In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 19-1404V
Filed: August 19, 2025

RONALD DAVISON, *Executor of the* ESTATE OF GARY DAVISON,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Edward Kraus, Kraus Law Group, LLC, Chicago, IL, for petitioner.
Jamica Marie Littles, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

On September 12, 2019, petitioner, Ronald Davison, filed a petition on behalf of his deceased brother, Gary Davison, under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that he suffered Guillain-Barré Syndrome ("GBS") caused-in-fact by a pneumococcal conjugate (Prevnar 13) vaccination he received on August 10, 2017. (ECF No. 1.) Petitioner also alleged that the decedent suffered depression leading to suicide as a consequence of his GBS. (*Id.* at 6.) For the reasons set forth below, I conclude that petitioner is entitled to an award of compensation.

[1] Because this document contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted 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 document 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, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] Within this ruling, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury or death; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury.

In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B). In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In that context, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

GBS is a Table Injury if onset occurs 3-42 days following receipt of a flu vaccine. 42 C.F.R. § 100.3(a)(XIV)(D). However, GBS is not a Table Injury relative to the Prevnar 13 vaccine at issue in this case. 42 C.F.R. § 100.3(a)(XII). To succeed on a claim that petitioner's Prevnar 13 vaccine caused GBS, petitioner must satisfy the burden of proof for "causation-in-fact."

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" with the logical sequence being supported by "reputable medical or scientific explanation." *Althen*, 418 F.3d at

1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. *Id*.

A petitioner may not receive a Vaccine Program award based solely on his or her assertions, but may support the petition with either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. § 300aa-13(b)(1). A petitioner may rely upon circumstantial evidence. *Althen*, 418 F.3d at 1280. In that regard, the *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3. Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 300aa-13(b)(1)(A). The special master is required to consider the entirety of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

Petitioner filed medical records, medical literature, and a declaration (ECF Nos. 10-11, 15; Exs. 1-21) and then filed a statement of completion in April of 2020. (ECF No. 16.) In November of 2020, respondent filed his Rule 4(c) Report, recommending against compensation. (ECF No. 25.) Respondent contested petitioner's ability to meet the three-part *Althen* test with respect to the decedent's GBS, but did not address the death claim.[3] (*Id*.) Petitioner filed an expert report by neurologist, Nizar Souayah, M.D., the following May. (ECF No. 28; Ex. 22; *see also* ECF Nos. 37-40; Exs. 23-55 (medical literature).) Respondent filed responsive expert reports by neurologist Peter Donofrio, M.D., and immunologist Harry Schroeder, Jr., M.D., Ph.D., in October of 2021. (ECF Nos. 32-33; Exs. A-D.) Although Dr. Souayah opined that the decedent's suicide was causally related to his GBS and provided supporting literature, neither Dr. Donofrio nor Dr. Schroeder addressed the decedent's death in their responsive reports.

Thereafter, a status conference was held, during which it was noted that neither the decedent's diagnosis nor the timing of onset appeared to be at issue, leaving primarily petitioner's theory of causation at issue. (ECF No. 34, p. 1.) I also instructed, with an eye toward addressing potential barriers to settlement discussions, that "if respondent intends to raise any legal or factual defense to petitioner's death claim (beyond the question of whether the decedent's GBS was vaccine-caused), he should do so now and not wait for any potential damages phase of litigation." (*Id.* at 2.) During a follow up status conference, respondent initially advised that he "wishes to develop the factual record with respect to the contributing factors underlying Mr. Davison's suicide and whether the suicide is attributable to depression stemming from his GBS as petitioner alleges," which he was permitted to do. (ECF No. 41.) However, respondent subsequently filed a motion to amend the schedule, advising that

> Respondent no longer wishes to develop the legal or factual record with respect to petitioner's claim that the decedent's suicide is attributable to depression stemming from the alleged vaccine injury of Guillain-Barré Syndrome ("GBS"). However, Respondent will continue to defend this case, contesting that the Prevnar 13 vaccine caused the decedent's GBS.

(ECF No. 46.)

[3] In order to state a claim for a vaccine-related injury under the Vaccine Act, a vaccinee must have either: (i) suffered the residual effects or complications of such illness, disability, injury, or condition for more than 6 months after the administration of the vaccine, or (ii) died from the administration of the vaccine, or (iii) suffered such illness, disability, injury or condition from the vaccine which resulted in inpatient hospitalization and surgical intervention. § 300aa-11(c)(1)(D). In this case, the decedent's death by suicide occurred less than six months post-vaccination. However, respondent did not challenge petitioner's satisfaction of the severity requirement. Notably, though, I find that the decedent's GBS alone meets the statutory severity requirement on the basis that he experienced inpatient hospitalization and surgical intervention, namely, a tracheostomy on August 31, 2017 (*see* Ex. 1, p. 875). *Accord Brandt v. Sec'y of Health & Human Servs.*, No. 23-1549V, slip op. at 3 (Fed. Cl. Spec. Mstr. July 30, 2025) (finding a tracheostomy is a surgical intervention based on respondent's report). Thus, petitioner's claim for the decedent's GBS is not dependent on the validity of the separate death claim.

An entitlement hearing had been set to be held in February of 2023; however, after the undersigned issued a ruling on entitlement in another case alleging that the Prevnar vaccine had caused GBS, the parties sought to introduce new experts and the entitlement hearing was cancelled. (ECF No. 52.) Petitioner then filed an expert report by neuroimmunologist Lawrence Steinman, M.D., in January of 2023 (ECF No. 54; Ex. 56; *see also* ECF Nos. 59-62; Exs. 57-83 (medical literature)), and respondent filed a responsive report by bioinformatician Yang Zhang, Ph.D., in June of 2023 (ECF No. 57; Exs. E-F). The parties also exchanged supplemental reports by Drs. Steinman and Zhang between October of 2023 and March of 2024. (ECF Nos. 63, 67-69; Exs. 90-103 (Steinman) and ECF No. 66; Exs. G-H (Zhang).) Dr. Steinman and Dr. Zhang primarily addressed petitioner's theory of causation with respect to the decedent's GBS.

Thereafter, the parties requested that the case be resolved based on written submissions and without a hearing. (ECF No. 70.) Petitioner filed a motion for a ruling on the written record on May 31, 2024. (ECF No. 73.) The motion was fully briefed as of September 12, 2024. (ECF Nos. 75-76.)

In light of the above, I have determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve entitlement on the existing record. *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *see also Kreizenbeck ex rel. C.J.K. v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record"). Accordingly, this matter is now ripe for resolution.

III. Factual History

The decedent received the Prevnar 13 (pneumococcal) vaccination at issue in this case on August 10, 2017. (Ex. 2, pp. 120-21.) He also received a Zostavax (shingles) vaccine. (*Id*.) He was 71 years old at the time with, *inter alia*, a prior history of three prior cerebral vascular accidents, which left him with mild left-side weakness and an unsteady gate. (Ex. 1, p. 290; Ex. 2, pp. 119, 136.) About ten days following these vaccinations, the decedent presented to the emergency department on August 20, 2017, with a three-day history of progressive weakness, numbness, and tingling in all of his extremities. (Ex. 1, p. 264; Ex. 2, p. 110.) He was also experiencing leg pain and difficulty breathing. (Ex. 1, p. 264; Ex. 2, p. 110.) On exam, he had absent deep tendon reflexes in his lower extremities and reduced vibration perception in his feet. (Ex. 2, p. 113.) After a neurology consult, GBS was suspected, IVIG treatment was started, and the decedent was transferred to the ICU for further evaluation. (Ex. 1, pp. 392-93.) During the course of his hospitalization, the decedent's GBS diagnosis was confirmed following a finding of elevated protein in cerebral spinal fluid on lumbar puncture and a finding of severe motor polyneuropathy on EMG/NCS study. (Ex. 1, pp. 161, 322-23, 922.) This GBS diagnosis is not contested.

While in the ICU, the decedent was intubated and sedated with propofol due to his respiratory difficulty and risk of aspiration. (Ex. 1, p. 354.) Although petitioner had

initially consented to full treatment, once intubated he "report[ed] he does not want any more treatment, would like to die. Is refusing all treatment . . . ." (*Id*. at 353.) This was attributed at least in part to an infectious disease consultation, which initially indicated (ultimately incorrectly) that he had tested positive for both HIV and syphilis. (Ex. 1, pp. 358-64; Ex. 2, p. 13.) Shortly after this, the decedent refused a recommended lumbar puncture, as well as any further treatment. (Ex. 1, p. 354.) He was reportedly "in shock" and stated that "he did not want to live anymore." (*Id*.) It was felt that the decedent was experiencing "likely situational depression," and a follow up psychiatry evaluation was ordered to evaluate the decedent's decision making capacity and his depressive symptoms. (*Id.*; Ex. 2, p. 13.) It was concluded that the decedent lacked medical decision-making capacity due to the decedent being "cognitively impaired, oriented to person and time somewhat and not completely place." (Ex. 1, pp. 355-56.) However, because the decedent's intubation limited him to yes or no questions (nodding or shaking of the head), his mood and thought content could not be assessed, making it impossible to evaluate his depressive symptoms. (*Id*. at 354-55.)

Petitioner remained in the ICU for about a month for treatment of GBS, aspiration pneumonia, hemolysis, rhabdomyolysis, atrial fibrillation, and hypernatremia. (Ex. 1, p. 264.) After two five-day courses of IVIG, the decedent began to see some improvement, though he was still suffering from quadriplegia with areflexia primarily affecting the lower extremities. (*Id*. at 264.) He was eventually extubated on September 21, 2017. (*Id*.) Thereafter, his decision-making capacity was reevaluated by the psychiatry department. (*Id*. at 289-90.) He regained his decision-making capacity; however, he continued to report feeling "down," though he denied symptoms of depression, expressed a preference for resuscitative efforts, and reported that he had no thoughts of harm to himself. (*Id*. at 290-92.) By this time, he was being treated with paroxetine (an anti-depressant) for his mood, which had been started on September 8, 2017, and which continued to be among his active medications through December of 2017. (*Id*. at 25; Ex. 19, p. 10.)

