

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 16-473V
(to be published)

ARTHUR L. TROLLINGER,

Petitioner,

v.

SECRETARY OF HEALTH
AND HUMAN SERVICES,

Respondent.

* Chief Special Master Corcoran
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* Dated: February 17, 2023
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Nancy Routh Meyers, Turning Point Litigation, Greensboro, NC, for Petitioner.
Mallori Browne Openchowski, U.S. Dep’t of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION¹

On April 14, 2016, Arthur Trollinger filed a petition for compensation under the National Vaccine and Injury Compensation Program (the “Vaccine Program”).² (ECF No. 1) (“Petition”). Petitioner alleges that the Prevnar-13 (“pneumococcal”) vaccine he received on July 17, 2015, caused him to incur Guillain-Barré syndrome (“GBS”). *Id.*

The parties have agreed that the matter could reasonably be resolved via ruling on the record and filed briefs in support of their respective positions. *See* Petitioner’s Motion, dated

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

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

January 14, 2022 (ECF No. 72) (“Mot.”); Respondent’s Opposition, dated April 29, 2022 (ECF No. 76) (“Opp.”); Petitioner’s Reply, dated June 2, 2022 (ECF No. 80) (“Reply”). Having reviewed the above plus the filed medical records, expert reports, and associated literature, I hereby deny an entitlement award. As discussed in greater detail below, Petitioner has not preponderantly established that the pneumococcal vaccine can cause GBS, or did so to him.

I. Factual Background

Petitioner, a 68-year-old man with a prior medical history of smoking and alcohol abuse (though he had remained sober for 24 years) and chronic lower back pain, received the pneumococcal vaccine during an annual wellness exam on July 17, 2015. Ex. 1 at 6–7; Ex. 10 at 2. A little more than a week later, he presented to the Emergency Room (“ER”) at Alamance Regional Medical Center in Burlington, North Carolina, on July 25, 2015, with complaints of abdominal pain. Ex. 3 at 2. After radiological and laboratory testing revealed no apparent cause of Petitioner’s pain, he was diagnosed with gastric reflux. Ex. 4 at 441–44, 450–51, 453–84.

Petitioner alleges that on July 27, 2015 (ten days post-vaccination), he woke up and discovered he had lost feeling in his hands, and later that morning was experiencing weakness plus pins and needles sensations in his legs. Pet. at 2. Mr. Trollinger’s wife drove him to a local walk-in medical provider, but the physician sent him back to the ER after developing numbness in his hands and feet, and ptosis³ of the right eye. Ex. 4 at 40–42, 72–73. He was admitted to Alamance Regional Medical Center and after MRIs of his brain and L-spine, a CT of his head, and lab work, he was diagnosed with GBS. *Id.* at 42, 67–74, 157, 319. He received five doses of IVIG,⁴ and his symptoms began to improve. *Id.* at 75–76, 78, 80, 165. At that time, treating neurologist Matthew Smith, M.D., stated that Petitioner’s GBS was “likely due to pneumonia shot” he had received one week earlier. *Id.* at 80. Petitioner was discharged from Alamance Regional Medical Center on August 3, 2015. *Id.* at 36–37.

Petitioner required rehabilitation and was transferred to Moses Cone Health—a long-term care facility—from August 3–10, 2015. Pet. at 2; Ex. 5 at 10–24. He was experiencing back pain, difficulty swallowing, fatigue, blurred vision, and weakness in his lower extremities, and needed minimum to moderate assistance with transfers and walking. Ex. 5 at 10–15. He received speech, physical, and occupational therapy, and was discharged one week later. Ex. 5 at 14. Petitioner then received at-home assistance through Advanced Home Care Agency from August 11, 2015 through August 27, 2015. Ex. 6 at 27–29, 35–49, 50–54. Petitioner was also referred for ongoing physical

³ Ptosis is “drooping of the upper eyelid.” *Ptosis*, Dorland’s Medical Dictionary Online, <https://www.dorlandonline.com/dorland/definition?id=42014> (last visited Feb. 17, 2023).

⁴ Intravenous immunoglobulin (“IVIG”) is a blood product used to treat patients with antibody deficiencies, including neurological disorders. Clinical Uses of Intravenous Immunoglobulin, NCBI (2005), <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1809480/> (last visited on February 17, 2023).

therapy, which he attended through Alamance Regional Medical Center's Outpatient Rehabilitation Center, from October 6, 2015 through the end of the year. Ex. 7 at 23–50, 60–97, 105–27.

On August 19, 2015, Mr. Trollinger saw his primary care physician (“PCP”) to follow up on his GBS diagnosis. Ex. 1 at 4–5. He was still attending physical therapy, and complained of difficulty with swallowing, back pain, and muscle spasms. *Id.* He was also experiencing tachycardia, chest pain, and shortness of breath, and was referred to a cardiologist. *Id.*

Petitioner was next seen by cardiologist Dwayne Callwood, M.D., on August 31, 2015. Ex. 9 at 6–13. Dr. Callwood recorded for history of present illness that Petitioner “developed Guillain-Barré syndrome after a pneumonia shot.” *Id.* at 6. He also recorded that Petitioner had angina⁵ symptoms the last three or four months, shortness of breath, and tachycardia, so he was referred for a cardiac evaluation. *Id.* Dr. Callwood stated that “[t]he multiple new symptoms [are] related to his Guillain-Barré.” *Id.* at 6.

Petitioner also followed up with neurology on September 21, 2015. Ex. 10 at 9–15. His records from that visit noted that he had received the pneumococcal vaccine in July 2015, but this record did not relate that event to his current GBS symptoms, which Petitioner reported were slowly improving. *Id.* at 9. The note did, however, list the pneumococcal vaccine as an allergic agent, and also noted numbness, shortness of breath, and chest pain. *Id.* at 10. Neurologist Hemang Shah, M.D. diagnosed Petitioner with GBS, prescribed Gabapentin, and advised Petitioner that he should hold off on driving. *Id.* at 15.

Petitioner underwent a nerve conduction study and EMG⁶ testing on October 2, 2015 (now more than ten weeks from his onset of neurologic symptoms). Ex. 10 at 2–3. At the time of the examination, he displayed bilateral weakness and numbness in his hands, feet, and legs. *Id.* at 2. Dr. Shah listed that Petitioner’s “[p]ast medical history [wa]s significant for pneumonia vaccine prior to gradual onset of symptoms.” *Id.* The impression was a “generalized polyneuropathy,” with sensory deficits predominant, but “no evidence of conduction block.” *Id.* at 3 (suggesting that Petitioner’s normal median motor response could represent “more proximal lesion”).

⁵ Angina is “any spasmodic, choking, or suffocative pain.” *Angina*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=2770&searchterm=angina> (last visited Feb. 17, 2023).

⁶ Electromyography is the process by which “an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation; performed using any of a variety of surface electrodes, needle electrodes, and devices for amplifying, transmitting, and recording the signals.” *Electromyography*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=15854&searchterm=electromyography> (last visited Feb. 17, 2023).

By January 20, 2016, Petitioner had improved enough to cut and haul wood, though he was still using a cane to walk. Ex. 13 at 5. In February, he asked to be cleared for his commercial driver's license, and his PCP referred him to neurology. *Id.* at 6. He also had a follow-up with cardiology in April 2016, where Dwayne Callwood, M.D., stated that his GBS was "possibly related to the pneumonia vaccine[.]" Ex. 11 at 15. On May 31, 2016, he again visited his PCP, where it was noted that his GBS symptoms were slowly improving and "is aware to never get another vaccine." Ex. 13 at 4–5.

Currently, Petitioner continues to suffer from numbness and tingling in his hands, up to his wrists, and in his feet, up to his calves. Pet. at 3. He also has tightening in his torso. *Id.* Petitioner continues to take Gabapentin and receives follow-up care from Dr. Shah. *Id.*

II. Expert Reports

A. Petitioner's Expert – Lawrence Steinman, M.D.

Dr. Steinman, an adult and pediatric neurologist, prepared four written reports for the Petitioner. Report, dated March 7, 2017, filed as Ex. 14 (ECF No. 26-1) ("Steinman First Rep."); Report, dated August 7, 2017, filed as Ex. 31 (ECF No. 35-1) ("Steinman Second Rep."); Report, dated August 6, 2021, filed as Ex. 40 (ECF No. 67-1) ("Steinman Third Rep."); Report, dated May 16, 2022, filed as Ex. 62 (ECF No. 79-1) ("Steinman Fourth Rep."). Altogether, Dr. Steinman offered 80 pages of expert opinion on this matter, not merely refining but almost substituting a second theory later in the claim's life. Ultimately, however (as discussed below and throughout this Decision), I do not conclude his effort was well spent.⁷

As shown in his CV, Dr. Steinman received his undergraduate degree from Dartmouth College, and his medical degree from Harvard Medical School. *Curriculum Vitae*, filed as Ex. 15 on March 7, 2017 (ECF No. 26-2) ("Steinman CV") at 1. He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past 37 years. *Id.*; Steinman First Rep. at 1. He is board certified in neurology from the American Board of Psychiatry and Neurology. Steinman CV at 2. Dr. Steinman has also published hundreds of peer-reviewed publications on neurology and autoimmune disease. *Id.* at 5–46. He holds several patents related to the diagnosis and treatment of autoimmune and demyelinating diseases. *Id.* at 2–3. He presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics at Stanford University. *Id.* at 1.

