In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: June 23, 2025

CHRISTOPHER BROWN,	*	PUBLISHED
Petitioner,	*	No. 18-1074V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Ruling on Entitlement; Pneumococcal Conjugate ("Prevnar 13") Vaccine; Guillain-Barré Syndrome ("GBS"); Motion to Strike.
Respondent.	*	

Jessica Anne Olins, mctlaw, Seattle, WA, for Petitioner.
Katherine Carr Esposito, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT AND MOTION TO STRIKE[1]

On July 23, 2018, Christopher Brown ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018),[2] alleging that as a result of a pneumococcal conjugate ("Prevnar 13") vaccine administered on August 18, 2015, he developed peripheral neuropathy and Guillain-Barré Syndrome ("GBS"). Petition at ¶¶ 1, 11, 15 (ECF No. 1). Respondent argued against compensation, recommending "compensation be denied." Respondent's Report ("Resp. Rept.") at 2, 12 (ECF No. 22).

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards,[3] the undersigned finds Petitioner has provided preponderant evidence that the Prevnar 13 vaccine he received caused his peripheral neuropathy and GBS, satisfying Petitioner's burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is entitled to compensation.

Additionally, the undersigned denies Petitioner's Motion to Strike the Supplemental Expert Report of Dr. Serota, for the reasons explained herein.

I. ISSUES TO BE DECIDED

The first disputed issue arises out of Petitioner's Motion to Strike the supplemental expert report submitted by Dr. Marc Serota. See Petitioner's ("Pet.") Motion ("Mot.") to Strike, filed Aug. 30, 2023 (ECF No. 102). Petitioner asks the undersigned to strike the expert report following a status conference during which the undersigned determined Dr. Serota plagiarized the work of another expert in the Program. Id. at 1; Order dated Aug. 30, 2023 (ECF No. 101). Respondent objected, arguing the report should remain part of the record. Resp. Response to Pet. Mot. to Strike ("Resp. Response to Mot. to Strike"), dated Aug. 31, 2023 (ECF No. 103). Petitioner disagreed with Respondent's position. Pet. Reply to Resp. Response to Pet. Mot. to Strike ("Pet. Reply to Mot. to Strike"), filed Sept. 1, 2023 (ECF No. 104).

Regarding the issue of entitlement, diagnosis is in dispute. Specifically, the parties disagree about whether Petitioner's "peripheral neuropathy was [GBS]." Joint Submission, filed May 20, 2024, at 1 (ECF No. 116). The parties do not dispute Petitioner received a Prevnar 13 vaccine on August 18, 2015 and a flu vaccine on October 8, 2015,[4] or that he experienced symptoms of peripheral neuropathy for more than six months from the date of administration of vaccination. Id. at 1-2.

Further, the parties dispute causation, specifically all three Althen prongs: (1) whether Petitioner presented preponderant evidence that the Prevnar 13 vaccine can cause peripheral

[3] While the undersigned has reviewed all the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[4] Petitioner does not assert that the flu vaccine he received on October 8, 2015 caused or significantly aggravated his GBS. See Petitioner's Memorandum in Support of Petitioner's Motion for Findings of Fact and Conclusions of Law Regarding Entitlement ("Pet. Memo."), filed May 20, 2024, at 1 (ECF No. 117); Joint Submission at 1-2.

neuropathy or GBS,[5] (2) whether Petitioner presented preponderant evidence of a logical sequence of cause and effect that the Prevnar 13 vaccine did cause Petitioner's peripheral neuropathy or GBS, and (3) whether Petitioner presented preponderant evidence that the onset of symptoms occurred within a medically appropriate timeframe and consistent with his causal theory. Joint Submission at 2.

II. PROCEDURAL HISTORY

Petitioner filed his petition on July 23, 2018, followed by medical records[6] from August to December 2018. Petition; Pet. Exhibits ("Exs.") 1-9. Respondent filed his Rule 4(c) Report on July 10, 2019, arguing against compensation. Resp. Rept. at 2, 12. This case was reassigned to the undersigned on October 3, 2019. Notice of Reassignment dated Oct. 3, 2019 (ECF No. 26).

Petitioner filed an expert report from Dr. Serota on September 20, 2021. Pet. Ex. 14. Respondent filed an expert report from Dr. Timothy Vartanian on February 17, 2022. Resp. Ex. A. Thereafter, the parties requested a Rule 5 conference, which was held on May 3, 2022. Joint Status Rept., filed Mar. 21, 2022 (ECF No. 84); Rule 5 Order dated May 4, 2022 (ECF No. 85). The undersigned preliminarily found Petitioner had GBS, with an onset on or about October 1 to October 5, 2015. Rule 5 Order at 2.

Thereafter, Petitioner filed a supplemental expert report from Dr. Serota on December 2, 2022 and Respondent filed an expert report from Dr. Thomas Leist on July 28, 2023. Pet. Ex. 34; Resp. Ex. C. For the reasons discussed in more detail below, the undersigned held a status conference on August 29, 2023 to address concerns of plagiarism. Order dated Aug. 30, 2023. To address said concerns, Petitioner filed a motion to strike the plagiarized expert report on August 30, 2023 and filed an expert report from Dr. Kazim A Sheikh on February 16, 2024. Pet. Mot. to Strike at 1; Pet. Ex. 66.

Following Petitioner's filing of the expert report from Dr. Sheikh, the parties requested a ruling on the record. Joint Status Rept., filed Mar. 21, 2024 (ECF No. 113).[7] Petitioner filed a motion for a ruling on the record on May 20, 2024, which was followed by Respondent's response on August 23, 2024. Pet. Memo.; Resp. Brief Opposing Entitlement, filed Aug. 23, 2024 (ECF No. 122). Petitioner did not file a reply.

This matter is now ripe for adjudication.

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[5] In their Joint Submission, the parties stated that the first issue of causation was "[w]hether Petitioner has preponderantly proven that administration of his Prevnar 13 vaccine caused his peripheral neuropathy." Joint Submission at 2. The undersigned has recharacterized this statement to accurately reflect Althen prong one, whether Petitioner has proven that the Prevnar 13 vaccine can cause peripheral neuropathy and GBS. See Althen, 418 F.3d at 1280.

[6] Medical records were filed throughout litigation.

[7] Respondent did not request to file a responsive expert report.

III. RULING ON PETITIONER'S MOTION TO STRIKE

A. Background

A Rule 5 conference[8] was held on May 3, 2022. Rule 5 Order. Thereafter, the parties filed additional post-Rule 5 conference expert reports. Pet. Ex. 34; Resp. Ex. C. In reviewing those reports, the undersigned discovered that Dr. Serota's supplemental expert report (Pet. Ex. 34) included expert opinions identical to expert reports written by Dr. Lawrence Steinman in other Prevnar 13/GBS cases. Order dated Aug. 30, 2023; see, e.g., Simeneta v. Sec'y of Health & Hum. Servs., No. 18-859V, 2024 WL 4881411 (Fed. Cl. Spec. Mstr. Oct. 31, 2024); Maloney v. Sec'y of Health & Hum. Servs., No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022). Dr. Serota's second expert report contained pages and paragraphs that were identical, word-for-word, to Dr. Steinman's reports from other cases, and Dr. Serota did not attribute the opinions to Dr. Steinman. Order dated Aug. 30, 2023, at 1. Thus, Dr. Serota plagiarized the work of Dr. Steinman. Id.

On August 29, 2023, a status conference was held with the parties to address the issue. Order dated Aug. 30, 2023. The undersigned advised that in determining how best to respond to the problem, she examined Raymo, a case where former Chief Special Master Vowell addressed the issue of a plagiarized expert report, and found it provided guidance here. Id. at 1 (citing Raymo v. Sec'y of Health & Hum. Servs., No. 11-0654V, 2014 WL 1092274 (Fed. Cl. Spec. Mstr. Feb. 24, 2014)).

At the entitlement hearing in Raymo, evidence was presented that Petitioner's expert, Dr. Becker, plagiarized his expert report from another expert, Dr. Kerr, in another case. Raymo, 2014 WL 1092274, at *13. The expert report was word-for-word the same and virtually identical. Id. at *13-14. Then Chief Special Master Vowell found by a preponderance of the evidence that Dr. Becker plagiarized his expert report and because of this, she did not rely on his opinions when making a determination in the case. Id. at *14. She wrote,

> It is clear that Dr. Becker presented the work product of Dr. Kerr as his own. It does not appear that he disclosed this fact to [P]etitioners or their attorney, at least before the issue was raised at hearing. It is this failure that I find the most concerning in deciding whether to credit any part of his testimony. Had Dr. Becker indicated that he had been provided a copy of Dr. Kerr's earlier report and agreed with the reasoning and conclusions therein and adopted them as his own, my concerns about his candor would be less pressing. However, whether for financial reasons, time constraints, or for the prestige attached to being an expert witness, Dr. Becker was willing to take a shortcut, pass another's work product off as his own and, more significantly, testify in a manner that attempted to

[8] Vaccine Rule 5(a) contemplates that a special master will hold a status conference to "review the materials submitted[,] [] evaluate the parties' respective positions," and "present tentative findings and conclusions" to facilitate the outlining of necessary proceedings for resolving the issues presented in the case.

> mislead the court about the origin of the opinions expressed in the report bearing his signature.

Id.

Here, the undersigned advised the parties she would follow the sound reasoning of the former Chief Special Master in Raymo and would not rely on the opinions in Dr. Serota's supplemental expert report (Pet. Ex. 34). Order dated Aug. 30, 2023, at 2. The undersigned allowed Petitioner to obtain another expert to provide an opinion in place of Dr. Serota's supplemental report so that Petitioner was not prejudiced by the plagiarized report. Id. An order memorializing this plan was issued on August 30, 2023. See id. at 1.

In response to the August 30, 2023 Order, Petitioner requested, on the same day, the Court strike Petitioner's Exhibit 34, the supplemental expert report of Dr. Serota. Pet. Mot. to Strike at 1. No explanation or basis, other than what was in the Court's Order, was provided. See id.

On August 31, 2023, Respondent filed a response objecting to the motion to strike and requesting the Court leave the report (Pet. Ex. 34) as part of the record in this case for any potential reviewing body. Resp. Response to Mot. to Strike at 1. Respondent argued "the exhibit is relevant to the credibility and weight to be given to the expert's testimony. Further, the special master is charged with 'creating a record sufficient to allow review of the special master's decision' pursuant to Vaccine Rule 3(b)(2)." Id. Respondent also noted that Petitioner did not request to strike Dr. Serota's initial expert report and that the undersigned stated she would not rely on only Dr. Serota's supplemental expert report. Id. at 1 n.1 (citing Order dated Aug. 30, 2023, at 2).

Petitioner filed a reply on September 1, 2023, arguing that striking the supplemental report from the record would not violate Vaccine Rule 3(b)(2). Pet. Reply to Mot. to Strike at 1. Specifically, Petitioner argued that in striking the report (Pet. Ex. 34) from the record and allowing Petitioner to supplement the record with another expert's report, "the Court is adhering to Vaccine Rule 3(b)(2), by affording each party a full and fair opportunity to present its case and creating a record sufficient to allow review of the special master's decision." Id. Petitioner added that "the issue of credibility and weight to be given to the expert's testimony is not currently ripe for a ruling in this matter." Id.

Because the undersigned had yet to be called upon to adjudicate entitlement, Petitioner's motion was deferred. Order dated Oct. 2, 2023 (ECF No. 105). Now that the undersigned has been asked by the parties to adjudicate entitlement, it is appropriate to rule on Petitioner's Motion to Strike.

B. Ruling Denying Petitioner's Motion to Strike

A special master must "afford[] each party a full and fair opportunity to present its case." Vaccine Rule 3(b)(2). Vaccine Rule 8(b)(1) provides that "[i]n receiving evidence, the special master will not be bound by common law or statutory rules of evidence but must consider all

relevant and reliable evidence governed by principles of fundamental fairness to both parties." This rule echoes the statutory requirement that a "special master . . . shall consider . . . all [] relevant medical and scientific evidence contained in the record." § 13(b)(1). Together, Vaccine Rule 8 and § 13 "direct[] the special master to consider all relevant and reliable evidence, unencumbered by traditional rules of admissibility, while being guided by principles of fairness." Hazelhurst v. Sec'y of Health & Hum. Servs., 604 F.3d 1343, 1349 (Fed. Cir. 2010); see also R.K. ex rel. A.K. v. Sec'y of Health & Hum. Servs., No. 03-0632V, 2015 WL 10911950, at *39-41 (Fed. Cl. Spec. Mstr. Sept. 28, 2015) (interpreting Vaccine Rule 8 and § 13 to grant special masters the discretionary authority to exclude unreliable evidence from the record but not requiring special masters to do so in any particular instance), mot. for rev. denied, 125 Fed. Cl. 57 (2016), aff'd per curiam, 671 F. App'x 792 (Fed. Cir. 2016); Hulbert ex rel. Hulbert v. Sec'y of Health & Hum. Servs., 49 Fed. Cl. 485 (2001), aff'd, 35 F. App'x 899 (Fed. Cir. 2002). The Vaccine Act further mandates "flexible and informal standards of admissibility of evidence." § 12(d)(2)(B).