The decedent was discharged from the hospital to inpatient rehabilitation on September 28, 2017, where he remained until being released home on November 30, 2017. (Ex. 1, pp. 264-65; Ex. 18, p. 24.) His rehabilitation intake diagnoses included GBS and "major depressive disorder." (Ex. 18, p. 27.) By the time of his discharge, the decedent had "done well" in rehabilitation and at discharge he had achieved sufficient independence to function at home, though he still required assistance and supervision with mobility. (*Id*. at 24.) He was noted to be "doing well" with his depression; however, depression remained among his discharge diagnoses. (*Id.*) He was hospitalized again from December 13, 2017, through December 22, 2017, for dizziness and difficulty breathing, likely either an exacerbation of COPD or pneumonia. (Ex. 19, pp. 14-16, 20, 28.)

The decedent expressed multiple times that he was concerned about the cost of his treatment and that he was concerned he did not have anyone to assist him.[4] (Ex. 1,

[4] The decedent lived with his brother, but suggested his brother was frequently out of town for work. (Ex. 19, p. 40.)

p. 265; Ex. 19, p. 43.) Shortly before his second hospital discharge, the decedent's social worker documented on December 19, 2017: "provided pt with financial assistances resources as he seemed depressed about his finances." (Ex. 19, p. 43.) The social worker noted that the decedent's financial constraints would be a "barrier" and that he would need a financial counselor once he received his bill. (*Id*.)

The petitioner, also the decedent's brother, recalled that

> [w]hen [the decedent] came home from the hospital on December 22, 2017, he had lost the progress he had made previously with his walking. He became very depressed because he was so weak and he did not think he would ever recover. He told me that he didn't think he would ever be well enough to care for himself or to leave the house on his own to visit friends or do any of the things he enjoyed before his GBS.

(Ex. 10, p. 3.) The decedent committed suicide on December 29, 2017, the day after completing his initial evaluation for at home physical therapy. (Ex. 3, pp. 46, 56-57; Ex. 16.)

IV. Summary of Expert Opinions

a. Petitioner's Expert, Nizar Souayah, M.D. [5]

Initially, petitioner filed a report by neurologist Nizar Souayah, M.D. (Ex. 22.) Following his review of the medical records, Dr. Souayah opined that the decedent suffered GBS, the onset of which occurred approximately ten days following his pneumococcal vaccination. (*Id*. at 6.) He explained that GBS is an autoimmune clinical syndrome affecting the peripheral nerves that is frequently triggered by an acute infectious process, with "[n]umerous" antecedent events recognized as triggers. (*Id*. at 8.) The most common form of GBS is acute inflammatory demyelinating polyneuropathy ("AIDP"), in which "invasion of the myelin sheath of the peripheral nerve by activated macrophages result[s] in damage to its intact myelin sheath, demyelination and secondary axonal loss." (*Id.* at 7-9.) Petitioner's condition is consistent with AIDP. (*Id*.)

[5] Dr. Souayah received his medical degree from Medical School of Tunis in Tunisia in 1990, before going on to complete a residency in primary care and family practice at Hospitals of the Medical School of Tunis in 1990; a residency in internal medicine at Hospitals of Medical School of Strasbourg, France, in 1997; a residency in internal medicine at University of Pennsylvania Health System, Presbyterian Medical Center, in 1999; and a residency in neurology at Temple University Hospital in Philadelphia, Pennsylvania, in 2002. (Ex. 104, pp. 1-2.) He is board certified in neurology with a subspecialty in neuromuscular medicine, and he maintains an active medical license in New Jersey. (*Id.* at 3-4.) He currently works as a professor of neurology and neurosciences, as well as an associate professor of pharmacology, physiology, and neurosciences, at Rutgers, The State University of New Jersey. (*Id.* at 2.) He has published 85 articles; 6 books, monographs, and chapters; and nearly 300 abstracts. (*Id.* at 23-47.)

The exact etiology of GBS is unknown, though it is considered autoimmune and post infectious and has been documented as following both upper respiratory and gastrointestinal infections. (Ex. 22, p. 8.)

> Six pathogens have been temporally associated with GBS in case-control studies: Camplyobacter jejuni [also referred to as *C. jejuni*], cytomegalovirus, hepatitis E virus, Mycoplasma pneumoniae, Epstein-Barr virus and Zika virus. Other pathogens are linked to GBS on the basis of evidence from case series or epidemiological studies, but their role in the pathogenesis of GBS is uncertain.

(*Id*.) Molecular mimicry, the most commonly proposed mechanism for autoimmune disease, has been demonstrated in the context of GBS following *C. jejuni*. (*Id*. at 9.) Although certain formulations of the flu vaccine have been epidemiologically linked to GBS, Dr. Souayah acknowledges that "[a]lthough several studies reported the occurrence of GBS after pneumococcal vaccination, the number of cases did not reach significance [] when compared to GBS in non-vaccinated patients." (*Id*. at 10-17.)

Noting that onset of the decedent's GBS occurred during the "peak" timeframe for post-vaccination GBS to occur (Ex. 22, p. 23), noting the decedent not to have had any relevant preceding respiratory or gastrointestinal illness (*Id*. at 8), and opining that the pneumococcal vaccine can result in molecular mimicry against myelin tissue notwithstanding the lack of associational data (*Id*. at 19), Dr. Souyah opines that the decedent's GBS was caused by his pneumococcal vaccination (*Id*. at 22-23).

Additionally, Dr. Souayah explains that GBS patients have a 2.6 to 4.8-fold increased risk for depression. (Ex. 22, p. 20 (citing Nian-Sheng Tzeng et al., *Risk of Psychiatric Disorders in Guillain-Barre Syndrome: A Nationwide, Population-Based, Cohort Study*, 381 J. NEUROLOGICAL SCIS. 88 (2017) (Ex. 47); Wafik Said Bahnasy et al., *Sleep and Psychiatric Abnormalities in Guillain Barré Syndrome*, 54 EGYPTIAN J. NEUROLOGY PSYCHIATRY & NEUROSURGERY 1 (2018) (Ex. 48)).) More severely disabled patients are more likely to develop psychological symptoms. (*Id*. (citing Bahnasy et al., *supra*, at Ex. 48; Christopher Hillyar & Anjan Nibber, *Psychiatric Sequelae of Guillain-Barré Syndrome: Towards a Multidisciplinary Team Approach*, 12 CUREUS e7051 (2020) (Ex. 49)).) Among severely disabled GBS patients, 67% have depression. (*Id*.) Erlnagsen et al. conducted a retrospective cohort study of over seven million people in Denmark to assess an association between neurologic disorders and higher rates of suicide. (*Id*. (citing Annette Erlangsen et al., *Association Between Neurological Disorders and Death by Suicide in Denmark*, 323 JAMA 444 (2020) (Ex. 50)).) Overall, suicide occurred more than twice as often among those with neurologic disorders (44 per 100,000 person-years versus 20.1 per 100,000 person-years among those without such a diagnosis). (*Id*.) The highest rate was between the first and third months after being diagnosed. (*Id*.) Overall, those diagnosed with neurologic disorders had an adjusted increased relative risk of suicide of 1.8 while GBS patients in particular had an adjusted increased relative risk of suicide of 2.2 (95% confidence interval from 1.2 to 4.1). (*Id.*) Dr. Souayah notes that even after being discharged home, the decedent still

had significant disability, with respiratory impairment, general deconditioning, bilateral partial foot drop, and the ability to walk or stand only with stand-by supervision. (*Id.* at 20-21.) Noting that the decedent had no prior history of depression or psychiatric issues, he opines that the decedent's December 29, 2017 suicide was the result of "severe disability and depression induced by GBS triggered by vaccination." (*Id*. at 21.)

b. Petitioner's Expert, Lawrence Steinman, M.D.[6]

Dr. Steinman filed three reports in this case. (Exs. 56, 90, 103.) Like Dr. Souayah, Dr. Steinman explains that molecular mimicry is a viable theory of causation for GBS. (Ex. 56, p. 5.) With regard to the Prevnar 13 vaccine in particular, he proposes two distinct molecular mimics. (*Id*. at 5-6.) First, Dr. Steinman explains that the Prevnar 13 vaccine contains phosphoglycerol molecules, which he opines have sufficient homology to cross-react with polar head groups within the lipids within myelin tissue. (*Id*. at 6-14.) Second, he explains that the *Streptococcus pneumoniae* ("*S. pneumonia*") strains included in the Prevnar 13 vaccine are conjugated to a diphtheria CRM197 protein. Using NIH "BLAST"[7] searching, Dr. Steinman proposes that CRM197 can be shown to be a molecular mimic to a molecule known as "Contactin-1," which he indicates is a known target of autoimmune attack in some cases of GBS. (*Id*. at 5, 14-26.) Because I have concluded that Dr. Steinman's theory based on his first proposed molecular mimic is sufficient to support petitioner's burden of proof under *Althen* prong one, the second theory will not be addressed further.[8]

Dr. Steinman explains that lipids constitute 70% of the myelin sheath. (Ex. 56, p. 6 (citing Peggy Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 6 SCI. TRANSLATIONAL MED. 137 (2012) (Ex. 64)).) Moreover, studies have shown that phospholipids within the myelin sheath are targeted by autoantibodies as part of the neuroinflammation associated with multiple sclerosis. (*Id*. (citing Jennifer Kanter et al., *Lipid Microarrays Identify Key Mediators of*

[6] Dr. Steinman received his medical degree from Harvard in 1973. (Ex. 59, p. 1.) He completed an internship and residency at Stanford University Hospital in 1973 and 1974, respectively, before going on to complete a neuroimmunology fellowship at the Weizmann Institute of Science in 1977. (*Id.*) He returned to Stanford University to complete an additional neurology residency in 1980. (*Id.*) Since then, Dr. Steinman has worked as a professor in the neurology department at Stanford University. (*Id.*) He is board certified in neurology. (Ex. 59, p. 2.) In his clinical capacity, Dr. Steinman has treated both adult and pediatric patients who suffered from various forms of autoimmune disease of the nervous system, including GBS. (Ex. 56, p. 1.) In his research capacity, he has published over 600 journal articles, many of which are in the area of molecular mimicry. (*Id.*; Ex. 59, pp. 5-52.) Relevant here, Dr. Steinman has also specifically published on the role of an immune response to lipids in the development of demyelinating disease. (Ex. 56, p. 4.)

