In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-761V

(to be published)

RAYMOND BIELAK,	*	
	*	Chief Special Master Corcoran
Petitioner,	*	Filed: December 9, 2022
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Joseph Alexander Vuckovich, Maglio Christopher & Toale, P.A., Washington, D.C., for Petitioner.

Ronalda Elnetta Kosh, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On May 31, 2018, Raymond Bielak filed this action seeking compensation under the National Vaccine Injury Compensation Program (the "Program").[2] ECF No. 1. Petitioner alleges that the pneumococcal vaccine (marketed under the tradename "Prevnar-13") he received on September 22, 2015, caused him to incur Guillain-Barré syndrome ("GBS"). A two-day entitlement hearing in the matter was held in Washington, D.C., from April 12-13, 2022.

Having reviewed the record, all expert reports and associated literature, and listened to those witnesses and experts who testified at the hearing, I hereby deny an entitlement award. As

[1] This Decision will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet**. As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Ruling's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire Decision will be available to the public in its current form. *Id.*

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

discussed in greater detail below, Petitioner has not preponderantly established that the pneumococcal vaccine can cause GBS, or did so to him.

I. Fact History

Pre-Vaccination History, Vaccination, and Onset of Symptoms

Prior to the vaccination at issue, Petitioner's medical history was significant for coronary artery disease with stents, gastroesophageal reflux disease, gout, hyperlipidemia, hypertension, hypothyroidism, and lumbosacral radiculopathy. Ex. 2 at 15. He had also experienced back pain secondary to trauma from a fall, plus muscle weakness and numbness in his left leg. Ex. 9 at 16, 58. And Petitioner received periodic cortisone injections for his lumbosacral radiculopathy. Ex. 2 at 18; Ex. 9 at 6, 40, 56.

On September 22, 2015, Mr. Bielak (then 65 years old) was administered a pneumococcal vaccine. Ex. 1 at 1. Two weeks later, at a cardiology appointment on October 6, 2015, he reported malaise, myalgia, and fatigue, along with decreased sensation to his fingers and foot (lateralization unspecified). Ex. 5 at 5. This was described in the cardiology note as "a reaction with the pneumonia immunization." *Id.*

Then, on October 9, 2015, Petitioner presented to a pain management clinic with complaints of "[b]ilateral hand and foot numbness and tingling" that he reported had been ongoing for approximately one week. Ex. 9 at 12. He stated that he was "unable 'to feel his feet' when standing and walking," and that he was experiencing progressive "'shooting' sensations radiating up into his forearms and calves . . . ." *Id.* He also indicated that he was curious whether the pneumococcal vaccine had been the cause of these symptoms. *Id.* Upon physical examination, Certified Physician Assistant ("PA") Mark Udy observed that Petitioner displayed diminished deep tendon reflexes in his lower extremities, and he informed Petitioner "that this may be a side effect from [his] recent [P]revnar vaccination although it is also possible that this could be Guillain-Barre Syndrome . . . ." *Id.* at 14–15. PA Udy therefore instructed Mr. Bielak to seek out an emergency department, as well as a neurologist for further evaluation. *Id.* at 15.

That same day Petitioner presented to Dixie Regional Medical Center, where he was first evaluated by physician Maria Petroulakis, M.D. Ex. 2 at 60. He complained of "[g]eneralized weakness that started mostly on the right side, and numbness, [and] tingling on the hands and feet." *Id.* Petitioner further reported that he:

> was in his usual state of health until 24 hours ago whe[n] he started developing muscle pain and some discoloration in the area o[f] the arm where he received a pneumonia vaccine 1 week prior to that. Following th[is] event, he developed paresthesias, mostly on the lower

extremities and paresthesias in the fingertips. He took Gabapentin[3] more than 3 days and he did not improve. He denies any other associated symptoms. He saw [a] doctor in his pain management clinic today and they suspected Guillain Barre syndrome and sent him here for further evaluation and management. The reason for the suspicion is because he had absent patellar and ankle jerks.

Id. Following the examination, Dr. Petroulakis mentioned the possibility of GBS, requesting a neurology consultation for confirmation. *Id.* at 61.

Corroboration of GBS Diagnosis

Petitioner was subsequently evaluated by neurologist Amalia Geller, M.D. Ex. 3 at 19. The history from this visit describes his condition's clinical course as follows:

> 3 weeks prior to admission, he was in his usual state of health up until he received the pneumococcal vaccination on September 22. Immediately following the vaccination, he did not experience any acute problems until 24 hours later he began noticing tightening in the shoulder and arm (left). Within 48 hours, he began developing discoloration of his arm that he describes as "red streaks"[4] going down from the shoulder to the elbow. This apparently took approximately 6 days to resolve. Following this, then he began to experience flu-like symptoms that he describes as body malaise, soreness, fatigue, [and] weakness that lasted approximately 4 days.
>
> One week now, after this vaccination, he began to develop progressive paresthesias that he describes as [a] burning sensation in his feet that progressed up to the balls of the feet and now up to the fingertips.
>
> This Sunday (5 days prior to [this hospital] admission), the numbness progressed. Four days prior to admission (Monday), the numbness now was involving the entire ball of his foot and he had an acute bout of diarrhea that lasted approximately 48 hours. The following day, he went to see his cardiologist, who felt this was unlikely to be a peripheral vascular disease and recommended to continue monitoring.
>
> The Wednesday of this week, the patient now is beginning to have significant numbness in the feet and the hands as well and increased flushing was noted in his face. Yesterday, the patient began experiencing gait instability and fell in the morning at home. He denies any loss of consciousness or any bodily injury from this fall.
>
> Today, Mr. Bielak went to see his pain specialist and saw the PA on staff this morning. The PA's examination showed absent deep tendon reflexes along the patella and ankle

[3] Gabapentin is administered for treatment of nerve pain. *See generally Gabapentin,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=19523 (last visited Dec. 9, 2022).

[4] This was the first mention of "red streaks" to treaters—this description was not given during Petitioner's first post-vaccination medical encounter on October 6, 2015. Ex. 3 at 19. During the hearing, Mr. Bielak did mention that his arm was red but did not mention the streaking. Tr. at 63.

> jerks. The patient was referred urgently to the emergency room for further evaluation for possible acute inflammatory demyelinating polyneuropathy.

Id. During her examination, Dr. Geller observed that Petitioner's patellar reflexes and ankle jerks were absent, no ankle clonus was elicited, and his plantar responses were mute bilaterally. *Id.* at 21. Based on these results, Dr. Geller expressed a "high suspicion for acute inflammatory demyelinating polyneuropathy/Guillain-Barre syndrome"[5] as the most likely explanation for Petitioner's symptoms. *Id.* at 22. She recommended a cerebrospinal fluid analysis ("CSF") to assess for cytoalbumino-dissociation, (a marker for GBS), initiation of a five-day course of IVIG,[6] and a physical therapy evaluation. *Id.*[7]

Petitioner had minimal improvement following IVIG, and was thus transferred to an acute in-patient rehabilitation facility on October 14, 2015, with a diagnosis of inflammatory demyelinating polyneuropathy. Ex. 3 at 13–14; Ex. 6 at 155. Petitioner participated in a five-day in-patient rehabilitation—consisting of speech therapy, physical therapy, and occupational therapy—during which time he "remained medically stable and had good functional improvement in his short rehab stay." Ex. 6 at 128. He was discharged from in-patient rehabilitation on October 19, 2015, with a diagnosis of GBS. *Id.*

Subsequent Treatment

After discharge, Petitioner continued to receive treatment for his GBS sequelae. Later evaluations provided additional confirmation of his initial diagnosis. *See, e.g.*, Ex. 3 at 6–7 (November 4, 2015 nerve conduction study testing). He was still experiencing some numbness and paresthesias into 2016. *Id.* at 1–4 (February 2016 treatment); Ex. 6 at 88–89 (April 2016 cardiology treatments, where some symptoms were deemed related to GBS). Petitioner's mobility and motor function improved throughout 2016, although not fully, with fatigue and other ongoing sensory issues still common. Ex. 3 at 26, 28. He has reported to treaters through 2019 that he also still experiences some related symptoms, although a second nerve conduction study performed in early

[5] Acute inflammatory demyelinating polyneuropathy, or "AIDP," is the most common GBS variant, so much so that it is often used interchangeably with "GBS" as a descriptive term. *See* Guillain-Barre Syndrome, Mayo Clinic, https://www mayoclinic.org/diseases-conditions/guillain-barre-syndrome/symptoms-causes/syc-20362793 (last visited on Dec. 9, 2022).

[6] Intravenous Immunoglobulin ("IVIG") therapy is used to treat immune system disorders. *See Primary Immunodeficiency*: *Treatment*, Mayo Clinic, https://www mayoclinic.org/diseases-conditions/primary-immunodeficiency/diagnosis-treatment/drc-20376910 (last visited on Dec. 9, 2022). During an IVIG treatment, immunoglobulin (a combination of antibody proteins) is injected into the body to help the immune system fight off infections. *Id.*

[7] Petitioner's CSF analysis confirmed "evidence of a normal white count in the presence of an elevated protein 3 times higher than normal range. This would be consistent with albuminocytologic dissociation." Ex. 3 at 16. It also noted an elevated CSF of 91. *Id.* at 17.

2019 did not identify any further evidence of an ongoing polyneuropathy. Ex. 23 at 4, 10. Since then, Petitioner has also reported some ongoing pain, although neurologic exams have resulted in the determination that his neuropathic symptoms are at least stable. Ex. 31 at 23–25 (August 2019 follow-up visit to neurologist).[8]

II. Witness Testimony

A. *Petitioner's Fact Witnesses*

1. Raymond Bielak – Petitioner testified at hearing on his own behalf. *See generally* Tr. at 60–124. He maintained that he was active and in good health prior to receiving the pneumococcal vaccine in September 2015. Tr. at 60–61. He acknowledged, however, a history of cardiovascular disease and lumbar pain due to a fall in 1971.[9] *Id.* at 95–96. He wore a back brace for the next six months after the accident, although his condition improved rapidly, and he was eventually able to continue with jobs that involved manual labor (like loading carts with hundred-pound sacks of potatoes) and standing on his feet for 12-14 hours a day. *Id.* at 96–97. He did, however, subsequently experience back pain, beginning after 2005, and he required pain management and some injections to help alleviate the associated symptoms. *Id.* at 62, 97–102, 104, 111–12.[10]

Petitioner overall deemed his back pain distinguishable from any GBS-related symptoms, with the former more acute but self-limiting, whereas his GBS symptoms were described as creeping that started in his toes and fingers and gradually became more like "needles and pins," working through his body before becoming very intense and long-lasting. Tr. at 62–63, 91, 97–98, 103. He was thus able to distinguish between these two different injuries, and even sought independent treatment for both after the start of his GBS. *Id.* at 79, 90–91.

Mr. Bielak specifically maintained that in the days immediately following his vaccination, his arm was sore and red, and that he felt sick, while experiencing fatigue and gait instability. Tr. at 63–64, 71, 112, 121–24. (He also described a bout of diarrhea that lasted one day around this time, but he could not remember how long after his vaccination it had occurred. *Id.* at 112). Five to six days from vaccination (by September 27th), he felt the needles and pins increasing in intensity, with the sensation working its way through his arms and legs to the point where he could

[8] Because Petitioner's diagnosis is not disputed, and his subsequent course does not bear on causation, I only provide herein a cursory overview of Petitioner's post-vaccination history.

[9] Mr. Bielak also noted that he had a heart attack in 2012, receiving medication in response. Tr. at 101–02. And at one point he sought a handicap placard because of issues with gout that would hit unexpectedly. *Id.* at 108, 123.

[10] Mr. Bielak used Narco to relieve back pain before he could get an injection. Tr. at 106, 111. He also used Gabapentin for the periods where his back pain would hit, but neither Narco nor Gabapentin were for long-term use, and he only used them as needed. *Id.* at 106–07, 111.

barely walk. *Id.* at 64, 113, 123–24. Petitioner also recalled around this time (though no specific date was provided) that he fell while working as a substitute teacher.[11] *Id.* at 64.

Shortly thereafter, Petitioner's primary care treater recommended he see a neurologist. Tr. at 65. He was also urged to seek emergency care, which resulted in his hospitalization for five days. *Id.* at 66–68, 114. While an in-patient, a lumbar puncture was performed, and Mr. Bielak was started on IVIG for 12-13 hours a day, which he recalled did nothing for his symptoms. *Id.* at 66–67. At this time, he was experiencing pain and very little sleep, requiring help to get up and walk to the restroom. *Id.*

After discharge from the hospital, Petitioner's symptoms included lack of coordination and balance, fatigue, weakness, dizziness, and gait. Tr. at 68. His discharge notes included his poor response to IVIG, as well as significant gait, balance, and functional deficits. *Id.* at 73. Petitioner then immediately began a course of acute inpatient physical therapy for the next five days, although he did not deem his efforts particularly successful. *Id.* at 69, 114–15. Petitioner also maintained that the symptoms he was now experiencing—lower extremity weakness, poor balance, severe pain in the hands, and decreased sensation to just above the knee bilaterally and into the hands with a shocking sensation up to his forearm—were distinguishable from his pre-vaccination symptoms. *Id.* at 71–73. He then proceeded with outpatient physical therapy, attending for four weeks until he mastered the exercises needed to complete this therapy at home. *Id.* at 69–70, 116–17.

Mr. Bielak otherwise described his medical visits from October 2015 to the winter of 2016, confirming what was documented in the medical records. Tr. at 74–84. During this time, he realized only minor improvements in how he felt. *Id.* at 76–77. At a January 27, 2016 visit to Dr. Swigert, Mr. Bielak contemplated another round of IVIG treatments as he was easing off Norco[12] and his symptoms were getting worse. *Id.* at 77, 119–20. Since then, his GBS symptoms have continued from spring of 2016 until the present day, plateauing somewhat in 2016, with only slight improvements in his gait. Tr. at 83–94, 117–18. He also reported that his activity level has decreased significantly due to his fatigue, but still tries to exercise. *Id.* at 89–90, 109. He has, however, had to make other lifestyle changes, such as how he uses knives in the kitchen, when he wears socks, and to take extra care when running. *Id.* at 93–94, 120–21.

[11] Petitioner had another falling instance while on a hike because he could not see the trees overgrown on the path—an event that occurred four to five years prior to this hearing. Tr. at 90, 92.

[12] Mr. Bielak continued using Gabapentin and Norco after onset of his GBS symptoms. Tr. at 118. He wanted to stop using Norco because it was a narcotic, but his GBS symptoms would intensify when he did not take this medication. *Id.* at 119–20. He also received a prescription for tremors, but he stopped taking it after a few years in favor of a different drug. *Id.* at 118.

2. Emily Droste-Bielak – Ms. Droste-Bielak (Mr. Bielak's wife) also testified. Tr. at 127–47. She is his caregiver and considers herself intimately familiar with Petitioner's medical history.[13] *Id.* at 128–29.

Before vaccination, Ms. Droste-Bielak recalled, Petitioner was active, enjoying golfing and hiking, as well as helping with house projects. Tr. At 128–29. He did use a cane 10-12 years before his GBS diagnosis, but this was due to an episode of gout that made it hard for him to walk. *Id.* at 141–42. She also noted that her husband had sleep apnea after his heart attack in 2012, and used a sleep apnea machine at night to treat it. *Id.* at 143–44. She acknowledged his pre-vaccination back pain, but noted that he only complained about it occasionally (and would also utilize a cane if needed). *Id.* at 129, 141–42. He had received intermittent treatment for it and did not consistently experience this pain. *Id.* at 129–30, 142.

Ms. Droste-Bielak then recalled when Petitioner received the pneumococcal vaccine on September 22, 2015. Tr. at 130. She had not noticed any changes in his health until they had friends over and he was not communicating, and his face turned red. *Id.* at 130–31. He did not at this time appear to be acting like his normal self, and he later told her that he had fallen at work. *Id.* at 131. In particular, he seemed dizzy, weak, tired, and quiet, so she decided to inspect his Gabapentin medication that he was using to see if there were any side effects. *Id.* at 131–32.

Ms. Droste-Bielak then went with Petitioner to his October 6, 2015 visit for initial treatment up to his hospitalization that same month. Tr. at 132–33. She stayed with him in the hospital every day and would only return home to sleep. *Id.* at 146. For the first few days he was in the hospital, he was unable to walk without two people helping him on either side. *Id.* at 133. She recalled that he had a lumbar puncture, was started on IVIG, hooked up to a heart monitor, and saw a swallowing specialist during this time. *Id.* at 133–34.