Although the Vaccine Rules do not specifically include a mechanism for a motion to strike testimony, in Vaccine Program cases, "exclusion from the record is an exceptional remedy, and should only be applied by the Court where the material sought to be excluded is so unreliable, it patently forfeits every trace of being helpful to the Court's consideration of the facts of the case." Veryzer v. Sec'y of Health & Hum. Servs., No. 06-522V, 2010 WL 2507791, at *21 (Fed. Cl. Spec. Mstr. June 15, 2010). "The Vaccine Rules favor broad inclusion, and 'the probative value of the evidence or the credibility of the witnesses . . . are matters within the purview of the fact finder.'" R.K., 2015 WL 10911950, at *36 (quoting Munn v. Sec'y of Health & Hum. Servs., 970 F.2d 863, 871 (Fed. Cir. 1992)). "Because special masters serve as both fact-finders and judicial officers, and because they have developed an expertise that lay jurors have not, they do not need the same procedural protection that excluding testimony at the outset provides." Id. at *41.

Further, if the Court is called upon to rule on entitlement, there will be nothing to cite to when referencing that the undersigned is not relying on Dr. Serota's supplemental opinions and the reason for not doing so. See Rogers v. Sec'y of Health & Hum. Servs., No. 94-89V, 2000 WL 1517675, at *3 (Fed. Cl. Spec. Mstr. Sept. 8, 2000) (denying Petitioner's motion to strike because it was a case of first impression and was highly controversial and striking the exhibit "might suggest the court is insecure in its decision"), dismissed, 47 F. App'x 579 (Fed. Cir. 2002). Accordingly, it should not be excluded as it is "helpful to the Court's consideration of the facts of the case." Veryzer, 2010 WL 2507791, at *21.

Additionally, during the status conference on August 29, 2023, the undersigned explained she looked to Raymo as guidance for how to handle a plagiarized expert report and there, the plagiarized report was not stricken from the record. See Order dated Aug. 30, 2023; Raymo, 2014 WL 1092274.

For these reasons, the undersigned finds it is not necessary to strike Dr. Serota's expert report. Pursuant to the Vaccine Rules and statute, which favor broad inclusion and principles of fundamental fairness to both parties, the undersigned **DENIES** Petitioner's motion to strike. Dr.

Serota's supplemental expert report (Pet. Ex. 34) will remain part of the record, but the undersigned will not consider it in her adjudication of this matter.

Moreover, in light of Dr. Serota's act of plagiarism, the undersigned has concerns about the credibility and veracity of Dr. Serota's opinions set forth in his initial expert report. Special masters have disregarded expert opinions entirely for improper conduct. See, e.g., McKown v. Sec'y of Health & Hum. Servs., No. 15-1451V, 2019 WL 4072113, *47-48 (Fed. Cl. Spec. Mstr. July 15, 2019); Raymo, 2014 WL 1092274, at *13-16 (rejecting entire medical opinion as unreliable where expert credibility issues included a plagiarized report and a failure to reveal a medical license suspension); see also Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1325-26 (Fed. Cir. 2010) (noting "[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010) (finding no error when the Special Master determined an expert witness lacked credibility when the Special Master explained his reason for so determining); see also Hanlon v. Sec'y of Health & Hum. Servs., 191 F.3d 1344, 1349 (Fed. Cir. 1999) (noting the Vaccine Act provides Special Masters with great deference in determining the credibility and reliability of expert witnesses and holding a Special Master's "credibility determinations are virtually unreviewable"). Therefore, Dr. Serota's first expert report will also not be considered by the undersigned in adjudication of entitlement, but it will remain in the record.

In summary, the undersigned **DENIES** Petitioner's motion to strike Dr. Serota's supplemental expert report filed as Petitioner's Exhibit 34. Neither of Dr. Serota's reports will be considered as evidence or referenced by the undersigned. However, both reports will remain in the record for the reasons asserted by Respondent.

IV. BACKGROUND RELEVANT TO ENTITLEMENT

A. Medical Terminology

GBS is "an acute polyradiculoneuropathy with a variable clinical presentation." Resp. Ex. C, Tab 3 at 1.[9] Criteria for the diagnosis were first published in 1978,[10] and included clinical features such as progression of symptoms, relative symmetry, usually mild sensory symptoms, abnormal levels of protein in cerebrospinal fluid ("CSF"), and an abnormal electrodiagnostic study. Resp. Ex. A, Tab 13 at 2. Over time the criteria have been revised and due to discovery of new subtypes, "the conceptual framework of GBS has become increasingly complex." Resp.

[9] Christiaan Fokke et al., Diagnosis of Guillain-Barré Syndrome and Validation of Brighton Criteria, 137 Brain 33 (2014).

[10] These criteria were published by the National Institute of Neurological and Communicative Diseases ("NINCDS") committee. Resp. Ex. A, Tab 13 at 2 (Anita McGrogan et al., The Epidemiology of Guillain-Barré Syndrome Worldwide: A Systematic Review, 32 Neuroepidemiology 150 (2009)).

Ex. A, Tab 23 at 1.[11] The Brighton criteria were developed by the Brighton Collaboration and sponsored by the World Health Organization in response to the H1N1 swine flu vaccination (2009/2010) and its association with GBS. Resp. Ex. C, Tab 3 at 2. The criteria are summarized below:[12]

	Level of diagnostic certainty			
Diagnostic criteria	1	2	3	4
Bilateral and flaccid weakness of limbs	+	+	+	+/−
Decreased or absent deep tendon reflexes in weak limbs	+	+	+	+/−
Monophasic course and time between onset-nadir 12 h to 28 days	+	+	+	+/−
CSF cell count < 50/µl	+	+[a]	−	+/−
CSF protein concentration > normal value	+	+/−[a]	−	+/−
NCS findings consistent with one of the subtypes of GBS	+	+/−	−	+/−
Absence of alternative diagnosis for weakness	+	+	+	+

+ present; − absent; +/− present or absent;.
NCS = nerve conduction studies; GBS = Guillain-Barré syndrome.
[a] If CSF is not collected or results not available, nerve electrophysiology results must be consistent with the diagnosis Guillain-Barré syndrome.

Id. at 2 tbl.1.

More recent studies have shown that GBS "consists of a spectrum of neuropathic disorders that may differ in the underlying pathogenesis and clinical manifestations." Resp. Ex. C, Tab 3 at 2. Further, "[t]here are no pathognomonic clinical characteristics . . . [and] no biomarkers" specific to GBS. Id.

B. Stipulated Facts

The parties agreed Petitioner was born on September 29, 1955. Joint Submission at 1. They also agree that he received a Prevnar 13 vaccine on August 18, 2015 and a flu vaccine on October 8, 2015. Id.

C. Summary of Medical Records[13]

Petitioner was 59 years old at the time of his Prevnar 13 vaccination on August 18, 2015. Pet. Ex. 1 at 2. Petitioner's active medical issues at the time of his vaccination included gastroesophageal reflux disease, essential hypertension, mixed hyperlipidemia, impaired fasting glucose, and degenerative spondylolisthesis of lumbar spine with pain radiating to right buttock and right posterior thigh. Pet. Ex. 2 at 141. Petitioner had no prior history of neurological or

[11] Benjamin R. Wakerley et al., Guillain-Barré and Miller Fisher Syndromes—New Diagnostic Classification, 10 Nature Revs. Neurology 537 (2014).

[12] These criteria "account for the level of diagnostic certainty based on the presenting findings at clinical and additional examinations, ranging from level 1 (highest level of diagnostic certainty) to level 4" (lowest level of diagnostic certainty). Resp. Ex. C, Tab 3 at 2.

[13] This summary is taken from Petitioner's Memorandum in Support of Entitlement and edited by the undersigned to include additional relevant information. See Pet. Memo. at 5-11.

demyelinating conditions. Id. at 141-44. Prior to vaccination, Petitioner was not diagnosed as diabetic. Id.

On October 8, 2015, Petitioner presented to his primary care provider ("PCP") Dr. Ronald McGee for his flu vaccine. Pet. Ex. 1 at 2; Pet. Ex. 6 at 40.

Four days later, on October 12, 2015, Petitioner returned to Dr. McGee with complaints of "a tingling sensation." Pet. Ex. 6 at 37. Dr. McGee's evaluation during this visit documented acute onset of sensory symptoms, sensory impairment on examination, and no history of preceding gastrointestinal or upper respiratory tract infections. Id. at 37-38. History of present illness documented,

> [Petitioner] complain[ed] of tingling sensation. [Petitioner] present[ed] with a diagnosis of tingling sensation. This was diagnosed 10 days ago. The course has been stable and nonprogressive. It is of mild intensity. He estimate[d] that the frequency of symptoms is every couple of minutes. Associated symptoms include paresthesias. He denie[d] arthralgias, chest pain, diarrhea, fever, headache, nausea, rash, sore throat[,] or tachycardia. [C]oncerned new [prescription] lipitor may contribute to symptoms. He has been on lipitor [six] weeks.

Id. at 37.

Neurologic examination revealed normal deep tendon reflexes ("DTRs") in his biceps, triceps, supinator, knee, and ankle jerk. Pet. Ex. 6 at 38. Petitioner also had "hyperesthesia in a 'stocking-glove' and bilateral ulnar nerve distribution." Id. Dr. McGee recommended laboratory work up, stop Lipitor for two weeks, and follow up in two weeks. Id. Laboratory work up showed elevated blood glucose of 116 (reference range 65-99 mg/dL), elevated hemoglobin ("Hgb") A1c of 6.3% (reference range < 5.7% of total Hgb),[14] and vitamin B12 and folate in normal range. Pet. Ex. 2 at 23-24.

On October 22, 2015, Petitioner returned to see Dr. McGee, who noted that Petitioner's tingling sensation "diagnosed [three] weeks ago" was of "moderate intensity" and that Petitioner was anxious. Pet. Ex. 6 at 32-33. Petitioner was advised to adhere to a 1500 calorie ADA diet, 10 pound weight loss, HgbA1c level checked quarterly, daily foot self-inspection, and decrease alcohol intake. Id. at 33. Petitioner was not prescribed medication to lower his blood glucose at this visit. See id.

[14] Petitioner's laboratory results provided an interpretive note explaining the significance of his result of 6.3%. Pet. Ex. 2 at 23-24. Results less than 5.7% were noted to be "[c]onsistent with the absence of diabetes" and results between 5.7-6.4% were noted to be "[c]onsistent with increased risk for diabetes (prediabetes)." Id. The level "[c]onsistent with diabetes" was results greater than or equal to 6.5%. Id. at 24. Thus, based on Petitioner's result, he was at increased risk for diabetes or prediabetic but not diabetic.

Petitioner presented to Mission Hospital emergency department ("ED") on November 3, 2015, with "complain[ts] of bilateral feet and hand numbness that began [three] weeks ago." Pet. Ex. 4 at 9. History taken by the ED physician (Dr. Gregory Lampe) noted,

> [Petitioner] state[d] that he went to bed, woke up in the middle of the night[,] and then had sudden numbness in his feet and hands. Patient went to see his [PCP], was diagnosed with peripheral neuropathy, and was started on Gabapentin.[15] However, numbness has gotten worse, with [Petitioner] stating that it is radiating upwards towards his arms and legs, but has not yet moved above his ankles. In addition, [Petitioner] state[d] that he has been dropping things due to the numbness in his hands. [Petitioner] note[d] that he still can sense pain, but sensation is what seems to be compromised. [Petitioner] report[ed] mild shortness of breath, but denies any head or neck pain. [Petitioner] report[ed] that he can walk in a straight line, but shuffles due to the numbness in his feet.

Id.

Neurologic examination revealed "intact right knee jerk, intact right bicep reflex, diminished left bicep reflex, diminished left knee jerk, ankle jerks diminished bilaterally, and oriented X4." Pet. Ex. 4 at 10. Brain computed tomography ("CT") and magnetic resonance imaging ("MRI") did not show any acute pathologies to explain Petitioner's symptoms. Id. at 11. The ED physician noted "[n]o obvious triggers [were] identified in [Petitioner's] history and [he] [did not] see an obvious pattern of ascending paralysis to suggest [GBS]." Id. at 14-15. Petitioner was diagnosed with peripheral neuropathy and discharged home with plans to see a neurologist. Id. at 15.

Two days later, on November 5, 2015, Petitioner saw neurologist Dr. Joey Gee. Pet. Ex. 3 at 7. Petitioner reported that four weeks prior, he awoke in the morning and his feet felt numb. Id. Recently, he has felt numbness in his hands, and he "turned his ankle a week ago." Id. He was taking gabapentin. Id. Also, he reported that his hands were weaker and his "feet [were] 75% numb from baseline." Id. Petitioner also reported neck pain but no headaches. Id. Petitioner also reported his Prevnar 13 vaccination about two months ago. Id. Review of systems was negative for preceding or ongoing gastrointestinal or respiratory infections. Id. at 8. Neurologic examination documented motor and sensory deficits and symmetric weakness of feet dorsiflexion (4/5) and plantar flexion (4/5), trace ankle reflexes, mild ataxia, and reduced vibration sensation in the feet (decreased by 50%; left more than right). Id. at 9-10.