[7] The Basic Local Alignment Search Tool (BLAST) "finds regions of local similarity between [nucleotide or protein] sequences" and "can be used to infer functional and evolutionary relationships between sequences as well as help identify members of gene families." *Basic Local Alignment Search Tool*, NAT'L LIBR. MED., https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited July 29, 2025).

[8] As noted in notes 12-13, *infra*, Dr. Steinman's CRM197 has been accepted in some instances and rejected in others.

Autoimmune Brain Inflammation, 12 NATURE MED. 138 (2006) (Ex. 63); Ho et al., *supra*, at Ex. 64.) Within these studies, it has been shown that it is the polar head group of these molecules that is targeted, including phosphatidylcholine and phosphatidylserine in particular. (*Id.* at 7 (citing Ho et al., *supra*, at Ex. 64).) While the Ho study pertained to multiple sclerosis, there is no debate in this case that autoimmune attack on the myelin sheath is relevant to the pathogenesis of GBS and a study by Nakos et al. demonstrated the presence of phospholipid antibodies within GBS patients, but not controls, including phosphatidylcholine and phosphatidylserine. (*Id*. at 6-7 (citing G. Nakos et al., *Anit-Phospholipid Antibodies in Serum from Patients with Guillain-Barré Syndrome*, 31 INTENSIVE CARE MED. 1401 (2005) (Ex. 68)).) Within the Nakos findings, phosphatidylcholine was among the main antigens. (*Id*.)

Separately, a study by Chang et al. demonstrates that phosphoglycerol linkages on the phosphate head group are "quite necessary" to the immunogenicity of the serotype 18C component of the Prevnar 13 vaccine. (Ex. 56, p. 7 (citing Janoi Chang et al., *Relevance of O-Acetyl and Phosphoglycerol Groups for the Antigenicity of* Streptococcus pneumonia *Serotype 18C Capsular Polysaccharide*, 30 VACCINE 7090 (2012) (Ex. 69)).) Citing the Prevnar 13 patent, Dr. Steinman indicates that phosphoglycerol is present in both the 18C and 23F serotypes within the vaccine. (*Id*. at 9 (citing U.S. Patent No. 9,492,559 B2 (issued Nov. 15, 2016) [hereinafter Prevnar 13 Patent] (Ex. 71)).) According to Dr. Steinman, a study by Bryson et al. shows in the context of the Pneumovax 23 vaccine (a different pneumococcal vaccine) that human antibodies target the 23F *S. pneumoniae* serotype via phosphoglycerol. (*Id*. at 10-11 (citing Steve Bryson et al., *Structures of Preferred Human IgV Genes-Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation*, 196 J. IMMUNOLOGY 4723 (2016) (Ex. 72)).) Although the same study has not been conducted relative to the Prevnar 13 vaccine, Dr. Steinman opines "[s]ince the 23F and 18C components of Prevnar 13 also contains the phosphoglycerol moiety . . . that is targeted by the antibodies generated by Pneumovax, it is very likely that the immune response to 23F and 18C components of Prevnar 13 vaccine also targets the phosphoglycerol moiety." (*Id*. at 13-14.)

In his second report, Dr. Steinman responded to Dr. Zhang's criticisms of both of his theories. (Ex. 90.) First, Dr. Steinman takes issues with what he sees as Dr. Zhang's broader suggestion that the adaptive immune system "recognize the entire protein or the entire lipid." (*Id*. at 1-2.) He characterizes the idea that the immune system detects fragments of proteins and lipids as "sound and reliable 'textbook principles.'" (*Id*. at 1.) Regarding the proposed molecular mimic based on phosphoglycerol, Dr. Steinman indicates that Dr. Zhang's bare assertion that the degree of structural similarity is inadequate is directly refuted by the Ho et al. study, which found that the phosphatidylcholine and phosphatidylserine were targeted in human myelin. (*Id*. at 2 (citing Ho et al., *supra*, at Ex. 64).) To the extent Dr. Zhang suggested that the phospholipid molecules found in patients with GBS are not necessarily pathologic, Dr. Steinman stresses that the Ho, Nakos, Chang, and Bryson studies should be considered in concert. (*Id*. at 6-9.) He suggests that Dr. Zhang's illustration within his report (Figure one, which is depicted below) is not informative because the

literature cited in Dr. Steinman's reports shows where the relevant binding occurs. (*Id*. 10.) Dr. Steinman charges that Dr. Zhang is wrong to discuss protein structure without discussing immunochemistry, because "he is not addressing how the immune system sees small domains of the antigens discussed in Petitioner's theory." (*Id*. at 30.)

Dr. Steinman's third report primarily addresses Dr. Zhang's criticisms of the CRM197 theory, which this decision does not reach. (Ex. 103.)

c. Respondent's Expert, Peter Donofrio, M.D.[9]

Dr. Donofrio agrees on respondent's behalf that the decedent suffered GBS beginning about ten days after his pneumococcal vaccine, but disagrees that the vaccine can be causally implicated. (Ex. A, pp. 1, 5.) Dr. Donofrio's report is limited to discussing a number of publications which he indicates show that the pneumococcal vaccine has not been associated with GBS. (*Id*. at 5-7.) He does not otherwise raise any issue with respect to the decedent's own clinical history. He concludes:

> The clinical and electrodiagnostic presentation of the petitioner and his response to therapy supports the diagnosis of GBS. Review of the literature does not confirm an association between the [Prevnar] 13 vaccination and GBS. Epidemiologic data does not exist on the development of GBS after receiving the pneumococcal vaccine. In the petitioner's case, GBS more likely occurred by chance. For this reason, more likely than not, it is my opinion that GBS in this petitioner is not related to the [Prevnar] 13 vaccination.

(*Id*. at 7.) Dr. Donofrio did not respond to Dr. Souayah's opinion that the decedent's suicide was a consequence of his GBS or otherwise discuss Dr. Souayah's observation that GBS is a cause of depression.

[9] Dr. Donofrio received his medical degree from Ohio State University School of Medicine in 1975, before going on to complete an internal medicine residency at Good Samaritan Hospital in Cincinnati, Ohio, in 1978; a neurology residency at the University of Michigan Medical Center in Ann Arbor, Michigan, in 1981; and a neuromuscular fellowship at the University of Michigan in Ann Arbor, Michigan, in 1982. (Ex. B, pp. 1-2.) From there, he worked as a professor of neurology. (*Id.* at 2-3.) He maintains positions as a Professor Emeritus of Neurology, as well as Chief of the Neuromuscular Section and Director of the MDA and ALS Clinics, at Vanderbilt University Medical Center. (Ex. A, p. 1.) He is board certified in neurology, internal medicine, electrodiagnostic medicine, and neuromuscular disorders. (Ex. B, p. 2; Ex. A, p. 1.) He has authored over 100 journal articles, a textbook, and several book chapters and abstracts, among other publications, that touched on GBS, CIDP, and other neuropathies. (Ex. B, pp. 12-62; Ex. A, p. 1.)

d. Respondent's Expert, Harry Schroeder, Jr., M.D., Ph.D.[10]

Dr. Schroeder cautions that:

> It is important to remember that while molecular mimicry can elicit autoimmune reactions in the host, these reactions are not random. GBS is a very specific disorder where there is a defined attack by the immune system on a specific set of antigens in neural tissue, which then yields a characteristic phenotype. Invocation of molecular mimicry requires that the offending foreign antigen contain a shape or a structure that can mimic that of a host self-antigen. As noted above (Ex11), the bacteria *Campylobacter jejuni* appear to include structures that can mimic the structures of gangliosides found on neural tissues. These findings have provided a causal link to GBS, a specific autoimmune disorder. We have an etiology as well as an epidemiologic association. Conversely, it then follows that if other microorganisms or vaccines both lack an epidemiologic association and also do not express molecules that can mimic gangliosides and elicit anti-ganglioside reactions, then [it] is highly unlikely that they will be associated with GBS. [Prevnar 13] has not been shown to contain molecules with structural similarities to the gangliosides associated with GBS.

(Ex. C, p. 7 (emphasis omitted).)

With regard to petitioner's case, Dr. Schroeder agrees that the decedent was appropriately diagnosed with GBS following his pneumococcal vaccination; however, like Dr. Donofrio, he does not agree that the condition can be attributed to the vaccination. (Ex. C, p. 9.) He cites three specific reasons for this conclusion. First, the Prevnar vaccine does not contain any ganglioside-like structures and is therefore unlikely to elicit the type of antiganglioside autoantibodies implicated in GBS, as has been evidenced in the case of *C. jejuni*. (*Id*.) Second, the decedent's medical work up, though sufficient to arrive at the GBS diagnosis, was not sufficient to rule out other possible causes of GBS, including prior infections, which could have been either unreported or subclinical. (*Id*. at 9-10.) And, third, the only evidence linking the vaccination to the decedent's GBS is the temporal proximity; however, the medical literature does not support the idea that this proximity is causally meaningful given that

[10] Dr. Schroeder received his medical degree and Ph.D. in Cell Biology from Baylor College of Medicine in Houton, Texas, in 1981 and 1979, respectively. (Ex. D, p. 1.) He went on to complete two genetics fellowships, as well as an internship and residency in internal medicine. (*Id.* at 2.) From there, Dr. Shroeder worked as a research associate at Howard Hughes Medical Institute at the University of Washington, before transitioning to the role of professor at the University of Alabama at Birmingham. (*Id.* at 2-4.) He is also the Director of the Program in Immunology and Training Program in Immunologic Disease and Basic Immunology at the University of Alabama. (*Id.* at 4; Ex. C, p. 1.) Dr. Shroeder is board certified in internal medicine and clinical genetics, and he maintains an active medical license in Texas and Alabama. (Ex. D, p. 4.) He has authored nearly 100 articles, 30 reviews and editorials, 28 book chapters, and 4 books, among other publications. (*Id.* at 24-36.)

no associational relationship has been detected. (*Id*. at 10.) Like Dr. Donofrio, Dr. Schroeder opines that the fact that the decedent's GBS arose post-vaccination is "due to chance alone." (*Id*.)