⁷ The excessive length of these reports is in some cases attributable to Dr. Steinman's propensity for cutting and pasting sections taken wholesale from prior reports—a practice for which he has been criticized in the past. *See, e.g., Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836, at *12–13 (Fed. Cl. Spec. Mstr. Jan. 19, 2022) (copying sections from prior reports); *Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651, at *15–18 (Fed. Cl. Spec. Mstr. Sept. 22, 2022) (same).

First Report

Dr. Steinman began his report with a summary of the medical records, accepting Petitioner’s GBS diagnosis. Steinman First Rep. at 3–5. He then outlined how the pneumococcal vaccine could trigger GBS. *Id.* at 5–8. Dr. Steinman’s initial theory was based on the contention that phospholipid⁸ molecular structures found in the vaccine’s antigens could, via the mechanism of molecular mimicry, prompt a cross-reactive autoimmune response resulting in GBS.

First, Dr. Steinman discussed the content of the vaccine itself. As the pneumococcal vaccine package insert indicates, the vaccine comprises a sterile suspension of saccharides of the capsular antigens of *Streptococcus pneumoniae* from 13 serotypes (1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F), individually linked to non-toxic diphtheria CRM₁₉₇ protein.⁹ *Id.* at 6 (citing Pneumococcal Package Insert, filed as Ex. 26 on Mar. 7, 2017 (ECF No. 27-5) (“Pneumococcal Package Insert”), at 24);¹⁰ *see generally* D. Wang et al., *Uncovering Cryptic Glycan Markers in Multiple Sclerosis (MS) and Experimental Autoimmune Encephalomyelitis (EAE)*, *Drug Dev. Res.* 172, 185–86 (2014), filed as Ex. 23 (ECF No. 27-2). Dr. Steinman also contended that certain phospholipids (phosphatidyl serine and phosphatidyl choline) are also present in the vaccine, expressed in association with its polysaccharide antigens. Steinman First Rep. at 7–8.

For example, the phospholipid phosphorylcholine is expressed in the 19A component, which is key in the pathophysiology of the pneumonia infection. Steinman First Rep. at 8; Yi-Ping Chuang et al., *Impact of the glpQ2 Gene on Virulence in a Streptococcus pneumoniae Serotype 19A Sequence Type 320 Strain*, 83 *Infection & Immunity* 682, 682 (2015), filed as Ex. 28 (ECF No. 27-7) (“Chuang”). Thus, in Dr. Steinman’s estimation, it is logical that a vaccine intended to immunize *against* this pneumococcus strain might also elicit “an immune response to lipids involved in neuroinflammation.” Steinman First Rep. at 8. In fact, the enzyme for metabolizing lipids and producing phosphorylcholine was present in strains 3, 6B, 19A, and 19F—with three of those strains (strains 3, 19A, and 19f) all included in the Prevnar formulation. Chuang at 682; B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with Guillain-Barre*

⁸ Phospholipids have been defined as “any lipid that contains phosphorus, including those with a glycerol backbone (phosphoglycerides and plasmalogens) or a backbone of sphingosine or related substance (sphingomyelins). Phospholipids are the major form of lipid in all cell membranes.” *Phospholipid*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=38759&searchterm=phospholipid> (last visited Feb. 17, 2023).

⁹ “CRM₁₉₇ is a nontoxic variant of diphtheria toxin isolated from cultures of *Corynebacterium diphtheriae* strain Cy (β197) grown in a casamino acids and yeast extract-based medium. CRM₁₉₇ is purified through ultrafiltration, ammonium sulfate precipitation, and ion-exchange chromatography.” Pneumococcal Package Insert at 24. It is included to induce “immunity through a T cell-dependent response.” *Deshler v. Sec’y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *19 (Fed. Cl. Spec. Mstr. July 1, 2020).

¹⁰ Also cited as Respondent’s Ex. M, Tab 5.

Syndrome: Cross-Reactive or Pathogenic? 16 *Autoimmunity* 1, 23–27 (1993), filed as Ex. 27 (ECF No. 27-6) (“Gilburd”) at 23, 25; *see also* J. Kornspan & S. Rottem, *The Phospholipid Profile of Mycoplasmas*, *J. Lipids* 1, 2 (2012), filed as Ex. 29 (ECF No. 27-8).

Second, Dr. Steinman offered scientific literature suggesting that antibodies to these same phospholipids were present in the blood sera of GBS patients. Steinman First Rep. at 6–7; Gilburd at 23–27 (two of the six GBS patients considered showed reactivity to phosphatidyl serine or phosphatidyl choline). Dr. Steinman's own research, he added, had shown that phospholipids are components of the nerve's myelin sheath in humans, and that they are targeted by antibodies in the context of existing neuroinflammation. Steinman First Rep. at 7; J. Kanter et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation*, 12 *Nat. Med.* 138 (2005), filed as Ex. 21 (ECF No. 26-8) (“Kanter”). Employing the same experimental animal model relied upon in another article (discussed herein), Kanter determined that (given the extent to which nerve myelin is comprised of lipids) lipid-specific autoantigens likely played *some* role in the pathogenesis of multiple sclerosis (“MS”), a demyelinating autoimmune disease of the central nervous system—although Kanter is not specific to peripheral neuropathies like GBS. Kanter at 138.

Another article offered by Dr. Steinman observed that MS autoantibodies “target a phosphate group in phosphatidyl serine and oxidized phosphatidyl choline derivatives.” Steinman First Rep. at 7; P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 4 *Sci. Translational Med.* 1, 1 (2012), filed as Ex. 22 (ECF No. 27-1) (“Ho”). But Ho only suggests that the studied antibodies might *contribute* to MS pathogenesis—not that they were the cross-reacting or an instigating spark for an autoimmune disease. Ho at 1. Dr. Steinman did not offer other evidence supporting the greater association between the *pneumococcal vaccine* and GBS.

Finally, Dr. Steinman endorsed the onset of Mr. Trollinger's GBS as having occurred in a medically-acceptable timeframe when measured from date of vaccination. Steinman First Rep. at 9–10. For support, he invoked an item of literature specific to the 1976 swine flu vaccine. Steinman First Rep. at 11; L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977*, 110 *Am. J. Epidemiol.* 105 (1979), filed as Ex. 30 (ECF No. 27-9) (“Schonberger”). Because Petitioner's onset occurred approximately ten days after vaccination, it fell within Schonberger's observed risk interval. Schonberger at 109. In so maintaining, Dr. Steinman proposed (erroneously) that what is known about timing in Schonberger is “often used as a surrogate” in other vaccine causation cases, adding that more detailed studies specific to GBS and the pneumococcal vaccine do not exist. Steinman First Rep. at 9.¹¹

¹¹ Dr. Steinman also cited to a “Reference 17” for support, but his first expert report only includes 16 citations.

Second Report

Dr. Steinman’s Second Report (only two pages in length)¹² provided four specific responses to some of the criticisms of Respondent’s first expert, Dr. Timothy Vartanian. First, Dr. Steinman disputed Dr. Vartanian’s arguments about the lack of EMG/NCS support for the existence of a demyelinating polyneuropathy. Steinman Second Rep. at 1. Dr. Steinman noted that the EMG had been performed three months after diagnosis, and in the wake of five days of IVIG treatment, all of which could likely diminish evidence of a motor conduction problem. *Id.* In addition, Dr. Shah (who performed the EMG) referred in the record memorializing the results of this testing to the existence of *motor* abnormalities and a proximal motor lesion. *Id.*; Ex. 10 at 2. (Dr. Steinman’s fourth rebuttal point was somewhat related, as he also maintained that the EMG results allegedly contrary to a finding of an existing demyelinating neuropathy actually showed the presence of abnormal F-wave latency supportive of the presence of demyelination. Steinman Second Rep. at 2.)