Dr. Gee performed same day electromyography ("EMG")/ nerve conduction study ("NCS") testing that showed evidence of "significant sensory and motor neuropathy" consistent with acute inflammatory demyelinating polyradiculoneuropathy ("AIDP"), a variant of GBS. Pet. Ex. 3 at 44-48. Impression was "concerning for [GBS], possibly and controversially induced by the pneumococcal vaccine. This vaccine was given within one month of the onset of sensory impairment." Id. at 7. Treatment with intravenous immune globulin ("IVIG") was recommended. Id.

[15] A record of this visit does not appear to be in the record.

On November 7, 2015, Petitioner was admitted to Mission for treatment and management of his GBS. Pet. Ex. 4 at 89-96. Admitting physician, Dr. Reza Bashtar, noted diminished reflexes/areflexia in the legs. Id. at 95. Impression and plan documented "[r]ecent history of pneumonia vaccination prior to initiation to the symptoms." Id. On November 8, 2015, Dr. Gee saw Petitioner and reexamination indicated no change in neurologic examination since prior examination on November 5. Id. at 98; see Pet. Ex. 3 at 9-10.

The following day, lumbar puncture and CSF studies were obtained, which showed elevated protein at 50 (reference range 15-45 mg/dL). Pet. Ex. 4 at 106, 150. Normal or negative studies included CSF glucose, herpes simplex virus ("HSV") 1 and 2, Lyme, West Nile, oligoclonal bands, myelin basic protein, CSF immunoglobulin G ("IgG") and IgG index, and viral and bacterial, including acid fast bacilli ("AFB"), cultures. Id. at 150-60. CSF white blood cell ("WBC") count was 2 (reference range 0-5). Id. at 146. MRI of the cervical spine showed mild degenerative changes but was otherwise unremarkable. Id. at 142. Petitioner received a five-day course of IVIG infusions, he remained stable, and he was discharged home on November 12, 2015. Id. at 85-87. At the time of discharge, Petitioner's symptoms had reached a plateau and had not "worsened or improved." Id. at 85. Petitioner's discharge diagnosis was GBS and his peripheral neuropathy was attributed to GBS. Id. at 86 ("Peripheral neuropathy – [GBS]."). During his hospital course, his treating providers documented "[a]llergy to pneumonia vaccine" under his problem list. See, e.g., id. at 104, 108, 127.

Petitioner followed up with his PCP, Dr. McGee, on November 18, 2015 for GBS following his recent hospitalization. Pet. Ex. 2 at 164. Dr. McGee noted that Petitioner's symptoms of GBS were resolving. Id. Petitioner also had diffuse foot joint pain that began three days before. Id. Examination revealed tenderness of the left metatarsophalangeal joint of the great toe. Id. at 165. Petitioner was taking gabapentin for neuropathic pain. Id. at 164.

On December 9, 2015, Petitioner followed up with neurologist, Dr. Gee, for his GBS. Pet. Ex. 3 at 5-6. Petitioner reported "right hand 'tingling' and decreased fine motor with bilateral feet numbness and hypersensitivity." Id. at 5. Dr. Gee noted Petitioner continued to have subjective paraesthesias and he provided counseling about "GBS recovery phase and routine exercise." Id. Petitioner remained on gabapentin. Id. at 6.

On December 28, 2015, Petitioner followed up with his PCP, Dr. McGee, for foot pain. Pet. Ex. 2 at 175-76. Dr. McGee documented bilateral lower extremity paraesthesias and questioned whether these were due to resolving GBS. Id. at 175. Petitioner remained on gabapentin. Id.

Petitioner followed up with his neurologist, Dr. Gee, on January 20, 2016. Pet. Ex. 3 at 30-38. EMG was repeated and again showed a "demyelinating pattern of neuropathy" with improvement and "some recovery of the patterns." Id. at 30.

In 2016, Petitioner saw his PCP, Dr. McGee several times. See generally Pet. Ex. 2. On June 9, 2016, he saw Dr. McGee for an ankle sprain and continued use of gabapentin was

documented. Id. at 180. On July 14, 2016, Petitioner returned to Dr. McGee for sinusitis. Pet. Ex. 6 at 15. Continued use of gabapentin was documented. Id.

On August 16, 2018, Petitioner saw Dr. David R. Zachary, for numbness and tingling in his hands and feet. Pet. Ex. 6 at 10-12. Dr. Zachary documented that Petitioner

> [complained of] numbness and tingling in hands and feet since he had [GBS] in late 2015. He has also had minimal weakness in his hands and feet. It has not gotten worse for over [two] years. Concerned that he may still have [GBS] and that it could reactivate.

Id. at 11. Dr. Zachary discussed "the natural course of [GBS] which may leave residual problems," explaining that Petitioner's "peripheral neuropathy has not progressed nor has the mild residual weakness in in the distal extremities." Id. at 12.

D. Affidavits

1. Petitioner

In his first affidavit, executed April 5, 2018, Petitioner addressed jurisdictional and statutory requirements. Pet. Ex. 5. He averred that he received a vaccine included in the Vaccine Injury Table and that it was administered within the United States. Id. at ¶¶ 1-2. He further stated his vaccine-related injuries lasted longer than six months and that he had not previously been awarded any money damages in a civil action arising out of a vaccine-related injury. Id. at ¶¶ 3-4.

Petitioner executed a supplemental affidavit on December 26, 2018, describing the chronology of events. Pet. Ex. 7. Petitioner averred that prior to vaccination, he had a "healthy and active lifestyle." Id. at ¶ 4. He was the manager of a restaurant and enjoyed sports, including golfing and surfing. Id.

He received a Prevnar 13 vaccination on August 18, 2015 and a flu vaccination on October 8, 2015. Pet. Ex. 7 at ¶¶ 2-3.

From September 29 to October 3, 2015, Petitioner and his wife went on a short vacation. Pet. Ex. 7 at ¶ 5. During this trip, he did not experience any symptoms of pain or numbness. Id. On approximately October 12, or "soon before that day," he woke and "suddenly felt numbness" in his feet. Id. at ¶¶ 6-7. When standing, "[he] felt an odd sensation . . . like . . . rocks on the bottom of [his] feet." Id. at ¶ 6. He also had numbness in both hands. Id. Petitioner's symptoms progressed, he fell, and "thought he was becoming paralyzed." Id. at ¶ 8. By Halloween Day, October 31, 2015, his condition was "horrible." Id. at ¶ 9. When he met with neurologist Dr. Gee, Dr. Gee ran tests and told Petitioner that his symptoms were "likely vaccine-related." Id. at ¶ 10. Dr. Gee instructed Petitioner to go to the ED if his condition worsened. Id.

Several days later, Petitioner went to the ED, where he was admitted, had diagnostic testing, and treatment (IVIG). Pet. Ex. 7 at ¶¶ 11-12. When he was discharged, Dr. Gee told Petitioner that there was no additional treatment and whether he would improve would be determined over time. Id. at ¶ 15. In spring 2016, Petitioner continued to have numbness in his arms, hands, legs, and feet. Id. at ¶ 16. His gait was awkward, and he was cautious to prevent falling or rolling his ankle. Id. By the winter, Petitioner's walking had improved but he continued to have pain when standing and had to take sitting breaks to manage his pain. Id. at ¶ 17.

Over time, Petitioner learned to adjust to his condition, which was "very difficult and frustrating." Pet. Ex. 7 at ¶ 18. When his pain, numbness, and weakness did not resolve, he realized that his health "would not return" to what it was prior to vaccination. Id. Petitioner saw Dr. Zachary in August 2018, and he was told there was no cure for his GBS. Id. at ¶ 19. As of the date his affidavit was executed in December 2018, he continued to have "general weakness and fatigue . . . [and] pain and numbness in both feet and hands." Id. at ¶ 20. Petitioner is the manager of a seafood restaurant and he is required to be very active and on his feet carrying trays with dishware. Id. at ¶ 21. He occasionally rolls his ankles, has difficulty bending and keeping his balance, and must use caution to avoid falls or drop things due to weakness. Id.

Petitioner explained that he is the primary provider in his family, and his wife has macular degeneration[16] and is dependent on him. Pet. Ex. 7 at ¶ 22. He expressed "constant fear" of not being able to work, pay the bills or the mortgage, and support his family. Id. He also feels stress due to the worry of becoming disabled and unable to take care of his wife. Id. He does not think that his condition will get any better in the future. Id. at ¶ 23.

2. Rebecca Brown

Rebecca Brown, Petitioner's wife, executed an affidavit on February 25, 2020. Pet. Ex. 10 at ¶ 2, 4. She has suffered from macular degeneration for years, and she and her husband spend a lot of time together, grocery shopping, running errands, and he does all the driving. Id. at ¶ 3.

Mrs. Brown recounted that one morning after her husband received the flu vaccine on October 8, 2015, he got up early around three or four o'clock in the morning and complained that his feet felt asleep. Pet. Ex. 10 at ¶¶ 8-9. He continued to complain of tingling in his feet, and this sensation worsened during the day. Id. at ¶ 10. After several days, Petitioner complained of numbness and tingling in his hands and feet. Id. at ¶ 11. On Halloween, the couple usually decorated their home and yard, but Petitioner was unable to help. Id. at ¶ 12. His hands were numb and his fingers curled up making it difficult for him to straighten them out. Id. Mrs.

[16] Macular degeneration refers to "degenerative changes in the macula lutea," which is "an irregular yellowish depression on the retina." Macular Degeneration, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=68886 (last visited June 12, 2025); Macula Lutea, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87699 (last visited June 12, 2025).

Brown also noted that Petitioner's gait was different; he shuffled his feet and rolled his ankles when walking. Id. at ¶ 13.

Petitioner was admitted to the hospital in November 2015, and he was diagnosed with GBS. Pet. Ex. 10 at ¶¶ 14-15. After he was discharged, Petitioner attended many outpatient examinations and received treatment for pain and numbness in his hands and feet. Id. at ¶ 16. Mrs. Brown explained that her husband continues to have numbness in his hands and feet and a shuffling gait. Id. at ¶ 18. When he gets home from work, he has soreness in his feet and ankles and he occasionally still rolls his ankles. Id. Mrs. Brown stated that "witnessing [her] husband suffer from GBS . . . has been both frightening and lifechanging." Id. at ¶ 19. She is hopeful that he will "gain full recovery in the near future." Id.

E. Expert Reports[17]

1. Petitioner's Expert, Dr. Kazim A. Sheikh[18]

a. Background and Qualifications

Dr. Sheikh is board certified in psychiatry and neurology with an additional qualification in muscle pathology and a subspecialty certification in clinical neuromuscular pathology. Pet. Ex. 66 at 1. Dr. Sheikh received his medical degree from King Edward Medical College in Pakistan. Pet. Ex. 67 at 1. Thereafter, he completed a neurology residency at the Neurological Institute at Columbia University in New York and was a postdoctoral fellow in peripheral nerve disorders at Johns Hopkins University School of Medicine in Baltimore, Maryland. Id. Dr. Sheikh is a tenured Professor of Neurology and the Director of the Neuromuscular Program at the University of Texas ("UT") Medical School. Pet. Ex. 66 at 1. He also serves as the Director of the Neuromuscular Disorders Center at the Mischer Neuroscience Institute at Memorial Hermann-Texas Medical Center and the Directors of the Muscle and Nerve Laboratory and of the GBS/chronic inflammatory demyelinating polyradiculopathy ("CIDP") Center of Excellence at The University of Texas Health Science Center at Houston. Id. He "ha[s] a longstanding research and clinical interest in immune neuropathies, particularly, in the pathogenesis of GBS and CIDP." Id. He has authored or co-authored over 250 publications related to inflammatory neuropathies and has served on numerous review panels related to immune and inflammatory neuropathies. Id. at 2. He has also served on numerous review panels related to immune and inflammatory neuropathies including GBS. Id.

[17] The undersigned does not discuss Dr. Serota's expert reports for the reasons discussed above. See supra Section III. Although the undersigned has reviewed all the expert reports, for the sake of brevity, this Ruling does not include every detail of the experts' opinions. Instead, the undersigned focuses on the experts' material opinions, as they relate to the relevant issue of causation.

[18] Dr. Sheikh submitted one expert report. Pet. Ex. 66.

b. Diagnosis Opinion

Dr. Sheikh opined that Petitioner's clinical course and laboratory findings are "consistent with demyelinating or AIDP variant of GBS" with an onset of approximately October 2, 2015, or 45 days after his Prevnar 13 vaccination. Pet. Ex. 66 at 5.

