at both proteins and glycolipids could be responsible for acute inflammatory demyelinating poly neuropathy immunopathology…*

Resp't. Ex. A, Tab 1.[29] This difference in the immunopathology between the two variants of GBS-AIDP and AMAN-is a critical distinction. The AMAN variant of GBS has a well identified immune target, while research on the AIDP-GBS variant have suggested several targets including those theorized by Dr. Steinman but the research has not reached the level of medical certainty, making it less likely that the petitioner in this case or any other AIDP-GBS case will be able to identify the specific immune target on the peripheral nerves . To require petitioner to demonstrate a theory by such specificity and scientific certainty is not required in the vaccine program. *Knudsen,* 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program.").

Further, Dr. Steinman provided support for his theory of molecular mimicry between the diphtheria protein and various proteins found in the peripheral nervous system. In his first report, he opined that there is molecular mimicry between the CRM-197 and contactin-1, a protein found at the nodal and paranodal locations on the peripheral nerves. Pet'r. Ex. 34 at 48-52. In his second report, Dr. Steinman expanded his search for molecular mimics between the diphtheria toxin and myelin antigens P0, P2, and PMP-22. Pet'r. Ex. 78 at 3-4. I have accepted Dr. Steinman's theory of molecular mimicry between CRM-197 and contactin-1 in other cases

[29] Willison, H. & van Doorn, P., *Guillain-Barré Syndrome,* 388 Lancet 717-27 (2016). [Resp't. Ex. A, Tab 1].

involving Prevnar-13 and GBS. *See Musick,* 2025 WL 2452232, at *38-39. Additionally, I have accepted molecular mimicry between the diphtheria toxoid and P2, P0, and PMP-22 in *McKenney,* another Prevnar-13-GBS case. *McKenney,* 2025 WL 4229212, at *11-12. Beyond acceptance in previous cases though, the medical literature referenced by Dr. Steinman supports his theory that autoantibodies to different proteins in the peripheral nervous system, are pathogenic in GBS.

The Appeltshauser article that Dr. Steinman referenced in his rebuttal report lends support to the mechanism of molecular mimicry between the diphtheria toxoid and contactin-1. Pet'r. Ex. 80 at 1. Dr. Jamieson correctly observed that some autoantibodies against the paranodal antigens contactin-1, Caspr-1, and neurofascin-155 have been associated with CIDP. Resp't. Ex. E at 2-3. But Appeltshauser found antiparanodal antibodies IgG2 and IgG3 in GBS patients, and that IgG3 autoantibodies reacted against contactin-1 and caspr-1 epitopes while IgG4 class autoantibodies were specific to Caspr-1, suggesting that different binding characteristics of the class of antiparanodal autoantibodies. *Id.* at 9. This article, along with the Devaux article, support Dr. Steinman's theory that nodal and paranodal structures in the peripheral nervous system are immune targets in GBS, along with other immune mediated neuropathic conditions. *See* Pet'r. Ex. 58.

The Sukenikova article demonstrates that autoreactive immune cells targeting myelin antigens, such as P0, P2, and PMP-22, have been found in patients with AIDP. *See* Pet'r. Ex. 81 at 8. The authors found CD4+ and CD8+ T-cells targeting P0, P2, and PMP-22 myelin antigens in the blood, CSF, and nerve tissue of a group of GBS patients with the demyelinating AIDP variant. *Id.* The authors wrote, "Our findings also identify common self-epitopes targeted across patients with AIDP, which are known to have a key physiological role."

Respondent's experts stress that there are no epidemiological studies that demonstrate an association between the Prevnar-13 vaccine and GBS. *See* Resp't. Ex. A at 16; Resp't. Ex. C at 4-5; Resp't. Ex. E at 5. Respondent's experts stressed the findings in the Haber, Baxter, and Tseng studies as evidence that Prevnar-13 does not cause GBS. A lack of epidemiological evidence is not dispositive. Where such evidence is submitted, "the special master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury." *Andreu,* 569 F.3d 1367,1379 (2009).