After Petitioner was discharged from the hospital, Ms. Droste-Bielak recalled, his condition seemed better, and he was now able to walk with assistance, but was still weak and unsteady. Tr. at 134. She found that he made progress while in acute rehabilitation to the point where he was able to walk unassisted, though he was not great at walking. *Id.* After his discharge from acute rehabilitation on October 9, 2015, until Thanksgiving that same year, he went to outpatient rehabilitation and became much stronger—he was learning how to walk better and was taught exercises that helped him. *Id.* at 134–35.

Ms. Droste-Bielak also testified about her husband's treatment in the years thereafter (although because the claim does not turn on facts relating to this timeframe, I do not include a

[13] Mrs. Droste-Bielak was also aware of Petitioner's pain medications—Gabapentin and Norco—and believed he took them regularly throughout the day until an injection helped alleviate the pain. Tr. at 142–43. Since Petitioner's GBS diagnosis, his medications are being monitored and he is taking them every four to six hours. *Id.* at 146.

summary of her statements regarding those matters). To date, Petitioner must exercise care when he walks, and he drops things on occasion and may cut himself while cooking and not realize it. Tr. at 140. She believed that he still has pins and needles though she does not ask him—she simply notices that he has troubles occasionally. *Id.* He is also only hiking on flat land now and does occasional projects around the house *Id.* at 141.

3. Elizabeth Remsburg-Bell – Ms. Remsburg-Bell (a long-standing family friend of the Bielaks) offered fact testimony in support of Petitioner's changed demeanor after his GBS diagnosis. *See generally* Tr. at 148–55. She had observed Petitioner in his weakened condition over the 2015 Thanksgiving holiday (and hence close in time to his hospitalization). *Id.* at 150. She also was aware of his slow but incomplete improvement over time thereafter. *Id.* at 151–52. But this witness did not possess direct knowledge of Petitioner's vaccination or immediate health leading up to his hospitalization and subsequent GBS diagnosis.

B. *Petitioner's Experts*

1. Sami Khella, M.D. – Dr. Khella, a neurologist, submitted an expert report and testified for Petitioner, offering the opinion that the pneumococcal vaccine likely caused Petitioner's GBS. *See generally* Tr. at 7–60; Report, dated Oct. 21, 2021, filed as Ex. 48 (ECF No. 53-2) ("Khella Rep.").

Dr. Khella attended the University of Pennsylvania for his undergraduate degree (in biology) and his medical degree. Tr. at 7; Curriculum Vitae, dated Oct. 21, 2021, filed as Ex. 49 (ECF No. 53-3) ("Khella CV"), at 1. Following medical school, he completed an internship and year-long residency at Penn Presbyterian Medical Center in Philadelphia. Tr. at 7; Khella CV at 1. He subsequently held a three-year residency in neurology at the Hospital of the University of Pennsylvania in Philadelphia. Tr. at 8; Khella CV at 1.

Currently, Dr. Khella serves as a Professor of Clinical Neurology at the University of Pennsylvania School of Medicine. Tr. at 10; Khella CV at 1. Dr. Khella is also the preceptor for outpatient HUP Family Medicine Residents and an attending physician for the neurology rounds at Penn Presbyterian Medical Center. Khella CV at 4. He is board certified by the American Board of Psychiatry and Neurology, American Board of Electrodiagnostic Medicine, and American Board of Psychiatry and Neurology: Added Qualification in Clinical Neurophysiology. *Id.* at 2; Tr. at 9. Dr. Khella has published articles focusing on demyelinating diseases in the peripheral nervous system. Tr. at 9, 38. He estimates that he treats 12-15 GBS patients a year. *Id.* at 11.

Dr. Khella briefly touched on the medical record, summarizing the key facts supporting his conclusions. Tr. at 12; Khella Rep. at 1–2. After Petitioner received the pneumococcal vaccine on September 22, 2015, he began experiencing numbness and tingling, plus gait unsteadiness. Tr. at 12; Khella Rep. at 1–2. In Dr. Khella's understanding, Mr. Bielak's malaise began on October 8th, although the record of the cardiology appointment on October 6th indicates that vaccine reaction-

like symptoms started even earlier. Tr. at 12; Ex. 1 at 1; Ex. 5 at 5. Petitioner reported to a physician on October 9th because of these symptoms and was subsequently hospitalized and placed in the ICU on October 10th. Tr. at 12; Ex. 2 at 60; Ex. 9 at 12.

Petitioner had a spinal fluid analysis that showed white blood cells and an elevated protein of 91 (well in excess of a normal reading of 45). Tr. at 13; Ex. 3 at 17–18. According to Dr. Khella, Petitioner was thereafter sent to inpatient rehabilitation on October 20th (though it actually occurred on October 14th), and in the following month underwent nerve conduction studies that were consistent with a demyelinating neuropathy. Tr. at 13; Ex. 3 at 6–7, 13–14; Ex. 6 at 155. Petitioner was not improving, however, so (according to Dr. Khella) treaters considered giving him additional IVIG on November 27th (though the record of such conversations is from February 2, 2016). Tr. at 13; Ex. 3 at 1–2.

During a visit on April 27, 2016, it was reported that Petitioner still had no feeling in his hands and could only walk 200 yards before needing a break (whereas he could previously hike for 12 miles). Tr. at 13; Ex. 2 at 3. He was then still exhibiting GBS symptoms on subsequent visits throughout 2016-2018. Tr. at 13–14; Ex. 3 at 26; Ex. 15 at 3, 18. Petitioner had an electromyography ("EMG")[14] on January 30, 2019, which suggested some recovery, although Dr. Khella noted that this did not mean all sequelae of Petitioner's GBS had passed—as evidenced by Petitioner's persistent neuropathic symptoms as late as August 12, 2019. Tr. at 14; Ex. 23 at 4, 10. Petitioner continues to require Gabapentin due to his ongoing neuropathic pain and symptoms. Tr. at 13–14.

Given the foregoing, Dr. Khella accepted Petitioner's GBS diagnosis. Tr. at 12, 16; Khella Rep. at 2–3. The medical record overall revealed that Petitioner had presented to the hospital with sudden onset of a new neuropathy that had demyelinating features (later confirmed by nerve conduction study/EMG and corroborated by evidence of high CSF proteins), and that was acute and monophasic but may have also resulted in persistent symptoms thereafter.[15] Tr. at 15–16; Ex. 3 at 17–18; Ex. 31 at 4–5. The phenotype of his GBS was weakness, unsteadiness, respiratory failure, and neuropathic pain. *Id.* at 15. Petitioner also had clearly suffered from lingering complications related to his GBS for more than six months. *Id.* at 12.

[14] An EMG is "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals." *Electromyography,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography (last visited Dec. 9, 2022).

[15] Petitioner also met the "Brighton criteria" (accepted factors in the broad medical community which can be employed in studies) for a diagnosis of GBS. Tr. at 16–17; Khella Rep. at 3; C. Fokke et al., *Diagnosis of Guillain-Barré Syndrome and Validation of Brighton Criteria*, Brain 33, 33 (2014), filed as Ex. 50 (ECF No. 53-4) ("Fokke").

Dr. Khella emphasized Petitioner's presentation, noting it was severe, ongoing, and that many of his symptoms remained unresolved or manifested later.[16] Tr. at 27–28, 31–36, 46, 53; Khella Rep. at 5–8; Ex. 2 at 7 (January 27, 2016 visit to Dr. Swigert discussing an assessment of GBS and another round of IVIG, indicating that Petitioner was impaired enough to warrant consideration of such treatment methods); Ex. 3 at 1–4, 14 (February 2, 2016 medical encounter, with the chief complaint that Petitioner's GBS symptoms were ongoing, with a discussion about more IVIG treatment and hospitalization if there was no improvement); Ex. 2 at 3–4 (April 27, 2016 visit to Dr. Swigert noting ongoing GBS symptoms, specifically an absent patellar Achilles reflexes and decreased sensation in his upper and lower extremities). Dr. Khella added that if he had seen a patient in Mr. Bielak's state as of the November 4, 2015 treatment encounter, he would not expect the associated symptoms to resolve. Tr. at 25–26; Khella Rep. at 7; Ex. 3 at 6. And Dr. Khella deemed it likely that Petitioner was experiencing these symptoms throughout his treatment. Tr. at 34–36.

Given the above, Dr. Khella objected to the characterization (mainly offered by one of Respondent's experts) of Petitioner's GBS as mild. Tr. at 46–47, 49–50; Khella at 6–7. GBS might be mild when a patient complained only of limited gait unsteadiness, numbness and tingling, but certainly not where the patient requires hospitalization with mechanical ventilation and endotracheal intubation, plus rehabilitation with continued use of Gabapentin and other drugs. Tr. at 47–48, 55; Khella Rep. at 7. Dr. Khella admitted, however, that a patient with long-standing comorbidities might present different concerns, and that the decision to hospitalize might come down to how the patient presented. Tr. at 48–49. He also acknowledged that GBS patients are *often* admitted to the hospital, although he did not know with what frequency. *Id.* at 49. He did not view the fact that Petitioner's physical therapy was completed within six months of onset as evidence of a mild course.[17] *Id*. at 47.

Dr. Khella next discussed whether the pneumococcal vaccine could trigger an autoimmune cross-reaction leading to the development of GBS. Tr. at 23. Dr. Khella answered the question in the affirmative, and although he largely deferred to Dr. Serota for Petitioner's causation opinion, he proposed a vaccine-induced autoimmune process as the most likely immunologic explanation. *Id.* at 17, 56–58; Khella Rep. at 3–4. It is well accepted in the field of neurology, he maintained, that demyelinating diseases of the peripheral nervous system often result from autoimmune-

[16] Petitioner later developed tremors, for example, which Dr. Khella noted is generally accepted by neuromuscular neurologists as a feature of peripheral neuropathies. Tr. at 37; Khella Rep. at 4; J. Fehmi et al., *Nodes, Paranodes, And Neuropathies*, 89 J. Neurology, Neurosurgery, & Psychiatry 61, 69 (2018), filed as Ex. 54 (ECF No. 53-8).

[17] On the contrary—Dr. Khella contended that Petitioner's overall course exceeded the Act's six-month severity requirement, pointing to the medical record proof of ongoing sequelae. Khella Rep. at 5–6; Ex. 31 at 26–27. Although Petitioner had resumed errands and teaching by April 2016, it was incorrect to assume that his GBS resolved by then, as many GBS patients engage in everyday activities while still experiencing chronic pain. Khella Rep. at 6. And even though Petitioner's January 30, 2019 EMG/NCV study was reported as "essentially normal," it did show some residual deficits and did not indicate Petitioner had an optimally functioning peripheral nervous system. *Id.*; Ex. 23 at 4, 10.

mediated processes. Tr. at 17. Prolonged inflammatory responses to infections are associated with the initiation and exacerbation of autoimmune diseases, facilitated by proinflammatory cytokines. *Id.* at 18. These proinflammatory cytokines are critical for clearance of pathogens in healthy patients, but for reasons that remain unknown, can also encourage autoimmune processes leading to illness (like GBS). *Id.*

Molecular mimicry, Dr. Khella noted, is widely accepted as a mechanism of autoimmunity in GBS, and is also reliably understood to be triggered by antigenic similarities between host tissues and *either* antigens for vaccines or wild infections. Tr. at 18–19. Literature in particular supports an association between the flu vaccine and GBS, allowing for the possibility that other antigenic stimuli (including other vaccines) may trigger a similar reaction. *Id.* at 19; Khella Rep. at 3–4; T. Lasky et al., *The Guillain–Barré Syndrome and the 1992-1993 and 1993-1994 Influenza Vaccines,* 339 New England J. Med. 1797, 1800–01 (1998), filed as Ex. 40 (ECF No. 46-7) ("Lasky").

Dr. Khella also referenced some case reports of GBS developing after receipt of different vaccines.[18] Tr. at 19, 56; Khella Rep. at 3–4. He deemed case studies useful in the analysis of disease causation and progression, since they provide initial insight as to causal mechanisms. Tr. at 25. By contrast, there is limited epidemiologic data regarding an association between the pneumococcal vaccine and GBS because such injuries post-vaccination are rare events, and what studies on the topic that do exist are underpowered, preventing any reliable causal conclusions. *Id.* at 24. And in any event, even if a particular epidemiologic study finds that an event does not occur at a statistically significant rate, this does not lead to the conclusion that the adverse event is impossible. *Id.* at 25.

In discussing causation, Dr. Khella emphasized the fact that the pneumococcal vaccine is a *conjugated* vaccine (specifically with diphtheria). Tr. at 19–20, 59. This conjugate, a bacterial entity, is distinguishable from some adjuvants, like aluminum, common to vaccines. *Id.* at 59. Nevertheless, its inclusion increases the immune response to the vaccine—and individuals susceptible to a strong or overactive response are more likely to experience an aberrant autoimmune process leading to disease (as was likely the case here). *Id.* at 19–20, 59.

[18] On cross examination, Dr. Khella discussed some citations within his expert report supporting different contentions. Tr. at 40–41; Khella Rep. at 3. He provided citations to his assertion that there is an elevated risk for GBS after receipt of the flu, Tdap, and/or HPV vaccines. Tr. at 40–41; Khella Rep. at 3; C. Vellozzi et al., *Guillain-Barré Syndrome, Influenza, and Influenza Vaccination: The Epidemiologic Evidence*, Clinical Infectious Diseases 1149, 1149 (2014), filed as Ex. 39 (ECF No. 46-6); H. Ammar, *Guillain-Barré Syndrome After Tetanus Toxoid, Reduced Diphtheria Toxoid, and Acellular Pertussis Vaccine: A Case Report*, 502 J. Med. Case Reports 1, 1 (2011), filed as Ex. 51 (ECF No. 53-5); N. Souayah et al., *Guillain–Barré Syndrome After Gardasil Vaccination: Data from Vaccine Adverse Event Reporting System 2006–2009*, 29 Vaccine 886, 888 (2011), filed as Ex. 52 (ECF No. 53-6) ("Souayah I"). Dr. Khella also deemed GBS one of the most common neurological sequela of vaccinations generally. Tr. at 41; Khella Rep. at 3; Souayah I at 886. However, he did not provide any case report citations relevant to GBS after the pneumococcal vaccine. Tr. at 41; Khella Rep. at 3.

Dr. Khella also discussed the function and composition of myelin (which wraps around nerve axons), and how it would play a role in the alleged autoimmune process. Tr. at 20–23. Myelin is vital for normal nerve function, because it increases the ability of a nerve to conduct electrical signals quickly, aiding numerous processes in the body. *Id.* at 20. In GBS, however, the myelin is disrupted, so rapid signal conduct does not proceed, impacting motor or sensory nerves and leading to dysfunction. *Id.* at 20–21.

Myelin is composed of proteins, lipids (including phospholipids), and polysaccharides, among other things. Tr. at 21; Y. Poitelon et al., *Myelin Fat Facts: An Overview of Lipids and Fatty Acid Metabolism*, 812 Cells 1, 2–3 (2020), filed as Ex. 67 (ECF No. 75-1) ("Poitelon").[19] Dr. Khella deemed phospholipids especially integral, noting that disruption in any part of these molecules will result in disturbances in function. Tr. at 22; Poitelon at 2–3. Poitelon, it should be noted, says nothing specific about GBS or other forms of neuropathic harm to myelin. Rather, it merely discusses (albeit in some detail) "the basic biology of myelin lipids" in order to "provide a foundation for future research characterizing the role of fatty acids and lipids in myelin biology and metabolic disorders affecting the central and peripheral nervous system." Poitelon at 1. The "metabolic disorders" Poitelon's authors had in mind, however, relate to the nervous system's consumption of oxygen in the production of myelin lipids—not autoimmune injuries triggered by external factors like infections or vaccines. *Id.* at 8–9.

Damage to the phospholipids found in myelin has been observed in GBS patients—although (as discussed below) the item of literature offered for that point does not quite squarely support the contention. Tr. at 22–23; G. Nakos et al., *Anti-Phospholipid Antibodies in Serum from Patients with Guillain-Barre Syndrome*, Intensive Care Med. 1401, 1401–02 (2005), filed as Ex. 63 (ECF No. 63-6) ("Nakos") (showing that autoantibodies against myelin phospholipids were found in patients with GBS). Thus, to the extent that the pneumococcal vaccine could cause production of antibodies likely to have a cross-reactive affinity for phospholipid components on the myelin (a part of Petitioner's theory, as discussed in greater detail below), these vaccine-instigated antibodies could then be a source of the attack on myelin to some degree. Tr. at 58–59.