Regarding the question of whether Petitioner's peripheral neuropathy was GBS, Dr. Sheikh opined in the affirmative, stating Petitioner's "acute neuropathic disease" was consistent with AIDP/GBS and was not caused by diabetes, hyperuricemia, or statins, as suggested by Respondent's expert, Dr. Leist. Pet. Ex. 66 at 5. In support of his opinion, Dr. Sheikh went through each of the Brighton diagnostic criteria for GBS and explained why Petitioner met all of the criteria at a level one, "diagnostic certainty," refuting Dr. Leist's opinions that Petitioner would not meet the lowest level of diagnostic certainty.[19] Id. at 6-8 (citing Resp. Ex. C at 7).

The first Brighton criterion is bilateral and flaccid weakness in the limbs. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 6. Dr. Sheikh explained that the Brighton criteria study cited by Dr. Leist acknowledged the significant variability in limb muscle weakness of patients with GBS, ranging from mild to complete paralysis. Pet. Ex. 66 at 6 (citing Resp. Ex. C, Tab 3 at 4 ("The severity of limb weakness at study entry and at nadir was highly variable. Weakness at nadir ranges from mild severity . . . to tetraparalytic . . . ."). Based on neurologist Dr. Gee's physical examination of Petitioner on November 5, 2015, Petitioner had bilateral distal ankle dorsiflexion weakness 4/5, satisfying this criterion. Id. (citing Pet. Ex. 3 at 9-10).

Criterion two is decreased or absent tendon reflexes in weak limbs. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 6. Dr. Sheikh noted that Petitioner had decreased ankle reflexes and lower limb weakness (ankle dorsiflexion and plantar flexion) documented by Dr. Gee on November 5, 2015, verifying that this criterion was met. Pet. Ex. 66 at 6 (citing Pet. Ex. 3 at 9-10).

The third criterion is a time frame between onset to nadir ranging from 12 hours to 28 days. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 6. Dr. Sheikh opined nadir was reached by November 5, 2015, as demonstrated by clinical observations and EMG, and assuming an onset between October 1 and October 5, 2015, such time frame between onset and nadir would be approximately four weeks. Pet. Ex. 66 at 6 (citing Pet. Ex. 3 at 9-10, 44-45). Additionally, this criterion requires a monophasic clinical course. Id. Dr. Sheikh explained that follow up EMG/NCS on January 20, 2015 showed improvement. Id. Petitioner's clinical course and the second EMG/NCS confirm that Petitioner had a single episode of GBS, which did not relapse, satisfying this aspect of the third criterion. Id. at 6-7.

The fourth and fifth criteria are based on CSF findings. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 7. Dr. Sheikh opined that Petitioner's CSF met these criteria because it revealed a normal WBC count and an elevated protein concentration (50; reference range 15-45). Pet. Ex. 66 at 7 (citing Pet. Ex. 4 at 150). He disagreed with Dr. Leist's assertions that Petitioner's protein level was only in the upper limit of normal and not elevated. See id. He maintained that

[19] For a more detailed explanation of the criteria and scoring system used, see Pet. Ex. 66 at 6-8; Resp. Ex. C, Tab 3.

although "CSF protein was mildly elevated," 30% of GBS patients do not have an increase in CSF protein or albuminocytologic dissociation "and absence of this finding does not exclude GBS diagnosis." Id. (citing Resp. Ex. C, Tab 3 at 5). Additionally, in a study he participated in, albuminocytologic dissociation was defined using a protein level greater than 45, the same cut off level used by the laboratory testing Petitioner's CSF. Id. (citing Pet. Ex. 68 (finding 70% of patients showed albuminocytologic dissociation and patients with "distal predominant weakness . . . were more likely to have lower CSF protein levels")).[20]

Criterion six requires that NCS be consistent with one of the subtypes of GBS. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 7. Dr. Sheikh explained that both of Petitioner's studies were consistent with the AIDP subtype of GBS, thus satisfying this criterion. Pet. Ex. 66 at 7-8.

The seventh and last criterion is the absence of an alternative diagnosis to explain weakness. Resp. Ex. C, Tab 3 at 2 tbl.1; Pet. Ex. 66 at 8. Dr. Sheikh listed all of the diagnostic tests done to rule out other causes of GBS and opined that there was no evidence of alternative causes. Pet. Ex. 66 at 8. He also responded to Dr. Leist's proposed alternative etiologies. Id. As for the idea that Petitioner's neuropathy was caused by diabetes, Dr. Sheikh explained that Petitioner did not have a diagnosis of diabetes but was prediabetic. Id. Next, Petitioner's electrodiagnostic studies (EMG/NCS) did not show features consistent with diabetes, or the other potential causes suggested, including hyperuricemia or statin neuropathy. Id. Further, the fact that Petitioner's follow-up EMG/NCS showed improvement weighed against diabetic neuropathy. Id. Dr. Sheikh concluded that Petitioner met this criterion. Id.

In conclusion, Dr. Sheikh opined that Petitioner met the Brighton diagnostic criteria with a diagnostic certainly of level one. Pet. Ex. 66 at 8. If the CSF protein level was considered in the normal range instead of elevated, then he opined Petitioner's diagnostic certainty level would be level two, which is still sufficient to support a diagnosis of GBS. Id.

c. Causation Opinion

Dr. Sheikh opined that the Prevnar 13 vaccine, "more likely than not, triggered [GBS] in the Petitioner." Pet. Ex. 66 at 14.

i. Althen Prong One

Dr. Sheikh offered molecular mimicry as the primary mechanism to explain how the Prevnar 13 vaccine can cause GBS. Pet. Ex. 66 at 14. He reiterated two examples of molecular mimicry taken from and attributed to his review of Dr. Serota's expert report based on cross

[20] Helle Al-Hakem et al., CSF Findings in Relation to Clinical Characteristics, Subtype, and Disease Course in Patients with Guillain-Barré Syndrome, 100 Neurology e2286 (2023).

reactivity between pneumococcal capsular polysaccharides ("CPS")[21] and phospholipids and CRM_{197} and contactin-1. Id. Dr. Sheikh also noted that "Dr. Lawrence Steinman has also proposed similar theoretical constructs regarding pneumococcal vaccination and GBS." Id. at 11. Based on his review of these theories, Dr. Sheikh opined that "these are carefully formulated credible constructs."[22] Id.

Additionally, Dr. Sheikh proposed two examples of molecular mimicry based on cross reactivity between "pneumococcal CPS and galactocerebroside [("Gal-C")[23]] enriched in peripheral nerves" and "CRM_{197} and P0 protein enriched in peripheral nerve myelin sheaths." Pet. Ex. 66 at 14.

1. Pneumococcal CPS and Gal-C in Peripheral Nerves

As explained by Dr. Sheikh, Prevnar 13 is a pneumococcal vaccine that contains CPS of 13 serotypes of *Streptococcus* ("*S.*") *pneumoniae* (1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F, and 23F). Pet. Ex. 66 at 11. Based on Dr. Sheikh's review of their biochemical profile, he opined that CPS contain repeat glycan (polysaccharide) motifs.[24] Id. at 12. Nine out of the 13

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[21] For the history and background information on CPS, see Pet. Ex. 71 (J.D. Grabenstein & K.P. Klugman, A Century of Pneumococcal Vaccination Research in Humans, 18 Clinical Microbiology & Infection 15 (2012)); Pet. Ex. 73 (K. Aaron Geno et al., Pneumococcal Capsules and Their Types: Past, Present, and Future, 28 Clinical Microbiology Revs. 871 (2015)).

[22] Since the undersigned is not considering Dr. Serota's opinions, she will also not consider opinions of Dr. Sheikh derived from his review of Dr. Serota's reports. The undersigned notes, however, that in his expert report, Dr. Sheikh indicated that he had reviewed Dr. Serota's reports and attributed the theories in such reports to Dr. Serota and Dr. Steinman. Thus, he did not "pass another's work product off as his own." Raymo, 2014 WL 1092274, at *14. Regardless, only the examples of homology independently proposed by Dr. Sheikh are discussed and considered by the undersigned.

[23] Galactocerebroside ("Gal-C") refers to "any of the cerebrosides in which the monosaccharide head group is galactose; they are abundant in the cell membranes of nervous tissue, particularly the myelin sheath." Galactocerebroside, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=19555 (last visited June 12, 2025). "Myelin is an electrical insulator that serves to speed the conduction of nerve impulses." Myelin Sheath, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=105841 (last visited June 12, 2025). Dr. Sheikh abbreviates galactocerebroside as GalC, however, the medical articles use the abbreviation Gal-C. For consistency, the undersigned uses the abbreviation Gal-C herein.

[24] Motif refers to "a structural element of a protein that is smaller than a domain and has a specific function." Motif, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=32233 (last visited June 12, 2025).

serotypes contain galactose monosaccharide in its pyranose[25] or furanose[26] form and five of these nine galactose residues are terminal (repeat or side chain). Id. (citing Pet. Ex. 73 at 4 tbl.1). Notably, Dr. Sheikh stated, "[t]he terminal residues can be more easily accessible for engagement with receptors or immune cells." Id.

In humans, peripheral nerve myelin is "enriched" with Gal-C, which Dr. Sheikh defined as a "galactose containing glycolipid with terminal galactose." Pet. Ex. 66 at 12. He explained that in AIDP/GBS, "the myelin sheath" is thought to be the "target of immune attack" in a "very small" number of patients. Id.

Based on this foundational information, Dr. Sheikh proposed that the CPS in the Prevnar 13 vaccine "could induce antibodies against galactose residue that are cross-reactive to [Gal-C]" causing nerve injury that manifests as GBS. Pet. Ex. 66 at 12.

The following observations and medical literature referenced by Dr. Sheikh provide additional foundation. To illustrate how molecular mimicry would work in the context of his proposed theory, he used the example of molecular mimicry relative to *Mycoplasma* ("*M.*") *pneumoniae* and GBS. Pet. Ex. 66 at 12. Jacobs et al.[27] reported that GBS occurs in patients after infection with *M. pneumoniae.* Id. (citing Pet. Ex. 74 at 1 ("[*M.*] *pneumoniae* infections occurred more often in GBS patients (5%) than in controls . . . .")).

Next, *M. pneumoniae* "express glycolipid antigens that cross-react with [Gal-C]." Pet. Ex. 66 at 12 (citing Pet. Ex. 75 at 1).[28] Kusunoki et al. stated Gal-C is "a major glycolipid antigen in the myelin of . . . [the] peripheral nervous system[]." Pet. Ex. 75 at 1. "Sensitization to Gal-C causes antibody-mediated demyelinating neuropathy, and anti-Gal-C antibody is a demyelinating factor." Id. The authors showed that a "Gal-C-like structure" was also present in *M. pneumoniae*, supporting the theory of "molecular mimicry between a major myelin glycolipid, Gal-C, and *M. pneumoniae*." Id.

[25] Pyranose is "any sugar containing a pyran ring structure, a cyclic form that ketoses and aldoses may take in solution." Pyranose, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=42367 (last visited June 12, 2025).

[26] Furanose is "any sugar containing a furan ring structure, a cyclic form that ketoses and aldoses may take in solution." Furanose, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=19459 (last visited June 12, 2025).

[27] B.C. Jacobs et al., The Spectrum of Antecedent Infections in Guillain-Barré Syndrome: A Case-Control Study, 51 Neurology 1110 (1998).

[28] Susumu Kusunoki et al., Anti-Gal-C Antibodies in GBS Subsequent to Mycoplasma Infection: Evidence of Molecular Mimicry, 57 Neurology 736 (2001).

Further, anti-Gal-C antibodies have been found in GBS patients, and these patients often have a "demyelinating/AIDP form of GBS." Pet. Ex. 66 at 12 (citing Pet. Ex. 76 at 1).[29] These antibodies are "probably produced by molecular mimicry" and "are considered an important factor in the pathogenesis of a subset of GBS." Pet. Ex. 76 at 1.

Lastly, Dr. Sheikh cited experimental animal studies that have shown that Gal-C and anti-Gal-C antibodies can induce demyelinating nerve damage. Pet. Ex. 66 at 12 (citing Pet. Ex. 77;[30] Pet. Ex. 79).[31]

Thus, Dr. Sheikh opined "[i]t is plausible that CPS in Prevnar 13 could induce antibodies against galactose residue that are cross-reactive to [Gal-C], which in turn can produce nerve injury." Pet. Ex. 66 at 12. He illustrated this theory using what is known about Gal-C in *M. pneumoniae* and its role in inducing GBS via molecular mimicry. Id. Relative to the vaccine here, the "galactose residues in [Gal-C] and galactose (pyranose form) contained in the CPS" (shown in the figure below) have a "close biochemical mimicry (resemblance)." Id.