Second, Dr. Steinman commented on the scientific criteria that Dr. Vartanian deemed necessary for the conclusion that molecular mimicry was the likely pathologic mechanism for a disease. Steinman Second Rep. at 1; N. Yuki, *Ganglioside Mimicry and Peripheral Nerve Disease*, *Muscle & Nerve* 691, 695 (2007), filed as Ex. Ex. F (ECF No. 33-6) (“Yuki”). One such factor was evidence of an epidemiologic association—but Dr. Steinman deemed it an impossibility to provide such evidence. Steinman Second Rep. at 1. He could not in this case retroactively ascertain whether there were T cells or antibodies to the mimics asserted in his theory, given that Petitioner was never so tested. *Id.* Nor was it reasonable to demand that the theory be supported by an animal model, since that would be “beyond the capacity of any expert” offering an opinion in a Program case. *Id.*

Third, Dr. Steinman took issue with Dr. Vartanian’s point that the mere presence of autoantibodies does not imply pathologic autoimmunity. Steinman Second Rep. at 2. Although it is well known that all people possess T cells reactive to self-antigens (including myelin basic protein), their interaction with pro-inflammatory cytokines (a different kind of immune cell relevant mostly to the immediate/innate immune response) is more frequent and aggressive in individuals suffering from a neuroinflammatory disease. *Id.*; K. Ota et al., *T Cell Recognition of an Immunodominant Myelin Basic Protein Epitope in Multiple Sclerosis*, 346 *Nature* 183, 183 (1990), filed as Ex. 32 (ECF No. 35-2); J. Zhang et al., *Increased Frequency of Interleukin 2-Responsive T Cells Specific for Myelin Basic Protein and Proteolipid Protein in Peripheral Blood and Cerebrospinal Fluid of Patients with Multiple Sclerosis*, 179 *J. Experimental Med.* 793, 980–83 (1994), filed as Ex. 33 (ECF No. 35-3). Thus, identifying mimics to myelin proteins is only the

¹² The end of Dr. Steinman’s second expert report attached (for no apparent reason) a copy of his previously-filed first expert report. Steinman Second Rep. at 3–14.

first step in understanding how autoimmunity occurs—but (again) requiring more is unreasonable in the context of a Vaccine Program case. Steinman Second Rep. at 2.

Third Report

The context in which Dr. Steinman’s third expert report was filed bears on its contents. Nearly *four years* separate Dr. Steinman’s second and third expert reports, and in that time period the case was progressing sluggishly, with little occurring save for adjudication of an interim fee request and a fruitless attempt at informal resolution. The case was also transferred twice from the original special master to whom it had been assigned, ending up with me in March 2021 (ECF Nos. 61-62). Not long after, I had asked the parties to propose a schedule for ruling on the record—but in the course of those efforts, Petitioner requested the opportunity to update Dr. Steinman’s opinion, since “the current analysis of the molecular mimicry in Prevnar 13 is far more extensive than in [Dr. Steinman’s] original expert report filed in this case in 2017.” *Status Report*, dated July 14, 2021 (ECF No. 66). I acceded to the request, and this report from Dr. Steinman was filed.

Dr. Steinman began by describing the recent “detective work” he had now performed to support his causation theory (and in particular to verify “whether the phosphoglycerol group, *common to the phospholipids* targeted in GBS, . . . were actually in the vaccine.”) (emphasis added)¹³ Steinman Third Rep. at 1, 2. Tellingly, however, that work consisted less of delving into new medical or scientific literature bearing on the purported pneumococcal vaccine-GBS association that his first two reports contended existed, and more reflected his revised consideration of *existing* evidence regarding the vaccine’s contents. Thus, he referenced an inquiry he had made to the Centers for Disease Control and Prevention (“CDC”),¹⁴ followed by a reference to the vaccine’s actual package insert, and then on to the vaccine’s patent. *Id.* at 2–6. Nowhere herein did Dr. Steinman explain why he had failed to perform this kind of “detective work” when preparing his initial expert report four years before—especially since the materials he ultimately relies upon appear to have *existed in 2017*, and thus could have been evaluated at the time of his first report.

As a result of these inquiries, Dr. Steinman was able to recast his opinion slightly, now maintaining that rather than phospholipids, the vaccine contained “phosphoglycerol,” a glycerol

¹³ Significantly, this assertion is somewhat inconsistent with the opinion expressed in Dr. Steinman’s first report: that “phospholipids ARE present in Prevnar 13.” Steinman First Report at 8. As Dr. Whitton noted (discussed below), phospholipids are not the same as phosphoglycerols. Whitton Rep. at 19.

¹⁴ Notably Dr. Steinman appears in this case to have referenced email communications generated in connection with a different Program case involving the pneumococcal vaccine. Steinman Third Rep. at 2–4; *Pierson v. Sec’y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836, at *13 n.16 (Fed. Cl. Spec. Mstr. Jan. 19, 2022) (describing how Dr. Steinman sought clarification on the chemistry of the pneumococcal vaccine).

compound of phosphoric acid (that is itself a *component* of phospholipids). Steinman Third Rep. at 6.

Dr. Steinman spent the rest of the third report discussing different possible molecular mimics to support his theory. Steinman Third Rep. at 6–21. He primarily focused on phosphoglycerol, which he maintained was contained in some of the vaccine’s polysaccharides taken from 13 individual pneumococcal strains, and allowed their conjugation to a protein carrier (CRM₁₉₇) that aids the vaccine’s immunogenicity. *Id.* at 6–7; Pneumococcal Package Insert at 24; Pneumococcal United States Patent, filed as Ex. 42 on Aug. 9, 2021 (ECF No. 68-2) (the “Vaccine Patent I”), at 34. Vaccine Patent I in particular confirmed that a glycerophosphate linkage is a central component of the 18C strain polysaccharide structure. Steinman Third Rep. at 7–8; J. Chang et al., *Relevance Of O-Acetyl and Phosphoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysaccharide*, 30 Vaccine 7090, 7091 (2012), filed as Ex. 43 (ECF No. 68-3) (“Chang”) (demonstrating that a phospholipid linkage is necessary for immunogenicity of these capsular polysaccharides); C. Lugowski & H. Jennings, *Structural Determination of the Capsular Polysaccharide of Streptococcus Pneumoniae Type 18C*, 131 Carbohydrate Res. (1984), filed as Ex. 44 (ECF No. 68-4) (same).

Other literature (some of which had already been filed) was referenced by Dr. Steinman to support the phosphoglycerol presence in the pneumococcal vaccine. Gilburd, for example, had found that six of the 16 patients with GBS included in the study possessed autoantibodies to various phospholipids. Gilburd at 24. Dr. Steinman admitted that Gilburd’s authors had acknowledged that “. . . no significant association was found between the presence of specific antiphospholipid antibodies (CL, PS, PE, PC) or anti-DNA antibodies and GBS when compared to controls. . . .” Gilburd at 26. He maintained, however, that an article published 12 years later occasioned a reconsideration of that statement. Steinman Third Rep. at 9–10; G. Nakos et al., *Anti-Phospholipid Antibodies in Serum from Patients with Guillain-Barré Syndrome*, Intensive Care Med. 1401, 1404 (2005), filed as Ex. 45 (ECF No. 68-5) (“Nakos”). In Nakos, Dr. Steinman maintained, “the phospholipids containing phosphoglycerol, all had higher levels of antibodies than the level of antibodies to ganglioside [a commonly understood target involved in GBS’s pathogenesis].” Steinman Third Rep. at 10. Thus, he read Nakos to better support his contention that these anti-phosphoglycerol antibodies were likely pathogenic in GBS.

A close evaluation of Nakos finds less support for the causation theory offered than Dr. Steinman proposed. In Nakos, a small sample of nine patients with the AIDP (meaning “acute inflammatory demyelinating polyneuropathy”) GBS variant had their blood tested for the presence of anti-phospholipid antibodies over a period of several days, with results revealing “a wide range” of these antibodies in comparison to controls. Nakos at 1405. Consistent with Dr. Steinman’s contention, Nakos did observe phosphatidylcholine (which has a phosphoglycerol component) as one of the main antigens in the tested sample. *Id.* at 1405–07. But Nakos’s authors did not purport

to determine the precise *role* of the autoantibodies in GBS’s pathogenesis, allowing that they could simply “represent a part of a more extensive immunoreaction that takes place in the GBS,” rather than a primary/initial causal factor. *Id.* at 1406. Thus, Nakos does not appreciably extend Gilburd’s findings.

In maintaining the potentiality for autoimmune cross-reactivity between phosphoglycerols in the vaccine antigens and phosphoglycerol groups contained in myelin phospholipids, Dr. Steinman seemed to concede that the latter was a literally small (in size) target for an antibody, but nevertheless maintained that the targeting could occur. Steinman Third Rep. at 10–11. He repeated his prior contentions regarding Ho, noting that the study showed how antibodies target the polar head group¹⁵ on phosphoglycerol (albeit only in the context of MS). Ho at 4. He also pointed to X-ray diffraction studies of monoclonal (meaning lab-created) antibodies, the results of which demonstrate that antibodies can recognize the polar head group on phosphocholine. Steinman Third Rep. at 11–13; E. Barbar et al., *Binding of Phenylphosphocholine-Carrier Conjugates to the Combining Site of Antibodies Maintains a Conformation of the Hapten*, 35 *Biochemistry* 2958, 2958 (1996), filed as Ex. 46 (ECF No. 68-6) (showing that antibodies have specificity for the polar head group). Thus, an antibody created in reaction to the polysaccharide from one pneumococcal strain (23F) had the capacity to bind to the phosphate group in the essential phosphoglycerol in the myelin.