Petitioner's GBS, Dr. Khella maintained, was most likely caused by the pneumococcal vaccine he had received. Tr. at 12; Khella Rep. at 7. Petitioner did not suffer from GBS prior to his vaccination. Tr. at 14. And Dr. Khella found it significant that no other potential infectious causes had been identified.[20] *Id.* at 24, 26; *see* Ex. 31 at 4 (November 4, 2015 doctor's visit, at which time it was stated there was no evidence of a systemic illness that could explain Petitioner's

[19] Poitelon was originally filed as Ex. 64, but Petitioner refiled it because one of its pages was unreadable after scanning.

[20] Dr. Khella noted there are other accepted causes of GBS in addition to vaccination and infection—although it can also be idiopathic (meaning no cause is identified) in origin as well. Tr. at 52.

demyelinating neuropathy). He also did not find it likely that Petitioner's pre-vaccination cardiovascular condition could have produced the neurological symptoms Petitioner experienced. Tr. at 55. And some of Petitioner's treaters had noted from the time of onset that he received the pneumococcal vaccines prior to his symptoms. *Id*. at 15.

The medical record, in Dr. Khella's reading, was unsupportive of any association between Petitioner's pre-vaccination back problems and his GBS. Tr. at 25–38. Treaters who saw Petitioner early on in his treatment had been made aware of Petitioner's history of lower back pain and disc protrusion in his lumbar spine, but there was nothing to suggest that they associated it with GBS. *Id.* at 25–27; Ex. 3 at 6. It would in fact be unusual that spinal disease would cause significant weakness, ataxia, and gait unsteadiness, as Petitioner had been experiencing in November 2015. Tr. at 26–27; Ex. 3 at 6. And several subsequent treater visits did not seem to add to the possibility of an association with Petitioner's lumbar spine issues. Tr. at 28–30, 32; Ex. 31 at 14. Dr. Khella also felt that treaters would not have recommended IVIG treatment if Petitioner's lumbar spine disc protrusion was thought to be the cause of his symptoms, since lumbar spine disease is treated (or cardiovascular disease for that matter) are not treated with IVIG. Tr. at 33–34. Dr. Khella himself would not attribute these symptoms (e.g., neuropathic pain, weakness, and ataxia) to a spinal injury. *Id.* at 30–31.

Dr. Khella nevertheless admitted that Petitioner had health problems prior to his GBS. Tr. at 43–44. He also noted that Petitioner was taking pain medication for some time before his onset of neurologic symptoms, and although he disclaimed expertise about pain management and associated pharmacologic treatments, he allowed that people who are taking those kinds of medications regularly (as was shown to be the case with Petitioner) likely have severe pain. *Id.* at 43–45. But he did not find it significant that Petitioner continued to be prescribed narcotics after his GBS symptoms. *Id.* at 53. Dr. Khella was also unaware that prior to vaccination Petitioner had required a cane to walk, had requested a handicap placard, or that he was receiving cortisone injections, but this did not change his opinion. *Id.* at 45–46, 53.

Dr. Khella finally opined that Mr. Bielak's neuropathic symptoms began approximately one week after vaccination. Tr. at 23. He deemed the temporal relationship between Petitioner's onset and date of vaccination consistent with the theory of causation. *Id*. at 22–23. In fact, he was especially persuaded of causation given the close timeframe. *Id.* at 57. He stated the likely range of time interval for such an immune-mediated inflammatory injury from the time of immune trigger until the first signs of onset was generally accepted to fall within a few days or weeks, as seen in his practice. *Id.* at 23. According to the Centers for Disease Control and Prevention ("CDC") and other authorities, the onset of GBS within two to six weeks of a vaccination makes it likely that the vaccine contributed to the development of GBS—though he did not cite any authority for this contention. Khella Rep. at 4–5.

2. Marc Serota, M.S., M.D., Ph.D. – Dr. Serota, an immunologist with a focus in dermatology and allergy/immunology, prepared two written reports and testified for Petitioner in support of the contention that the pneumococcal vaccine can cause GBS. *See generally* Tr. at 156–233; Report, dated Feb. 22, 2021, filed as Ex. 35 (ECF No. 46-2) ("Serota First Rep."); Report, dated Sept. 29, 2021, filed as Ex. 47 (ECF No. 52-2) ("Serota Second Rep.").

Dr. Serota attended the University of Missouri-Kansas City for his undergraduate and medical degree. Tr. at 157; Curriculum Vitae, dated Feb. 22, 2021, filed as Exhibit 36 (ECF No. 46-3) ("Serota CV") at 1. He then completed a residency in dermatology at the University of Colorado in Denver, a pediatric residency at Cohen Children's Hospital in New York, and an allergy/immunology fellowship at Children's Mercy in Kansas City. Tr. at 157; Serota CV at 1. Dr. Serota sees patients with issues arising under his primary specialties—dermatology and allergy/immunology—although he could not estimate how much time he spent on either, since their issues often overlap. Tr. at 199–200.

Dr. Serota is currently working at the Veteran's Affairs Hospital in Denver, and the University of Colorado as a physician. Serota CV at 1–2. He also practices telemedicine for dermatology and general medicine. *Id.* He has treated patients with GBS and other autoimmune neurological diseases.[21] Tr. at 161, 196. He is board certified in allergy/immunology, dermatology and pediatrics, and is licensed to practice medicine in Colorado. *Id.* at 158–59, 195; Serota CV at 3; Serota First Rep. at 1; Serota Second Rep. at 1. He has published a few peer-reviewed articles and textbook chapters on various autoimmune mechanisms.[22] Tr. at 160, 201.

Dr. Serota agreed with Petitioner's GBS diagnosis, but deferred to Dr. Khella's expertise on that subject. Tr. at 166, 195; Serota First Rep. at 9–10. Dr. Serota nevertheless explained GBS and the factors believed to drive it. Tr. at 166–67; Serota First Rep at 6. Any autoimmune disease

[21] On cross examination, when asked how much of his research and clinical work focused on immunologic diseases, Dr. Serota noted it was about 15-20% through lecturing and seeing patients. Tr. at 195. Treatment of immune-oriented conditions mostly arose during his allergy/immunology fellowship, where he worked with neuroimmunology patients including adults and children. *Id.* at 196–97. During this time, he estimated that he treated 5-10 GBS patients. *Id.* at 197. He would also consult on patients as part of a multidisciplinary team, usually by taking a history, examining the individual, determining possible triggers, and administering IVIG. *Id.* at 197–99. He could not recall whether these were primarily outpatients. *Id.* at 199.

[22] Specifically, Dr. Serota has published nine peer-reviewed articles, which he discussed in further detail on cross examination. Tr. at 200–05. He admitted that he had never published articles on the immune response to an infection or vaccination or demyelinating diseases, but claimed he had possibly written about molecular mimicry in some textbook chapters. *Id.* at 205. He was unable to describe the peer-review process, because each journal goes through different rigors in determining whether an article should be published and since he was not first author on many of these articles, so he was unaware of the process. *Id.* at 202–04. He also stated that the first author on a publication has more prestige, but author order is not representative of the amount of work performed on a given article. *Id.* at 203–04. None of his lectures, presentations, or teaching experiences relate to neuroimmunology, but some referenced immunologic issues in the context of discussing allergy and dermatology. *Id.* at 200, 205.

(which includes GBS) features an aberrant immune attack on the self, usually attributable to the human immune system mistaking a self protein or tissue as foreign. Tr. at 166. GBS specifically involves an immune attack on the myelin sheath (and in particular the protein and lipids that constitute the sheath) that envelops the nerve axons of the peripheral nerves. *Id.* at 166–67; Serota First Rep. at 6. It typically presents as a "progressive, fairly symmetric muscle weakness accompanied by absent or depressed deep tendon reflexes," developing acutely over a period of two weeks, with most patients reaching nadir by around four weeks from onset. Serota First Rep. at 6. Some patients have developed GBS due to *Campylobacter jejuni* bacterial infection, cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, and/or the Zika virus. *Id.* Other triggering events can include surgery, trauma, and bone-marrow transplants—as well as vaccination, Dr. Serota maintained. *Id.*

Dr. Serota went on to discuss how vaccines impact the immune system. Vaccination causes stimulation of both the innate and acquired/adaptive immune system, and is safe for the vast majority of people.[23] Tr. at 165, 187; *see generally* Serota First Rep. at 9–10 (reinforcing the importance of vaccines). Vaccines "work" by exposing the immune system to a foreign antigen, thereby triggering an initial, innate response, during which the antigen is "seen" while cytokines (immune system messenger cells integral to the immune response) are released, which can cause an individual to feel a post-vaccination malaise. Tr. at 187. This initial step is required to create an acquired immune system response (the ultimate goal of the vaccine), but it does not occur in isolation. *Id.*

The goal of vaccination against *streptococcus pneumoniae*—a bacteria that causes upper respiratory infections, like pneumonia—is to prevent serious illness if a patient is exposed to the wild bacterial infection later. Tr. at 169–70. The pneumococcal vaccine is engineered to achieve this result by its inclusion of saccharides,[24] often called "sugars," derived from the capsular antigens[25] of different strains of *streptococcus pneumonia* bacteria. Tr. at 170; Pneumococcal Package Insert, filed as Ex. 65 on Mar. 15, 2022 (ECF No. 63-8) ("Pneumococcal Package Insert"), at 19. The pneumococcal vaccine contains 13 different "serotypes"[26] of the underlying bacterium,

[23] Indeed, Dr. Serota emphasized that vaccines are an important part of patient care, adding that he recommends vaccines to almost all his patients absent extenuating circumstances. Tr. at 193.

[24] A saccharide is defined as a unit structure of carbohydrates. Tr. at 170; *Saccharide,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=44348 (last visited Dec. 9, 2022).

[25] Capsular antigens are defined as "a surface antigen occurring on the bacterial capsule. . . ." *K Antigen,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56931 (last visited Dec. 9, 2022).

[26] Serotypes of an antigen are variations on the underlying molecular sequence. Prevnar includes what are thought to be the 13 most prevalent variants that the immune system can see—any number of them would form a reaction. Tr. at 170–71. One of these variants is serotype 18C, but the phosphoglycerol component is also critical for the antigenicity of other serotypes, too. *Id.* at 171, 175–76; Pneumococcal Package Insert at 19; Pneumococcal United States Patent, filed as Ex. 66 on Mar. 15, 2022 (ECF No. 63-9) ("Pneumococcal U.S. Patent"), at 27. A vaccine containing more

to increase the likelihood of a successful immune response in the future. Tr. at 170–71; Pneumococcal Package Insert at 19.

The glycerol phosphate[27] component of the saccharide obtained from the pneumococcal bacteria capsid particularly enhances the immunogenicity of the vaccine—meaning a better immune response occurs in reaction. Tr. at 171–73; Pneumococcal United States Patent, filed as Ex. 66 on Mar. 15, 2022 (ECF No. 63-9) ("Pneumococcal U.S. Patent"), at 19. Antibodies formed in response to the vaccine are intended to target the phosphoglycerol component of the capsular polysaccharide for the wild bacterium. Tr. 173–74, 176; J. Chang et al., *Relevance Of O-Acetyl and Phosphoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysaccharide*, 30 Vaccine 7090, 7090 (2012), filed as Ex. 59 (ECF No. 63-2) ("Chang") (observing the existence of a phosphoglycerol group that is part of the serotype 18C capsular polysaccharide).

Dr. Serota maintained that vaccine-caused neuropathic injuries can occur in rare circumstances—particularly when the individual possesses a genetic susceptibility. Tr. at 165; Serota Second Rep. at 6. They result from an immune response to the vaccine triggering a cross-reactive attack by antibodies[28] on self (here, peripheral nerve components). Tr. at 167; Serota First Rep. at 6. And GBS can be mediated by a vaccine—via the mechanism of molecular mimicry. Tr. at 165. Molecular mimicry is a scientific concept in which a vaccine's antigens can appear similar to self structures, either due to sequential homology (in the case of amino acid sequences that make up proteins) common to both, or outright molecular structure "fit." *Id*. at 167–68; Serota First Rep. at 7; M. Cusick et al., *Molecular Mimicry as a Mechanism of Autoimmune Disease,* Clinical Rev.'s Allergy & Immunology 102, 103, 105 (2012), filed as Ex. 37 (ECF No. 46-4) ("Cusick") (summarizing molecular mimicry). In a genetically predisposed person, the immune system mistakes a self protein or tissue for a foreign antigen it has been "trained" to attack, eventually producing symptoms of an autoimmune disease due to the attack's effects.[29] Tr. at 168, 188–89.

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than one serotype of a particular antigen is more likely to trigger an immune reaction. Tr. at 171, 175–76; Pneumococcal U.S. Patent at 19.

[27] Glycerol phosphate is a sugar that can modify the overall structure of the bacterial antigen and is part of the larger polysaccharide making up the capsid for the bacterium. Tr. at 172–73; Pneumococcal U.S. Patent at 19. It is important to maintain the overall structural integrity of the antigen in the vaccine, so when the immune system samples the vaccine, it could recognize the real bacteria if ever exposed. Tr. at 172–73; Pneumococcal U.S. Patent at 19.

[28] Dr. Serota clarified later that there was not a direct one-to-one correlation between the titer of antibodies (and thus measured degree of antibody positivity) and the severity of resulting disease. Tr. at 212. Hence, the "number" of antibodies produced could not be shown to relate to the degree of subsequent disease.

[29] In most cases, a vaccine can be administered safely, without this cross-attack occurring, due to a lack of individual susceptibility. Tr. at 188–89.

In Dr. Serota's view, the antigenic components of the pneumococcal vaccine had the capacity to produce a disease-causing cross-reaction leading to GBS. Tr. at 169–76. In particular, he opined that the phosphoglycerol groups contained in the pneumococcal vaccine's antigens could mimic a similar-appearing molecule found in a patient's myelin, resulting in a cross-reaction by antibodies produced in response to the vaccine. *Id.* at 182–83. Such antibodies would thus erroneously attack the phosphoglycerol molecule that is part of the phospholipids of the peripheral myelin, resulting in GBS.[30] *Id.* at 183, 211. In this respect, the causation theory offered in this case differs from what has been proposed with respect to the GBS-flu vaccine association. That association relies on the theory that the *protein* antigenic components of the flu virus contained in the vaccine lead to production of antibodies that cross-react with *protein* components of the myelin. The present theory, by contrast, involves antibodies generated against carbohydrate structures in the vaccine that would interact with lipid/sugar structures found on the myelin sheaths around the nerves—none of which are protein-based. *Id.* at 168, 226–28.

To illustrate this aspect of his opinion, Dr. Serota referenced several items of literature. Tr. at 176–83; Poitelon at 2. First, he noted studies establishing that GBS patients had (in small studies) been shown to possess antibodies to myelin-associated phospholipid molecules. *See, e.g.*, Nakos at 1405–07. Additional articles (discussed in detail below) suggested that antiphospholipid antibodies are present in *other* neuroinflammatory demyelinating diseases—and that they can be pathogenic, allowing the same possibility in the context of GBS. Tr. at 178–82, 212.

Second, Dr. Serota highlighted case reports. Tr. at 183–86, 228–29, 231; Serota First Rep. at 10. One directly involved a reported instance of GBS after receipt of the pneumococcal vaccine. N. Ravishankar, *Guillain-Barre Syndrome Following PCV Vaccine*, 4 J. Neurology & Neurosurgery 1 (2017), filed as Ex. 26 (ECF No. 32-3) ("Ravishankar"). Ravishankar reported an instance of a 66-year-old woman who received two doses of pneumococcal vaccine over a seven-month period, with onset of lower limb/knee weakness a month after the second dose. Ravishankar at 1. She presented to the ER two months after the second dose, with her presumed GBS resolved not long after, but then leading to additional sequelae over the ensuing year. *Id.* Ravishankar noted, however, that in comparison to the flu vaccine-GBS association, "far fewer cases" had been reported connecting the pneumococcal vaccine to this neuropathic injury. *Id.* at 2.