Dr. Schroeder also did not respond to Dr. Souayah's opinion that the decedent's suicide was a consequence of his GBS or otherwise discuss Dr. Souayah's observation that GBS is a cause of depression.

e. Respondent's Expert, Yang Zhang, Ph.D.[11]

Significant portions of Dr. Zang's reports are dedicated to seeking to refute Dr. Steinman's proposed molecular mimic between CRM197 and Contactin-1. (Exs. E, G.) Indeed, Dr. Zhang's supplemental report is dedicated exclusively to seeking to rebut Dr. Steinman's CRM197-based theory based primarily on the distinction between linear and conformational epitopes. (Ex. G.) However, as noted above, this ruling will not reach that theory of causation. With regard to Dr. Steinman's proposed phosphoglycerol theory, Dr. Zhang raises two objections: (1) he opines that, although phospholipid molecules are found in patients with GBS, "there is no evidence establishing that these molecules are causative agents of GBS"; and (2) comparison of the proposed mimic using "stringent bioinformatics criteria," shows no structural similarity. (Ex. E, pp. 12-13.) Ultimately, Dr. Zhang opines that Dr. Steinman's opinion includes a "critical logic chain flaw" and "lacks direct evidence" to support his conclusion. (Ex. G, p. 6.)

Dr. Zhang opines that Dr. Steinman's theory is logically flawed because the fact that phospholipid molecules are observed in GBS patients "does not necessarily mean" that they cause disease. (Ex. E, p. 3.) However, this is the full extent of his analysis on that point. He does not specifically discuss any of Dr. Steinman's more specific observations regarding the potential pathogenicity of phospholipid antibodies in GBS or any of the literature he relied upon. In particular, Dr. Zhang includes no mention of the study by Ho et al., which Dr. Steinman relied upon as providing a basis for revisiting the pathogenicity of these antibodies.

[11] Dr. Zhang received his Ph.D. in physics from the Institute of Particle Physics at Central China Normal University in Wuhan, China, in 1996. (Ex. H, p. 1.) From there, Dr. Zhang completed a fellowship in the Physics Department at Free University Berlin in Berlin, Germany. (*Id.*) In 1999, he accepted a position as a postdoctoral associate at the Institute of Theoretical Physics, Chinese Academy of Sciences, in Beijing, China. (*Id.*) The following year, he moved to the United States and accepted a position as a postdoctoral associate at Donald Danforth Plant Science Center in St. Louis, Missouri. (*Id.*) From there, Dr. Zhang worked as a research associate at Center of Excellence in Bioinformatics, University at Buffalo, in Buffalo, New York. (*Id.*) He accepted a position as an assistant professor in the Department of Molecular Biosciences at the University of Kansas in Lawrence, Kansas, in 2005. (*Id.*) In 2009, Dr. Zhang transitioned to associate professor in the Department of Computational Medicine and Bioinformatics at the University of Michigan in Ann Arbor, Michigan. (*Id.*) He remained at the University of Michigan in Ann Arbor for several years and was eventually promoted to professor in the Department of Computational Medicine and Bioinformatics, the Department of Biological Chemistry, and the Department of Macromolecular Sciences and Engineering. (*Id.*; Ex. E, p. 1.) In 2022, Dr. Zhang accepted a position as professor in the Departments of Computer Sciences and Biochemistry at the National University of Singapore. (*Id.*) He has authored over 250 peer-reviewed scientific publications. (Ex. E, p. 1; Ex. H, pp. 14-25.)

Regarding the structural comparison, Dr. Zhang explains that "[t]he biological function of small molecules is closely linked to their structure," such that "[e]ven slight differences can lead to entirely different biological functions." (Ex. E, p. 3.) He indicates that the two phospholipid molecules present in the Prevnar 13 vaccine (in serotypes 18C and 23F) "differ in global structure and molecular formula" from the four major phospholipid antigens observed in GBS patients. (*Id.* at 3-4.) Thus, there is not "sufficient 'mimicry' to classify the phospholipid molecules in the Prevnar 13 vaccine as antigens for GBS." (*Id.* at 4.) He provided the following figure:

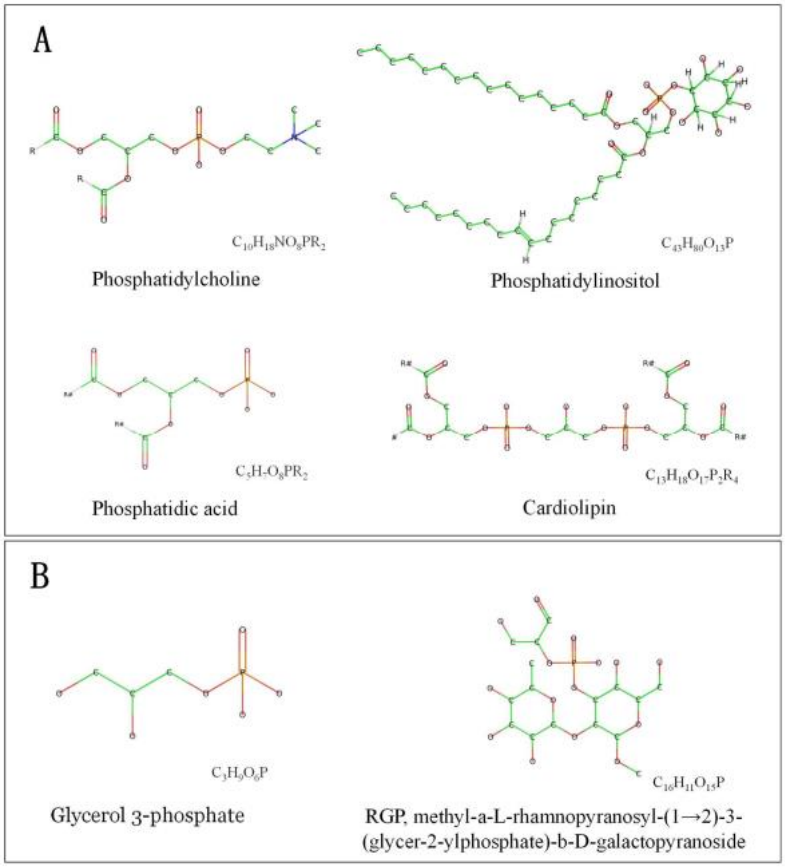

Figure 1. **(A)** 2D structures of the four major phospholipid molecules present in GBS: Phosphatidylinositol, Cardiolipin, Phosphatidylcholine, and Phosphatidic acid. **(B)** 2D structures of the two phospholipid molecules found in Prevnar 13 vaccine.

(*Id*.) Dr. Zhang's discussion of Dr. Steinman's phosphoglycerol theory does not include any citation to literature and does not explain how the above figure was generated. Although Dr. Steinman provided significant supplemental information seeking to rebut Dr. Zhang's conclusion that there is insufficient mimicry, Dr. Zhang's subsequent supplemental report did not address that discussion. (*Compare* Ex. 90, *with* Ex. G.)

V. Discussion

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," showing that the subject vaccine can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004)). Such a theory need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *See Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [the proposed causal] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Boatmon*, 941 F.3d at 1359 (citation omitted) (first quoting *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010); then quoting *Knudsen*, 35 F.3d at 548-49).

As with many cases in the Program, petitioner's theory involves molecular mimicry. Molecular mimicry is a concept whereby a susceptible host encounters a foreign antigen that has sufficient similarity ("homology") with components of host tissue such that the immune system "cross-reacts," producing antibodies that attack the host tissue instead of the foreign antigen to ultimately cause disease or injury. (Robert S. Fujinami et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 CLINICAL MICROBIOLOGY REVS. 80 (2006) (Ex. 27); Lawrence Steinman, *Autoimmune Disease: Misguided Assaults on the Self Produce Multiple Sclerosis, Juvenile Diabetes and Other Chronic Illnesses. Promising Therapies Are Emerging*, 269 SCI. AM. 106 (1993) (Ex. 66).) Molecular mimicry "is a generally accepted scientific principle, [but] mere invocation of the scientific term does not carry a petitioner's burden in a Program case." *Deshler v. Sec'y of Health & Human Servs*., No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec'y of Health & Human Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019)). Moreover, "the finding of sequence homology does not necessarily mean the similarity has significance to the immune system." *Tullio v. Sec'y of Health & Human Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *aff'd*, 149 Fed. Cl. 448 (2020); *see also Caredio ex rel. D.C. v. Sec'y of Health & Human Servs.*, No. 17-0079V, 2021 WL 4100294, at *31 (Fed. Cl. Spec. Mstr. July 30, 2021) ("[D]emonstration of homology alone is not enough to establish a preponderant causation theory." (emphasis omitted) (citing *Schultz v. Sec'y of Health & Human Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n.24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020))), *mot. for rev. denied*, No. 17-79V, 2021 WL 6058835 (Fed. Cl. Dec. 3, 2021).