An alternative source for mimicry leading to autoimmune cross reactivity, according to Dr. Steinman, was the CRM₁₉₇ protein conjugate. Steinman Third Rep. at 14. To identify a possible molecular congruence, Dr. Steinman conducted a “BLAST” search¹⁶ focusing on contactin-I, a molecule that some studies had identified as a target in the myelin damage central to GBS. *Id.*; Y. Miura et al., *Contactin I IgG4 Associates to Chronic Inflammatory Demyelinating Polyneuropathy with Sensory Ataxia*, 138 *Brain* 1484, 1486 (2015), filed as Ex. 49 (ECF No. 68-9). He then performed the same tabletop computer analysis consistent with what he has done in countless Program cases¹⁷ where he seeks to establish an autoimmune process driven by molecular mimicry.

¹⁵ Not defined by Dr. Steinman, but he has previously described polar head groups as “the lipid components targeted by the auto-antibodies.” *Koller v. Sec’y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947, at *10 (Fed. Cl. Spec. Mstr. Oct. 8, 2021).

¹⁶ Basic Local Alignment Search Tool (“BLAST”) is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, <https://blast.ncbi.nlm.nih.gov/Blast.cgi> (last visited Feb. 17, 2023). A BLAST search involves review of an online database to “compare[] nucleotide and protein sequences, to search for a homology between the . . . vaccine and [the body’s myelin basic protein].” *Montgomery v. Sec’y of Health & Hum. Servs.*, No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019).

¹⁷ *Schilling v. Sec’y of Health & Hum. Servs.*, No. 16-527V, 2022 WL 1101597, at *5 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (“[Dr. Steinman] also (as he has done many times before) performed online BLAST searches to identify amino acid sequence homology. . .”).

Specifically, he (a) attempted to propose the degree of amino acid homology that would be necessary, arguing that five out of a twelve amino acid string is sufficient (*Id.* at 14) (b) performed searches with the BLAST online government database for the components of the pneumococcal vaccine, to identify the amino acids comprising them, and determine whether other researchers identified the same epitope(s)¹⁸ (*Id.* at 17–19), and (c) compared them to the molecular composition in the relevant GBS target. *Id.* at 19–20. He concluded from the foregoing that “a compelling case” existed in support of his contention that “molecular mimics in the [pneumococcal vaccine] can cause GBS.” *Id.* at 19.

An additional factor, Dr. Steinman maintained, was the previously-described cross-reactive process encouraged by the pneumococcal vaccine’s alum adjuvant. Steinman Third Rep. at 20–21. By stimulating certain pro-inflammatory cytokines (IL-1 and IL-18) released in association with the innate/initial immune response, alum had the capacity to “contribute[] to the pathogenesis of GBS.” *Id.* at 20; S. Eisenbarth et al., *Crucial Role for the Nalp3 Inflammasome in the Immunostimulatory Properties of Aluminum Adjuvants*, 453 *Nature* 1122, 1122 (2008), filed as Ex. 55 (ECF No. 68-15) (“Eisenbarth”). Literature involving animal models established that IL-1 and IL-18 are upregulated during active GBS, with levels declining as the disease (which is understood to be acute and monophasic) resolves. Steinman Third Rep. at 20–21; K. Nyati et al., *Correlation of Matrix Metalloproteinases-2 and -9 with Proinflammatory Cytokines in Guillain-Barré Syndrome*, 88 *J. Neuroscience Res.* 3540, 3542 (2010), filed as Ex. 59 (ECF No. 68-19); S. Jander & G. Stoll, *Interleukin-18 Is Induced in Acute Inflammatory Demyelinating Polyneuropathy*, 114 *J. Neuroimmunology* 253, 253 (2001), filed as Ex. 60 (ECF No. 68-20). In effect, the adjuvant sets an inflammatory “stage” for pathogenesis, and therefore these pieces of literature provided “a strong scientific foundation for how the alum in the pneumococcal vaccine would lead to an inflammatory polyneuropathy.” Steinman Third Rep. at 21. Dr. Steinman did not, however, offer any items of literature showing that the alum alone, in the tiny amounts included in a vaccine, could raise cytokine levels to a pathogenic level, simply on the basis of the fact that alum activates an intracellular innate immune response. Eisenbarth at 1122.

As should be evident from the citations above, *none* of the literature offered in support of Dr. Steinman’s revised causation theory had been published in the 2017-21 timeframe, with many (if not most) of the newly-filed items published even *before* this case’s initiation. Dr. Steinman’s thinking on the causal theory in this case may have changed in four years, but nothing in the scientific or medical community’s understanding on the topic of the pneumococcal vaccine and GBS was (at this time) shown to have similarly progressed.

¹⁸ Dr. Steinman first tested the sequence WEQAKALSVE and identified it as an area of alignment between contactin-1 and CRM₁₉₇, determining it is “an epitope in diphtheria toxin, which provide the basis for CRM₁₉₇.” Steinman Third Rep. at 18. Dr. Steinman tested a second sequence, EYMAQACAGNRVRR, which also has “known cross-reactivity with epitopes described in humans and on the c. diphtheria microbe that is the basis for CRM₁₉₇.” *Id.* at 18–19.

Fourth Report

Dr. Steinman’s final expert report responds to the opinion of Respondent’s second expert, Dr. J. Lindsay Whitton—and (notably for a rebuttal opinion) is the longest report of any he submitted in the entirety of this case (calling into question why his initial efforts were thinner).¹⁹

At the outset, Dr. Steinman makes no effort to defend against Dr. Whitton’s contention that the pneumococcal vaccine does not *itself* contain phospholipids. He only devotes himself to bulwarking the contention from his third report: that *some* of the polysaccharides of the 13 *S. pneumoniae* strains in the vaccine contain *phosphoglycerol*, and that other evidence establishes an immune response to it capable of producing cross-reactive antibodies. *See, e.g.*, Steinman Fourth Rep. at 1–9.

Even with respect to this contention, however, Dr. Steinman’s arguments ignored or sidestepped a number of Dr. Whitton’s points. For example, Dr. Steinman expansively discussed literature establishing that an immune response was mounted to the 23F strain in the pneumococcal vaccine. S. 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, 4725, 4727–28 (2016), filed as Ex. 65 (ECF No. 79-4) (“Bryson”); Steinman Fourth Rep. at 1–7. Even though Bryson involved antibodies generated against antigens contained in the non-covered Pneumovax 23 version of the vaccine, both include the 23F serotype. Steinman Fourth Rep. at 2, 4, 7. And he reemphasized that phosphoglycerol is critical to the vaccine’s capacity to induce any immune response. Steinman Fourth Rep. at 5, 7–12; Chang at 7091; Pneumococcal United States Patent, filed as Ex. 63 on May 16, 2022 (ECF No. 79-2), at 15.

In so maintaining, however, Dr. Steinman made no mention of Dr. Whitton’s contention that the phosphoglycerol molecule’s small size meant that it had to be joined to a larger molecule to make the reaction possible—and in fact, that Bryson confirmed this was what occurred. Bryson at 4727–29; Whitton Rep. at 18–19. Thus, even if evidence established that *some* immune reaction to this aspect of single included *S. pneumoniae* strain was likely, this did not establish the pathologic capacity of a more targeted response to the phosphoglycerol alone (besides the fact that an antibody responding to the “phosphoglycerol plus” molecule would find and attack the same structure in the myelin phospholipids).

Independent of Dr. Whitton’s criticism, Bryson in fact says even less about causation herein than Dr. Steinman proposes. The focus of Bryson was not on the immunogenicity of the pneumococcal vaccine, how antibodies with affinity for phospholipid components might

¹⁹ I would also note that in this fourth report, Dr. Steinman repeated much of the same information discussed in his previous expert reports, rather than succinctly raising new points in light of Dr. Whitton’s criticisms.

cause/contribute to GBS, or any other issue directly relevant to this case. Rather, its authors clearly indicated that their study’s intent was to evaluate the role of *preferred genes responsible for coding antibodies to certain pathogens*. Bryson at 4724. Thus, Bryson considered two particular antibodies—one formed in response to the 23F antigenic serotype, and one formed in reaction to the wild cytomegalovirus—choosing them specifically because *both* rely on a “pair of . . . preferred genes” for their generation, even though the two pathogenic incitements are distinguishable. *Id.* at 4723–24. It authors subsequently considered the specific structures of the antibodies, permitting them to “provide structural evidence that the same . . . genes . . . can assume different roles in protective [antibodies]” due to distinct configurations—in turn establishing that “evolutionary pressure by pathogens to retain certain . . . genes can collaborate with two stochastic processes.” *Id.* at 4724. Bryson’s authors concluded that their findings “provide an example that illustrates how a limited set of human germline genes can contribute to the creation of binding suites for a variety of highly diverse [antigens].” *Id.* at 4729. Bryson seems to have only been cited by Dr. Steinman because it had a chart showing that the 23F serotype includes phosphoglycerol.