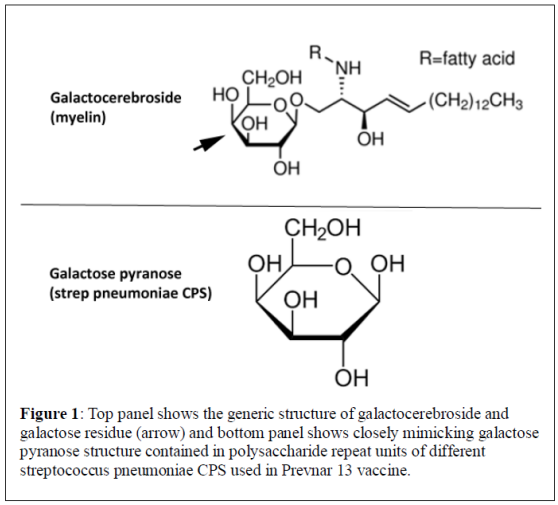

Figure 1: Top panel shows the generic structure of galactocerebroside and galactose residue (arrow) and bottom panel shows closely mimicking galactose pyranose structure contained in polysaccharide repeat units of different streptococcus pneumoniae CPS used in Prevnar 13 vaccine.

Id.

[29] Makoto Samukawa et al., Clinical Features in Guillain-Barré Syndrome with Anti-Gal-C Antibody, 337 J. Neurological Scis. 55 (2014).

[30] Takahiko Saida et al., Experimental Allergic Neuritis Induced by Sensitization with Galactocerebroside, 204 Science 1103 (1979).

[31] Kyoko Saida et al., In Vivo Demyelination Induced by Intraneural Injection of Anti-Galactocerebroside Serum, 95 Am. J. Pathology 99 (1979).

2. CRM_{197} and Myelin Protein P Zero ("P0")[32]

Dr. Sheikh's second example of molecular mimicry is based on proposed cross-reactivity between CRM_{197} ("genetically detoxified form of diphtheria toxin")[33] in the Prevnar 13 vaccine and peripheral nerve myelin protein P0. Pet. Ex. 66 at 12-13. Dr. Sheikh provided the following supportive evidence.

Myelin protein P0 "is unique to peripheral nerve" and "its sequence and structural similarities" have been implicated in the pathogenesis of leprosy after infection with *Mycobacterium* ("*M.*") *leprae*.[34] Pet. Ex. 80 at 1-2. Vardhini et al., using bioinformatic search tools,[35] found that "myelin P0 had sequence/structural similarities to . . . diphtheria toxin." Id. at 1-2. Diphtheria toxin was identified as a structural neighbor of myelin P0. Id. at 6. They noted that "[t]he key pathological event in leprosy and other neuropathies is demyelination, which could be a result of molecular mimicry/altered host signaling/host-pathogen binding and the consequent events." Id. Further, they explained that "[r]ecent reports have shown that . . . the diphtheria toxin . . . could induce [GBS] in certain proportion of immunized individuals." Id. The authors concluded that their study "support[ed] the hypothesis that there could be common molecular mechanisms underlying neurodegeneration." Id. at 7.

Next, Dr. Sheikh explained that in animal studies, "antibody and/or T-cell responses against P0 protein produce inflammatory demyelinating neuropathy resembling AIDP." Pet. Ex. 66 at 13. Dr. Sheikh cited several studies supporting this premise. See, e.g., Pet. Ex. 81 at 1, 5 (reporting "antibodies to P0 in the serum of GBS . . . patients");[36] Pet. Ex. 82 at 1 ("Antibodies to P0 . . . occur in GBS but are uncommon. . . . They might be part of a harmful pathogenic

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[32] "Myelin P0 is the most abundant and uniquely expressed peripheral nerve glycoprotein." Pet. Ex. 80 at 2 (Deena Vardhini et al., Comparative Proteomics of the Mycobacterium Leprae Binding Protein Myelin P0: Its Implication in Leprosy and Other Neurodegenerative Diseases, 4 Infection Genetics & Evolution 21 (2004)).

[33] Protein carrier "CRM197 is a nontoxic variant of diphtheria toxin isolated from cultures of *Corynebacterium diphtheriae* strain C7 (β197) grown in a casamino acids and yeast extract-based medium." Pet. Ex. 35 at 24 (Prevnar 13 package insert).

[34] "In vivo, myelin P0 exists as tetramers [four polymer units] which interleave with the tetramers of the adjacent membrane . . . linking the myelin membranes leading to compaction. Disruption of these P0-P0 interactions by competitive binding of structurally similar molecules could disrupt myelin compaction." Pet. Ex. 80 at 6 (internal citations omitted). This occurs in leprosy and is called "contact dependent demyelination." Id.

[35] For a description of the search tools/databases used, see Pet. Ex. 80 at 2.

[36] A. Khalili-Shirazi et al., Antibody Responses to P_0 and P_2 Myelin Proteins in Guillain-Barré Syndrome and Chronic Idiopathic Demyelinating Polyradiculopathy, 46 J. Neuroimmunology 245 (1993).

process or represent a regulatory response.");[37] Pet. Ex. 83 at 1 (proposing, following an animal study, that "molecular mimicry between T cell epitopes on pathogen derived antigens and the P0 protein may play [a role] in the pathogenesis of [GBS]");[38] Pet. Ex. 84 at 1 (showing via an animal study that in an autoimmune peripheral polyneuropathy in mice, the pathogenesis included "autoreactive T-cells and autoantibodies directed against myelin protein zero [P0]").[39] Dr. Sheikh noted the study from Yan et al.[40] showed that injection of anti-P0 antibodies from patients with inflammatory demyelinating neuropathy can induce inflammatory demyelinating nerve injuries resembling CIDP ("the chronic counterpart of acute . . . [GBS]") in animals. Pet. Ex. 66 at 13; Pet. Ex. 86 at 1.

In summary, Dr. Sheikh opined that clinical and experimental studies support the theory that CRM_{197} protein in the Prevnar 13 vaccine can "induce P0 protein specific autoimmunity by molecular mimicry." Pet. Ex. 66 at 13.

3. Adjuvant Induced Inflammation and Autoimmune Response

In addition to the above examples of molecular mimicry, Dr. Sheikh proposed that "[t]he vaccination process itself can also induce inflammation and enhanced autoimmune responsiveness" due to the alum adjuvant in the vaccine. Pet. Ex. 66 at 13. More specifically, Dr. Sheikh opined that when a vaccine is injected, the needle tract transects muscle and nerve fibers, leading to the "release of self-nerve antigens in the milieu of the vaccine and adjuvant." Id. This results in recruitment of "inflammatory cells . . . including macrophages (antigen presenting cells)." Id. The self-antigens caused by trauma and the foreign antigens from the vaccine "can be processed by the cells infiltrating the injection cite and induce immune responses." Id.

Dr. Sheikh also proposed that "alum in the Prevnar [13] vaccine can contribute to the development of GBS via stimulation of [] cytokines[] [interleukin ("IL")]-1 and IL-18." Pet. Ex. 66 at 13. He cited Eisenbarth et al.,[41] who explain that alum adjuvants "activate an intracellular

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[37] A. Makowska et al., Immune Responses to Myelin Proteins in Guillain-Barré Syndrome, 79 J. Neurology Neurosurgery & Psychiatry 664 (2008).

[38] Martin Adelmann & Christopher Linington, Molecular Mimicry and the Autoimmune Response to the Peripheral Nerve Myelin P0 Glycoprotein, 17 Neurochemical Rsch. 887 (1992).

[39] Eroboghene E. Ubogu et al., Behavioral, Electrophysiological, and Histopathological Characterization of a Severe Murine Chronic Demyelinating Polyneuritis Model, 17 J. Peripheral Nervous Sys. 53 (2012).

[40] Wei Xing Yan et al., P0 Protein Is a Target Antigen in Chronic Inflammatory Demyelinating Polyradiculopathy, 50 Annals Neurology 286 (2001).

[41] Stephanie C. Eisenbarth et al., Crucial Role for the Nalp3 Inflammasome in the Immunostimulatory Properties of Aluminium Adjuvants, 452 Nature 1122 (2008).

innate immune response system called the Nalp3 . . . inflammasome" which plays a role in "[p]roduction of [] pro-inflammatory cytokines." Pet. Ex. 88 at 1. Dr. Sheikh asserted that pro-inflammatory cytokines IL-1 and IL-18 are "relevant" because they are "strongly upregulated during the acute phase of GBS" and decline as the illness resolves. Pet. Ex. 66 at 14.

Jander and Stoll[42] reported that "T helper cell-dependent activation of macrophages is the principal mechanism underling immune-mediated myelin destruction in GBS and [experimental autoimmune neuritis ("EAN")]."[43] Pet. Ex. 91 at 1. They explained that T helper cells have two major subtypes: Th1 cells, which secrete proinflammatory cytokines IFN-γ and tumor necrosis factor-α, and Th2 cells, which secrete anti-inflammatory cytokines including IL-4 and IL-10. Id. "[I]t is a widely accepted view that disease induction in the EAN model as well as in human GBS follows a Th1-dependent immunopathogenesis." Id. Further, in EAN, there is a "coordinated increase of IL-18 . . . during active disease progression[,] suggesting a crucial role" for this cytokine. Id. at 2 (internal citations omitted).

Based on the literature cited, Dr. Sheikh concluded that the alum adjuvant in the vaccine and the effects of intramuscular injection could lead to an "inflammatory neuropathic disease process." Pet. Ex. 66 at 14. He also posited that the adjuvant could lead to the "breakdown of tolerance against self-antigens." Id.

ii. Althen Prong Two

Dr. Sheikh opined there was a logical sequence of cause and effect given the mechanistic theories discussed above. Pet. Ex. 66 at 14-15. Further, he explained there was no evidence of an alternative cause for Petitioner's GBS. Id. Petitioner did not have an antecedent upper respiratory infection, a diarrheal illness, or prior medial event, and the only triggering event was vaccination. Id.

Moreover, Dr. Sheikh opined Petitioner's treating physicians noted the association of Petitioner's Prevnar 13 vaccination with his GBS. Pet. Ex. 66 at 15. For example, Dr. Gee, on November 5, 2015, documented in Petitioner's history of present illness that "[h]e had a pneumonia shot about two months ago." Pet. Ex. 3 at 7. Dr. Gee's impression stated, "With the timing of symptoms, this is concerning for [GBS], possibly and controversially induced by the pneumococcal vaccine. The vaccine was given within one month of the onset of sensory impairment." Id. Petitioner also reported the vaccination to the ED physician upon admission on November 7, 2015, indicating the vaccination occurred "prior to the initial onset of his numbness." Pet. Ex. 4 at 89. Admitting physician, Dr. Bashtar noted, "[r]ecent history of pneumonia vaccination prior to initiation of the symptoms" in her impression. Id. at 95.

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[42] Sebastian Jander & Guido Stoll, Interleukin-18 Is Induced in Acute Inflammatory Demyelinating Polyneuropathy, 114 J. Neuroimmunology 253 (2001).

[43] "Experimental autoimmune neuritis (EAN) is the laboratory model of GBS." Pet. Ex. 93 at 1 (Shup Yu et al., Neutralizing Antibodies to IL-18 Ameliorate Experimental Autoimmune Neuritis by Counter-Regulation of Autoreactive Th1 Responses to Peripheral Myelin Antigen, 61 J. Neuropathology & Experimental Neurology 614 (2002)).

Additionally, Dr. Sheikh noted Petitioner's treating physicians also documented that Petitioner was allergic to the pneumococcal vaccination. Pet. Ex. 66 at 15 (citing Pet. Ex. 4 at 104, 108, 127 ("Allergy to pneumonia vaccine.")).

Dr. Sheikh summarized that in a susceptible host, the trigger of vaccination may lead to the breakdown of tolerance, and through the mechanisms of molecular mimicry and/or adjuvant, lead to GBS. Pet. Ex. 66 at 15.

iii. Althen Prong Three

Dr. Sheikh opined there was a temporal association between Petitioner's Prevnar 13 vaccination and his GBS. Pet. Ex. 66 at 15. In support of this opinion, he cited Langmuir et al.,[44] also cited by Dr. Leist, "as a surrogate for the Prevnar 13 vaccine and onset of GBS." Id. (citing Resp. Ex. C, Tab 7). Langmuir et al. reported a risk window of six-weeks for the development of GBS following administration of the swine flu vaccine. Resp. Ex. C, Tab 7 at 1, 24.

2. Respondent's Expert, Timothy Vartanian, M.D., Ph.D.[45]

a. Background and Qualifications

Dr. Vartanian is a board-certified neurologist specializing in the care of people with inflammatory demyelinating diseases. Resp. Ex. A at 1. He obtained his M.D. and Ph.D. from the University of Chicago, after which he completed a neurology residency at Massachusetts General Hospital and two fellowships at Beth Israel Hospital and Harvard Medical School in Boston, Massachusetts. Resp. Ex. B at 1-2. He taught at Harvard Medical School from 1992 to 2009, worked as an Attending Neurologist at Beth Israel Deaconess Medical Center from 1992 until 2009, and was the Director of the Division on Demyelinating Diseases at Beth Israel Deaconess Medical Center from 1996 until 2009. Id. at 2; Resp. Ex. A at 1. In 2009, Dr. Vartanian moved to head the Weill Cornell Division of Neuroimmunology at Weill Cornell Medical College and New York Presbyterian Hospital in New York City. Resp. Ex. A at 1. "[His] research focuses on the relationship between innate immunity and [central nervous system] injury." Id. He has also "cared for people with autoimmune demyelinating diseases as [his] primary clinical and research efforts since completing residency." Id. at 2. He has authored or co-authored approximately 60 publications throughout his career. Resp. Ex. B at 11-24.