Each case report was in Dr. Serota's opinion a data point useful in determining causation. Tr. at 207. Evidence from the Vaccine Adverse Event Reporting System ("VAERS")[31] was also

[30] Dr. Serota emphasized that his theory was not that the phospholipids were *themselves* a specific vaccine component, but rather that the phosphoglycerol molecule was *found* in the make-up of the pneumococcal bacteria antigens. Tr. at 219.

[31] VAERS is a database maintained by the CDC to compile information from reports about reactions to immunizations listed on the Vaccine Injury Table, 42 U.S.C. § 300aa–14(a). *See About VAERS*, VAERS, https://vaers.hhs.gov/about.html (last visited Dec. 9, 2022). As Dr. Serota explained, VAERS is used to monitor reported instances of after-vaccination adverse effects. Tr. at 206–07.

supportive of his theory, he argued. *Id.* at 206–08, 210–11. In so doing, he acknowledged that he could not point to published literature that has *concluded* that the streptococcus pneumoniae bacterium can cause GBS. *Id.* at 207. He also admitted that here (unlike in the context of what was scientifically known and accepted about the GBS-flu vaccine association) there was no epidemiological evidence or experimental animal models to corroborate a pneumococcal vaccine-GBS link. *Id*. at 229–30. And Dr. Serota was unable to confirm or deny whether the medical community associated the wild *streptococcus pneumoniae* infection with GBS, further reducing the probative value of case reports. *Id.* at 206.

Dr. Serota otherwise admitted that case reports are considered weaker evidence as a general scientific matter, but argued that in the setting of rare injuries, they can be helpful in suggesting possibilities of associated adverse events. Tr. at 185–86, 231. Epidemiologic evidence cannot, he maintained, always provide a useful "signal" when evaluating the likelihood of extremely uncommon events. *Id.* at 186, 229–30; Serota Second Rep. at 3–5. Thus, reliance on case reports alone was an imperfect but justifiable approach to substantiating causation under the circumstances. He also noted that his theory gained some heft from the evidence of Petitioner's reported acute, vigorous immune response shortly after vaccination. Tr. at 230–32. And because vaccines are intended to elicit a "controlled" immune response comparable to a wild infection, evidence of autoimmune disease in the wake of such an infection had obvious applicability to the context of vaccination, as well. *Id.* at 184–85; Serota First Rep. at 7. In fact, Dr. Serota maintained that the immunogenicity of a vaccine could in some cases exceed the response to a wild infection, since vaccines require a robust response if they are to perform their intended function. Tr. at 220.[32]

The fact that the pneumococcal vaccine is conjugated also, in Dr. Serota's view, increased the possibility of an aberrant immune response (for susceptible individuals). Tr. at 183–84, 205–06, 208–09; Serota First Rep. at 8; Ravishankar at 2. Conjugation involves attaching the vaccine's primary antigenic components to another molecule or antigen to help improve antigenicity (by sparking an immune response to the conjugate itself, thereby creating an environment favorable for response to the primary vaccine antigens). Tr. at 183–84; Serota First Rep. at 8; Pneumococcal Package Insert at 19. The pneumococcal vaccine's conjugate, he stated, is diphtheria.[33] Tr. at 213. But Dr. Serota offered no independent evidence, outside of his own opinion, establishing that this aspect of the vaccine's design had any possible adverse impact on anyone, let alone susceptible individuals.

[32] At the same time, Dr. Serota acknowledged that a wild infection is inherently more dangerous and naturally destructive than a vaccination. Tr. at 220–21.

[33] In fact, the pneumococcal vaccine conjugate is "a non-toxic, *genetically modified variant* of the diphtheria toxin." *Deshler v. Sec'y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *1 n.3 (Fed. Cl. Spec. Mstr. July 1, 2020) (emphasis added).

Petitioner's medical history provided further support for Dr. Serota's causation opinion. Mr. Bielak reported a short-term reaction to the vaccine, which included malaise (attributable to the systemic response from cytokines) plus a rash on his arm. Tr. at 186, 223–24; Serota First Rep. at 8, 10; Serota Second Rep. at 6. Though malaise is a common side effect to vaccines (as noted in the pneumococcal vaccine package insert), Dr. Serota found it relevant that Petitioner *also* experienced a localized immunologic response at the site of vaccination, which is less common. Tr. at 223–24; Pneumococcal Package Insert at 1. This suggested to Dr. Serota that Mr. Bielak's subsequent GBS was more likely caused by the vaccine, since the record established that Petitioner had experienced a robust innate response that likely contributed to the subsequent autoimmune process. Tr. at 187–88, 230–31; Serota First Rep. at 8. Of course, Dr. Serota's logic relied (as he admitted) on the assumption that Petitioner was predisposed to an aberrant immune reaction—as evidenced (in circular fashion) by the fact of his ultimate injury. Tr. at 225–26. No other evidence was provided to show why a clinically-obvious immediate vaccine reaction increased the likelihood of an aberrant autoimmune response.

In addition, Dr. Serota maintained that the absence of other explanations for Petitioner's GBS supported the finding that the pneumococcal vaccine had been causal. Serota First Rep. at 8, 10. At most, there was some speculation in the record of a possible cardiac etiology, but Dr. Serota disputed that this could reasonably account for Petitioner's neurological symptoms. *Id*. By contrast, some of Petitioner's initial treating physicians had pointed to the pneumococcal vaccine as a possible cause. Serota First Rep. at 8; Ex. 2 at 60 (October 9, 2015 admittance to Dixie Regional Medical Center); Ex. 5 at 5 (October 6, 2015 cardiologist visit). The timing of Petitioner's reaction and development of GBS post-vaccination was also consistent with the known timeframe associated with vaccine-related GBS, although he cited no literature or other independent evidence to support this contention (and did not even identify a specific onset date). Serota First Rep. at 8.

Finally, Dr. Serota endeavored to rebut points made by Respondent's primary immunologic expert, Dr. Robert Fujinami. Tr. at 189–92. Dr. Serota disagreed with Dr. Fujinami's contention that pathologic molecular mimicry leading to an autoimmune disease, like GBS, was improbable in the context of vaccination, arguing in reaction that logically the same immune process by which autoantibodies would be produced post-infection would also occur after vaccination. *Id.* at 189–90. In fact, he argued, wild infections aimed to *sidestep* the immune system, whereas vaccines are intended to alert the body to their presence, in order to provoke immune memory of the presenting antigen in the future—and thus it made no sense to Dr. Serota that an aberrant response could not also occur after vaccination. *Id.* at 190–91.

Dr. Serota also claimed that literature supported his contention on this subject—although he pointed primarily to articles involving the flu vaccine's/virus's GBS association. Tr. at 191–92; Fujinami First Rep. at 4–5; Serota First Rep. at 7, 10; H. Lehmann et al., *Guillain-Barre Syndrome After Exposure to Influenza Virus*, 10 Lancet Infectious Disease 643, 646 (2010), filed as Ex. 38

(ECF No. 46-5) (noting that GBS was associated with swine flu in 1976) ("Lehmann");[34] C. Vellozzi et al., *Guillain-Barre Syndrome, Influenza, and Influenza Vaccination: The Epidemiologic Evidence*, Clinical Infectious Diseases, 1149, 1153 (2014), filed as Ex. 39 (ECF No. 46-6) ("Vellozzi") (showing an increased risk of GBS following flu infections and vaccines associated with swine flu in 1976); D. Salmon et al., *Association Between Guillain-Barre Syndrome and Influenza A (H1N1) 2009 Monovalent Inactivated Vaccines in the USA: A Meta-Analysis*, 381 Lancet 1, 6–7 (2013), filed as Ex. 41 (ECF No. 46-8) (finding the H1N1 influenza A vaccine used in the U.S. was associated with a small increased risk in GBS) ("Salmon").

C. *Expert Testimony Relying on Late-Filed Literature*

During the hearing, Respondent highlighted Petitioner's late filing of medical literature he seemed to consider important to his causation theory. Tr. at 215–18. Approximately one month prior to the mid-April 2022 hearing, Petitioner filed six new items of literature not previously offered (plus two more routine items specific to the pneumococcal vaccine's formulation or its administration). *See generally* Exs. 59–66. All had been published at the time of Dr. Serota's reports—meaning they could well have been filed much earlier in the case's life. Dr. Serota's testimony relied heavily (although not exclusively) on these "new" items of literature. Tr. at 22–23, 169–83, 213–19, 226–28.

In cross-examination, Respondent observed that these six items of literature had not previously been referenced or identified in the filed written reports. Tr. at 213–17. Dr. Serota could not provide any insight as to why this literature was filed late, or why he did not produce another expert report addressing the items (besides the fact that he was not asked to do so). *Id.* at 215, 217–18. He also could not recall whether he was aware of the theory relating to the new articles being used in other cases in the Vaccine Program. *Id.* at 218. Dr. Serota maintained, however, that this newly-filed literature was supportive of the theory behind which the pneumococcal vaccine could cause GBS, although he took many different data points to draw this conclusion. *Id.* at 213–15, 218.

Given that my amended pre-hearing order extended the deadline to file record evidence to March 15, 2022—the day these items were filed—they were not submitted late in any technical sense.[35] And Respondent and his experts had not only advance notice of the new items but were able to discuss them at hearing. Thus, I include these items in my analysis—but because they seemed to be relied upon to a larger extent than the previously-filed articles (and have in fact also

[34] Also filed as Respondent's Ex. A, Tab 3.

[35] My initial prehearing order set March 1, 2022, as the deadline to submit record evidence. ECF No. 55. But I granted Respondent's Motion for an extension of time, which in turned allowed additional medical literature filings until March 15, 2022. *Scheduling Order*, dated Jan. 28, 2022.

been cited in several more recent pneumococcal vaccine-GBS cases), I discuss them in some detail below.

1. Chang

Petitioner offered Chang to establish the immunologic significance of the serotype 18C polysaccharide found in the pneumococcal vaccine's antigenic components, in order to bulwark the contention that the vaccine can cause the production of antibodies "targeting phosphoglycerol groups" found in the myelin. Petitioner's Explanation of Additional Medical Literature, dated March 15, 2022 (ECF No. 66) ("Lit. Exp.") at 1.

Chang observed that the pneumococcal vaccine (to be effective) needs to induce the production of anti-capsular polysaccharide antibodies. Chang at 7090. However, the *streptococcus pneumoniae* bacteria's polysaccharide antigens can have their immunogenicity affected by the manner in which the conjugate version of the vaccine (deemed the "vaccine of choice to target child protection") is manufactured/formulated. *Id.* Chang's authors therefore conducted an animal study in which the relevant capsular polysaccharide used in most versions of the vaccine was treated and prepared in different ways, then injected into the animal subjects, in order to evaluate "a suitable modification-conjugation procedure" that would ensure preservation of the most important antigenic features of the vaccine. *Id.* at 7095; *see also* 7091–92. Chang concludes that preservation of the glycerol phosphate group is more important than the O-acetyl group. *Id.* at 7095–96.

Thus, Chang merely speaks to the importance of not diminishing the vaccine's immunogenicity—but in highlighting the most significant sub-aspect of the vaccine antigens, says *nothing about those antigens being pathogenic*. At best, it establishes the glycerol phosphate group contained in the capsid antigen that is most likely to spur the immune system to produce antibodies.

2. B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with Guillain-Barre Syndrome: Cross-Reactive or Pathogenic?* 16 Autoimmunity 1, 23–27 (1993), filed as Ex. 60 (ECF No. 63-3) ("Gilburd")

Petitioner cites Gilburd to show that the pneumococcal vaccine can cause the production of autoantibodies that are allegedly pathogenic in the context of GBS. Lit. Exp. at 1–2. But (and putting aside the fact that it was published nearly 30 years ago, yet does not appear to have encouraged much, if anything, in the way of follow-up research) Gilburd is as notable for its caveats and limitations as its purported findings. Gilburd admits at its outset that "the autoantigen of GBS has not yet been identified"—a fact that remains true today. Gilburd at 23. Further, its authors note something even more fundamental—that "it is not settled whether the autoantibodies in GBS induce the nerve damage *or are induced by the demyelination and liberation of*

autoantigens." *Id.* (emphasis added). This second factor, as discussed in greater detail below, has central importance in evaluating the success of Petitioner's causation showing.

Otherwise, Gilburd's findings are quite modest. Gilburd's authors tested the blood of 16 GBS patients for the presence of certain autoantibodies. Gilburd at 24. In particular, they looked for anti-phospholipid antibodies, noting their association with other kinds of conditions and neurologic syndromes, many of which are not specifically thought to be autoimmune in character. *Id.* at 26. Indeed, Gilburd noted that "only a few reports" even associated this antibody with GBS. *Id.* Ultimately, Gilburd did find that these autoantibodies were present in the blood serum of some tested subjects, although "our results do not show a significant increase in any specific antiphospholipid antibody" in patients with GBS, and therefore Gilburd's authors deemed their presence potentially attributable to myelin damage or other autoimmune processes. *Id.* at 27. If so, they would be the *product* of an autoimmune process—not the cause.

3. P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation,* Sci. Translational Med. 1, 3, 7 (2012), filed as Ex. 61 (ECF No. 63-4) ("Ho")

Ho does not involve GBS, but instead multiple sclerosis ("MS")—a neuroinflammatory disorder of the central nervous system that also features demyelination, but is otherwise wholly distinct from GBS in numerous important regards. Noting the extent to which lipids are components of myelin, Ho's authors began by identifying (from CSF samples, since MS's immediate harm begins in the central nervous system) a number of anti-lipid antibodies that were not present in control patients, and then tested them using a mouse model for MS. Ho at 2–3. This assisted Ho's authors in identifying potential myelin lipid targets of the autoantibodies in question. They sought to do so because the effectiveness of these lipids in warding off the primary autoimmune attack from T cells in MS could be diminished by autoantibodies that reduced the "anti-inflammatory effect" of the myelin lipids. *Id.* at 8. At most, then, Ho suggested that the studied antibodies might *contribute* to MS pathogenesis—but *not* that they were the cross-reacting/instigating spark for an autoimmune disease. *Id.*

4. J. Kanter et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation,* 12 Nat. Med. 1, 138–43 (2005), filed as Ex. 62 (ECF No. 63-5) ("Kanter")

Kanter is another MS-specific study involving several of Ho's co-authors (in fact, it pre-dates Ho and was cited therein). *See* Ho at 2 n.3. Employing the same experimental animal model relied upon in Ho, Kanter merely determined that (given the extent to which nerve myelin is comprised of lipids) lipid-specific autoantigens likely played *some* role in MS's pathogenesis. Kanter at 142. But it did not suggest this role was initial or primary. To the contrary, Kanter's

authors also noted that prior studies had suggested that the lipid-oriented attacks might be secondary to ongoing autoimmune-mediated damage, occurring as a result of "epitope spreading" encouraged by other antibodies. *Id.* at 141.

5. Nakos

Nakos is a 2005 study that attempted to evaluate whether lipid antigens could be GBS autoantibody targets. Nakos at 1402. It thus is parallel to Kanter (and somewhat anticipates Ho). In Nakos, nine patients with the AIDP (meaning "acute inflammatory demyelinating polyneuropathy") GBS variant had their blood tested for the presence of anti-phospholipid antibodies over a period of several days, with results revealing "a wide range" of these antibodies in comparison to controls. *Id.* at 1405. But (and consistent with other literature mentioned herein), Nakos's authors did not purport to determine the precise *role* of these autoantibodies in GBS's pathogenesis, allowing that they could simply "represent a part of a more extensive immunoreaction that takes place in the GBS." *Id.* at 1406. Nakos also found that phosphatidylcholine in particular (which has a phosphoglycerol component) was one of the main antigens in the small sample of tested GBS patients. Tr. at 178–79; Nakos at 1405–07.

D. *Respondent's Experts*

1. Robert Fujinami, Ph.D. – Dr. Fujinami, a neuroimmunologist, testified on Respondent's behalf and prepared two written reports, as well. *See generally* Tr. at 234–368; Report, dated June 30, 2021, filed s Ex. C (ECF No. 50-15) ("Fujinami First Rep."); Report, dated Jan. 3, 2022, filed as Ex. F (ECF No. 57-4) ("Fujinami Second Rep."). He sought to rebut Petitioner's argument that the pneumococcal vaccine can cause GBS.