As further support for his theory, Dr. Steinman introduced a number of actual “new” articles—although all of them only discussed molecular mimics for MS and the Epstein-Barr virus infection. Steinman Fourth Rep. at 13–15; T. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis rather Bind EBV EBNA I and GlialCAM*, 603 *Nature* 1, 1 (2021), filed as Ex. 66 (ECF No. 79-5) (“Lanz”); W. Robinson & L. Steinman, *Epstein-Barr Virus and Multiple Sclerosis*, 375 *Science* 1, 1 (2020), filed as Ex. 67 (ECF No. 79-6) (“Robinson”); H. Wekerle, *Multiple Sclerosis Sparked by Virus-led Autoimmunity*, 603 *Nature* 1, 1 (2022), filed as Ex. 68 (ECF No. 79-7) (“Wekerle”). Dr. Steinman appears to have offered these articles to bulwark his endorsement of molecular mimicry as a “sound and reliable theory”—although they say nothing about its application *in the specific context of this case*, and otherwise do not make it more likely than not that the pneumococcal vaccine could cause GBS via molecular mimicry.

Dr. Steinman also devoted extensive portions of his final report to justifying his use of “BLAST” searches to identify a basis for the purported mimicry. Steinman Fourth Rep. at 15–24. Dr. Whitton had contended that one article established a proper use of these searches (in the context of identifying allergens), but revealed the importance of long homologous sequences—and thus Dr. Steinman’s willingness to embrace homology as evidence of potential cross-reactivity based on far shorter sequences (amino acids in the context of a protein such as CRM₁₉₇) was ruining his analysis due to the risk of “garbage in garbage out.” Whitton Rep. at 32–38, 40; A. Silvanovich et al., *The Value of Short Amino Acid Sequence Matches for Prediction of Protein Allergenicity*, 90 *Toxicological Sci.’s* 252, 258 (2006), filed as Ex. M, Tab 29 (ECF No. 79-29) (“Silvanovich”). In response, Dr. Steinman maintained that the criteria deemed important in Silvanovich to ensuring the reliability of homology findings were “irrelevant to how the adaptive immune system processes antigens,” (Steinman Fourth Rep. at 17), and otherwise that Dr. Steinman’s “filtration process” for

evaluating the significance of the homologies he identifies provided sufficient protection of his conclusions. Steinman Fourth Rep. at 17–24.

Regarding the argument about alum’s putative role in causing GBS, Dr. Steinman ignored Dr. Whitton’s argument that this amounted to an embrace of the discredited ASIA causation theory.²⁰ Steinman Fourth Rep. at 26. Instead, Dr. Steinman merely reiterated his prior argument that the kind of pro-inflammatory cytokines upregulated due to the inclusion of an alum adjuvant in a vaccine had been shown to be elevated in GBS patients. *Id.* Otherwise, and throughout his final report, Dr. Steinman defended the overall quality of his evidence (and indirectly the fact that he offered very little specific to the pneumococcal vaccine’s intersection with GBS). He maintained instead that he could do no more and should not be asked to in a Vaccine Program case. Steinman Fourth Rep. at 6 (stating that demonstration of antibody response to 23F antigen “takes us about as “close” as we can get to certainty”), 15 (“[t]he best that can be done in providing a sound and reliable theory is to do the in-silica computer searches that have been performed”).

B. Respondent’s Experts

1. *Timothy Vartanian, M.D., PhD.* – Dr. Vartanian submitted two expert reports on behalf of Respondent. Report, filed as Ex. A on May 26, 2017 (ECF No. 33-1) (“Vartanian First Rep.”); Report, filed as Ex. L on October 27, 2017 (ECF No. 38-1) (“Vartanian Second Rep.”). Dr. Vartanian contested Dr. Steinman’s initial medical theory, and also questioned whether the record even evinced a true neuropathy (or at least one that could be linked to the Petitioner’s receipt of the pneumococcal vaccine). Vartanian First Rep. at 10.

Dr. Vartanian received his bachelor’s degree from Oakland University, along with his medical and doctorate degree from the University of Chicago. *Curriculum Vitae*, filed as Ex. B on May 26, 2017 (ECF No. 33-2) (“Vartanian CV”) at 1; Vartanian First Rep. at 1. He completed a residency at Massachusetts General Hospital in Neurology. Vartanian CV at 2; Vartanian First Rep. at 1. He then completed two fellowships, the first at Beth Israel Hospital and the second at Harvard Medical School. Vartanian CV at 2. Since 2009, Dr. Vartanian holds positions as a Professor at Weill Cornell Medicine. *Id.* and an attending neurologist at New York Presbyterian Hospital. Vartanian CV at 2; Vartanian First Rep. at 1. He is licensed to practice medicine in New York, and is board certified by the American Board of Psychiatry and Neurology and Adult

²⁰ “ASIA” stands for “autoimmune/autoinflammatory syndrome induced by adjuvants.” Dr. Whitton is not the only person to question ASIA as a credible or scientifically-reliable causal theory—for numerous special masters have reached the very same conclusion (including myself). *See, e.g., McGuinness v. Sec’y of Health & Hum. Servs.*, No. 17-0954V, 2021 WL 5292343, at *17 n.17 (Fed. Cl. Spec. Mstr. Oct. 20, 2021); *Morris v. Sec’y of Health & Human Servs.*, No. 12-415V, 2016 WL 3022141, at * 12 (Fed. Cl. Spec. Mstr. Apr. 1, 2016); *Rowan v. Sec’y of Health & Human Servs.*, No. 10-272V, 2014 WL 7465661, at * 16 (Fed. Cl. Spec. Mstr. Dec. 8, 2014), *mot. for review den’d*, 2015 WL 3562409 (Fed. Cl. May 18, 2015); *D’Angiolini v. Sec’y of Health & Human Servs.*, No. 99-578V, 2014 WL 1678145, at *60 (Fed. Cl. Spec. Mstr. Mar. 27, 2014), *mot. for review den’d*, 122 Fed. Cl. 86 (2015), *aff’d*, 645 F. App’x 1002 (Fed. Cir. 2016).

Neurology. Vartanian CV at 3. Dr. Vartanian conducts research centered around pattern recognition receptors, specifically in the central nervous system. *Id.* at 8–10; Vartanian First Rep. at 1. He has published a substantial number of peer-reviewed articles. Vartanian CV at 13–20.

First Report

After consideration of the medical record, Dr. Vartanian explained the relevant disease and diagnosis. Vartanian First Rep. at 2–3. GBS (or the AIDP variant thought to be most common) and Chronic Inflammatory Demyelinating Polyneuropathy (“CIDP”) are related neuropathies characterized by focal segmental demyelination of peripheral nerves, which EMG/NCS studies can identify with evidence of signal “conduction block.” *Id.* at 3–4. Demyelination occurs in GBS most commonly due to an autoimmune response directed against gangliosides (molecular structures on the myelin surface), or other lipids, proteins, or carbohydrates present on myelin. *Id.* at 4. Demyelinating neuropathies show marked slowing of conduction velocities and marked increases in latencies. *Id.* at 4–5.

Although Dr. Vartanian’s report did not directly contest Petitioner’s diagnosis, he did raise several questions about its trustworthiness. First, he noted discrepancies between the EMG/NCS testing Petitioner underwent in October 2015, and what would normally be expected for persons experiencing a true demyelinating neuropathy. For example, Dr. Vartanian observed that reduced amplitudes of action potentials are seen in demyelinating neuropathies only when there is conduction block. Vartanian First Rep. at 5. But Petitioner’s EMG/NCS testing revealed no conduction block, but instead a decrease in the amplitude of the action potentials only in the sensory fibers (his motor fibers were normal). *Id.* at 4.

Second, Dr. Vartanian highlighted other findings that suggested alternative explanations for Petitioner’s illness. On November 25, 2015, there was a discussion among treaters about the existence of hyponatremia²¹ and hypoosmolality based on testing results.²² Vartanian First Rep. at 6.; Ex. 12 at 2. Hyponatremia is known to have a negative effect on neuropathies, and “proton pump inhibitor” drugs intended to treat acid reflux or other gastric disorders (and which Petitioner was then receiving) are specifically associated with hyponatremia. Vartanian First Rep. at 6. Thus, Dr. Vartanian deemed it unwise to have placed Petitioner on such a drug, since it likely exacerbated his condition. *Id.*; G. Bahat, *Risk of Proton Pump Inhibitor–Induced Mild Hyponatremia in Older Adults*, 62 *J. Am. Geriatrics Soc’y* 1206, 1206–07 (2014), filed as Ex. C (ECF No. 33-3); M.

²¹ Hyponatremia is a “deficiency of sodium in the blood.” *Hyponatremia*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=24305&searchterm=hyponatremia> (last visited Feb. 17, 2023).

²² Hyposmolality is “abnormally decreased osmolar concentration.” *Hyposmolality*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=24393&searchterm=hyposmolality> (last visited Feb. 17, 2023).

Naherty et al., *Pantoprazole Sodium–Induced Hyponatremia in a Frail Elderly Adult*, 62 J. Am. Geriatrics Soc’y 787, 787–88 (2014), filed as Ex. D (ECF No. 33-4); G. Liamis et al., *A Review of Drug-Induced Hyponatremia*, 52 Am. J. Kidney Diseases 144, 150 (2008), filed as Ex. E (ECF No. 33-5). Petitioner’s treaters also observed evidence of increased Coproporphyrin I and Coproporphyrin III²³—and although Dr. Vartanian characterized this to be a finding of unknown significance, he also emphasized that elevated porphyrin levels are associated with diseases of the peripheral and central nervous systems. Vartanian First Rep. at 6; Ex. 4 at 113; Ex. 10 at 3.