As noted above, however, petitioners in this Program are not required to establish scientific certainty. *See, e.g.*, *Gross v. Sec'y of Health & Human Servs.*, No. 17-1075V, 2022 WL 9669651, at *36 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (finding that criteria, including supportive epidemiology, identification of antibodies directed against human antigens, identification of the mimics of the target antigen, and reproduction in an animal model, is tantamount to "require[ing] scientific certainty, which is a bar too high"). With regard to the application of molecular mimicry, prior cases have expressed that "[t]he line must be drawn somewhere between speculation and certainty." *Brayboy v. Sec'y of Health & Human Servs.*, No. 15-183V, 2021 WL 4453146, at *19 (Fed. Cl. Spec. Mstr. Aug. 30, 2021). Thus, for example, in *Brayboy*, an omnibus proceeding addressing autoimmune premature ovarian insufficiency, the special master found it sufficient that the petitioners "identified cross-reaction between components of the vaccine and proteins in the body that are directly responsible for the health and productivity of the organ at issue" and further expressed that requiring additional steps, or insisting on direct, testable evidence would impermissibly heighten petitioner's burden of proof. *Id.*

GBS is an acute inflammatory polyneuropathy affecting the peripheral nerves and, in the case of AIDP, most notably affecting myelin tissue. (*Guillain-Barré Syndrome Fact Sheet*, NAT'L INST. NEUROLOGICAL DISORDERS & STROKE (last updated Aug. 26, 2014) (Ex. 65).) While the condition is believed to be of an autoimmune etiology, the pathogenesis of GBS is incompletely understood. (Ex. 22, p. 9; *see also* Nakos et al., *supra*, at Ex. 68.) Although there is no established association between *S. pneumoniae* infection and GBS (Ex. C, p. 7), it is generally accepted that a number of different infectious antigens can cause GBS, including unspecified respiratory infections (J.B. Winer, *An Update in Guillain-Barré Syndrome*, AUTOIMMUNE DISEASES, Jan. 2014, at 1 (Ex. 23); Baxter et al., *supra*, at Ex. 40, pp. 1, 4). This includes both viral and bacterial infections. (Ex. 22, p. 9; Winer, *supra*, at Ex. 23, p. 2; Baxter et al., *supra*, at Ex. 40, p. 1.) Thus, there is little question that multiple antigens are implicated as causes of GBS. Additionally, for at least one of these antigens, *C. jejuni*, there is sufficient proof to conclude that molecular mimicry is the mechanism of causation leading to GBS, albeit resulting primarily in the axonal subtype of GBS and involving a molecular mimic not at issue here. (Ex. 22, p. 9; Winer, *supra*, at Ex. 23, p. 3.) At least some formulations of the flu vaccine have been identified as a cause of GBS, suggesting that antigenic triggers for GBS are not limited to active infections. (Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979) (Ex. 29).) Petitioner's experts opine that this understanding of GBS provides some basic support for the opinion that the Prevnar 13 vaccine can cause GBS. (Ex. 22, pp. 8-10; Ex. 56, pp. 5, 26.)

In some prior cases, this background information has partly informed the special masters' analysis of a petitioner's theory of causation with respect to GBS. *See, e.g.*, *J.G. v. Sec'y of Health & Human Servs.*, No. 20-664V, 2023 WL 2752634, at *30 (Fed. Cl. Spec. Mstr. Feb. 13, 2023) (observing that "[t]he experts do not dispute the theory of molecular mimicry, or that it is a sound and reliable theory generally as it relates to

GBS" and that "[m]olecular mimicry has been accepted as a sound and reliable theory in many Vaccine Program cases dealing with demyelinating conditions, including GBS"); *Osso v. Sec'y of Health & Human Servs.*, No. 18-575V, 2023 WL 5016473, at *21 (Fed. Cl. Spec. Mstr. July 13, 2023) (molecular mimicry accepted as a "sound and reliable theory"); *Harris v. Sec'y of Health & Human Servs.*, No. 18-944V, 2023 WL 2583393, at *22 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (finding that "the fact that GBS is well accepted as an autoimmune condition with a wide variety of suspected antigenic triggers, inclusive of antigens from both infection and vaccination, provides meaningful evidence supporting petitioner's burden of proof with respect to *Althen* prong one"). *But see Trollinger v. Sec'y of Health & Human Servs.*, No. 16-473V, 2023 WL 2521912, at *30 (Fed. Cl. Spec. Mstr. Feb. 17, 2023) (finding that "Dr. Steinman's theory had a one-size-fits-all quality, in which he strained to shoehorn the science behind the flu-GBS association into the context of the pneumococcal vaccine" and further noting that, "[i]f this were sufficient to establish that this particular vaccine 'can cause' GBS, it is hard to imagine the theory not also applying to *each and every one* of the sixteen Program-covered vaccines/vaccine antigenic components"), *mot. for rev. denied*, 167 Fed. Cl. 127 (2023). Although only the flu vaccine is presumed to be a cause of GBS in this Program (42 C.F.R. § 100.3(a)), petitioners have been found entitled to compensation in at least isolated instances for GBS caused by many other vaccines. This includes vaccines that target both viruses and bacteria. *See, e.g.*, *Salmins v. Sec'y of Health & Human Servs.*, No. 11-140V, 2014 WL 1569478, at *14 (Fed. Cl. Spec. Mstr. Mar. 31, 2014) (finding that HPV vaccine "can cause" GBS); *Peugh v. Sec'y of Health & Human Servs.*, No. 99-638V, 2007 WL 1531666, at *17 (Fed. Cl. Spec. Mstr. May 8, 2007) (finding as part of an omnibus proceeding that hepatitis B vaccine can GBS); *Whitener v. Sec'y of Health & Human Servs.*, No. 06-0477V, 2009 WL 3007380, at *20 (Fed. Cl. Spec. Mstr. Sept. 2, 2009) (finding that meningococcal vaccine can cause GBS); *Koller v. Sec'y of Health & Human Servs.*, No. 16-439V, 2021 WL 5027947, at *7-20 (Fed. Cl. Spec. Mstr. Oct. 8, 2021) (finding that pneumococcal conjugate vaccine, Prevnar 13, can cause GBS); *Mohamad v. Sec'y of Health & Human Servs*., No. 16-1075V, 2022 WL 711604, at *9-18 (Fed. Cl. Spec. Mstr. Jan. 27, 2022) (finding that Tdap vaccine can cause GBS); *J.G.*, 2023 WL 2752634, at *29-32 (finding that hepatitis A vaccine can cause GBS). In fact, given the nature of the condition, molecular mimicry has been accepted as a theory of causation for GBS even in the absence of *any* demonstration of homology and cross-reaction. *Salmins*, 2014 WL 1569478, at *14.

Within that context, Dr. Steinman opines that the Prevnar 13 vaccine can be considered among the causes of GBS, presenting several pieces of medical literature to demonstrate that (1) the Prevnar 13 vaccine contains phosphoglycerol groups that are necessary to the vaccine's immunogenicity (Ex. 56, pp. 7-12 (citing Chang et al., *supra*, at Ex. 69; Prevnar 13 Patent, *supra*, at Ex. 71; Bryson et al., *supra*, at Ex. 72)); (2) the phosphate portion of the phospholipid molecule has immune reactivity in myelin tissue, albeit demonstrated in the context of a different demyelinating condition (multiple sclerosis) (*Id.* at 6 (citing Ho et al., *supra*, at Ex. 64)); (3) GBS patients develop anti-phospholipid antibodies (*Id.* at 7 (citing Nakos et al., *supra*, at Ex. 68)), and (4) these antibodies are cross-reactive with phospholipids present in myelin tissue (*Id.* at 6 (citing Ho et al., *supra*, at Ex. 64)).

Dr. Steinman's theory does not involve the antiganglioside antibodies that are most commonly associated with GBS. However, literature filed in this case confirms that this is not very significant. (Winer, *supra*, at Ex. 23, p. 2 (noting that, while the association between antiganglioside antibodies and GBS is "strong" with respect to MFS and AMAN, its "only rarely associated" with AIDP); Tony Ho & John Griffin, *Guillain-Barre Syndrome*, 12 CURRENT OP. NEUROLOGY 389 (1999) (Ex. 25, p. 4) ("In AIDP, the nature of the target remains unresolved. The possibility of a glycolipid antigen remains, but no candidate glycolipid to date has had high degrees of sensitivity or specificity.").) We do not actually know the full scope of the antibodies that may be implicated in the pathology of GBS, leaving little reason to doubt Dr. Steinman's theory on that basis. (*See, e.g.*, Winer, *supra*, at Ex. 23, p. 3 ("Although antiganglioside antibodies are the most commonly reported antibody in GBS there are other reports of antibodies that might be pathogenic in a small number of patients."); *see also Gross*, 2022 WL 9669651, at *36 (indicating that "the literature filed by the parties does not support the notion that gangliosides are the only player in the game of molecular mimicry").) Afterall, the "S" in GBS stands for "syndrome" and "GBS variants are generally believed to have a multitude of both clinical presentations and causes," *McGill v. Sec'y of Health & Human Servs.*, No. 15-1485V, 2023 WL 3813524, at *27 n.16 (Fed. Cl. Spec. Mstr. May 11, 2023), raising the question of whether a single causal theory can explain every instance of GBS, post-vaccination or otherwise. (*See* Anil K. Jasti et al., *Guillain-Barré Syndrome: Causes, Immunopathogenic Mechanisms and Treatment*, 12 EXPERT REV. CLINICAL IMMUNOLOGY 1175 (2016) (Ex. C, Tab 1, p. 1) (expert commentary stating that "we are convinced that the syndrome results from a permissive genetic background on which environmental factors, including infections, vaccination and the influence of aging, lead to disease").)