Dr. Fujinami received his undergraduate degree from the University of Utah and doctorate from Northwestern University. Tr. at 234–35; Curriculum Vitae, dated June 30, 2021, filed as Ex. D (ECF No. 50-21) ("Fujinami CV") at 1; Fujinami First Rep. at 1. He completed his postdoctoral training at The Scripps Research Institute and advanced to Assistant Professor investigating how viruses/infections could induce autoimmune disease. Tr. at 235; Fujinami CV at 1; Fujinami First Rep. at 1. While at Scripps, Dr. Michael Oldstone and Dr. Fujinami first introduced molecular mimicry as a theoretical concept in the early 1980's, with respect to viruses and their putative role in the propagation of autoimmunity. Tr. at 235; Fujinami First Rep. at 1. He is accordingly one of the scientific "fathers" of the concept (although he is not a medical doctor).

Dr. Fujinami is currently a Professor in the Department of Pathology, Division of Microbiology and Immunology, and an Adjunct Professor in the Department of Neurology at the University of Utah School of Medicine. Tr. at 236, 244–45; Fujinami CV at 1; Fujinami First Rep. at 1. He has published hundreds of articles about microbial infection, vaccines, and autoimmune

disease. Tr. at 236, 238–39. Dr. Fujinami has also served on various panels for the National Institute of Health, Institute of Medicine, National Academy of Sciences, National Science Foundation, and National Multiple Sclerosis Society. Fujinami First Rep. at 2. He acknowledged on cross-examination, however, that he has not specifically studied, or published on, GBS, the pneumococcal vaccine, or other specific aspects of Petitioner's causation theory. Tr. at 303–04.

Dr. Fujinami deferred to Respondent's other expert, Dr. Jamieson, as to any matters pertaining to Mr. Bielak's diagnosis, medical course, or the manner in which GBS is believed to occur biologically. Tr. at 247, 308, 313. He instead focused on the science behind Petitioner's theory. The Prevnar vaccine, Dr. Fujinami observed, contains 13 serotypes of the *streptococcus pneumoniae* bacteria, included to increase the likelihood that the immune system will recognize different versions of the infection, with different antibodies responding specifically to each. Tr. at 288–89. Each serotype is a portion of the strain's "capsid," or carbohydrate shell which serves to protect the bacteria. *Id.* at 349.

The vaccine is also conjugated to a nontoxic version of diphtheria (a different bacteria) and includes some alum as an adjuvant. Tr. at 289–90. The conjugate is included because (in Dr. Fujinami's words) it is "really hard" to stimulate an immune response against the polysaccharides from the bacterial strain capsids alone, so the conjugate acts as a "carrier protein" that permits the immune system to better recognize the pneumococcal bacterial antigens (in the wake of a cell-mediated response to the conjugate) and manufacture antibodies in the future. *Id.* at 366–67. The vaccine contains no "live" components and does not also contain phospholipids as direct ingredients. *Id.* at 290. Thus, like other vaccines,[36] the pneumococcal vaccine ultimately acts as "a tool that we use to generate antibodies" that can fight wild infections upon future exposures. *Id.* at 314.

As Dr. Fujinami understood it, Dr. Serota was proposing that the pneumococcal vaccine's antigens—the carbohydrate polysaccharide taken from the capsid of the 13 different strains of the streptococcus bacteria—instigated an immune response, resulting in the production of "pathogenic antibodies against the carbohydrate part, particularly the phosphoglycerol part of the sugar molecule" from the antigen, but that these antibodies subsequently cross-react with nerve myelin, resulting in GBS. Tr. at 249, 295. Dr. Fujinami agreed that some of these antigens shared molecular elements or characteristics with certain myelin, lipid-specific components. *Id.* at 312. But Dr. Fujinami strongly disputed that the pneumococcal vaccine could cause GBS—especially via molecular mimicry alone. *Id.* at 247, 312, 355 ("you need additional factors" besides mimicry between antigens and self structures for autoimmune disease to occur), 362–63 (differentiating between the vaccine's intended immunogenic effect and the potentiality for pathogenicity), 368.

[36] Dr. Fujinami stressed that vaccines have different compositions and functions differently, as well—with different intended direct impacts on the immune system, even if the aim of any vaccine was to "train" the immune system to fight a specific pathogen. Tr. at 315–16.

And although he expressed the view that the theory did not have widespread medical community support,[37] he relied on his personal expertise for his conclusion—offering several substantiated explanations for his opinion based on his own overall experience. *Id.* at 247–48, 251, 325 ("I would say nothing in the literature, nothing that I do in my work" supports Petitioner's causation theory), 343–44.

First, Dr. Fujinami maintained that some of the underlying scientific models used to confirm the existence of molecular mimicry, as well as its capacity to mediate autoimmune disease, did not support causation. In particular, he noted that one model (known as "experimental autoimmune encephalomyelitis ("EAE"))[38] referenced in many of Petitioner's filed items of literature has been used to show that taking a neuro-antigen (in this case, "something that has cross-reactivity between the phospholipids and myelin"), mixing it with adjuvants, and directly immunizing animals with it can induce production of antibodies. Tr. at 250. But the response generated is what he deemed a "Th2-type[39] immune response"—which only encourages production of antibodies, whereas "organ-specific autoimmune diseases" are more understood to be "cell-mediated," or driven by attacking T cells. *Id.* at 250, 364. Thus, while production of antibodies can be confirmed after exposure to the tested antigen, this does not mean they are primarily pathogenic. Indeed, Dr. Fujinami maintained, the EAE model in some cases revealed that direct immunization of the neuro-antigens believed to stimulate antibodies would be *protective against* autoimmune disease. *Id.* at 250–51.

Dr. Fujinami next provided his interpretation of some of Petitioner's case reports. As a general matter, he did not deem case reports to be good causation evidence, noting that their observations were usually based on a single instance ("kind of an N-of-1") and thus of limited experimental value, as recognized by the scientific community. Tr. at 260–61. But he went on to point out specific deficiencies in the case reports offered. For example, one observed a temporal relationship between a streptococcus infection and development of GBS. G. Bianchi & G. Domenighetti, *Pneumococcus Pneumoniae Infection and Guillain-Barré Syndrome: Fortuitous or Specific Association,* Intensive Care Med. 338, 338–39 (2006), filed as Ex. 27 (ECF No. 32-4) ("Bianchi") (proposing that the patient's infection could induce a predominant motor form of GBS, associated with anti-ganglioside antibodies). But Dr. Fujinami disputed that Bianchi supported the

[37] Dr. Fujinami also noted the limited number of adverse reporting events involving GBS following the pneumococcal vaccine. Tr. at 293–94. But given that I routinely observe that this kind of passive surveillance proof is no more probative than case reports, I do not give this aspect of Dr. Fujinami's argument significant weight.

[38] EAE models are used to mirror MS, which is a disease of the central nervous system. Tr. at 305–07. This is different from experimental autoimmune neuritis ("EAN"), which is one of the animal models used for GBS. *Id.* at 303, 307–08.

[39] Th2 cells are a type of "T helper cell" that secondarily stimulate antibody production by B cells, rather than directly engage with pathogens, like other forms of T cell. Tr. at 367; *Perekotiy v. Sec'y of Health & Hum. Servs.*, No. 16-997V, 2020 WL 12904810, at *4 n.12 (Fed. Cl. Spec. Mstr. Apr. 20, 2020), *mot. for review denied,* No. 16-997V, 2020 WL 5887548 (Fed. Cl. Sept. 17, 2020)

conclusion that the wild infection could result in production of "anti-ganglioside antibodies,"[40] noting that medical science had not observed any connection between the wild bacterium and GBS (nor did the report's authors conclude otherwise). Tr. at 252–54; *see also* Tr. at 343 ("infection with streptococcus pneumonia, it doesn't induce [GBS]"). Bianchi thus went against the weight of understanding about the lack of a pneumococcal bacterium/GBS connection.

A second case report (also involving a wild infection) was similarly unsupportive, with Dr. Fujinami emphasizing that the patient subject had been experiencing "a very systemic infection," which would not be consistent with the results of vaccination. Tr. at 254–56; H. El Khatib et al., *Case Report: Guillain-Barré Syndrome with Pneumococcus: A New Association in Pediatrics*, ID Cases 36, 36–37 (2018), filed as Ex. 28 (ECF No. 32-5) ("El Khatib"). And a third, similar report (which also observed a temporal association between a wild pneumococcal infection and GBS) specifically *tested* for the purportedly-pathogenic anti-ganglioside antibodies, but obtained negative results. Tr. at 256–58; B. White et al., *A Novel Pneumococcus with a New Association,* Travel Med. & Infectious Disease 84, 85 (2011), filed as Ex. 29 (ECF No. 23-6).

In discussing such case report literature, Dr. Fujinami criticized Dr. Serota's assumption that vaccination and infection are likely to impact the immune system identically, when in fact "the vaccines we are talking about don't approximate what an infection would do." Tr. at 296. This was especially so given that the pneumococcal vaccine was not a "live attenuated vaccine," in which the immune response might be more akin to an infectious process. *Id.* at 315–16. Thus, the case reports Petitioner invoked often involved demonstrably "robust, systemic infections," in which a live organism would replicate in the body, causing independent damage far exceeding in immunologic impact how the body would react to a vaccine. *Id.* at 296–98. This would not, Dr. Fujinami maintained, occur with vaccination. And only one case report—Ravishankar—involved the pneumococcal vaccine itself, yet Ravishankar's author had expressly admitted that fewer reports of post-vaccination GBS existed for the pneumococcal vaccine than for the flu vaccine. Tr. at 258–59; Ravishankar at 2.[41]

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[40] Before the hearing (and filing of new literature discussed above), Petitioner's causation theory was more focused on establishing that the pneumococcal vaccine could cause the production of anti-ganglioside antibodies of the sort that the flu vaccine is thought to produce. *See, e.g.*, Serota First Rep. at 7. But these kind of antibodies are more usually deemed to be potentially stimulated into production by viral antigens, which would be distinguishable from a bacterium-oriented vaccine like Prevnar. *Chinea v. Sec'y of Health & Hum. Servs.*, No. 15-095V, 2019 WL 1873322, at *15 (Fed. Cl. Spec. Mstr. Mar. 15, 2019) (theorizing petitioner's GBS was initiated by the hemagglutinin component of the flu vaccine, which is known to induce antiganglioside antibodies). The causal theory offered at trial, however, focused on phospholipid-oriented antigenic targets for cross-reactive attack rather than gangliosides (although Dr. Fujinami nevertheless addressed this earlier iteration of Petitioner's theory).

[41] Dr Fujinami also observed that the Ravishankar patient had received *two* versions of the vaccine: Prevnar in January 2015, and then Pneumovax-23 (a non-covered vaccine) in August that year. Tr. at 259; Ravishankar at 1. And many months passed between the two vaccinations, with onset taking an additional month after the second vaccination. Ravishankar at 1. Petitioner's circumstances were distinguishable. Tr. at 260.

Dr. Fujinami also provided his reading of the late-filed literature items.[42] He noted, for example, that Chang had been cited by Petitioner to prove the pathogenic nature of the antibodies generated in response to the pneumococcal vaccine. Tr. at 270. But the very experiment performed in Chang (intended to show that "when you immunize animals with the 18C [carbohydrate] moiety, you see a marked increase in antibodies to the phospholipids") resulted over time in huge increases in the measurable amount of these antibodies—but with no disease appearing in the tested animal subjects, thus implicitly undermining the conclusion that they are likely pathogenic. *Id.* at 271–72. For Dr. Fujinami, this simply highlighted the fact that "one can have autoreactive antibodies, but that doesn't mean and automatically lead to develop autoimmune disease." *Id.* at 273–74, 345.[43]

Gilburd, Dr. Fujinami maintained, was also less supportive of Petitioner's theory than proposed. It concluded that "[t]he phospholipid antibodies are a result of the initial myelin damage not the cause of the demyelination," and thus the generation of these purportedly pathogenic antibodies was "what they call an epiphenomenon, that it's found in association [with] but not the cause" of GBS. Tr. at 275–76; Gilburd at 27 ("in GBS the autoantibody production is more likely an epiphenomenon *resulting from the myelin damage* and the liberation of various myelin antigens into the circulation") (emphasis added)). In fact, Dr. Fujinami proposed (on cross-examination) that Gilburd to some extent "answered" the contention that genetic variance or predisposition explained why not all individuals possessing the purportedly-pathogenic antibodies would become sick—noting that its authors had determined that the antibodies were present in control subjects as well as those with GBS (meaning the antibodies themselves were not likely the factor causing disease). Tr. at 347–48; Gilburd at 26. He admitted, however, that antibody counts or amounts could not be relied upon alone as suggesting an increased or decreased likelihood of disease. Tr. at 348.

Ho did observe that antibodies found in MS patients were "directed to phospholipid components of the central nervous system," but Dr. Fujinami maintained that the study did not otherwise "get at whether they are the cause or an effect of the neuroinflammation." Tr. at 277. Nakos, Dr. Fujinami noted, was primarily concerned with whether "antibodies against phospholipids can be used as a biomarker of GBS"—meaning evidence that GBS was diagnostically the disease at issue. Tr. at 284–85. This was not the same as an inquiry into whether the antibodies were *pathogenic*. *Id.* Otherwise, Nakos seemed to confirm that antibodies to the phospholipids were arising in the context of an existing autoimmune process involving other, more

[42] Although Respondent had been provided with these articles before the hearing, Dr. Fujinami only learned of Dr. Serota's specific interpretation of this evidence from his testimony at hearing. Tr. at 356.

[43] Dr. Fujinami was asked on cross-examination whether individual genetic susceptibility or predisposition was the "x factor" that, in the presence of large numbers of putatively-pathogenic autoantibodies, could lead to an autoimmune disease. Tr. at 345–46. But he responded that the very *fact* of antibody production was evidence of the proposed predisposition (which he termed possession of the "correct major histocompatibility molecules")—and thus the failure for disease to occur in the presence of large antibody titers still undercut their allegedly causal role. *Id.* at 346.

likely instigating antibodies, and hence the purportedly-causal phospholipid antibodies were a result of existing damage to myelin, not a cause. *Id.* at 286–87.

Ho also utilized the EAE animal model to simulate MS, and in so doing determined that likely lipid targets of phospholipid autoantibodies were ameliorative of MS if directly injected into the animal subjects. Tr. at 278–79 ("the lipids are actually trying to suppress the neuroinflammation that's going on"). But the interference by autoantibodies with such neuroinflammation-modulating lipids did *not* mean the autoantibodies were causal of MS; rather it suggested that they played a secondary role in its pathogenesis after some other instigating factor. *Id.* at 279.

Dr. Fujinami made another point in reference to Ho that has particular significance in this case. As he noted, the EAE animal model requires use of an extremely powerful adjuvant[44] to generate results useful to scientists. Tr. at 279. The pneumococcal vaccine, by contrast, includes only alum as an adjuvant. *Id.* at 280; Pneumococcal Package Insert at 19. As Dr. Fujinami explained, "antigens mixed with alum tend to favor or skew the immune response towards an antibody or Th2-type response" with the effect of encouraging antibody production. Tr. at 280, 318–20, 321, 323. Dr. Fujinami deemed that kind of immune response "very different" from the autoimmune response believed to drive MS itself (or GBS for that matter)—a "cell-mediated" response (which is not antibody-dependent, but instead involves direct attack by a different class of T cells). *Id.*; *see also id.* at 364. Thus, it could not simply be assumed that research results from this experimental context (no matter how generally interesting) reliably predicted what a vaccine with an entirely different expected impact on the immune system would do.

Other literature filed late but relied upon by Dr. Serota was no more persuasive in Dr. Fujinami's view. Kanter also involved use of the EAE model and discovered the presence of certain lipid-specific antibodies in the studied sample animals (again in the context of MS rather than GBS). Tr. at 281. Dr. Fujinami felt Kanter's results to be consistent with Gilburd, but noted that although Kanter reports that the injection of antibody (which was in fact *different* from the purportedly-causal phospholipid antibody at issue) "worsens" disease (Kanter at 141), review of the article's figure showing the antibody's introduction into mouse subjects actually revealed that the EAE's "clinical severity" only worsened "at the very end," many days after the disease's

[44] Freund adjuvant is used in EAE. It is defined as "a water-in-oil emulsion incorporating antigen, in the aqueous phase, into lightweight paraffin oil with the aid of an emulsifying agent. On injection, this mixture *(Freund incomplete a.)* induces strong persistent antibody formation. The addition of killed, dried mycobacteria, e.g., *Mycobacterium butyricum,* to the oil phase *(Freund complete a.)* elicits cell-mediated immunity (delayed hypersensitivity), as well as humoral antibody formation." *Freund adjuvant,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=55029&searchterm=Freund%20adjuvant (last visited Dec. 9, 2022).

peak[45]—thus again suggesting that "these antibodies by themselves don't cause the initial demyelinating disease," as alleged in this case. Tr. at 282–83; Kantor at 141 (Figure 4C).