Despite the above, Dr. Vartanian’s questioning of the neuropathy diagnosis ultimately turned on his interpretation of the EMG/NCS testing. Vartanian First Rep. at 9. By the time the EMG/NCS was performed, and given the nature of Petitioner’s symptoms, there should have been clear evidence of a demyelinating neuropathy. Yet, the EMG/NCS evidence indicated an *axonal* neuropathy. *Id.*

Dr. Vartanian went on to discuss Dr. Steinman’s initial theory. While he did not review the process of molecular mimicry in detail, he acknowledged that the concept finds medical/scientific acceptance as relevant in *some* pathologic processes. Vartanian First Rep. at 6. He also agreed that the general immunologic mechanisms leading to autoimmunity following vaccination would entail the same mechanisms leading to autoimmunity following infection. *Id.* However, he listed four criteria that must be satisfied in order to reliably conclude that the mechanism of molecular mimicry has explanatory power in understanding a disease process: (1) establishment of an underlying epidemiological association between the infectious agent and the disease in question; (2) identification of T cells or antibodies directed against target antigens associated with the disease; (3) identification of microbial mimics of the target antigen introduced by the exogenous antigen; and (4) reproduction of the theoretic disease process in an animal model. *Id.*; Yuki at 706. The contention that an infectious agent (or vaccine) contains one or more epitopes that share structural characteristics with epitopes on host molecules may have a baseline plausibility, but such a possibility alone cannot *prove* pathophysiologic causality without other factors being established. Vartanian First Rep. at 7.

Dr. Vartanian also raised three key issues relevant to contentions that an autoimmune disease process (whether driven by B or T cells) is occurring. Vartanian First Rep. at 7. First, he observed that autoreactive cells and antibodies are widespread in humans. *Id.* at 7–8; E. Ellwardt et al., *Understanding the Role of T Cells in CNS Homeostasis*, 37 Trends Immunology 154, 160 (2016), filed as Ex. G (ECF No. 33-7); D. Richards et al., *Re-Examining the Nature and Function*

²³ Coproporphyrin is defined as “a porphyrin produced by oxidation of the methylene bridges in coproporphyrinogen. Four isomers are possible, but only two exist naturally. *Coproporphyrin III* is excreted in the feces and urine in hereditary coproporphyria and variegate porphyria, particularly during acute attacks. *Coproporphyrin I* is excreted in the feces and urine in congenital erythropoietic porphyria.” *Coproporphyrin*, Dorland’s Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=11082&searchterm=coproporphyrin> (last visited Feb. 17, 2023).

of Self-Reactive T Cells, 37 Trends Immunology 114, 116–17 (2016), filed as Ex. H (ECF No. 33-8). But their mere presence does not automatically connote pathogenicity. Vartanian First Rep. at 8. Second, he noted that naturally-occurring, self-reactive antibodies are actually thought to *protect* against many of the toxic compounds that emerge with aging. *Id.* One study found that self-reactive antibodies against beta amyloid²⁴ (the proposed host-produced molecule that is pathogenic in Alzheimer's disease), are protective, helping to clear away toxic protein. S. Sollvander et al., *Increased Number of Plasma 8 Cells Producing Autoantibodies Against A β ₄₂ Protofibrils in Alzheimer's Disease*, 48 J. Alzheimer's Disease 63, 67–69 (2015), filed as Ex. J (ECF No. 33-10). And third, these same autoreactive cells and antibodies are also present in people with autoimmune diseases. Vartanian First Rep. at 8. As a result, “fishing for auto-reactive cells or antibodies in someone with bona fide autoimmune disease such as GBS is scientifically insufficient” to prove that such immune cells are driving the disease process. *Id.* at 8–9; Gilburd at 23 (citing the abstract).

Dr. Vartanian did not discuss Petitioner's onset, other than to note that the time between vaccine administration and the onset of his symptoms was within a reasonable range, although this was meaningless given his view that the underlying theory had not been adequately substantiated. Vartanian First Rep. at 9.

Second Report

Dr. Vartanian's second report addressed succinctly some of the contentions in Dr. Steinman's supplemental report.

First, Dr. Vartanian revisited questions about the findings from Petitioner's October 2015 EMG, completed three months after diagnosis and also after five days of IVIG treatment. Ex. 10 at 3; Vartanian Second Rep. at 1. In reaction to Dr. Steinman's argument that the timing of this testing (coupled with IVIG treatment) meant that it inherently was less likely to reveal neuropathic signal/conduction issues, Dr. Vartanian maintained that even weeks or months after neuropathy onset, EMG/NCS testing should yield abnormal results if the patient remains symptomatic, as here. *Id.*

Dr. Vartanian also disagreed with Dr. Steinman's assertion that treater interpretations of the EMG results were consistent with neuropathy. In Dr. Vartanian's view, Dr. Shah had referred to the diagnosis as sensory predominant solely because in his interpretation of the data, sensory fiber abnormalities dominated the pathology. Vartanian Second Rep. at 1. This did not mean that the results otherwise showed motor conduction loss, as well. He also observed that Dr. Shah had

²⁴ Amyloid beta is defined as “an abnormal, neurotoxic peptide, either 40 or 42 amino acids in length, found in aggregates in the cerebrovascular walls and the cores of the plaques in Alzheimer disease.” *A β amyloid*, Dorland's Medical Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=55861> (last visited Feb. 17, 2023).

written that the f-wave latencies could suggest a more proximal lesion, but in Dr. Vartanian's understanding a diagnosis from an EMG/NCS is not made based on a singly inquiry of f-waves, h-reflexes, or compound motor action potential amplitudes, but is instead the aggregate of findings and the clinical setting. *Id.* at 2. Thus, Dr. Vartanian maintained again that the overall EMG findings were not fully supportive of an AIDP-like neuropathy.

Second, Dr. Vartanian defended his prior assertions that insufficient evidence supported molecular mimicry as a reliable explanation in this context for Petitioner's alleged vaccine-caused injury. Vartanian Second Rep. at 1–2. He was not demanding that Dr. Steinman provide research-oriented findings specific to the Petitioner that were impossible to obtain, but rather was noting an absence of independent evidence supporting Dr. Steinman's causal contentions. *Id.* at 2.

2. *J. Lindsay Whitton, M.D., Ph.D.* – Dr. Whitton, an immunologist, submitted an expert report for the Respondent in support of the argument that the pneumococcal vaccine cannot cause GBS. Report, dated April 29, 2022, filed as Ex. M (ECF No. 77-1) (“Whitton Rep.”). Dr. Whitton opined that the initial version of Dr. Steinman's theory was foundationally erroneous, and he deemed baseless the contention that the phosphoglycerol/glycerophosphate-containing²⁵ polysaccharides in certain pneumococcal vaccine antigens could induce an antibody response that causes GBS.

Dr. Whitton obtained his undergraduate, medical, and doctorate degrees from the University of Glasgow in Scotland. *Curriculum Vitae*, filed as Ex. N on April 29, 2022 (ECF No. 77-2) (“Whitton CV”) at 1; Whitton Rep. at 1. He then began working as a senior research associate at the Scripps Research Institute in La Jolla, California, where he studied immunology, vaccinology, and viral pathogenesis. Whitton CV at 1; Whitton Rep. at 1. Dr. Whitton has conducted extensive research in these subject areas and has published numerous articles on the subjects. Whitton CV at 2–15. In addition to his research, Dr. Whitton also serves as a professor in the department of Immunology and Microbial Science at Scripps Research Institute. Whitton CV at 1. In both his research and teaching, Dr. Whitton has focused on viral pathogenesis, innate and adaptive immune responses, and molecular mimicry. Whitton Rep. at 1–2. Dr. Whitton does not have board certification in the United States to practice medicine. *Id.* at 2.

Dr. Whitton did not offer an opinion as to the propriety of Petitioner's diagnosis,²⁶ but instead focused on the issue of causation. He began with a lengthy discussion of the pneumococcal

²⁵ Dr. Steinman's reports frequently employ the term phosphoglycerol, but Dr. Whitton argued that he actually meant to refer to *glycerophosphate*. Whitton Rep. at 5 n.e.