[44] Alexander D. Languir et al., An Epidemiological and Clinical Evaluation of Guillain-Barré Syndrome Reported in Association with the Administration of Swine Influenza Vaccines, 119 Am. J. Epidemiology 841 (1984).

[45] Dr. Vartanian submitted one expert report. Resp. Ex. A.

b. Diagnosis Opinion

Dr. Vartanian agreed that Petitioner had GBS. Resp. Ex. A at 14-15. He opined that "the diagnosis of GBS" in Petitioner "is not questioned." Id. at 18. Further, he opined the diagnosis of GBS was supported by Petitioner's EMG/NCS findings. Id. at 15. The study done on November 5, 2015 revealed "evidence of conduction block and reduced or absent F-waves which are classic findings in [GBS]." Id. While Petitioner's CSF was "essentially normal" showing only a "minimally elevated protein of 50," this alone would not support a diagnosis of GBS; however, Petitioner's GBS diagnosis was "supported by [his] EMG/NCS findings." Id.

c. Causation Opinion

i. Althen Prong One

Dr. Vartanian endorsed the mechanism of molecular mimicry to explain the pathogenesis of GBS in the context of infections, but not as a mechanism to explain GBS following flu or pneumococcal vaccinations. Resp. Ex. A at 10-14. He relied on epidemiology studies to support his opinion that there is no evidence that these vaccinations cause GBS. Id. at 14.

Dr. Vartanian opined there is an "[a]bsense of a causal relationship" between the flu vaccine and GBS. Resp. Ex. A at 13. Baxter et al.,[46] a large retrospective study, did not report an increased risk of GBS after vaccination of any kind. Id. at 14. Dr. Vartanian also cited Hall et al.,[47] another large study that failed to find a causal relationship between the flu vaccine and GBS. Id. Lastly, he cited the 2012 Institute of Medicine ("IOM") Report,[48] which concluded that "the weight of epidemiologic evidence does not support a causal link" between the flu vaccination and GBS. Id.

Specific to the pneumococcal vaccination and GBS, Dr. Vartanian opined "there are a handful of case reports which lack validity in attempting to establish a causal connection." Resp. Ex. A at 14. Dr. Vartanian did not cite or discuss any case reports for support.

Dr. Vartanian did not submit a report responding to Dr. Sheikh's expert report or opinions.

ii. Althen Prong Two

As described above, Dr. Vartanian agreed with the diagnosis of GBS based on the EMG/NCS findings. Resp. Ex. A at 14-15, 18. He noted that "[r]elevant anti ganglioside and

[46] This study was not filed.

[47] This study was not filed.

[48] While this report or section of the report was not filed, the undersigned is familiar with the 2012 IOM report.

sulfatide antibodies . . . were all negative." Id. at 15. He did not explain the significance of these findings in the context of an antecedent vaccination. See id.

Dr. Vartanian did not opine as to any alternative cause of Petitioner's GBS. See Resp. Ex. A.

iii. Althen Prong Three

Dr. Vartanian opined that "[t]he timing of vaccination" for either the pneumococcal or flu vaccinations "does not make logical sense" given the onset of Petitioner's symptoms. Resp. Ex. A at 18. Based on the medical records, Dr. Vartanian opined that the onset of Petitioner's symptoms occurred between October 1 and October 7, 2015. Id. at 16. The flu vaccine was administered on October 8, which was after the onset of symptoms. Id. Further, he opined the pneumococcal vaccine was administered on August 18, 2015 and that "timing is far too late being [six] weeks following vaccination." Id.

He explained that it generally takes four days for the adaptive immune system to respond to a pathogen, if the human host has previously encounter the pathogen. Resp. Ex. A at 15. The outside window of causation is six weeks following vaccination. Id. at 16.

3. Respondent's Expert, Thomas P. Leist, M.D., Ph.D.[49]

a. Background and Qualifications

Dr. Leist is a board-certified neurologist. Resp. Ex. C at 1. He received a Ph.D. in Biochemistry from the University of Zurich in Switzerland and an M.D. from the University of Miami in Florida. Resp. Ex. D at 1. Thereafter, he completed an internal medicine internship at the University of Miami and a neurology residency at Cornell Medical Center/Sloan Kettering Memorial Cancer Center in New York. Id. Dr. Leist is a neurology professor at Thomas Jefferson University, where he also directs the Division of Clinical Neuroimmunology. Id.; Resp. Ex. C at 1. Additionally, he holds multiple hospital and administrative appointments. Resp. Ex. D at 1. He is "regularly involved in the care of patients with neurological, neuroimmunological[,] and neurovirological conditions including multiple sclerosis, neuromyelitis optica spectrum disorder, [GBS], [CIDP] as well as infectious conditions of the central nervous system." Resp. Ex. C at 1. Dr. Leist has authored or co-authored various publications on immunology, neuroimmunology, and of the central nervous system. Id., Resp. Ex. B at 6-16.

b. Diagnosis Opinion

Unlike Dr. Vartanian, Respondent's second expert, Dr. Leist, questioned the diagnosis of GBS based on his interpretation and application of the Brighton criteria. Resp. Ex. C at 7. Regarding the first criterion, Dr. Leist opined Petitioner did not have flaccid weakness. Id.

[49] Dr. Leist submitted one expert report. Resp. Ex. C.

Second, he opined Petitioner had "full lower extremity strength" when he had "reduced or absent ankle reflexes," and thus, the second criterion was "at most only partially met." Id.

Next, Dr. Leist disagreed Petitioner had a monophasic course or that the time from onset to nadir was within 28 days. Resp. Ex. C at 7. He opined that onset occurred between October 1 and 5, and "new subjective symptoms [were reported] on November 7, 2015." Id. at 7-8. He explained that Dr. McGee did not document any progression of symptoms from the time of onset until October 12, or between October 12 and October 22, 2015. Id. at 7. Assuming Petitioner's condition plateaued on October 22, and he had new symptoms by November 7, 2015, Dr. Leist opined Petitioner's course progressed beyond 28 days and was not monophasic. Id. at 8. Thus, Petitioner did not have a typical clinical course of GBS. In support of this opinion, Dr. Leist cited Fokke et al., who reported that in GBS, the progressive phase lasted less than four weeks in most patients (97%). Id. at 13 (citing Resp. Ex. C, Tab 3 at 5).

Fourth, Dr. Leist opined that Petitioner's CSF protein level was on the lower limit of abnormal when tested after one dose of IVIG. Resp. Ex. C at 8. Dr. Leist suggested IVIG could have elevated Petitioner's protein level. Id. Alternatively, Dr. Leist explained the protein level may have been increased due to Petitioner's degenerative spine disease. Id. Regardless of the cause of the mildly elevated CSF protein level, Dr. Leist argued it was not high enough to support a diagnosis of GBS. Id. He concluded this criterion was "likely not met." Id.

Next, Dr. Leist asserted the NCS performed November 5, 2015 was of "low technical quality" which did "not allow conclusions" to be reached as to the diagnosis of GBS. Resp. Ex. C at 8. And lastly, Dr. Leist opined that Petitioner's longstanding degenerative lumbar spine disease was an alternative explanation for Petitioner's weakness. Id.

In summary, while Dr. Leist agreed Petitioner had "some symptoms of GBS," he opined that Petitioner did not meet "Level 4 of diagnostic certainty, the lowest level" of the Brighton criteria. Resp. Ex. C at 7.

c. Causation Opinion[50]

Assuming, however, that Petitioner's diagnosis was GBS, Dr. Leist opined there is no causal association between the Prevnar 13 vaccine and GBS, and Petitioner's GBS was not caused by his August 18, 2025 Prevnar 13 vaccine. Resp. Ex. C at 16.

[50] In his expert report, Dr. Leist responded to the opinions of Dr. Serota and did not submit an expert report or expert opinions in response to the expert report from Dr. Sheikh. Since the undersigned is not considering the opinions of Dr. Serota, Dr. Leist's opinions responsive to Dr. Serota's opinions are not discussed. Even if the undersigned had considered Dr. Leist's opinions related to Dr. Serota, it would not change the outcome reached herein.

i. Althen Prong One

Dr. Leist explained that "[i]f a component of a pathogen is thought to be able to cause GBS, then it [would] be expected that the whole pathogen would do so." Resp. Ex. C at 8. He asserted there is no literature to show that infection with the live bacteria *S. pneumoniae* causes GBS. Id. at 8-9. Since the pneumococcal vaccine is given to protect against *S. pneumoniae* infections, and these infections are not known to cause GBS, Dr. Leist opined that the vaccine does not cause GBS. Id.

According to Dr. Leist, a Medline search[51] only identified "simple case reports documenting a temporal relationship between pneumococcal infection and GBS," which he attributed to coincidence. Resp. Ex. C at 9. He also noted that the package insert for Prevnar 13 does not identify GBS as an adverse reaction associated with the vaccine. Id. at 12.

ii. Althen Prong Two

Regarding a logical sequence of cause and effect, Dr. Leist offered several opinions.[52] First, he questioned whether there was an alternative explanation for Petitioner's symptoms. Resp. Ex. C at 8. He opined that Petitioner had "longstanding low back issues" and mild weakness in the muscles of his lower legs due to degenerative lumbar spine disease. Id. Dr. Leist also asserted that Petitioner's GBS symptoms did not improve with IVIG treatment. Id. at 8, 15. Based on these observations, he questioned the diagnosis of GBS, as described above.

Next, Dr. Leist opined Petitioner had risk factors for peripheral neuropathy, including prediabetes and diabetes. Resp. Ex. C at 15. On October 14, 2015, Petitioner's HbA1c was 6.3%, and values 6.5% or greater are "consistent with diabetes." Id. Dr. Leist opined that Petitioner's consumption of "beer and carbohydrates" adversely affected his ability to control his glucose; he had "'associated symptoms' includ[ing] paraesthesias." Id. (citing Pet. Ex. 6 at 32, 41). Thus, Dr. Leist opined Petitioner had "longstanding impaired glucose control/prediabetes" and his symptoms reported to Dr. McGee on October 12, 2015 were due to "resulting length dependent neuropathy." Id.

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[51] "MEDLINE is the National Library of Medicine's (NLM) premier bibliographic database that contains more than 31 million references to journal articles in life sciences with a concentration on biomedicine. MEDLINE is a primary component of PubMed, a literature database developed and maintained by the NLM National Center for Biotechnology Information (NCBI)." MEDLINE Overview, Nat'l Libr. Med., https://www.nlm.nih.gov/medline/medline_overview.html (last visited June 12, 2025).

[52] In response to Dr. Serota's assertion that the flu vaccination aggravated Petitioner's condition, Dr. Leist disagreed, opining there was no evidence of significant aggravation. See Resp. Ex. C at 14-15. Since Dr. Sheikh did not opine that the flu vaccine caused a significant aggravation, the undersigned does not discuss Dr. Leist's opinions related to significant aggravation here. Further, significant aggravation was not identified by the parties as an issue in this matter. See Joint Submission.

In combination with prediabetes, Dr. Leist opined that Petitioner's elevated uric acid levels (hyperuricemia) combined to elevate Petitioner's risk of diabetic peripheral neuropathy. Resp. Ex. C at 15. For support, Dr. Leist cited Zhang et al.,[53] who found that elevated serum uric acid levels were "significantly associated with a higher risk of developing [diabetic peripheral neuropathy]" but this was in patients with type 2 diabetes. Resp. Ex. C, Tab 12 at 1. Although Petitioner's A1c level was "[c]onsistent with increased risk for diabetes (prediabetes)," Petitioner was never diagnosed with type 2 diabetes. Pet. Ex. 2 at 23-24.

Lastly, Dr. Leist noted that Dr. McGee documented concern that Petitioner's statin medication (atorvastatin) may have "contributed to his symptoms." Resp. Ex. C at 16. Dr. Leist explained that although a causal association has not been established, there have been post-marketing case reports of peripheral neuropathy associated with atorvastatin. Id. Kristensen et al., for example, found "[n]ew [statin] users had a slightly increased [diabetic polyneuropathy] risk during the first year [of use]." Resp. Ex. C, Tab 6 at 1.[54] However, Kristensen et al., like Zhang et al., examined the risk in patients diagnosed with type 2 diabetes, not prediabetics. Id. Therefore, it is not clear whether the study supports Dr. Leist's opinions.