By contrast, Dr. Fujinami pointed to other studies that he proposed established the low likelihood that the pneumococcal vaccine could provoke an autoimmune process leading to GBS. One item showed the results of directly injecting myelin in the context of the EAE model. M. Sicotte et al., *Immunization with Myelin or Recombinant Nogo-66/MAG in Alum Promotes Axon Regeneration and Sprouting After Corticospinal Tract Lesions in the Spinal Cord,* Molecular & Cell Neurosciences 251, 254–55, 261 (2003), filed as Ex. F, Tab 1 (ECF No. 57-5) ("Sicotte"). Although the aim of Sicotte was in part to evaluate the effectiveness of different kinds of adjuvants, its authors also observed that the tested "immunization" could be protective of myelin. Tr. at 291, 358; Sicotte at 254–55. At most, Dr. Fujinami noted, the right adjuvant balance caused subjects to "develop antibodies against myelin components," but without resulting disease. Tr. at 291. Sicotte therefore, in Dr. Fujinami's estimation, undercut Dr. Serota's theory.

A second article was offered by Dr. Fujinami as further evidence of the likely-protective character of direct myelin immunization. M. Wallberg et al., *Vaccination with Myelin Oligodendrocyte Glycoprotein Adsorbed to Alum Effectively Protects DBA/1 Mice from Experimental Autoimmune Encephalomyelitis*, Eur. J. Immunology 1539, 1545 (2003), filed as Ex. F, Tab 2 (ECF No. 57-6) ("Wallberg"). Like Sicotte, Wallberg involved injection of a neuronal, myelin-associated antigen[46] into animal subjects, also observing a protective effect against EAE. Walberg at 1544–54; Tr. at 292–93, 358, 361. Sicotte and Wallberg thus, in Dr. Fujinami's estimation, contradicted the assumption of Petitioner's theory that a cross-reaction to antigenic components of a vaccine would be necessarily pathogenic. *Id.* at 358–59. If *direct* injection of the myelin itself was not harmful (or actually proved protective), regardless of whether it stimulated antibody development, then the mere exposure to the antigen via the immune system's reaction to the pneumococcal vaccine antigens, was even less likely to be pathogenic even if antibodies were generated. *Id.* at 292. Dr. Fujinami acknowledged, however, that the myelin antigens used in these studies (which were animal-derived) could be somewhat distinguished from a "pathogen-derived" antigen, and therefore might inherently produce a somewhat more muted response (especially to the extent mature animals develop immune tolerance over time, further reducing the chance of cross-reactive autoimmune processes). *Id.* at 333–37

[45] Kantor plainly notes that the introduction of the autoantibody occurred "just after the peak of disease"—and thus not that they were injected into animal subjects who were disease-free. Kantor at 141.

[46] On cross-examination, Dr. Fujinami admitted that the antigens in Walberg and Sicotte were protein-based, distinguishing them from the putative pathologic antigen at issue in this case. Tr. at 329–30. But, Dr. Fujinami reasoned that since Petitioner was contending that phospholipids in the pneumococcal vaccine were also found in myelin, then myelin-based antigens *of any kind* would still also contain the purportedly-pathogenic phospholipids. *Id.* at 330–31.

Dr. Fujinami's testimony also included some comments specific to Petitioner's medical history. For example, he noted Petitioner's claim of an immediate post-vaccination reaction. But he maintained (based on his lab experience testing animals) that any initial, innate immune response to a vaccine would not be enough to trigger a neuroinflammatory disease like GBS. Tr. at 298–99. Rather, an innate response to a vaccine resulting in some symptoms was not unexpected, and even if severe could not be deemed to inherently lead to an autoimmune disease. *Id.* at 300.

2. Dara Jamieson, M.D. – Dr. Jamieson, a neurologist, prepared two written reports and testified in support of the contention that there is not a casual association between the pneumococcal vaccine and GBS. *See generally* Tr. at 370–419. Report, dated June 30, 2021, filed as Ex. A (ECF No. 50-1) ("Jamieson First Rep."); Report, dated Jan. 3, 2022, filed as Ex. E (ECF No. 57-1) ("Jamieson Second Rep.").

Dr. Jamieson received her undergraduate degree from George Washington University, and her medical degree from the University of Pennsylvania School of Medicine. Tr. at 370; Curriculum Vitae, dated June 30, 2021, filed as Exhibit B (ECF No. 50-14 ("Jamieson CV") at 1; Jamieson First Rep. at 1. She then proceeded to a neurology residency and a cerebrovascular fellowship at the Hospital of the University of Pennsylvania. Tr. at 370–71; Jamieson CV at 1; Jamieson First Rep. at 1. Dr. Jamieson was a practicing neurologist for 32 years in academic medical centers before transitioning to a teaching appointment as the Clinical Professor of Neurology at Weill Cornell Medicine in New York, New York. Jamieson CV at 1; Jamieson First Rep. at 1.

Dr. Jamieson received specialty board certificates in neurology and vascular neurology by the American Board of Psychiatry and Neurology, neurosonology by the American Society of Neuroimaging, and headache medicine by the United Council for Neurological Subspecialties. Tr. at 371; Jamieson CV at 2; Jamieson First Rep. at 1–2. Dr. Jamieson has published papers in peer reviewed journals, authored two books and other book chapters, and reviewed articles of multiple neurological topics. Tr. at 372; Jamieson CV at 10–15; Jamieson First Rep. at 2. She also has ample experience treating peripheral neuropathic conditions like GBS. Jamieson First Rep. at 1.

Dr. Jamieson began with a discussion of Mr. Bielak's medical history prior to his GBS diagnosis, describing his health overall as significantly impaired. Tr. at 373–78; Jamieson First Rep. at 15, 19. As she noted, Petitioner had suffered from multiple medical conditions—coronary artery disease with vascular risk factors,[47] gout,[48] obstructive sleep apnea, hypothyroidism, and

[47] These risk factors included hyperlipidemia, hypertension, which has resulted in significant coronary artery disease having had a myocardial infarction. Tr. at 373.

[48] The medical record indicated that gout was not a significant cause of his disability, but he was taking medication to keep it in check. Tr. at 374. According to a January 9, 2015 medical record, Petitioner was taking medications for his gout, but his last flare-up was a year prior. Tr. at 376–77; Ex. 2 at 20.

Meniere disease.[49] Tr. at 373–74; Jamieson First Rep. at 19. Most prominently, Petitioner suffered from chronic pain syndrome for which he had been prescribed narcotics,[50] and Gabapentin for back pain, leg pain with weakness and numbness, and shoulder pain. Tr. at 374; Jamieson First Rep. at 15, 18.

Although Petitioner testified that he was an active person, this did not comport with Dr. Jamieson's reading of the medical records. Tr. at 375; Jamieson First Rep. at 15. For example, she highlighted a progress note from Dr. Udy on March 23, 2015 (prior to vaccination and GBS onset), indicating that Mr. Bielak was suffering from significant muscle weakness that interfered with activities like sitting, standing, walking, lying down and sleeping. Tr. at 375; Ex. 9 at 23. Petitioner proceeded to a March 26, 2015 evaluation for whether he should receive a handicap placard[51] in connection with his lumbar disc disease (for which Petitioner was receiving epidural steroid injections, among other things). Tr. at 375–77; Jamieson First Rep. at 15; Ex. 2 at 18. In addition, the medical record revealed that Petitioner used a cane for support and was taking medication for pain. Tr. at 376; Ex. 2 at 18.

Dr. Jamieson largely agreed with Petitioner's GBS diagnosis,[52] specifying that he most likely suffered from the AIDP variant[53] (which she noted lacks a known antibody biomarker). Tr. at 378–80; Jamieson First Rep. at 15–16; Jamieson Second Rep. at 2. However, she took issue with Dr. Khella's assessment that Petitioner's course had been severe. Tr. at 387, 400; Khella Rep. at 7. Severe GBS, she argued, would involve circumstances where (as set forth in one of Petitioner's case report filings) the individual was on a ventilator and/or in intensive care for weeks. Tr. at 389–99; Bianchi, at 338–39 (finding a 78-year-old incurred GBS in the midst of an active pneumococcal infection featuring a fever and requiring hospitalization prior to onset of neurologic symptoms).

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[49] Meniere disease is "hearing loss, tinnitus, and vertigo resulting from nonsuppurative disease of the labyrinth with edema." Tr. at 373; *Meniere Disease,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70588&searchterm=Meniere%20disease (last visited Dec. 9, 2022). For Petitioner, Meniere disease caused him to lose hearing in one ear. Tr. at 373–74.

[50] Dr. Jamieson was critical of Petitioner's use of narcotic medication. Tr. at 374–75, 377–78; Jamieson First Rep. at 18. However, the issue is tertiary to the claim's resolution, and I therefore do not give the matter extensive consideration herein.

[51] Though gout was listed under Petitioner's past medical history, there was nothing to indicate that he was symptomatic with gout associated with this visit. Tr. at 376; Ex. 2 at 18.

[52] Dr. Jamieson did observe that not all the general diagnostic criteria were met—in particular, Petitioner did not manifest bilateral and flaccid weakness of his limbs. Tr. at 387–88; Fokke at 34. But ultimately, she did not deny the appropriateness of the diagnosis, but instead invoked such non-corroborative findings as proof of the mild character of Petitioner's GBS course.

[53] Each GBS variant, Dr. Jamieson noted, is associated with distinct clinical and immunological features. Tr. at 392; Jamieson Second Rep. at 2.

Petitioner, however, had never experienced that degree of severity. His GBS manifested with mainly sensory findings, with Dr. Jamieson opining that even his gait issues were more likely the product of sensory disturbances rather than motor dysfunction. Tr. at 388; Jamieson First Rep. at 14. Petitioner also did not have any autonomic, respiratory, or bulbar complications.[54] Tr. at 389; Jamieson First Rep. at 14. He never needed a feeding tube, and his blood pressure and pulse rate never became so abnormal as to require medication. Tr. at 389. The mere fact Petitioner had been hospitalized was not in Dr. Jamieson's estimation proof of severity. *Id*. at 389–90. And after Mr. Bielak's discharge, he made good progress (especially considering his prior condition). *Id*. at 393. Thus, when Petitioner went back to his neurologist in February 2016, his examination produced normal results, at which point Dr. Jamieson felt he had largely recovered. *Id*. at 389, 393–94, 414; Jamieson First Rep. at 14; Jamieson Second Rep. at 4; Ex. 3 at 1–3.[55]

Although Dr. Jamieson acknowledged that causation was an issue beyond her primary expertise, she did not accept that Petitioner's GBS was likely vaccine-caused. Tr. at 391, 400–01; Jamieson First Rep. at 18–19; Jamieson Second Rep. at 1. First, she emphasized the lack of epidemiological evidence showing an increased risk of GBS associated with the pneumococcal vaccine. Tr. at 380–81, 391; Jamieson First Rep. at 16; R. Baxter et al., *Lack of Association of Guillain-Barré Syndrome with Vaccinations,* Clinical Infectious Diseases 197, 200 (2013), filed as Ex. A, Tab 9 (ECF No. 50-10) (finding no evidence of an increased risk of GBS following vaccinations of any kind, in a large retrospective study of 415 incident cases of GBS patients from 1995 through 2006). A more recent study had looked for adverse events based on a sample of 300,000 patients receiving the 13-valent pneumococcal conjugate vaccine (and 200,000 who had received the 23-valent pneumococcal polysaccharide vaccine, which is not at issue herein), but identified only *four* cases of post-vaccination GBS for the Prevnar version studied—not nearly

[54] In so arguing, Dr. Jamieson questioned some of Dr. Khella's clinical highlights as inaccurate. Petitioner's wife and nurse, for example, did not remember he was ever in the intensive care unit or had respiratory problems, despite Dr. Khella's contention, nor did the medical record itself so establish. Tr. at 387.

[55] In support of one of Respondent's contentions, Dr. Jamieson attempted also to demonstrate that Petitioner's symptoms could not meet the Act's six-month severity requirement. *See generally* Tr. at 402–06, 419; Jamieson First Rep. at 14, 18; Jamieson Second Rep. at 4. To that end, she maintained that tremors Petitioner reported in August 2016 were likely normal/physiologic rather than GBS sequelae. Tr. at 395–96; Jamieson Second Rep. at 4; Ex. 3 at 26–27. She similarly argued that other lingering symptoms were more likely associated with Petitioner's many pre-vaccination conditions (although some pre-vaccination records do not support the ongoing existence of certain specific symptoms, like fatigue). Tr. at 396–98, 406–10, 414; Jamieson First Rep. at 14, 18; *compare* Ex. 9 at 17 (June 24, 2015 visit where Petitioner was denying symptoms of fatigue) *and* Ex. 2 at 16 (September 9, 2015 record reporting that Petitioner was experiencing more refreshing sleep) *with* Ex. 5 at 5 (October 6, 2015 notation that following vaccination Petitioner was experiencing fatigue) *and* Ex. 6.1 at 94 (April 21, 2016 cardiac catheterization reporting continued fatigue six months post-vaccination).

My resolution of this case does not, however, turn on severity. Indeed, I can find on this record that there is preponderant evidence of six months of post-GBS onset sequelae. It is Petitioner's inability to establish the "can cause" prong that is fatal to the claim.

enough to establish a statistically significant risk. Tr. at 380–81; Jamieson First Rep. at 17; H. F. Tseng et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults,* Open Forum Infectious Diseases 1, 1, 4, 6–7 (2018), filed as Ex. A, Tab 8 (ECF No. 50-9) ("Tseng"). In particular, Tseng concluded that the data did not support an increased risk of adverse events following the vaccine's administration in individuals 65 and older—a cohort that would include the Petitioner. Tr. at 381; Tseng at 7.

Dr. Jamieson also noted that few case reports showed an increased risk of GBS associated with the pneumococcal vaccine. Tr. at 381, 391. And those that had been offered as evidence in this case were (in her estimation) deficient or unpersuasive. Tr. at 381–85; Jamieson First Rep. at 17–18. Ravishankar, for example, was authored by a student at a private for-profit medical school, who published it in two "predatory" journals (where the author pays to publish in a journal that is not subject to peer review and is not cited in PubMed—the primary online repository of medical literature).[56] Tr. at 382, 384; Jamieson First Rep. at 17–18. In addition, (and although the description of the case subject's medical history was not clear) Dr. Jamieson questioned whether the GBS diagnosis was even reliable or medically accurate.[57] Tr. at 383–85; Jamieson First Rep. at 18; Ravishankar at 1.

A second case report of post-vaccine GBS was in Dr. Jamieson's opinion no better. Tr. at 385–86; Jamieson Second Rep. at 3; N. Souayah et al., *Guillain–Barre Syndrome After Vaccination in United States: A Report from the CDC/FDA Vaccine Adverse Event Reporting System,* Vaccine 5253, 5253–255 (2007), filed as Ex. 53 (ECF No. 53-7) ("Souyah II"). Souyah II relied on adverse event passive surveillance reporting, finding that out of 54 cases reporting *any* association between vaccines and GBS for a single year (2004),[58] only *one* involved the pneumococcal vaccine, while the majority involved the flu vaccine (an association already recognized in the Vaccine Program). Souayah II at 5254. In addition, Souayah II's authors provided no information as to time period between receipt of the pneumococcal vaccine and onset of symptoms (whereas they were able to observe some timeframes for the other instances of post-vaccination GBS involving different vaccines). Tr. at 385–86; Jamieson Second Rep. at 3.

[56] *See generally McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *35 n.66 (Fed. Cl. Spec. Mstr. July 15, 2019) (defining predatory journals as "open-access (i.e., they charge authors a price to publish the article) and are not effectively peer-reviewed prior to publication").

[57] For example, Dr. Jamieson noted that the patient's onset began in October 2015, but clinical nadir occurred in January 2016—a three-month timeframe inconsistent with GBS's known acute/monophasic course (at least in the context of the AIDP variant). Tr. at 384–83; Ravishankar at 1.