Haber examined VAERS reports to examine the safety profile of the Prevnar-13 vaccine and found 11 cases of GBS reported within 42-days of vaccination. Resp't. Ex. F, Tab 11. But as explained above, and as recognized by Dr. Steinman, Haber and co-authors explained that there are limitations with VAERS, including underreporting, varying quality of data, and lack of an unvaccinated comparison group, making it "extremely difficult to determine causal associations between vaccines and AEs." *Id.* at 5. Baxter examined GBS cases from the Kaiser Permanent Northern California ("KPNC") patient population and only identified 2 cases of GBS within six weeks of a polysaccharide pneumococcal (as opposed to the conjugate) vaccine. Resp't. Ex. A, Tab 9. The study "did not find any association between influenza vaccine or any other vaccine and the development of GBS within six weeks following vaccination," but they noted that they had "limited power to fully assess the risk of GBS following vaccination due to the rarity of the outcome." *Id.* at 8. Finally, Tseng assessed adverse events following administration of the polysaccharide pneumococcal vaccine against adverse events following the

conjugate pneumococcal vaccine. Resp't. Ex. A, Tab 13. The study identified 4 cases of GBS after administration of the conjugate pneumococcal compared to the 8 GBS cases following the polysaccharide vaccine. *Id.* at 6. The authors concluded that the risk of GBS following the conjugate vaccine was no greater than the risk following administration of the polysaccharide vaccine. *Id.* at 7. However, the authors did assess the background rate of GBS following the polysaccharide against any control. *Id.*

I have considered these three studies relied upon by respondent and find that they do not undermine Dr. Steinman's theory. Each of these studies conclude that there is no *increased* risk of GBS post-vaccination, not that vaccines are not the cause of GBS in rare instances. Further, the lack of identifying an increased risk of GBS after vaccination does not answer the question of whether petitioner has provided a sound and reliable *theory* for how the vaccine can cause the type of injury being alleged. It is not petitioner's burden to demonstrate that the risk of the alleged injury is significantly increased by a vaccine, but instead, how the vaccine could cause the injury being alleged. "While epidemiological studies can establish an increased incidence, and this increased incidence can support an inference of causation, epidemiological studies cannot absolutely refute a causal connection. Epidemiological studies cannot prove a negative. *Harris v. Sec'y of Health & Hum. Servs.,* 2014 WL 3159377, at *11 (Fed. Cl. Spec. Mstr. June 10, 2014), *mot. for review dismissed,* 2015 WL 2129036. A vaccine injury is a rare event that cannot be disproved because a vaccinee did not experience a response consistent with that of the general population. *Walls v. Sec'y of Health & Hum. Servs.* No. 16-557V, 2020 WL 13801342, at *16 (Fed. Cl. Spec. Mstr. June 23, 2020). Petitioner does not need to provide epidemiological evidence to show that a vaccine can cause an injury. *See Andreu,* 569 F.3d at 1378 (quoting *Capizzano,* 440 F.3d at 1325-26); *see also Althen,* 418 F.3d at 1280 (noting that "close calls" are resolved in a petitioner's favor).

After considering the evidence as a whole, I find that petitioner has provided by preponderant evidence a sound and reliable theory for how 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).

Petitioner received the Prevnar-13 vaccine on February 12, 2019, and on March 2, 2019, petitioner reported symptoms of tingling in her fingers for four days. Pet'r. Ex. 3 at 81. This would put the onset of petitioner's symptoms at approximately fourteen days post-vaccination. The parties do not dispute that the onset of petitioner's symptoms began approximately 18 days

after vaccination, not do they dispute petitioner's diagnosis. However, respondent's expert. Dr. Fujinami argues that the onset of petitioner's GBS is simply coincidental to her vaccination and that the reliance on Schonberger is not relevant, as it examines a different vaccine. Resp't. Ex. C at 7; Resp't. Ex. F at 11.