²⁶ Dr. Whitton did include a review of the features of GBS. *See* Whitton Rep. at 8–11. I will refer elsewhere to some of the points he made therein, although otherwise this aspect of his opinion does not require summary (and clinical aspects of GBS are topics beyond his expertise in any event).

vaccine itself. Whitton Rep. at 3–8. The vaccine aims to immunize against a bacterial infection caused by *Streptococcus pneumoniae*, or “pneumococcus.” *Id.* at 3. The *S. pneumoniae* bacterium is surrounded by a “capsule” made of polysaccharides.²⁷ *Id.* at 3–4. There are close to 100 serotypes²⁸ of pneumococcus, which are distinguished based on their having structurally-different polysaccharide capsules. *Id.* at 5. Some *S. pneumoniae* polysaccharides (including strains included in the Prevnar form of the pneumococcal vaccine) contain modifications, such as the chemical linkage of glycerophosphate (which Dr. Whitton deemed a “small molecule”). *Id.*

In addition, Dr. Whitton explained the “conjugated” character of the pneumococcal vaccine. Although adults can mount a robust immune response when exposed simply to the naked bacterial polysaccharides, it has been found that children (especially infants) do not. Whitton Rep. at 6. As a result, to boost the immune response (and in particular the intended production of antibodies to the *S. pneumoniae* bacterium), the vaccine’s antigenic components are linked, or conjugated, to a protein that “can trigger a reasonably strong T cell response” (and in this case Dr. Whitton referred to “T helper cells”—a lymphocyte that assists B cells in production of antibodies).²⁹

Only the conjugated version of the pneumococcal vaccine is “covered” by the Vaccine Program,³⁰ and many studies have noted not only its general safety, but the fact that it (or the antigenic components common to both versions) is not associated with an increased risk of disease—including GBS. Whitton Rep. at 7-8; R. Baxter et al., *Lack of Association of Guillain-Barre Syndrome with Vaccinations*, 57 *Clinical Infection Diseases* 197, 201 (2013), filed as Ex. M, Tab 6 (ECF No. 78-6) (“Baxter”) (Pneumovax 23 vaccine (containing 12 of the 13 Prevnar antigenic components) poses no increased risk of GBS); P. 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–*

²⁷ Polysaccharides are made up of sugar molecules and occur throughout nature. Whitton Rep. at 3–4. This kind of molecule is distinguishable from a protein (comprised of chains of amino acids). *Id.* Amino acids contain only two attachment points, so the amino acid chains have a linear character. *Id.* at 4. Sugars, on the other hand, have several different points at which they can link, resulting in different molecular orientations. *Id.* at 4–5; J. Prestegard et al., *Oligosaccharides and Polysaccharides*, *Essentials of Glycobiology* 1, 4 (2017), filed as Ex. M, Tab 1 (ECF No. 78-1). Thus, the pneumococcal vaccine will “teach” the immune system to respond to specific strains it “sees” (based on the vaccine’s formulation), but not others. Whitton Rep. at 5.

²⁸ According to Dr. Whitton, serotype means “this organism is distinguished from related organisms on the basis of how it is recognized by the antibody response.” Whitton Rep. at 5.

²⁹ For a discussion of the conjugate’s function, see *Bielak v. Sec’y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *31 (Fed. Cl. Spec. Mstr. Dec. 9, 2022).

³⁰ Another version—Pneumovax 23—is not similarly conjugated, and because it is only recommended for adults, cannot be the basis of a Program claim. Whitton Rep. at 6; *Louvaris v. Sec’y of Health & Hum. Servs.*, No. 21-416V, 2021 WL 4955690, at *1–2 (Fed. Cl. Spec. Mstr. Sept. 27, 2021)

December 31, 2015, 34 Vaccine 6330, 6330, 6333–34 (2016), filed as Ex. M, Tab 7 (ECF No. 78-7) (“Haber”) (noting no reported relationship between the pneumococcal vaccine and GBS through a data mining analysis of VAERS³¹ data); H. Tseng et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults*, *Open Forum Infectious Diseases* 1, 1 (2018), filed as Ex. M, Tab 8 (ECF No. 78-8) (“Tseng”) (“[t]hese data do not support an increased rate of adverse events following PCV13 administration in elders compared with PPSV23 and should provide reassurance regarding continued use of PCV13”).

Dr. Whitton then turned to the primary contentions contained in Dr. Steinman’s reports. He spent little time with the first iteration of Petitioner’s causal theory: that “phospholipids” in the pneumococcal vaccine could mimic phospholipid components or structures in the nerve myelin, leading to an autoimmune cross-reactive process. In Dr. Whitton’s estimation, this version of the theory was utterly baseless, for the simple reason that the vaccine could not reasonably said to *contain* phospholipids themselves. Whitton Rep. at 14–15. Rather, Dr. Steinman appeared to have assumed that materials present “in the intact bacterium” were also found in the vaccine, when they were not, or had confused the names of different kinds of molecules (specifically, phosphocholine—not a phospholipid, with phosphatidyl choline (which is)). *Id.* at 15. And Dr. Whitton otherwise noted that Dr. Steinman had provided no affirmative proof for his underlying contention that phospholipids were found in the vaccine. *Id.*

By contrast, Dr. Whitton admitted that between two and four of the Prevnar vaccine’s 13 strains possessed a *glycerophosphate*-containing polysaccharide—opening the door to Dr. Steinman’s theory (set forth in his revised third report) that this molecule could cause an immune response, resulting in the production of antibodies to it that would in turn cross-react with mimicking structures *contained* in myelin phospholipids. Whitton Rep. at 17.³² But Dr. Whitton still found significant fault in the theory—for reasons both general and specific.

First, Dr. Whitton raised a fundamental question: if Dr. Steinman’s theory about the pneumococcal vaccine had validity, a wild *S. pneumoniae* infection should also be capable of causing GBS (presumably by triggering the same autoimmune response via molecular mimicry, since the glycerophosphates should be found in the wild bacterium antigens). Whitton Rep. at 6. However, there is no convincing scientific evidence linking prior *S. pneumoniae* infection to GBS.

³¹ VAERS is a database maintained by the CDC to compile information from reports about reactions to immunizations listed on the Vaccine Injury Table, 42 U.S.C. § 300aa–14(a). *See About VAERS*, VAERS, <https://vaers.hhs.gov/about.html> (last visited Feb. 17, 2023).

³² Dr. Whitton noted, however, that Dr. Steinman baselessly contended that *all* the vaccine’s 13 polysaccharide components were conjugated to CRM₁₉₇ “via phosphoglycerol.” Whitton Rep. at 22. But if the phosphoglycerol served this specific function, it would be altered in the process—thus eliminating the vaccine’s antigenicity (since Dr. Steinman had otherwise maintained that conservation of “glycerol-phosphate” in the vaccine was critical to its function). *Id.* at 23 (*citing* Chang at 7090).

Id. at 6, 9. Rather, a large number of *Jasti* kinds of infections, viral or bacterial, had been associated with GBS, but no mention was ever made of pneumococcus. *Id.* at 9–10. He differentiated pneumococcus from another bacterium, *C. jejuni*, far more understood to be associated with GBS. *Id.* at 9; A. Jasti et al., *Guillain-Barre Syndrome: Causes, Immunopathogenic Mechanisms and Treatment*, 12 *Expert Rev. Clinical Immunology* 1175, 1176 (2016), filed as Ex. M, Tab 19 (ECF No. 78-19) (“Jasti”) at 1176 (finding that a *C. jejuni* infection is believed to precede 30–40 percent of GBS cases).³³

This raised another problem, from Dr. Whitton’s perspective, for a causal theory proposing that antigens derived from *S. pneumoniae* could be as causal of GBS as different bacteria like *C. jejuni*. Some bacteria known to be associated with GBS are “gram negative,”³⁴ (meaning their poly saccharide capsule is “thin”) but *S. pneumoniae* is “gram positive,” or thick. Whitton Rep. at 10. Indeed, all but one of the wild bacteria already understood to be potentially causal of GBS are gram negative. Whitton Rep. at 10; Jasti at 1176. The absence of any standard gram-positive bacteria from the list is striking, according to Dr. Whitton, and is consistent with the notion that bacteria like *S. pneumoniae* do not contain the molecules necessary to trigger GBS. Whitton Rep. at 11.

Second, Dr. Whitton questioned whether the molecular mimicry mechanism relied upon by Dr. Steinman could be applied in every disease context. He did not dispute the theory’s core scientific reliability, or that GBS can in some cases be mediated by a molecular mimicry process involving cross-reactive antibodies to different pathogens. Whitton Rep. at 8. But molecular component homology (especially when only short sequences were considered) is common in nature, with “no meaningful biologic effect.” *Id.* at 36. As a result (and somewhat parallel to what Dr. Vartanian had noted), a number of intermediate “steps” were required for molecular mimicry not only to occur *but also* to result in disease. Whitton Rep. at 12–13. This made it “extraordinarily difficult” for molecular mimicry to result in autoimmune disease. Dr. Whitton noted that well-regarded experts on the topic (such as the late Dr. Noel Rose, the “father of autoimmunity”) had

³³ Dr. Whitton also provided an intriguing hypothesis for why the *C. jejuni* bacterium would be causal of GBS. As he explained, gangliosides on nerve myelin (which are understood to be a common target for cross-reactive antibodies in GBS) might be mimicked by “ganglioside-like structures” that *C. jejuni* causes to be expressed—meaning that antibodies the body generates to an infection of that bacterium end up cross-reacting with the host gangliosides. Whitton Rep. at 9; I. Nachamkin et al., *Campylobacter Species and Guillain-Barré Syndrome*, 11 *Clinical Microbiology Rev.*’s 555, 557 (1998), filed as Ex. M, Tab 20 (ECF No. 78-19). This offers yet another distinction between infectious bacteria known to be associated with GBS and *S. Pneumoniae*—since Dr. Steinman has not proposed in this case that the pneumococcus bacterium does the same (and in fact proposes a different target antigen entirely).