I have previously concluded that Dr. Steinman's phosphoglycerol theory is sound and reliable, and preponderantly supports a legally probable, though not scientifically certain, theory of causation sufficient to satisfy petitioner's burden of proof under *Althen* prong one, based on the same medical literature cited in this case. *Pierson v. Sec'y of Health & Human Servs.*, No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Bartoszek v. Sec'y of Health & Human Servs.*, No. 17-1254V, 2024 WL 4263604, at *17-22 (Fed. Cl. Spec. Mstr. Aug. 27, 2024); *Datte v. Sec'y of Health & Human Servs*., No. 18-2V, 2025 WL 1565894, at *14-19 (Fed. Cl. Spec. Mstr. May 9, 2025); *see also Cooper v. Sec'y of Health & Human Servs.*, No. 18-1885V, 2024 WL 1522331, at *13-18 (Fed. Cl. Spec. Mstr. Mar. 12, 2024). I again reach the same conclusion based on the evidence of record in this case. Additionally, other special masters have reached similar conclusions based on substantially similar underlying evidence. *Koller*, 2021 WL 5027947, at *16-20 (Gowen); *Maloney v. Sec'y of Health & Human Servs*., No. 19-1713V, 2022 WL 1074087, at *30-31 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (Dorsey); *Gross*, 2022 WL 9669651, at *35-36 (Dorsey); *Sprenger v. Sec'y of Health & Human Servs*., No. 18-279V, 2023 WL 8543435, at *18-19 (Fed. Cl. Spec. Mstr. Nov. 14, 2023) (Dorsey); *Parker v. Sec'y of Health & Human Servs.*, No. 20-411V, 2023 WL 9261248, at *20-22 (Fed. Cl. Spec. Mstr. Dec. 20, 2023) (Dorsey); *Anderson v. Sec'y of Health & Human Servs.*, No. 18-484V, 2024 WL 557052, at *30-31 (Fed. Cl.

Spec. Mstr. Jan. 17, 2024) (Dorsey); *Simeneta v. Sec'y of Health & Human Servs.*, No. 18-859V, 2024 WL 4881411, at *30-31 (Fed. Cl. Spec. Mstr. Oct. 31, 2024) (Dorsey); *Brown v. Sec'y of Health & Human Servs*., No. 18-1074V, 2025 WL 2017882, at *25-27 (Fed. Cl. Spec. Mstr. June 23, 2025) (Dorsey); *see also Tracy ex rel. R.S. v. Sec'y of Health & Human Servs*., No. 16-213V, 2022 WL 1125281, at *29-32 (Fed. Cl. Spec. Mstr. Mar. 30, 2022) (Special Master Young accepting a similar theory in the context of transverse myelitis).[12]

However, acceptance of this theory has not been unanimous among special masters.[13] *Bielak v. Sec'y of Health & Human Servs.*, No. 18-761V, 2023 WL 35509, at *33-37 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (Corcoran); *Trollinger*, 2023 WL 2521912, at *26-30 (Corcoran); *Gamboa-Avila v. Sec'y of Health & Human Servs*., No. 18-925V, 2023 WL 6536207, at *25-29 (Fed. Cl. Spec. Mstr. Sept. 11, 2023) (Corcoran), *mot. for rev. denied*, 170 Fed. Cl. 441 (2024), *appeal filed*, No. 24-1765 (Fed. Cir. May 1, 2024); *Jaye v. Sec'y of Health & Human Servs.*, No. 20-672V, 2024 WL 3691413, at *14-17 (Fed. Cl. Spec. Mstr. July 18, 2024) (Corcoran); *Morrison v. Sec'y of Health & Human Servs.*, No. 18-386V, 2024 WL 3738934, at *19-23 (Fed. Cl. Spec. Mstr. July 18, 2024) (Oler). These contrary decisions are not binding on me. *Boatmon*, 941 F.3d at 1358-59; *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). Nonetheless, I have considered the points raised by these decisions. I simply reach a different conclusion based on my overall weighing of the evidence on this record.[14] Notably, even when reaching a different result, there has still been agreement that record evidence comparable to what has been presented in this case at a minimum

[12] Other special masters have also found that petitioners have preponderantly established that the Prevnar 13 vaccine case cause GBS based on the other causal theory Dr. Steinman presented. *Diponziano v. Sec'y of Health & Human Servs*., No. 17-1130V, 2025 WL 942744, at *18-25 (Fed. Cl. Spec. Mstr. Feb. 11, 2025) (Gowen) (accepting petitioner's causal theory based on molecular mimicry between CRM197 and Contactin-1); *Byrd v. Sec'y of Health & Human Servs.*, No. 20-1476V, 2024 WL 4003061, at *21-26 (Fed. Cl. Spec. Mstr. July 8, 2024) (Gowen) (same); *Anderson*, 2024 WL 557052, at *31-32 (Dorsey) (same); *Sprenger*, 2023 WL 8543435, at *19-20 (Dorsey) (same); *Gross*, 2022 WL 9669651, at *36-37 (Dorsey) (same); *Maloney*, 2022 WL 1074087, at *32 (Dorsey) (same).

[13] Dr. Steinman's alternative CRM197 and Contactin-1 theory has likewise also been rejected on some occasions. *Deshler*, 2020 WL 4593162, at *19-21; *Trollinger*, 2023 WL 2521912, at *29; *Gamboa-Avila*, 2023 WL 6536207, at *27; Jaye, 2024 WL 3738934, at *14-17; *Morrison*, 2024 WL 3738934, at *21-23. An additional case was resolved against the petitioner based on a different theory of causation. *McConnell v. Sec'y of Health & Human Servs.*, No. 18-1051V, 2022 WL 4008238, at *7-9 (Fed. Cl. Spec. Mstr. Aug. 19, 2022).

[14] There have been some significant differences in the record evidence of the various decisions weighing the theory Dr. Steinman presents in this case. For instance, while some prior cases denying compensation for GBS based on this theory found significance in the distinction between B- and T-cell responses, respondent's expert in *Cooper* specifically disclaimed a hardline distinction between such responses, testifying instead that "nothing is pure B cell/Tcell". *Compare Cooper*, 2024 WL 1522331, at *17, *with Deshler*, 2020 WL4593162, at *19-20, *and Bielak*, 2023 WL 35509, at *29-37. The *Morrison* decision turned in part on acceptance of the pathological role of antiganglioside antibodies in GBS as suggested by respondent's expert, Dr. Whitton. 2024 WL 3738934, at *19-21. Yet, respondent's expert in *Bartoszek*, Dr. Kedl, asserted to the contrary that this is "tenuous, speculative and clinically unproven," and that "the vast majority of literature stands in sharp contrast to the assertion that there is any causal relationship between ganglioside-specific antibodies and GBS." 2024 WL 4263604, at *20 n.16.

> *does* offer reliable support for the conclusion that phosphoglycerol is found in the pneumococcal vaccine; that the immune system produces antibodies in reaction to the relevant antigens containing the phosphoglycerol; and that individuals with neuropathies (although some suffer from the distinguishable disease MS) have been shown in small sample studies to possess antibodies specific to myelin-containing phospholipids.

Trollinger, 2023 WL 2521912, at *28.

I have also considered the issues raised by respondent in this particular case, but do not find that they cast sufficient doubt on Dr. Steinman's theory such that they would prevent petitioner from meeting her burden of proof under *Althen* prong one.

As a general matter, respondent argues that the fact that the wild *S. pneumoniae* infection has not been shown to cause GBS counsels against a finding that the vaccine can cause GBS. (ECF No. 75, pp. 21-22.) However, parallels to wild infection are never strong evidence. *E.g.*, *Gaskin v. Sec'y of Health & Human Servs*., No. 21-835V, 2025 WL 786306, at *12 (Fed. Cl. Spec. Mstr. Feb. 11, 2025) (noting that the IOM concludes that evidence of parallels to natural infection, though some evidence, "is never sufficient" to either accept or reject a causal relationship). Moreover, the clarity of this issue is reduced in this context, given that, as noted above, *S. pneumoniae* causes respiratory infections and *unspecified* upper respiratory infections in turn are generally accepted as a cause of GBS. Ultimately, a petitioner need not "demonstrate that a vaccine's infectious counterpart is a known-disease trigger." *Morrison*, 2024 WL 3738934, at *18. Respondent also argues that the epidemiologic evidence weighs against petitioner's theory. (ECF No. 75, pp. 22-26.) The Federal Circuit has previously stressed that a petitioner is not obligated to prove a case with epidemiology. *Capizzano*, 440 F.3d at 1325. Yet, "[n]othing in *Althen* or *Capizzano* requires the Special Master to ignore probative epidemiological evidence that undermines petitioner's theory." *D'Tiole v. Sec'y of Health & Human Servs.*, 726 F. App'x 809, 811 (Fed. Cir. 2018). I have considered all the epidemiology submitted in this case, and although I give some weight to these studies highlighted by respondent, I do not find that they cast significant doubt on the viability of Dr. Steinman's theory.

Regarding the specifics of Dr. Steinman's phosphoglycerol theory, respondent argues that (1) there are no published studies examining the specific cross-reactivity he has proposed between phosphoglycerol moieties (ECF No. 75, p. 28); (2) the theory is inconsistent with literature proposing gangliosides as the target of autoimmune attack in GBS (*Id*. at 29); (3) the presence of phospholipid antibodies in GBS patients does not establish their relevance to the pathogenesis of the disease (*Id*.); and (4) Dr. Steinman overstated the significance of tangentially related studies (*Id*. at 29-30). I have considered these arguments, but they are all retreads of arguments I have addressed in prior decisions. *Pierson*, 2022 WL 322836, at *27-31; *Bartoszek*, 2024 WL 4263604, at *20-22; *Datte*, 2025 WL 1565894, at *18-19; *Cooper*, 2024 WL 1522331, at *16-18. All of these arguments can be summed up as an objection to the fact that Dr. Steinman's

theory is only circumstantially supported. However, as I have previously explained, petitioners are permitted to rely on circumstantial evidence. *Pierson*, 2022 WL 322836, at *29. In this particular case, respondent's suggestion that these arguments poke any significant hole in Dr. Steinman's reasoning is especially unpersuasive given the cursory nature of his own expert's competing analyses.

Accordingly, petitioner has met his preponderant burden of proof under *Althen* prong one, having presented a sound and reliable theory of causation demonstrating that the Prevnar 13 vaccine can cause GBS.

b. *Althen* prong three

Under the third *Althen* prong, a petitioner must demonstrate a "proximate temporal relationship" between the subject vaccination and the alleged injury. *Althen*, 418 F.3d at 1278. To do this, petitioner must provide "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008).

Respondent disputes that petitioner has met his burden of proof under *Althen* prong three for two reasons. First, petitioner relied primarily on a study by Schonberger et al., which examined the timeframe for development of GBS following an unrelated vaccine. Respondent notes, in particular, that the Prevnar vaccine is conjugated and contains an alum adjuvant. (ECF No. 75, p. 35.) Second, petitioner failed under *Althen* prong one to set forth a theory from which a relevant timeframe could be drawn. (*Id*.) These arguments are not persuasive.