Even though the Schonberger article does involve the 1976-77 swine flu vaccine and GBS, the timeframe of six-weeks was utilized in assessing risk periods in some of the studies referenced in this case. *See* Resp't. Ex. A, Tab 9; Pet'r. Ex. 19 at 2. In the Haber article, 11 cases of GBS following administration of the Prevnar-13 vaccine were reported within the six-week timeframe, and the median onset interval was nine days. The Yih article, which examined GBS after administration of the Tdap vaccine, stated that "the exposure window of interest was 1-42 days after vaccination," because "For GBS, 6 weeks is considered the period of elevated risk after administration of other vaccines," and cited to Center for Disease Control and Prevention's work regarding GBS following administration of the Menactra Meningococcal conjugate vaccine. Pet'r. Ex. 19 at 2.

Additionally, the onset of petitioner's symptoms, occurring fourteen days post-vaccination has been acknowledged as appropriate in other Vaccine cases in which molecular mimicry has been proffered as the causal mechanism. *See e.g. Diponziano,* 2025 WL 942744, at *28 (finding a 11-day onset of GBS after administration of Prevnar-13 an appropriate timeframe); *Sprenger,* 2023 WL 8543435, at *22 (finding a 15-day onset of GBS after administration of the Prevnar-13 an appropriate timeframe); *Gross,* 2022 WL 9669651, at *38-39 (finding a GBS onset of 13 days after Prevnar-13 vaccination to be appropriate).

Accordingly, the undersigned finds that petitioner has provided preponderant evidence that the onset of her symptoms began within a medically acceptable time frame, 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.").

There is no disagreement between the parties that petitioner suffered from GBS with secondary PRES. Pet'r. Ex. 12 at 5; Resp't. Ex. A at 19. As I have concluded above, petitioner that proffered a sound and reliable theories of vaccine causation and the onset of her symptoms was approximately 14-days post-vaccination, an appropriate time frame in which molecular mimicry could cause GBS. "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 that a vaccine was the cause of her AIDP and subsequent PRES. During her hospitalization from March 14, 2019 through April 1, 2019, in a progress note, Dr. Shah wrote, "65 y/o woman with a complex neuromuscular course and PRES/seizures. The current though is that she had a vaccine provoking GBS provoking autonomic dysregulation causing PRES and seizures." Pet'r. Ex. 4 at 522. When petitioner was discharged from Swedish Medical Center Rehab on April 11, 2019, her discharge summary includes a description of her course treatment, which provides, "3/26: Pt with hallucinations. EEG showed intermittent delta activity without seizures. BP was elevated to 203/94. Neurology working etiology is felt to be vaccine that provoked GBS which provoked autonomic dysregulation and PRES with seizures." *Id.* at 282. At her rehabilitation medical appointment on August 27, 2019, it was noted that petitioner's "[t]rigger for the Guillain-Barre is unclear, may have been related to recent vaccine." Pet'r. Ex. 4 at 257. Further, other possible etiologies for the cause of petitioner's GBS and PRES were ruled out. *See* Pet'r. Ex. 4 at 452 (negative meningitis panel negative, negative lyme IgG/IgM; *see also* Pet'r. Ex. 4 at 566 (Plasma metanephrine and catecholamines not consistent with pheochromocytoma). Nor do Drs. Jamieson and Fujinami offer another possible explanation for the cause of petitioner developing GBS and subsequent PRES.

While petitioner's case is somewhat unique as that she developed PRES secondary to the GBS, her clinical course is consistent with other GBS cases following a Prevnar-13 vaccination. Most notably the petitioners in *Byrd, Diponziano,* and *Datte* all required treatment in intensive care units and then experienced a protracted recovery, which included time in rehabilitation facilities and the need for extensive physical and occupational therapy. The petitioner in *Byrd* began to experience GBS symptoms approximately four days after the Prevnar-13 vaccination and progressed to being unable to walk, requiring treatment in the intensive care unit, intubation, five days of IVIG, and then transfer to a long-care nursing facility. *Byrd,* 2024 WL 4003061, at *26-28. 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. Similarly, the petitioner in *Datte,* 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.

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.

## IV. Conclusion

For the reasons discussed above, the undersigned finds that petitioner has established by preponderant evidence that her Prevnar-13 vaccine she received on February 12, 2019, caused her GBS and subsequent PRES. 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