Although Dr. Leist identified risk factors related to prediabetes, hyperuricemia, and use of statins, he did not opine that any of these factors individually or in combination, more likely than not caused Petitioner's GBS. See Resp. Ex. C.

iii. Althen Prong Three

Petitioner presented to Dr. McGee on October 12, 2015, complaining of a 10-day history of numbness and tingling in his hands and feet. Resp. Ex. C at 6. Based on these facts, Dr. Leist opined that onset of symptoms occurred "on or about October 3, 2015, or about 46 days" after Prevnar 13 vaccination. Id. He also opined that onset could have occurred within the range from October 1 to October 5, 2015, or 44 to 48 days after vaccination. Id. at 7, 13. Using these parameters, Dr. Leist opined that Petitioner's onset "occurred outside the hypothetical 42-day risk period" accepted as appropriate for GBS following vaccination. Id. at 13.

In support of the 42-day risk window, Dr. Leist cited data from Langmuir et al., also cited by Dr. Sheikh, observed in those vaccinated for the swine flu in 1976/1977. Resp. Ex. C at 13 (citing Resp. Ex. C, Tab 7). Although Dr. Leist noted that "it is not known what an appropriate timeframe between pneumococcal conjugate vaccine and GBS would be," he found the time interval of 42 days informative based on Langmuir et al. Id.

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[53] Wanli Zhang et al., Association of Elevated Serum Uric Acid with Nerve Conduction Function and Peripheral Neuropathy Stratified by Gender and Age in Type 2 Diabetes Patients, 12 Brain Scis. 1 (2022).

[54] Frederik P. Kristensen et al., Statin Therapy and Risk of Polyneuropathy in Type 2 Diabetes: A Danish Cohort Study, 43 Diabetes Care 2945 (2020).

V. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly, 592 F.3d at 1322 n.2. Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, a petitioner must prove 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)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 57 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed. Cir. 2020).

There are situations in which compelling testimony 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) ("[L]ike 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 v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 57 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 v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to §

13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received actually caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(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 a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is

by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

VI. ANALYSIS

A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury," determining facts relating to the claimed injury can be significant. Id. Here, the parties disagree as to the nature of Petitioner's peripheral neuropathy and whether it was GBS. Joint Submission at 1.

Based on the facts and evidence presented, the undersigned finds there is preponderant evidence that Petitioner's peripheral neuropathy was due to his GBS. This finding is based on the opinions of Petitioner's treating physicians and both parties' experts, Dr. Sheikh and Dr. Vartanian.

Petitioner received his Prevnar 13 vaccination on August 18, 2015. He had no prior history of neurological or demyelinating illnesses. On October 12, 2015, he saw his PCP, Dr. McGee, complaining of a tingling sensation and parasthesias that began 10 days before. Petitioner presented to the ED on November 3, 2015, with progressive numbness radiating upwards towards his arms and legs. He was diagnosed with peripheral neuropathy and referred to a neurologist. Petitioner saw neurologist Dr. Gee two days later, on November 5. At this point, Petitioner's hands were weaker and he had symmetric weakness of his feet, with only trace ankle reflexes. EMG/NCS performed that day showed significant sensory and motor nerve injury and conduction abnormalities consistent with AIDP/GBS. Dr. Gee's diagnosis was "concerning for [GBS]." Pet. Ex. 3 at 7.

On November 7, 2015, Petitioner was admitted to the hospital for treatment of GBS. His discharge diagnosis on November 12 was GBS. Thereafter, Petitioner saw both Dr. McGee (PCP) and Dr. Gee (neurologist), and both continued to note Petitioner's history and diagnosis of GBS. In 2018, Petitioner continued to have numbness and tingling in his hands and feet which were diagnosed as "residual" from GBS. Pet. Ex. 6 at 10-12. From the onset of his neuropathic symptoms in 2015, and throughout Petitioner's subsequent clinical course, his diagnosis remained GBS. After he was diagnosed with GBS, Petitioner's peripheral neuropathy was not attributed to any condition other than GBS.

In addition to the evidence in his medical records and the diagnosis of his treating physicians, both Dr. Sheikh and Dr. Vartanian attribute Petitioner's peripheral neuropathy to his GBS. Only Dr. Leist questioned other causes for Petitioner's peripheral neuropathy. These other causes included diabetes, hyperuricemia, and avorastatin. Only one of these causes, avorastin, was questioned by Petitioner's physicians, but after Petitioner's EMG/NCS showed AIDP/GBS, avorastatin was not mentioned again as a potential causal factor.

Regarding diabetes and hyperuricemia, based on the medical literature referenced by Dr. Leist, both of these risk factors presume a diagnosis of diabetes. However, Petitioner was not diagnosed diabetic. In October 2015, his HgA1c level was 6.3%, and therefore, he had an "increased risk for diabetes (prediabetes)," but he was not diabetic. Pet. Ex. 2 at 23-24. Since Petitioner was not diabetic, the risk factors attended to diabetics are not applicable. Even assuming Petitioner did have risk factors for peripheral neuropathy, these would not explain the specific findings found on nerve testing (EMG/NCS), since the abnormalities were described as consistent with AIDP/GBS, and not diabetic peripheral neuropathy. Lastly, Petitioner's treating physicians did not attribute his peripheral neuropathy to diabetes but instead to his GBS.

The Brighton Criteria also support the diagnosis of GBS. Dr. Sheikh disagrees with Dr. Leist's interpretation and application of the criteria as too rigid, asserting Dr. Leist fails to acknowledge the significant variability in patient presentations, especially related to limb weakness. The Fokke et al. study cited by Dr. Leist and Dr. Sheikh found that weakness in the limb muscles of GBS patients was "highly variable" and ranged from mild to complete paralysis. Resp. Ex. C, Tab 3 at 4 ("The severity of limb weakness at study entry and at nadir was highly variable. Weakness at nadir ranges from mild severity . . . to tetraparalytic . . . ."). Dr. Gee noted Petitioner had symmetric distal ankle dorsiflexion weakness as 4/5 on November 5, 2015. Dr. Sheikh opines this degree of weakness in ankle dorsiflexor muscles satisfies criterion one based on Fokke et al., and therefore, that criterion was met.

Criterion three, monophasic course and time between onset and nadir (12 hours to 28 days), was also questioned by Dr. Leist. In response, Dr. Sheikh opines that a monophasic course was "objectively confirmed" when follow-up EMG/NCS testing on January 20, 2015 showed improvement as compared to the first study. Pet. Ex. 66 at 6. He explains that nadir was reached by November 5, 2015, when Dr. Gee documented ankle dorsiflexion weakness and mild ataxia. And given onset was between October 1 and 5, 2015, nadir was reached in approximately four weeks.

Dr. Leist also questions Petitioner's CSF protein level of 50 (reference range 15-45). Although the level was higher than the range as defined by the laboratory that performed and reported the results, he argued Petitioner would not meet the reference range used in Fokke et al.. See Resp. Ex. C, Tab 3 at 5-6. Dr. Sheikh cited a study he participated in that defined albuminocytologic dissociation as a protein level greater than 45 in the absence of an elevated WBC count. And in this study, "patients with distal predominant weakness, as in [Petitioner's] case, were more likely to have lower CSF protein levels." Pet. Ex. 66 at 7 (citing Pet. Ex. 68 at 1). Dr. Sheikh concludes Petitioner's CSF was "mildly elevated." Id. He further opines "[albuminocytologic dissociation] is not found in 30% of GBS cases," and thus, its absence "does not exclude [a] GBS diagnosis." Id. Assuming Petitioner's elevated CSF of 50 is "accepted at face value," Petitioner meets the diagnostic certainly level 1, and if not, diagnostic certainty level 2 is met. Id. at 8.

Dr. Sheikh disagrees with Dr. Leist's assertion that Petitioner's first dose of IVIG artificially increased the level of his CSF protein. Although Dr. Sheikh acknowledges that IVIG can increase the protein level in the CSF, because only one dose of IVIG was administered

before Petitioner's CSF was tested and there was a short time interval between IVIG and the lumbar puncture, he opines IVIG was unlikely a "confounding factor." Pet. Ex. 66 at 7.

The last and seventh criterion is the absence of an alternative explanation for weakness. As described above, Dr. Leist suggests several alternative causes for Petitioner's weakness. However, Dr. Sheikh notes Petitioner underwent extensive blood and CSF testing, brain CT and MRI, and MRI of the cervical spine, and these studies did not reveal another cause.

In summary Dr. Sheikh concludes that Petitioner met diagnostic certainly level one of the Brighton criteria.

Respondent's expert, Dr. Vartanian, does not question the diagnosis of GBS. Instead of relying on the Brighton criteria, he opines the diagnosis is based on "clinical grounds with important ancillary evidence from [EMG/NCS] and CSF analysis." Resp. Ex. A at 14. Dr. Vartanian finds Petitioner's initial EMG/NCS showed "classic findings" for GBS and described the CSF protein level as "minimally elevated." Id. at 15. He explains that Petitioner's CSF results, "in the absence of the EMG/NCS findings[,] would not support [the] diagnosis of GBS." Id. However, given the EMG/NCS findings, Dr. Vartanian supports the diagnosis of GBS. Dr. Vartanian does not offer any alternative opinion as to the cause of Petitioner's peripheral neuropathy.

Therefore, for the above mentioned reasons, the undersigned finds there is preponderant evidence that Petitioner's peripheral neuropathy was due to his GBS.

B. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds Petitioner has provided, by preponderant evidence, a sound and reliable theory by which the Prevnar 13 vaccine can cause GBS, and therefore, Petitioner has satisfied the first Althen prong. There are three reasons for this finding. First, molecular

mimicry has long been invoked as a sound and reliable causal mechanism for GBS. Second, Dr. Sheikh posits two examples of how the Prevnar 13 vaccine can cause GBS via molecular mimicry and the foundational evidence supporting these examples is derived from peer-reviewed medical literature.[55] Third, Respondent did not offer any responsive expert opinion refuting Dr. Sheikh's theories setting forth specific examples of homology.

Molecular mimicry has been accepted as a sound and reliable theory in many demyelinating conditions, including GBS, in the Vaccine Program, forming the basis for petitioners to be entitled to compensation. See, e.g., Conte v. Sec'y of Health & Hum. Servs., No. 17-403V, 2020 WL 5743696, at *57 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program"); Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *8-9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting molecular mimicry "has been accepted in other Program cases as a reliable medical explanation for how various autoimmune conditions could develop after the receipt of different kinds of vaccinations"). Petitioners have also been found to be entitled to compensation for GBS caused by various vaccines. See, e.g., Salmins v. Sec'y of Health & Hum. Servs., No. 11-140V, 2014 WL 1569478, at *14 (Fed. Cl. Spec. Mstr. Mar. 31, 2014) (finding human papillomavirus vaccine can cause GBS); Peugh v. Sec'y of Health & Hum. Servs., No. 99-638V, 2007 WL 1531666, at *17 (Fed. Cl. Spec. Mstr. May 8, 2007) (finding hepatitis B vaccine can GBS); Whitener v. Sec'y of Health & Hum. Servs., No. 06-0477V, 2009 WL 3007380, at *20 (Fed. Cl. Spec. Mstr. Sept. 2, 2009) (finding meningococcal vaccine can cause GBS); Koller v. Sec'y of Health & Hum. Servs., No. 16-439V, 2021 WL 5027947, at *7-20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021) (finding Prevnar 13 can cause GBS); Mohamad v. Sec'y of Health & Hum. Servs., No. 16-1075V, 2022 WL 711604, at *9-18 (Fed. Cl. Spec. Mstr. Jan. 27, 2022) (finding tetanus-diphtheria-acellular pertussis vaccine can cause GBS); J.G. v. Sec'y of Health & Hum. Servs., No. 20-664V, 2023 WL 2752634, at *29-32 (Fed. Cl. Spec. Mstr. Feb. 13, 2023) (finding hepatitis A vaccine can cause GBS). And even in cases where there has been no evidence of homology, molecular mimicry has been accepted as a theory of causation. See, e.g., Salmins, 2014 WL 1569478, at *14.

Next, Dr. Sheikh's examples of homology supporting the theory of molecular mimicry are supported by peer-reviewed medical literature. He accurately cites the relevant literature and reaches appropriate inferences. He identifies the proteins and/or structures in the vaccine and the nerve myelin that are structurally similar, provides evidence of the significance of these mimics, identifies relevant antibodies cited in the literature, and provides relevant examples of animal studies. In summary, Dr. Sheikh identifies components of the vaccine that could initiate development of antibodies that could cross-react with structures within peripheral nerve myelin.

[55] Dr. Sheikh also offered opinions related to the alum adjuvant. The undersigned does not find that aspect of Dr. Sheikh's opinion to be well developed or persuasive. Thus, the undersigned's ruling is not based on this aspect of Dr. Sheikh's opinion. Respondent asserts that this aspect of Dr. Sheikh's opinion was borrowed from Dr. Steinman's opinion in Trollinger and other Prevnar 13/GBS cases. Resp. Brief Opposing Entitlement at 18 (citing Trollinger v. Sec'y of Health & Hum. Servs., No. 16-473V, 2023 WL 2521912 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), mot. for rev. denied, 167 Fed. Cl. 127). The undersigned has no knowledge of the expert reports filed in Trollinger and makes no finding with respect to this assertion.

He has identified components of the Prevnar 13 vaccine that could trigger a human antibody response.