While Dr. Steinman did rely primarily on the Schonberger study with respect to timing, Dr. Souayah also explained on petitioner's behalf that two-thirds of GBS patients experienced symptoms of infection within the 6 weeks prior to onset of their GBS, which is generally viewed as consistent with it having a post-infectious antibody-driven pathogenesis. (Ex. 22, pp. 8-9.) This point is not in dispute. For example, Dr. Schroeder similarly opined on respondent's behalf that GBS "often occurs a few days or weeks after respiratory or gastrointestinal microbial infections" and that, in the case of *C. jejuni*, this has been shown to be the result of molecular mimicry leading to attack on neuronal tissue. (Ex. C, pp. 6-7.) Further to this, Dr. Souayah observed that the ten-day onset at issue in this case coincides with the expected time for the body's immune system to produce antibodies in response to vaccination. (Ex. 22, p. 13.)

Respondent is correct in noting that the Schonberger study involved a vaccine different than the one at issue in this case; however, the findings of this study have been utilized in assessing risk periods in subsequent studies that have looked at other potential causes of GBS. (Roger Baxter et al., *Lack of Association of Guillain-Barré Syndrome with Vaccinations*, 57 CLINICAL INFECTIOUS DISEASES 197 (2013) (Ex. 40, pp. 7-8) (referencing the 1976 swine flu vaccine study as helpful for selecting an appropriate time interval); *see also* Penina Haber et al., *Post-Licensure Surveillance of*

13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged ≥ 19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012-December 31, 2015, 34 VACCINE 6330 (2016) (Ex. 88, p. 5) (noting that the study results "verified 11 GBS reports with symptoms onset within 42 days of [Prevnar 13] vaccination" and referencing the Schonberger study in the bibliography).) Other special masters have also referenced Schonberger et al. in evaluating whether petitioners, who have alleged that they suffered GBS following Prevnar 13 vaccinations, have met their burden of proof under the third *Althen* prong. *See, e.g.*, *Godfrey v. Sec'y of Health & Human Servs.*, No. 17-1419V, 2025 WL 896840, at *28 (Fed. Cl. Spec. Mstr. Feb. 26, 2025) (Dorsey); *Koller*, 2021 WL 5027947, at *22-23 (Gowen). Moreover, respondent has not established that the fact that the Prevnar vaccine is conjugated and adjuvanted would be meaningful to assessing the timing of expected onset of GBS. Respondent's argument is limited to merely noting the existence of this distinction and he has not pointed to any medical evidence suggesting these are meaningful distinctions vis-à-vis the expected timing of onset. Moreover, the period of onset in this case – approximately ten days post-vaccination – is not at the edge of the expected timeframe cited by petitioner. Schonberger et al. found increased risk of onset of GBS primarily within five weeks of vaccination with the 1976 swine flu vaccine. (Schonberger et al., *supra*, at Ex. 29.) Accordingly, a marginal difference in the expected timing would not be significant.

Accordingly, petitioner has preponderantly shown under the third *Althen* prong that onset of the decedent's GBS occurred within a medically acceptable timeframe from which vaccine causation can be inferred.

c. *Althen* prong two

The second *Althen* prong requires preponderant proof of a logical sequence of cause and effect, which is usually supported by facts derived from petitioner's medical records.[15] *Althen*, 418 F.3d 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. While the opinions of treating physicians are often favored, *Capizzano*, 440 F.3d at 1326, a petitioner may support a cause-in-fact claim through presentation of either medical records or an expert medical opinion. *See* § 300aa-13(a).

[15] Medical records are generally viewed as trustworthy evidence. *Cucuras*, 993 F.2d at 1528. These records are generally contemporaneous to the medical events and "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.* However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (reasoning that "nothing . . . mandates that the testimony of a treating physician is sacrosanct—that is must be accepted in its entirety and cannot be rebutted").

Respondent stresses the Federal Circuit's caution in *Capizzano* that the second prong of *Althen* "is not without meaning." (ECF No. 75, p. 34 (quoting 440 F.3d at 1326-27).) He argues that

> petitioner has presented no evidence that the manifestation of [the decedent's] GBS was somehow aberrant from a typical manifestation of GBS absent vaccination. The evidence does not support that *S. pneumoniae* or *diphtheria* is generally implicated in causing GBS, weighing against the logic of concluding that pneumococcal vaccination acts as an inciting prodrome event.

(*Id*.) However, contrary to what respondent argues, I have concluded that petitioner has presented a sound and reliable theory explaining that the Prevnar vaccine can cause GBS. Although respondent is correct that the second *Althen* prong "is not without meaning," the Federal Circuit also explained in *Capizzano* that the petitioner's satisfaction of *Althen* prongs one and three is probative with respect to *Althen* prong two. *Capizzano*, 440 F.3d at 1326. Thus, it is significant to petitioner's *Athen* prong two showing that he has demonstrated that the Prevnar vaccine can cause GBS and that the onset of symptoms in this case occurred within a timeframe from which causation can be inferred.[16]

Moreover, as I have observed in prior cases, "[o]rdinary medical care offers little beyond clinical history that would confirm the cause of GBS." *Datte*, 2025 WL 1565894, at *21 (alteration in original) (quoting *Bartoszek*, 2024 WL 4263604, at *23). "The presence of preceding events is frequent, but they are not essential to the diagnosis." (Arthur K. Asbury & David R. Cornblath, *Assessment of Current Diagnostic Criteria for Guillain-Barré Syndrome*, 27 ANNALS NEUROLOGY S21 (1990) (Ex. 24, p. 1).) Thus, petitioner's theory of causation simply posits that the vaccine at issue is among the potential causes of GBS. Contrary to what respondent suggests, it does not anticipate any aberrant manifestation of GBS. Nor has respondent otherwise substantiated that any aberrant manifestation should be expected. For example, the Qualifications and Aids to Interpretation accompanying the Vaccine Injury Table for flu-vaccine related GBS are consistent with the ordinary diagnostic standards for GBS. 42 C.F.R. § 100.3(c)(15).

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[16] The *Capizzano* Court described the circumstances in which *Althen* prong two may be a stumbling block as follows:

> There may well be a circumstance where it is found that a vaccine *can* cause the injury at issue and where the injury was temporally proximate to the vaccination, but it is illogical to conclude that the injury was actually caused by the vaccine. A claimant could satisfy the first and third prongs without satisfying the second prong when medical records and medical opinions do not suggest that the vaccine caused the injury, or where the probability of coincidence or another cause prevents the claimant from proving the vaccine caused the injury by preponderant evidence.

440 F.3d at 1327.

In addition to the decedent's GBS arising within a period during which a causal inference is appropriate, it is also the case that no other cause is evident for the decedent's GBS. This is also probative with respect to *Althen* prong two. *Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1151 (Fed. Cir. 2007) (noting that "a petitioner is certainly permitted to use evidence eliminating other potential causes to help carry the burden on causation and may find it necessary to do so when the other evidence on causation is insufficient to make out a prima facie case"). Although Dr. Schroeder opined on respondent's behalf that the decedent's medical work up did not actually rule out other causes because an infectious illness may have gone unreported or been present only subclinically (Ex. C, pp. 9-10), this is rank speculation. None of respondent's experts opined that any infectious trigger, or any other cause of the decedent's GBS, was more likely than not present.

Of course, the *Althen* court held that "neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." 418 F.3d at 1278. Treating physician opinion on the cause of the decedent's GBS is not robust, consisting primarily of the fact that the decedent's prior pneumococcal vaccination was documented as a consideration as they assessed the likelihood of the GBS diagnosis. (ECF No. 73, p. 26 (discussing Ex. 1, p. 868).) However, petitioner stresses in his motion for a ruling on the written record that he has demonstrated actual causation with affirmative expert medical opinion by both Drs. Souayah and Steinman based on their review of the facts of this case, as well as their experience and qualifications as neurologists and their familiarity with GBS in particular. (*Id.* at 25-26.) Although respondent submitted competing medical opinions by Drs. Donofrio and Schroeder, who are also qualified to opine regarding the causes of GBS, their conclusion that the decedent's GBS was merely coincidental to vaccination is based exclusively on their disbelief as to general causation. However, as discussed under *Althen* prong one, I found petitioner's evidence more persuasive with respect to general causation. Accordingly, their opinions are by extension also less persuasive with respect to *Althen* prong two.

Petitioner also argues that he has established a logical sequence of cause and effect whereby the decedent's vaccination resulted in his GBS "leaving him unable to walk or adequately care for himself, and severely depressed, ultimately resulting in his suicide." (ECF No. 73, p. 25.) That is, petitioner argues that the decedent's death is a part of the vaccine injury at issue because the decedent's GBS was a but for cause of his depression and suicide. (ECF No. 76, p. 4.) In response, respondent argues, similar to his argument with respect to the underlying GBS, that "none of [the decedent's] treating physicians opined that [his] GBS caused him to become depressed such that he eventually died by suicide."[17] (ECF No. 75, p. 34.) Based on my review of

[17] Petitioner's reply argues that respondent has waived this argument. (ECF No. 76, p. 4.) Petitioner's wavier argument stems from the October 20, 2021 status conference referenced above in the procedural history. (*Id*. (quoting ECF No. 34, p. 2).) At that time, I instructed respondent to raise any legal or factual defense to petitioner's death claim "now" (meaning during the entitlement phase). (ECF No. 34, p. 2.) Thereafter, respondent confirmed he would not develop the record with respect to the suicide. (ECF No. 46, p. 1.) However, I cannot agree that this constitutes a waiver of the argument actually raised.

the evidence, petitioner has preponderantly established that the decedent's GBS was a substantial contributing factor and but for cause of his death.