[58] I also observe that Souayah II does not indicate how many *total* vaccinations were administered for that single year, further reducing the probative value of its findings.

Dr. Jamieson did acknowledge that reliable medical/scientific evidence supported an association between *Campylobacter jejuni* bacterial infections and GBS—although the resultant GBS variant tended to be axonal in nature rather than mainly demyelinating, as in the case of AIDP. Tr. at 392, 399, 401; Jamieson First Rep. at 16; Bianchi at 338–39; Lehmann at 643; N. Yuki & H-P Hartung, *Guillain–Barré Syndrome,* New England J. Med. 2294, 2297–300 (2012), filed as Ex. Ex. A, Tab 4 (ECF No. 50-5) ("Yuki"). And there was little evidence in Petitioner's records showing that he had suffered from such an infection. Tr. at 401.[59] Dr. Jamieson was otherwise unable to identify an alternative factor that could have triggered Petitioner's GBS (although she noted that a third of GBS cases had an unknown/idiopathic cause). *Id*. at 401–02. In the end, the only connection between Petitioner's vaccination and GBS was temporal—something that Dr. Jamieson deemed insufficient to establish causation by itself. *Id.* at 391, 401–02; Jamieson Second Rep. at 3–4.

III. Procedural History

After the case's initiation in May 2018, the matter was originally assigned to the "special processing unit" (the "SPU"), based on the presumption that it was likely to settle, given how often GBS is alleged as a vaccine injury. Petitioner filed medical records, affidavits, and a statement of completion by June 2018. Respondent's Rule 4(c) Report was then filed on April 24, 2019. ECF No. 22. The matter was then transferred out of SPU and reassigned to another special master before coming to me. Petitioner filed his expert report from Dr. Serota on February 22, 2021. ECF No. 46. Respondent thereafter filed expert reports from Drs. Jamieson and Fujinami on June 30, 2021. ECF No. 50. Petitioner was subsequently ordered to file a supplemental expert report from Dr. Serota, which was filed on September 30, 2021. ECF No. 52. Petitioner also filed another expert report from Dr. Khella on October 21, 2021. ECF No. 53. Respondent then filed supplemental expert reports on January 3, 2022. ECF No. 57.

A prehearing order set a two-day entitlement hearing to begin on April 12, 2022. ECF No. 55. The trial occurred as scheduled, and the matter is now ripe for resolution.

IV. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time

[59] As to Petitioner's diarrhea in the days following his vaccination (though unspecified), Dr. Jamieson agreed there was insufficient information from the record for her to make any determination as to whether it might have reflected a pre-vaccination, *Campylobacter*-like infection that could be causal. Tr. at 392, 401.

or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[60] In this case, Petitioner cannot assert a Table claim based on the pneumococcal vaccine causing GBS—there is no such claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

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

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

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

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

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates

that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

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

B. *Legal Standards Governing Factual Determinations*

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

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

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

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

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

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

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

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

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of

expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

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

D. *Consideration of Medical Literature*

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

ANALYSIS

I. Overview of Relevant Medical Terms and Applicable Prior Decisions

It is well understood that GBS is an acute, monophasic peripheral neuropathy involving rapidly progressive and ascending motor neuron paralysis, which is thought to have an autoimmune mechanism. Lehmann at 643–44; *Guillain-Barré Syndrome in Adults: Pathogenesis, Clinical Features, and Diagnosis,* UpToDate, https://www.uptodate.com/contents/guillain-barre-syndrome-in-adults-clinical-features-and-diagnosis?search=guillain%20barre&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1 (last visited Dec. 9, 2022). Although its etiology is mostly unknown, two-thirds of GBS cases follow an antecedent infection (typically an upper respiratory tract or gastrointestinal infection) beginning a few weeks prior to symptoms onset. Vellozzi at 1149. A GBS diagnosis requires a thorough medical assessment involving the patient's clinical presentation, plus other kinds of diagnostic testing, such as nerve conduction studies or CSF analysis. Fokke at 34.

GBS's primary clinical features are generalized muscle weakness combined with sensory symptoms. P. Donofrio, *Guillain-Barré Syndrome*, 23 Continuum 1295, 1296 (2017), filed as Ex. A, Tab 2 (ECF No. 50-3) ("Donofrio"). GBS typically begins abruptly with paresthesia in the feet, progressing to a flaccid paralysis of the lower limbs and ascending to the trunk, upper limbs, and face (although some cases involve paresthesia in all four limbs simultaneously or paresthesia beginning in the upper limbs and descending downward). Donofrio at 1296. Weakness of the facial muscles is common and is frequently bilateral, and respiratory weakness can also occur (requiring arterial ventilation in severe cases). *Id.* Increased protein levels in the cerebral spinal fluid without a corresponding increase in cells is often featured in GBS. *Id.* at 1295. The AIDP variant (consistent with Petitioner's diagnosis) is the most common form of GBS, accounting for approximately ninety percent of cases in the United States. Lehmann at 643.

Much is known about not only GBS's likely pathogenesis (albeit in defined circumstances pertinent to certain variants), but also its association with one particular vaccine covered by the Program: the flu vaccine. There are several evidentiary components supporting this association. First, reliable science has established not only that a specific subvariant of GBS[61] can occur in the wake of a specific bacterial intestinal infection (*Campylobacter jejuni*), but also that an autoantibody generated in response to that infection drives the subsequent neuropathic disease process, cross-reacting with ganglioside structures[62] on the myelin that mimic the bacterial antigen

[61] The relevant GBS subvariant associated with *Campylobacter jejuni* is acute motor axonal neuropathy ("AMAN"). *Acute Motor Axonal Neuropathy,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92651 (last visited Dec. 9, 2022).

[62] Gangliosides are "any of a group of glycosphingolipids in which the polar head group on ceramide is a sialic acid–containing oligosaccharide linked via a glucose residue; they occur predominantly in tissues of the central nervous

and then causing autoimmune damage. Yuki at 2294–95, 2299. It is thus believed that molecular mimicry could also be the biologic mechanism driving *other* forms of GBS, as well—although no specific autoantibody has been identified as associated with AIDP. Second, viral infections have also been found associated with GBS—further supporting the contention that the flu vaccine could be causal. Lehmann at 644–48. And there is reliable evidence that the amino acid peptide sequences that make up different proteins could mimic aspects of myelin basic protein. *see generally* Cusick at 104–06. Finally, (and of special importance) a reliable item of epidemiologic evidence (albeit now more than 40 years old)[63] established a higher incidence of GBS after receipt of a different form of flu vaccine when compared to the unvaccinated population.

All these evidentiary components have been deemed sufficient to preponderantly demonstrate the flu vaccine can likely cause GBS.[64] *Mason v. Sec'y of Health & Hum. Servs.,* No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (noting that the flu vaccine-GBS association is supported by a "mix of (a) knowledge about how molecular mimicry "works" in GBS's pathogenesis, (b) trustworthy animal experiments that model demyelinating injuries in the context of the molecular mimicry mechanism, and (c) solid (if somewhat old) epidemiologic evidence . . . establishing a higher incidence of GBS after vaccination when compared to an unvaccinated population"). As a result, too many well-reasoned decisions to count have found the issue resolved (at least for purposes of deciding Program cases). *See, e.g.*, *Chinea v. Sec'y of Health & Human Servs*., No. 15-095V, 2019 WL 1873322 (Fed. Cl. Spec. Mstr. Mar. 15, 2019); *Strong v. Sec'y of Health & Human Servs*., No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018); *Stitt v. Sec'y of Health & Human Servs*., No. 09-653V, 2013 WL 3356791 (Fed. Cl. Spec. Mstr. May 31, 2013); *Stewart v. Sec'y of Health & Human Servs*., No. 06-777V, 2011 WL 3241585, at *16 (Fed. Cl. Spec. Mstr. July 8, 2011). Such cases often also rely on the theory of molecular mimicry, proposing that antibodies produced by B cells in response to a vaccine's viral antigen components can cross-attack the myelin sheath (because the target antigen and gangliosides of the myelin sheath share structural homology), thereby causing demyelination of peripheral nerves. *Chinea*, 2019 WL 1873322, at *15.[65]

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system." *Ganglioside,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=19729&searchterm=ganglioside (last visited Dec. 9, 2022).

[63] The item of literature at issue was not filed in this case, and is not directly relevant to whether the pneumococcal vaccine can cause GBS, and I therefore do not cite it—but it has been repeatedly referenced in cases in which the flu vaccine's association with peripheral neuropathies like GBS is at issue. *See, e.g.*, *Rowan v. Sec'y of Health & Hum. Servs.*, No. 17-760V, 2020 WL 2954954, at *16 (Fed. Cl. Spec. Mstr. Apr. 28, 2020) (deeming the item the "'Ur-text' of scientific articles for purposes of all Vaccine Act claims").

[64] Of course, it is axiomatic that prior decisions do not bind my determination herein. Rather, I decide this case based on the evidence before me. However, it is not only useful, but prudent, to take into account prior determinations in reaching my decision.

[65] Indeed, the relevant science was convincing enough to the Government that GBS was added in 2017 as a Table Claim for the flu vaccine. See 42 C.F.R. § 100.3(a).

Importantly for present purposes, however, *the same is not true for the pneumococcal vaccine* (which does *not* also have a counterpart Table claim for GBS), for several reasons. First, the vaccine's composition is distinguishable in significant regards from the flu vaccine. The form of flu vaccine most commonly administered in the U.S. is "unadjuvanted"—meaning it does not include an ingredient like alum which aids in the vaccine's immunogenicity. *A.K. v. Sec'y of Health & Hum. Servs.*, No. 17-792V, 2022 WL 2678877, at *22 (Fed. Cl. Spec. Mstr. June 17, 2022), *mot. for review denied*, No. 17-792V, slip op. (Fed Cl. Oct. 27, 2022). As a result, the flu vaccine aims solely to expose the human immune system to its viral antigens, in the hope/expectation that immune memory will subsequently occur. The Prevnar version of the pneumococcal vaccine, by contrast, includes *both* an adjuvant as well as an additional component—CRM197, a synthesized version of diphtheria—to which the vaccine's bacterial components are "conjugated," or attached. Pneumococcal Package Insert at 19; *see also Deshler v. Sec'y of Health & Hum. Servs.,* No. 16-1070V, 2020 WL 4593162, at *19–20 (Fed. Cl. Spec. Mstr. July 1, 2020) (discussing purpose of conjugate in Prevnar). This encourages a more robust immune response—something that is needed because the pneumococcal vaccine's antigens would be less likely to trigger immune memory if simply exposed by themselves to the immune system.

Second, the manner in which the pneumococcal vaccine prompts an immune reaction is wholly different from the flu vaccine—in part due to the inclusion of the conjugate. Prevnar seeks to induce longer-term immunity *first* by prompting an initial T cell-dependent response to the diphtheria conjugate (with production of bacteria-specific antibodies occurring later, in the wake of that response). Pneumococcal Package Insert at 19 (noting that the conjugate "elicits a T-cell dependent immune response. Protein carrier-specific T-cells provide the signals needed for maturation of the B-cell response"). An unconjugated/unadjuvanted vaccine like the flu vaccine, by contrast, seeks to induce immunity *directly* through B cell antibody production, resulting simply from the immune system's exposure to vaccine antigens (making such vaccines "T cell independent"). Given these facial differences between how the two vaccines actually "work," it is as a threshold matter questionable whether the pneumococcal vaccine—engineered specifically to rely on a T cell process to *secondarily* promote B cell recognition of the vaccine's antigens—is as likely to cause a pathogenic process driven by molecular mimicry, since that theory depends on antibodies, generated *directly* by a vaccine, cross-reacting.

Given the above, there are far fewer reasoned decisions discussing whether the pneumococcal vaccine could cause GBS. The past three years have seen an increase in the number of such determinations—and at present, more favor causation than not.

I issued the first reasoned decision on the topic two years ago and denied entitlement. *Deshler*, 2020 WL 4593162. But the *Deshler* petitioner relied on a theory that the conjugate component of the Prevnar-13 vaccine *itself* had caused an aberrant, mimicry-driven autoimmune

process relating to the vaccine's antigens. *Deshler*, 2020 WL 4593162, at *27. Petitioner's expert did opine, as here, that the subsequent B cell reaction was driven by the polysaccharide component of the vaccination, although (unlike this case) he conceded that he could not demonstrate mimicry between the streptococcus pneumoniae polysaccharides and self-structures. *Id*.[66] Respondent's expert argued in reaction that the polysaccharides contained in the vaccine did not share structural homology with self-structures of the peripheral nervous system, and thus could not contribute to the pathogenesis of GBS via a molecular mimicry-driven cross-reaction to the vaccine's antigens. *Id*. at *27. I concurred with Respondent, while also finding that the petitioner relied too heavily on the temporal association between vaccination and onset as evidence of causation (and that there was another potential explanation for the claimant's GBS that had not been rebutted). *Id.* at *22, 27.

A year later, another special master found in favor of a petitioner alleging the pneumococcal vaccine could cause GBS. *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). The *Koller* petitioner relied on a different causation expert than in this case (as did Respondent), but that expert invoked arguments about the phosphoglycerol component of the pneumococcal polysaccharide antigen fairly close to what has been contended in this matter. *Koller*, 2021 WL 5027947, at *10. Indeed, the *Koller* expert specifically relied on many of the same articles offered in this action, like Ho, Chang, and Gilburd. The special master found the claimant had satisfied the first *Althen* prong. *Koller*, 2021 WL 5027947, at *20.

In 2022 alone, there have been four reasoned decisions involving the pneumococcal vaccine and GBS—the majority of which were also favorable to the relevant claimant. *See, e.g., Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); *Maloney v. Sec'y of Health & Hum. Servs.*, No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); *Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836 (Fed. Cl. Spec. Mstr. Jan. 19, 2022). Yet all involved the same expert who offered the opinion for the petitioner in *Koller*. Nevertheless, the same general theory was advanced and relied on almost all the same items of literature that Petitioner in this case filed in March 2022. *See, e.g.*, *Gross*, 2022 WL 9669651 at *15–16, 28 (referencing Nakos, Ho, Kanter, Gilburd, and Chang); *Pierson,* 2022 WL 322836, at *12, 28, 31 (same); *Maloney,* 2022 WL 1074087, at *16, 24 (same). Even *Koller* discusses at some length a subset of these items of literature. *Koller,* 2021 WL 5027947, at *20 (discussing Ho, Gilburd, and Chang).

By contrast, one reasoned decision in 2022 denied entitlement. *McConnell v. Sec'y of Health & Hum. Servs.*, No. 18-1051V, 2022 WL 4008238 (Fed. Cl. Spec. Mstr. Aug. 19, 2022).

[66] In this case, Petitioner's experts focus on the vaccine's *streptococcus* antigens as the direct cause of injury, with the conjugate merely amplifying the immune response (and thus encouraging an otherwise-aberrant immune reaction). *See, e.g.*, Tr. at 19–20, 59, 169–76.

The petitioner in *McConnell* used a different expert from the petitioner in *Koller*, however, and offered a "generic molecular mimicry theory," rather than what was proposed in the previous 2022 decisions (and in this case herein, as well). *McConnell*, 2022 WL 4008238, at *8. The *McConnell* petitioner also only referenced a few pieces of the literature mentioned in this case. *McConnell*, 2022 WL 4008238, at *7–9 (addressing El Khatib and Ravishankar).

II. Petitioner Has not Carried His Burden of Proof[67]

A. *Althen Prong One*

Petitioner's GBS diagnosis is not reasonably contested,[68] so this case largely turns on the fact that Petitioner was unable to preponderantly establish that the pneumococcal vaccine likely "can cause" GBS. Even though Petitioner's theory has reliable components, and has been deemed in prior cases enough to satisfy preponderance, my close reading of the literature and theory presented, along with expert opinions offered, lead me to the opposite conclusion.

As discussed above, the mechanism Petitioner embraces—molecular mimicry—is well-established in the Vaccine Program as providing a reliable pathogenic process for how GBS may *often* occur. *See Chinea*, 2019 WL 1873322, at *29. Molecular mimicry is predominantly driven by B cell activity, occurring when antibodies are produced in response to antigenic components of the vaccine—but which (due to mimicry between the presenting vaccine antigens and self-tissues) cause harmful cross-reactions, by mistakenly attacking the self antigens. Cusick at 103, 105. Program cases alleging GBS after receipt of the flu vaccine have often involved successful demonstrations by petitioners that the wild flu viral components of the vaccine contain amino acid sequences that share sequential and structural homology to self-structures (gangliosides) that would be the putative target of autoimmune attack. *See generally Pierson*, 2022 WL 322836, at *24 (citing *Chinea*, 2019 WL 1873322, at *15).