Moreover, other examples of homology have been offered by Dr. Lawrence Steinman, who has testified frequently in these cases, and these theories have previously been accepted as sound and reliable in a number of other Prevnar 13 cases, decided by different special masters, including the undersigned. See, e.g., Simeneta, 2024 WL 4881411, at *30-33; Bartoszek v. Sec'y of Health & Hum. Servs., No. 17-1254V, 2024 WL 4263604, at *17-22 (Fed. Cl. Spec. Mstr. Aug. 27, 2024);[56] Byrd v. Sec'y of Health & Hum. Servs., No. 20-1476V, 2024 WL 2003061, at *21-26 (Fed. Cl. Spec. Mstr. July 8, 2024); Cooper v. Sec'y of Health & Hum. Servs., No. 18-1885V, 2024 WL 1522331, at *14-18 (Fed. Cl. Spec. Mstr. Mar. 12, 2024); Anderson ex rel. Meyer v. Sec'y of Health & Hum. Servs., No. 18-484V, 2024 WL 557052, at *30-32 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); Parker v. Sec'y of Health & Hum. Servs., No. 20-411V, 2023 WL 9261248, at *20-22 (Fed. Cl. Spec. Mstr. Dec. 20, 2023); Sprenger v. Sec'y of Health & Hum. Servs., No. 18-279V, 2023 WL 8543435, at *18-20 (Fed. Cl. Spec. Mstr. Nov. 14, 2023); Gross v. Sec'y of Health & Hum. Servs., No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); Maloney, 2022 WL 1074087; Pierson v. Sec'y of Health & Hum. Servs., No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); Koller, 2021 WL 5027947, at *18. While prior decisions are not binding on the undersigned, they can be considered by the undersigned in forming her opinions. See Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); Boatmon, 941 F.3d at 1358. The undersigned agrees with the reasoning offered by her colleagues in these other cases, and for many of the same reasons finds the Petitioner's theory here sound and reliable and proven by preponderant evidence.

The undersigned recognizes there is not uniformity between the special masters in decisions addressing the Prevnar 13 vaccine and GBS. In some of these cases, there are factual issues that affected the outcome. See, e.g., McConnell v. Sec'y of Health & Hum. Servs., No. 18-1051V, 2022 WL 4008238, at *5-11 (Fed. Cl. Spec. Mstr. Aug. 19, 2022) (noting disputes with diagnosis and onset, but ultimately finding Petitioner did not provide preponderant evidence in support of Althen prongs). And in others, the special master did not accept Petitioner's theory, finding Petitioner did not provide preponderant evidence in support of Althen prong one. See, e.g., Deshler v. Sec'y of Health & Hum. Servs., No. 16-1070V, 2020 WL 4593162, at *19-21 (Fed. Cl. Spec. Mstr. July 1, 2020); Trollinger, 2023 WL 2521912, at *27-30, mot. for rev. denied, 167 Fed. Cl. 127; Bielak v. Sec'y of Health & Hum. Servs., No. 18-761V, 2023 WL 35509, at *33-37 (Fed. Cl. Spec. Mstr. Jan. 3, 2023); Gamboa-Avila v. Sec'y of Health & Hum.

[56] In Bartoszek, Dr. Sheikh presented two of the same theories: "(1) a structural mimic between the carbohydrates in the Prevnar 13 vaccine and a major glycolipid in the myelin sheath, and (2) a sequential mimic between myelin protein P0 and the diphtheria toxoid protein conjugate portion of the Prevnar 13 vaccine (CRM197)." Bartoszek, 2024 WL 4263604, at *9. However, the special master did not address the specific molecular mimics proposed by Dr. Sheikh. Id. Because Dr. Steinman also provided an expert opinion in Bartoszek that the special master had previously accepted as sound and reliable in other cases, satisfying Althen prong one, the special master found it was not necessary to determine whether Dr. Sheikh's proposed mimics also satisfied Althen prong one. Id.

Servs., No. 18-925V, 2023 WL 6536207, at *26-32 (Fed. Cl. Spec. Mstr. Sept. 11, 2023), mot. for rev. denied, 170 Fed. Cl. 441 (2024), appeal filed, No. 2024-1765 (Fed. Cir. May 1, 2024); Morrison v. Sec'y of Health & Hum. Servs., No. 18-386V, 2024 WL 3738934, at *17-24 (Fed. Cl. Spec. Mstr. July 18, 2024). The undersigned acknowledges these cases but also notes that these decisions are not binding on the undersigned. Boatmon, 941 F.3d at 1358; Hanlon, 40 Fed. Cl. at 630.

Lastly, Respondent did not submit a responsive expert opinion to refute the opinions of Dr. Sheikh. Instead, Respondent, in his brief, argued Dr. Sheikh's opinions related to molecular mimicry and his posited examples of molecular mimicry were flawed and not supported by the medical literature. See Resp. Brief Opposing Entitlement at 24-28. Counsel's argument, however, is not a substitute for an expert report. And because Petitioner has offered preponderant evidence in support of his claim, including well-explained and sound and reliable theories from Dr. Sheikh, the undersigned finds Respondent's counsel's assertions alone are not sufficient to overcome the evidence presented by Dr. Sheikh. See, e.g., Barone, 2014 WL 6834557, at *9 (noting Respondent did not provide an expert report responsive to Petitioner and finding "that because Petitioner has offered adequate preponderant proof in support of her claim, including an adequate expert report, Respondent needed to do more than simply stand on her thinly-substantiated objections"); Goza v. Sec'y of Health & Hum. Servs., No. 07-290V, 2008 WL 6082761, at *4-5 (Fed. Cl. Spec. Mstr. Aug. 1, 2018) (finding petitioner submitted "sufficient [evidence] to support a finding of a causal connection," which was not rebutted by any submission from Respondent); see also Lankford v. Sec'y of Health & Hum. Servs., 37 Fed. Cl. 723, 726 (1996); Gerhardt v. Sec'y of Health & Hum. Servs., No. 09-180V, 2014 WL 4712690 (Fed. Cl. Spec. Mstr. Aug. 29, 2014) (granting motion for ruling on the record in favor of Petitioner where Respondent offered no competing expert opinion to rebut Petitioner's case).

For these reasons, the undersigned finds that Petitioner has proven by preponderant evidence a sound and reliable causal theory establishing that the Prevnar 13 vaccine can cause GBS, satisfying Althen prong one.

2. Althen Prong Two

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

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

There are three reasons why the undersigned finds preponderant evidence of a logical sequence of cause and effect establishing that the Prevnar 13 vaccination administered to

Petitioner was the cause of his GBS. First, Petitioner was appropriately diagnosed with GBS, and Petitioner has proffered a sound and reliable mechanism of vaccine causation.

Second, the undersigned finds that preponderant evidence does not support an alternative cause of Petitioner's GBS. Although Respondent's expert, Dr. Leist, questioned whether there was an alternative explanation for Petitioner's symptoms, including longstanding lower back issues, longstanding impaired glucose control/prediabetes, elevated uric acid levels (hyperuricemia), and use of statins, he did not opine that any of these factors individually or in combination, more likely than not caused Petitioner's GBS. Additionally, testing did not support Dr. Leist's arguments. And even if Petitioner did have risk factors, testing (EMG/NCS) showed abnormalities consistent with AIDP/GBS, not another type of peripheral neuropathy (e.g., diabetic).

The third reason for finding that Petitioner has proven prong two is based on the statements and opinions by Petitioner's treating physicians. Dr. Gee, Petitioner's neurologist, documented Petitioner's history of "a pneumonia shot about two months ago" during a visit on November 5, 2015. Pet. Ex. 3 at 7. At this visit, his assessment noted, "With the timing of symptoms, this is concerning for [GBS], possibly and controversially induced by the pneumococcal vaccine. The vaccine was given within one month of the onset of sensory impairment." Id. The ED physician, upon admission on November 7, 2015, documented the vaccination occurred "prior to the initial onset of his numbness." Pet. Ex. 4 at 89. Admitting physician, Dr. Bashtar noted Petitioner's "[r]ecent history of pneumonia vaccination prior to initiation of the symptoms" in her impression. Id. at 95. And during this hospitalization, Petitioner's treating physicians documented the vaccine as an allergy in his medical records. See, e.g., id. at 104, 108, 127 ("Allergy to pneumonia vaccine.")). Although the statements and opinions offered by Petitioner's treating physicians alone do not constitute preponderant evidence, when combined with the other evidence herein, they support a finding in favor of Petitioner.

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court").

In conclusion, the undersigned finds that Petitioner has proven by preponderant evidence a logical sequence of cause and effect establishing that the Prevnar 13 vaccination caused Petitioner's GBS. Thus, Petitioner has satisfied the second Althen prong.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

Petitioner received his Prevnar 13 vaccine on August 18, 2015. He presented to his PCP, Dr. McGee, on October 12, 2015 complaining of a "tingling sensation" which was "diagnosed 10 days ago." This is the first relevant record by a health care provider, and it places onset on or about October 1 or 2, 2015.

Petitioner returned to Dr. McGee again on October 22, still complaining of tingling. The neurological examination was unremarkable. Dr. McGee recommended B-12 supplements and to decrease alcohol intake. At that visit, Dr. McGee stated these symptoms were "diagnosed [three] weeks ago," which the undersigned interprets as onset three weeks prior. This is consistent with Dr. McGee's earlier record placing onset in early October 2015. Thus, there is consistency in two different records, placing onset about October 1 or 2.

Petitioner was seen in the ED November 3, 2015. Dr. Lampe recorded a history of bilateral numbness beginning three weeks ago. This record places onset on or about October 13. On November 5, 2015, Petitioner presented to Dr. Gee. Dr. Gee placed onset at four weeks before, on or about October 5. At that point, Petitioner had mildly diminished reflexes and mild ataxia. Dr. Gee's assessment was acute sensory neuropathy, and he considered the diagnosis of GBS. EMG/NCS showed findings consistent with GBS.

In summary, Petitioner reported his symptoms to several physicians who documented onset. While the records are not consistent as to a specific date, taken together, the history reported by Petitioner and recorded by his health care providers establish onset occurred in a range from October 1 to October 5, 2015. This is 44 to 48 days after Petitioner received the Prevnar 13 vaccination.

The undersigned finds that Petitioner's and his wife's affidavits are not necessarily inconsistent with the medical records. The language in Petitioner's affidavit suggests he has no clear memory when his symptoms began. Petitioner's wife remembers Petitioner shuffling his feet a week or so before Halloween. To the extent there are inconsistencies between the medical records and the affidavits, the undersigned finds the medical records to be more accurate, as they were recorded closer in time to the salient events.

The undersigned disagrees with Respondents' experts' opinions that onset is too far out to be associated with the pneumococcal vaccine. In the Vaccine Program, and in the

undersigned's experience, there are a number of GBS cases where onset occurs at six weeks, or 42 days. This timeframe is not too late for the mechanism proposed and well within eight weeks that the undersigned considers a more definitive end period for the vaccine to be causally related. See, e.g., Barone, 2014 WL 6834557, at *13 (noting two months is the longest reasonable timeframe for a flu/GBS injury); De La Cruz v. Sec'y of Health & Hum. Servs., No. 17-783V, 2018 WL 945834, at *1 (Fed. Cl. Spec. Mstr. Jan. 23, 2013) (finding an onset of GBS more than two months after flu vaccination not compensable); Aguayo v. Sec'y of Health & Hum. Servs., No. 12-563V, 2013 WL 441013, at *3 (Fed. Cl. Spec. Mstr. Jan. 15, 2013) (rejecting an onset of three-and-one-half months in a flu/GBS case); Corder v. Sec'y of Health & Hum. Servs., No. 08-228V, 2011 WL 2469736, at *27-29 (Fed. Cl. Spec. Mstr. May 31, 2011) (finding petitioner failed to prove that the flu vaccine can cause GBS four months after vaccination).

Here, an onset of 44 to 48 days is exceedingly close to the 42-day period discussed by Dr. Leist, and well within eight weeks. See Paluck v. Sec'y of Health & Hum. Servs., 786 F.3d 1373, 1383-84 (Fed. Cir. 2015) (finding the "special master [] erred in setting a hard and fast deadline . . . between vaccination and [] onset"); Spayde v. Sec'y of Health & Hum. Servs., No. 16-1499V, 2021 WL 686682, at *18-19 (Fed. Cl. Spec. Mstr. Jan. 27, 2021) (finding an onset of 60 days "exceedingly close" to the generally accepted timeframe of 56 days and appropriate given the mechanism of molecular mimicry). Therefore, it reasonable and appropriate to find that the onset of Petitioner's GBS is within the appropriate timeframe given the mechanism of molecular mimicry.

Therefore, undersigned finds that Petitioner has met her burden of proof as to Althen prong three.

VII. CONCLUSION

Based on the record, and for the reasons discussed above, the undersigned finds there is preponderant evidence to satisfy all three Althen prongs and to establish that Petitioner's Prevnar 13 vaccination caused him to develop GBS. Thus, the undersigned finds that Petitioner is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master