In his affidavit, petitioner asserts that his brother, the decedent, had no history of depression. (Ex. 10.) This is corroborated insofar as the medical records that are available do not suggest otherwise. Nor has respondent asserted that the decedent had any history of depression or suicidal ideation.[18] However, during the course of his hospitalization for his GBS, the decedent's physicians recorded that he at one point refused treatment and stated that "he did not want to live anymore."[19] (Ex. 1, pp. 353-354; Ex. 2, p. 13.) He was believed to be suffering "likely situational depression." (Ex. 2, p. 13.) A follow up screening found the decedent did not have decision-making capacity; however, a complete screening for his depressive symptoms could not be completed because he was intubated at the time. (Ex. 1, pp. 354-56.) After the decedent was extubated and regained his decision-making capacity, he continued to report feeling "down," but denied symptoms of depression, expressed a preference for resuscitative efforts, and reported that he had no thoughts of harm to himself. (*Id.* at 290.) Nonetheless, he continued to be treated with antidepressant medication and after being discharged from the hospital, his inpatient rehabilitation records later characterize him as experiencing a "major depressive disorder" (Ex. 3, p. 43) and depression remained among his discharge diagnoses. Although the decedent provided no first-hand account of the reason for his suicide (*Id.* at 44; Ex. 16, p. 3), his brother averred that, when he returned home, he

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Although respondent declined to develop the record further on this point, he bases his argument on the otherwise existing record. Therefore, while I would have been disinclined to entertain any request to delay resolution of the issue or belatedly develop the record further for the reason raised by petitioner, respondent's argument as couched in his motion response is not precluded by the prior procedural history.

[18] Respondent does note that the decedent had a prior history of refusing medical treatment (ECF No. 75, pp. 2-3 (citing Ex. 2, pp. 120-22, 125, 136)), though he stops short of arguing that this is relevant to assessing the decedent's state of mind following onset of his GBS (*Id*. at 34-35). Specifically, the decedent had two surgeries to remove a polyp in his throat in 2014 and 2015. (*E.g.*, Ex. 2, p. 131.) After the second surgery, he was told that the polyp, which was suspected to be a verruca carcinoma, was growing back and radiation therapy was recommended. (*Id.*) The decedent refused this further treatment, including as recently as an August 10, 2017 check up. (*Id*. at 121-22.) It was noted at that time, however, that there had been no changes in his medical condition. (*Id*.) Verruca carcinoma is a form of squamous cell cancer that is generally slow growing and for which metastasis is rare. *See Verrucous Carcinoma*, WIKIPEDIA, https://en.wikipedia.org/wiki/Verrucous_carcinoma (last visited July 31, 2025). Accordingly, especially given the decedent's age, it is not readily apparent that his decision to forgo radiation is comparable to or predictive of his reaction to his GBS. The medical records cited by respondent do not discuss the details of the decedent's discussions with his physicians regarding the risks and benefits of the proposed radiation therapy, simply noting that he was aware of the risks associated with his decision. (Ex. 2, pp. 122.)

[19] The medical records suggest that this may have been prompted at least in part by the decedent having been incorrectly informed that he was HIV positive (this was ultimately a false positive). (Ex. 1, pp. 353-54.) However, it is also notable that this occurred close to the nadir of his GBS and at a time when he was intubated. Moreover, the treatment he was refusing was for the GBS, not HIV.

> became very depressed because he was so weak and he did not think he would ever recover. He told me that he didn't think he would ever be well enough to care for himself or to leave the house on his own to visit friends or do any of the things he enjoyed before his GBS.

(Ex. 10, p. 3.)

Further to this, petitioner has provided a medical opinion by Dr. Souayah. Dr. Souayah opines that GBS patients are at increased risk of major depressive disorders which, in turn, are a risk factor for suicide. (Ex. 22, p. 20 (citing Bahnasy et al., supra, at Ex. 48); *see also Does Depression Increase the Risk for Suicide?*, U.S. DEP'T OF HEALTH & HUM. SERVS. (last updated Sept. 14, 2014) (Ex. 15).) In fact, the literature filed in this case indicates that GBS patients in particular have small, but elevated, risk of suicide. (Ex. 22, p. 20 (citing Erlangsen et al., *supra*, at Ex. 50).) Thus, Dr. Souayah opines that this decedent's suicide was causally related to his GBS, stressing that the decedent had no prior history of depression and became depressed as a result of his GBS and ongoing disability. (*Id.* at 20-21.) Erlangsen, et al., found that among neurologic patients overall, increased incidences of suicide were highest between one to three months after diagnosis as well as being correlated to cumulative hospital contacts. (Erlangsen et al., *supra*, at Ex. 50, p. 4.) This decedent's suicide is broadly consistent with this pattern. His GBS diagnosis was confirmed after his EMG study on September 7, and his suicide occurred less than four months later, on December 29, and shortly after release from a second hospitalization, which itself followed a prolong hospitalization and inpatient rehabilitation. (Ex. 1, pp. 322-23; Ex. 16.)

All of this together tends to show that petitioner was not simply despondent regarding his circumstances, but that his state of mind was likely a direct result of his condition, inclusive of GBS and a related depressive disorder. Although Dr. Souayah is not a psychiatrist or psychologist, his qualifications and experience as a clinical neurologist permit him to offer an opinion as to the likely sequela of GBS. In any event, Dr. Souayah's medical opinion on this point is entirely unrebutted. None of respondent's experts offered any opinion contesting that the decedent's death was casually related to his GBS.

Nonetheless, after noting that none of the treating physicians specifically concluded that the decedent suffered GBS-related depression leading to suicide, respondent argues that "[i]nstead, the medical evidence reveals that less than two weeks before his death, [the decedent] told his treating physician and a hospital social worker about his concerns regarding his inability to care for himself and pay for post-hospitalization GBS treatment." (ECF No. 75, pp. 34-35 (citing Ex. 1, pp. 264-65, 432; Ex. 19, p. 43).) To the extent respondent appears to be arguing that financial concerns represent an alternative or mutually exclusive explanation for the decedent's suicide, this is not persuasive without more. The fact that the decedent had specific concrete concerns with respect to the consequences of his injury does not undermine the allegation that his state of mind was due to GBS-related depression. Indeed, in addressing the decedent's financial concerns, the social worker specifically remarked

on December 19, 2017, just ten days prior to his death, that he "seemed depressed." (Ex. 19, p. 43.)

Accordingly, petitioner has preponderantly demonstrated a logical sequence of cause and effect under the second *Althen* prong whereby he has shown that the decedent's GBS was caused-in-fact by his August 10, 2017 Prevnar 13 vaccination and that his vaccine-caused GBS was in turn a substantial contributing factor and a but for cause of his subsequent death.[20]

d. Factor unrelated to vaccination

Once petitioner has satisfied his own burden of proof, the burden shifts to respondent to demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to vaccination. § 300aa-13(a)(1)(B); *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367-69 (Fed. Cir. 2013). Respondent has not presented any factor unrelated to vaccination as a potential cause of the decedent's GBS. (ECF Nos. 25, 75.)

[20] Despite this conclusion in this case, the question of whether a death by suicide is cognizable under the Vaccine Act as a "vaccine-related death" or a death "from the administration of the vaccine" is probably still best viewed as an open or unresolved question. 42 U.S.C. § 300aa-15(a)(2) and 11(c)(1)(D)(ii). I am not aware of any prior case that has adjudicated this question within the Program, and, in this particular case, respondent has not raised this question at any point, even after prompting. (ECF Nos. 25, 46, 75.) Thus, apart from the factual issue raised by respondent, which I have resolved, the petitioner's ability to assert a claim based on this decedent's death has not been contested as a legal matter. Therefore, I opt not to delve into that *potential* issue sua sponte, allowing petitioner the benefit of the remaining ambiguity. *Accord Lowry v. Sec'y of Health & Human Servs*., 189 F.3d 1378, 1381 (Fed. Cir. 1999) (explaining the Vaccine Program is intended to compensate petitioners "quickly" and "with 'generosity'" to keep them out of the traditional tort system (quoting H.R. REP. NO. 99-908 (1986))); Vaccine Rule 8(f) (arguments "must be raised specifically in the record before the special master"). Liability for suicide resulting from tortious injury is not treated uniformly in jurisdictions throughout the country. *Compare, e.g.*, *White v. Lawrence*, 975 S.W.2d 525, 530 (Tenn. 1998) (finding that "leading risk factors for suicide include physical illness and depression. The decedent suffered from both. The plaintiff presented medical proof that the decedent's suicide was reasonably foreseeable from a medical standpoint, and that the defendant's conduct was a substantial factor in bringing about the suicide."), *and Kivland v. Columbia Orthopaedic Grp., LLP*, 331 S.W.3d 299, 309 (Mo. 2011) (indicating that "[a] plaintiff can show that the defendant's negligence was the proximate cause of the decedent's suicide by presenting evidence that the decedent's suicide was the 'natural and probable consequence' of the injury he suffered at the hands of the defendant"), *with Krieg v. Massey*, 781 P.2d 277, 278 (Mont. 1989) (stating as the general rule that "[n]egligence actions for the suicide of another will generally not lie since the act of suicide is considered a deliberate intervening act"), *and McPeake v. William T. Cannon, Esquire, P.C*., 553 A.2d 439, 441 (Pa. 1989) (stating that "suicide constitutes an independent intervening act so extraordinary as not to have been reasonably foreseeable by the original tortfeasor"). The so called "suicide rule" – that suicide inherently breaks the causal chain – remains the traditional approach; however, the modern trend has been to move away from brightline application of the traditional rule. *See* Alex B. Long, *Abolishing the Suicide Rule*, 113 NW. U.L. REV. 767 (2019). Accordingly, absent direct appellate authority, there is no readily available answer as to how this evolving tort background would apply in this specific program, a remedial no-fault system with no private tortfeasor.

VI. Conclusion

After weighing the evidence of record within the context of this Program, I find by preponderant evidence that the decedent suffered GBS caused-in-fact by the Prevnar 13 vaccination he received on August 10, 2017, and further that his death was causally related to his GBS. Accordingly, petitioner is entitled to compensation. A separate damages order will be issued.

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master