Much of the literature offered in this matter establishes that exposure to certain viruses, or even other bacteria such as *Campylobacter jejuni*, is associated with an increased risk of developing GBS via molecular mimicry. *See, e.g.*, Bianchi at 338; El Khatib at 36; Lasky at 1800; Lehmann at 643; Vellozzi at 1149–50; Yuki at 2294. But no such association between wild *S. pneumoniae* bacterial infections and the subsequent development of GBS has been demonstrated—and indeed, Respondent successfully established that such an association likely *does not exist*. Tr. at 27, 40, 106, 147, 215. Thus, the remaining question is whether vaccines derived from the

[67] Because I find that Petitioner could not satisfy the first two *Althen* prongs, I include no discussion of the third.

[68] Drs. Khella and Jamieson mostly addressed diagnostic issues in the case that were ultimately not dispositive, and I therefore do not further consider their testimony specifically (although some items of evidence they offered are evaluated below).

polysaccharide capsids of *S. pneumoniae* possess sufficient homology to initiate cross-reactivity when the wild bacteria itself does not.

Having reviewed the filed literature and considered the expert arguments, I find this has not been preponderantly established. In part, this is because Petitioner's causal theory borrows liberally from the ultimately-inapplicable GBS/flu vaccine "roadmap." Taken as a whole, Petitioner's experts have endeavored to link the pneumococcal vaccine to GBS based on the same reasoning used to link the flu vaccine with GBS. But because the two vaccines have as their antigenic basis two different pathogens (with, moreover, one being a virus and the other a bacterium), and do not even function in the same manner, the theory must identify different commonalities between the vaccine and the peripheral nerve myelin and mechanisms specific to the vaccine's distinguishable formulation. What "works" for the flu vaccine does not *per se* apply to a completely different vaccine.

Petitioner's experts did endeavor to offer evidence specific to the pneumococcal vaccine. Thus, they point to phospholipids common to the bacterial capsid antigens and the lipid content of myelin, attempting further to show the potential for cross-reactive harm by identifying anti-phospholipid antibodies in the blood serum of GBS patients, or other individuals experiencing somewhat-comparable demyelinating diseases (thus suggesting that those antibodies play some pathogenic role in GBS's progression or initiation). *See generally* Tr. at 168, 183, 211, 226–28. Dr. Fujinami did not dispute that some homologic commonalities were demonstrated through this aspect of Petitioner's case.

Deficiencies in Petitioner's theory, however, can be ascertained by considering both "ends" of the proposed pathogenic process. First, the evidence does not preponderate in favor of the conclusion that the pneumococcal vaccine can cause the purportedly-pathogenic antibodies to come into existence. Of Petitioner's two experts, Dr. Serota's testimony and opinion did an overall better job of attempting to address this question—but ultimately he could only establish a *plausible, putative* connection between vaccine and antibodies, based on studies like Chang (which stood more for the general proposition about the kinds of phospholipid antibodies the vaccine *intends* to promote, rather than their potential pathogenic character), or Ho and Kanter—which not only involved the distinguishable illness MS but only showed the *presence* of anti-lipid antibodies in tested samples—not that they drive the disease. Gilburd was admittedly specific to GBS, but otherwise is consistent with other items of literature to the extent it does not sufficiently "connect" the pneumococcal vaccine to pathology.

In addition, as Dr. Fujinami persuasively established, it is more likely that these antibodies specific to phospholipid structures are merely the *product* of an existing and ongoing demyelinating process—not the instigating cause of it. Tr. at 275–76, 355, 362–63; Gilburd at 27. Thus, the second portion of this aspect of Petitioner's theory—that the phospholipid antibodies

likely cross-react with antigenically similar structures on the myelin, causing GBS—is also not demonstrated. There is a difference between antibodies that *initiate* harm (critical to any claim that vaccine caused an injury) and antibodies that secondarily encourage *an already-existing* pathogenic demyelinating process.[69] Dr. Fujinami also demonstrated that given the manner in which the pneumococcal vaccine relies on a T-cell dependent conjugate process to *later* cause antibodies specific to *streptococcus pneumoniae* bacteria to be generated, it was ultimately less likely to generate directly-causal autoantibodies than a T-cell independent vaccine (like the flu vaccine), further reducing the likelihood of a molecular mimicry-driven cross-reaction. And he noted experimental instances in which excess antibody production resulted in no disease.

Otherwise, I do not deem evidence regarding the flu vaccine and GBS to be helpful in this case, or to suggest that the outcome of this case should simply follow what has been found in that other context. As discussed above, far more is known about the former situation—the kinds of cross-reactive, homologous autoantibodies generated by infections and flu vaccines, plus the manner in which they can drive damage to the myelin on the peripheral nerves. And there is even reliable epidemiologic evidence supporting that association.[70] Here, by contrast, Petitioner's theory only speculates that a different form of autoantibody (comprised of carbohydrates and not amino acids) is *also* likely to drive disease, but with far less reliable evidence on the point. In effect, all Dr. Serota seems to have done is tried to show that molecular "stuff" in the vaccine is also found in the myelin, and that antibodies believed to cross-react at some time in the GBS pathogenic process (not likely at the outset of disease) are sometimes seen in the blood sera of GBS patients.

Petitioner also relies on several case reports as indirect proof of a causal association. But such evidence is only weakly probative of causation when offered in Vaccine Program cases. *See, e.g.*, *Pearson v. Sec'y of Health & Human Servs.*, No. 17-489V, 2019 WL 1150044, at *11 (Fed. Cl. Spec. Mstr. Feb. 7, 2019) (concluding that case reports receive only limited evidentiary weight and cannot cure *Althen* prong one deficiencies); *Harris v. Sec'y of Health & Human Servs.*, No. 10-322V, 2014 WL 3159377, at *18 (Fed. Cl. Spec. Mstr. June 10, 2014) ("case reports are generally not a valuable form of evidence"). And many were not specific to the pneumococcal vaccine, further diminishing their evidentiary value. *See, e.g.*, Lehmann at 646; Salmon at 6–7;

[69] In theory, a claimant *could* argue that even if a vaccine did not directly spark a demyelinating disease, it could promote production of pathogenic autoantibodies that worsen the disease or assist its chronic nature. But this is effectively a claim for significant aggravation—and not only does Petitioner not allege such a claim, but the evidence does not support the finding that Petitioner's GBS likely predated the vaccination.

[70] I do not *require* Petitioner in this case to offer epidemiologic evidence to support his claim—and were I to do so I would commit legal error. *Perekotiy v. Sec'y of Health & Hum. Servs.*, No. 16-997V, 2020 WL 12904810, at *13 (Fed. Cl. Spec. Mstr. Apr. 20, 2020), *mot. for review denied*, No. 16-997V, 2020 WL 5887548 (Fed. Cl. Sept. 17, 2020) (citing *Andreu*, 569 F.3d at 1378–79) ("[p]etitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory."). I merely note that the overall mix of evidence supporting the flu vaccine-GBS association is far more robust than what was offered herein.

Vellozzi at 1150. The one most on-point, Ravishankar, was not only problematic from a foundational reliability standpoint (since, as Respondent points out, it was offered by a person lacking in full medical credentials, and was also published in a less-respected publication), but was also factually distinguishable, since the patient in question had received two doses of different forms of the pneumococcal vaccine over a lengthy period of time. Ravishankar at 1. More broadly, if I am to give weight to case reports offered herein as at least allowing for *some* possibility of causation, then I must contrast them with larger studies relying on similar passive surveillance temporal observations, like Tseng, but which see little to no relationship between the pneumococcal vaccine and GBS. Tseng at 7.

At the same time, Respondent's experts (primarily Dr. Fujinami) raised unrebutted points about the low probability that the pneumococcal vaccine promotes an aberrant autoimmune process mediated by molecular mimicry. He convincingly showed through his testimony that pathologic autoimmunity is not a given simply because of the potential for cross-reactivity, and that vaccination is categorically less likely to spark aberrant autoimmunity in the first place. He persuasively testified about studies in which direct injection of a cross-reactive neuro-antigen capable of stimulating antibodies either caused no neuropathic/demyelinating disease experimentally, or was protective—thus greatly undermining the contention that the possibility of phospholipid-specific antibodies after vaccination makes disease likely. *See* Sicotte at 254–55; *see also* Wallberg at 1544–54. As one of the scientists recognized to have first expounded on the concept of molecular mimicry in the context of autoimmunity, Dr. Fujinami's background and personal expertise made him exceedingly well-qualified to address these issues—giving his testimony additional trustworthiness and heft. (Dr. Serota, by contrast, while qualified to offer the opinion he did, demonstrably lacked the same degree of extensive direct research experience on the topics in contention).

My causation determination is admittedly contrary to several recent reasoned decisions referenced above—even though the causal theory offered herein closely parallels what was offered in those other cases, based on much of the same literature, as well. But despite the temptation to submit to "majority rule," I will not follow those prior decisions for two reasons.

First (and as already noted), special masters are never bound by the decisions of their colleagues. And here, despite the overlap in causation theories, the same mix of evidence relied upon in the prior determinations—of facts as well as expert input—*is not present in this case*. In particular, a completely different set of experts testified herein. Dr. Fujinami—a recognized pioneer in elucidating the contours of molecular mimicry, and a persuasive expert—did *not* testify in any of the prior cases. His reading of the items of literature common to all the most recent pneumococcal vaccine-GBS cases, coupled with his deep, demonstrated knowledge of autoimmune processes theorized to proceed via molecular mimicry, carried significant weight in leading me to reject Petitioner's causation showing. The Program not only tolerates but expects

that for certain kinds of causation theories (especially where medical science is not in agreement), the results of individual cases may vary, depending on the evidence offered. *Lampe*, 219 F.3d at 1361–62.

Second, *I do not find the theory accepted in these prior contrary determinations to be reliable*. Having read carefully the same items of literature that were offered in those other recent cases, and absorbed Dr. Fujinami's take on the same, I do not discern reliable scientific or medical support for the theory that the antibodies the pneumococcal vaccine is *intended* to cause the immune system to produce are in turn likely to be pathogenic in any critical manner for GBS to occur—or that an unintended effect of the vaccine in rare cases is the production of other cross-reactive antibodies capable of instigating GBS. At most, the literature shows that myelin is composed in part of certain lipids, and that some antibodies to these lipid-predominant aspects of the myelin are apparent, and/or may play a role in GBS's progression.

But *when*, and *what* role? And more fundamentally, what evidence shows that the antibodies the vaccine is intended to stimulate are primarily driving GBS—as *must* be the case if the vaccine is likely causal? Not only do articles relied upon in those prior cases (i.e., Ho, Nakos, Chang, and Gilburd) not satisfactorily answer these questions, they do not even *address* them. And some of these items (especially when read in conjunction with articles offered by Respondent, like Sicotte or Wallberg) actually suggest that the phospholipid antigenic targets on the myelin either serve a protective, immune-modulating role, or at worst are damaged by an ongoing autoimmune process only *after* it has begun—thus greatly undermining the contention that antibodies to these lipid myelin components *initiate* it.

Importantly, the theory deemed meritorious in the flu vaccine-GBS context relies on antibodies generated in response to *viral* particles (comprised of proteins "built" of amino acid sequences) cross-reacting against protein components in the myelin. Here, by contrast, Petitioner's theory cannot identify amino acid sequences in a bacterium-oriented vaccine—and so instead effectively looks for an alternative triggering target for GBS. In effect, this causation theory seems driven by the need to propose an alternative process of how GBS unfolds, simply because the vaccine at issue could not result in a cross-reaction to distinguishable molecules. But it is the height of conclusory reasoning to maintain that *any* vaccine administered before the onset of GBS can cause it—so long as a clever enough expert connects the vaccine's components and a presumed autoimmune target in the myelin with some molecular "links." And Dr. Fujinami persuasively established that molecular mimicry was not likely relevant in the context of GBS occurring due to the pneumococcal vaccine.

I give no weight to Petitioner's explanations for the thin quality of his theory—contentions about the "rarity" of vaccine injuries making it is difficult to identify reliable proof, for example, or the fact that medical science cannot precisely explain how immune-mediated diseases work (a

concept captured by the oft-quoted phrase from *Althen,* "a field bereft of complete and direct proof of how vaccines affect the human body"). *Althen,* 418 F.3d at 1280. As the Court has recognized, "the standard of proof does not operate as a sliding scale that varies depending upon the quantity and quality of the scientific evidence that is available." *Caves v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 143 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012). Special masters are expressly tasked with weighing evidence pro and con, and then concluding whether the result of that weighing preponderates in the claimant's favor. To find that this process did not favor causation in this case is not akin to requiring a petitioner to prove causation with certainty. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 962 (Fed. Cir. 1993) (affirming denial of entitlement).

Finally, I stress that based on my evaluation of the experts' competing opinions plus the literature filed, *this is not a close case.* Petitioner has not preponderantly shown the pneumococcal vaccine can cause GBS. His expert cannot simply identify some similarities between the vaccine's antigens and aspects of nerve myelin, and then declare victory after hoisting the molecular mimicry "flag." *Yalacki v. Sec'y of Health & Hum. Servs.*, No. 14-278V, 2019 WL 1061429, at *34 (Fed. Cl. Spec. Mstr. Jan. 31, 2019), *mot. for review denied*, 146 Fed. Cl. 80 (2019). Nor does establishing the presence of the putative pathogenic autoantibody in GBS patients, without some other reliable proof associating that autoantibody with *initiation* of disease pathogenesis in some regard, carry his burden. The Petitioner's showing only rises to the level of plausibility, albeit substantiated with some reliable items of proof—but that is not enough to be preponderant.

B. *Althen Prong Two*

The record in this case does not preponderate in favor of the conclusion that the pneumococcal vaccine "did cause" Petitioner's GBS (assuming it could—a finding I expressly do not reach, as discussed above). I note that my determination does not turn on the extensive evidence of Petitioner's pre-vaccination conditions. Although ample evidence was offered as to the many kinds of debilitating medical problems Petitioner faced, it was not demonstrated that these illnesses or health issues were causally related to his GBS. Some record evidence suggests a possible gastrointestinal infection not long after vaccination that arguably could be causal of GBS, but I cannot conclude preponderantly that this is likely.

Nevertheless, there is insufficient evidence connecting the vaccine to the demyelinating disease process that transpired thereafter. Treater statements associating the vaccine with his GBS occurred early in his course, or were memorialized based on what he told them, rather than evidence of the suspicion of an association—and certainly his overall treatment record does not support the contention that his neurologic care providers concluded the vaccine was causal. There are also no testing results that would corroborate Petitioner's possession of the purportedly-causal

autoantibodies identified in Dr. Serota's theory. And no other medical exam evidence offers any indirect proof that an aberrant immune process triggered by vaccination was to blame.

At most, there is record evidence, bulwarked by Petitioner's credible testimony, that he experienced a transient reaction to the vaccine. But general post-vaccination malaise is not unexpected. And although Petitioner's complaints of skin streaks are somewhat unusual, they were not demonstrated to be associated with, or an expected precursor for GBS. Dr. Serota otherwise did not persuasively establish through his testimony that post-vaccination malaise inevitably means an individual is experiencing a vaccine-driven autoimmune process that will soon result in disease. To so conclude is to give unwarranted weight to the temporal relationship between vaccination and onset.

CONCLUSION

It cannot be assumed that because GBS is closely associated with a single vaccine (here, the flu vaccine), that it is likely similarly attributable to *other* vaccines, and in the same general way. Rather, claimants must carry their burden of proof—here, by preponderantly establishing, via an offering of sufficient evidence *specific to the vaccine in question*, how it could cause GBS. This has not been accomplished in this case—the pneumococcal vaccine has not been shown to likely lead to GBS.

I enjoyed meeting (if virtually) the Petitioner and found him an honest individual. He has my sympathies in his suffering, and I regret that I cannot award him damages. But my evaluation of the evidence, in light of the applicable legal standard, compels me to dismiss this claim. In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[71]

IT IS SO ORDERED.

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

[71] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.