³⁴ Bacteria can be subdivided into two groups based on their cell walls, using a procedure called gram staining. Whitton Rep. at 10–11. Gram-positive bacteria have thick capsules with no outer membrane, and *S. pneumoniae* in particular is composed mainly of polysaccharides that are used in the pneumococcal vaccine. *Id.*; G. Zhang et al., *On the Essentiality of Lipopolysaccharide to Gram-Negative Bacteria*, 16 *Current Op. Microbiology* 779, 779 (2013), filed as Ex. M, Tab 21 (ECF No. 78-21) (“Zhang II”). In contrast, gram-negative bacteria have a flimsy capsule that is surrounded by an outer membrane. Whitton Rep. at 10–11; Zhang II at 779.

expressed skepticism about how widely the theory could be applied, even if the theory had underlying reliability. N. Rose & I. Mackay, *Molecular Mimicry: A Critical Look at Exemplary Instances in Human Diseases*, 57 Cellular & Molecular Life Sci.'s 542, 542 (2000), filed as Ex. M, Tab 22 (ECF No. 78-22) (“[t]here are, as yet, no firm instances of molecular mimicry by microorganisms serving as the initiating agents of human autoimmune disease, although a great deal of attention has been paid to this issue”).

Another particularly important sub-component of any theory relying on molecular mimicry as causing a disease like GBS was the need to identify a host target for the autoimmune attack—a locus containing some molecular structure resembling the antigenic component of an infection (or, as alleged here, vaccine). Whitton Rep. at 12–13. But Dr. Steinman had not established that phospholipids associated with nerve myelin *were* such likely targets. *Id.* at 19–21. To support this contention, Dr. Steinman had referenced Ho, even though it was specific to MS. *Id.* at 19; Ho at 1. A second such article, Kanter, not only also involved only MS, but actually differentiated between the antigenic target in GBS and other autoimmune diseases. Kanter at 138 (“[a]utoimmune responses directed against phospholipids and gangliosides contribute to the pathogenesis in systemic lupus erythematosus and [GBS], *respectively*”) (emphasis added).³⁵

In fact, Dr. Whitton maintained, it was well understood by medical science that gangliosides were a likely target for cross-reactive antibodies in the pathogenesis of GBS. Whitton Rep. at 9; Jasti at 1177; R. Hughes & J. Rees, *Clinical and Epidemiologic Features of Guillain-Barre Syndrome*, 176 J. Infectious Diseases S92, S92 (1997), filed as Ex. M, Tab 13 (ECF No. 78-13) (“Hughes I”); J. Kwan & S. Biliciler, *Guillain-Barre Syndrome and Other Acute Polyneuropathies*, 37 Clinics Geriatric Med. 313, 318 (2021), filed as Ex. M, Tab 26 (ECF No. 78-26) (“Kwan”). By contrast, such authorities made no mention at all of phospholipids or phosphoglycerol as a putative autoimmune target. Whitton Rep. at 30; R. Hughes et al., *Guillain-Barré Syndrome in the 100 Years Since its Description by Guillain, Barré and Strohl*, 139 Brain 3041 (2016), filed as Ex. M, Tab 24 (ECF No. 78-24) (“Hughes II”); (numerous mention of anti-ganglioside antibodies as driving GBS, but no mention at all of antibodies targeting phosphoglycerols); Kwan at 318 (“[i]t is postulated that the epitopes on the surface of pathogens mimic components of the *peripheral nerve ganglioside*, triggering an aberrant activation of the immune system”) (emphasis added). Otherwise, Dr. Steinman’s evidence only showed that anti-phospholipid antibodies could be *found* in the blood sera of GBS patients—*not* that these antibodies were primarily causal of GBS at its outset, or could even be said to have any demonstrated relationship to disease severity. *See generally* Whitton Rep. at 29; Gilburd at 27; Nakos at 1401, 1406.

³⁵ The term “respectively,” it should be noted, functions in the quoted sentence from Kanter to distinguish between the antigenic target in lupus (phospholipids) and that of GBS (gangliosides). It cannot be reasonably parsed to mean that *both* targets are equally relevant to these two disparate autoimmune diseases.

Dr. Whitton also focused specifically on Dr. Steinman’s core contention reflected in his third report: that phosphoglycerol/glycerolphosphate found in a minority of the *S. pneumoniae* strains contained in the vaccine could prompt an autoimmune antibody reaction. Whitton Rep. at 17–30. First, he observed that the relatively small size of the phosphoglycerol molecule made it “extremely unlikely” that it could trigger an immune response leading to production of an antibody that would in turn (mistakenly) recognize a host tissue mimic on the myelin. *Id.* at 17. The established size of the molecule was inconsistent with what science viewed as necessary to induce an antibody response. *Id.* at 17–18. Dr. Whitton conceded that attachment of phosphoglycerol to a larger molecule made such a reaction more possible, and that some literature offered by Petitioner (Bryson, for example) supported the contention—but then the purported cross-reaction would require an antibody to recognize the same “phosphoglycerol plus carrier molecule” combination in a host tissue (something he maintained Dr. Steinman had not at all demonstrated). *Id.* at 18–19.

Second, Dr. Whitton deemed it speculative for Dr. Steinman to presume that phosphoglycerol was the ultimate target for the proposed cross-reactive autoimmune attack. Much of the literature Dr. Steinman relied upon, like Ho, was specific to MS and “brain lipids,” and thus was being stretched to cover the distinguishable context of a peripheral neuropathy. Whitton Rep. at 23. Indeed, Ho made no mention at all of phosphoglycerol. *Id.* And although Dr. Steinman employed an imprecise array of terms to cover the antigens he deemed responsible for this mimicking autoimmune process (phosphocholine, phosphoglycerol/glycerolphosphate, etc.), only one was “truly common to some phospholipids and to the glycerolphosphate-containing bacterial polysaccharides”—but it was also so “ubiquitous in biology” that the theory in effect led to the possibility of “many billions of potential targets” and far more widespread occurrences of disease due to molecular mimicry than were actually experienced. *Id.* at 24.

In fact, Dr. Whitton maintained, Dr. Steinman had not persuasively established *any* likely target host antigen for this purported cross-reaction by antibodies responding to the vaccine’s phosphoglycerol sub-components. Whitton Rep. at 19–30. Dr. Steinman (relying on articles like Ho, Gilburd, or Nakos) maintained that the sera of GBS patients could be shown to include antibodies to “various phospholipids.” Nakos at 1405–07.³⁶ But Dr. Whitton proposed in response (comparable to his argument that the vaccine did not literally contain “phospholipids”) that the host tissue phospholipids themselves *likely did not contain* the purportedly cross-reacting phosphoglycerol. Whitton Rep. at 25. Putting aside the generally low possibility that phosphoglycerol found in a *polysaccharide* would generate an antibody that would in turn react to phosphoglycerol found in a *lipid*,³⁷ the alleged target host phospholipids were, at best, “built” from

³⁶ Even when an anti-phospholipid antibody could be demonstrated to exist, it was not clear, Dr. Whitton contended, why some antibodies arguably “recognized” phospholipids while others did not. Whitton Rep. at 29–30; Ho at 8.

³⁷ A lipid, it bears noting, is a class of organic compounds including fatty acids or their derivatives that are insoluble in water but soluble in organic solvents. *Lipid*, Dorland's Medical Dictionary Online,

glycerophosphate whose chemical structure was altered in the course, meaning that “what is left is no longer glycerophosphate.” *Id.* at 25, 26. In effect, like an egg used to bake a cake, the molecular “ingredients” were transformed into something different than their original constituency. *Id.* at 25. Thus, any glycerophosphate attached to one of the vaccine’s polysaccharides would not possess a mimic in the host tissue phospholipid. *Id.* at 27–28. At bottom, “Dr. Steinman’s approach to biochemistry, chemistry, and molecular structure . . . is inaccurate.” *Id.* at 28.

Dr. Whitton called into question the remaining aspects of Dr. Steinman’s theory. He disputed, for example, the contention that the vaccine’s conjugate contained a mimic to contactin-I (a nervous system protein) sufficient for antibodies generated in response to the vaccine to cross-react. Whitton Rep. at 30–31. In so doing, he commented at length on the limitations of Dr. Steinman’s use of BLAST searches³⁸ to identify homologous amino acid sequences, noting that (a) the BLAST database was arguably being improperly used by Dr. Steinman “to try to identify possibly-immunologically-significant tracts of protein,” when the database served a more general purpose, and (b) the short homologic sequences that Dr. Steinman looked for and defended as meaningful were not deserving of “immunological weight.” *Id.* at 32–33. But Dr. Whitton also argued that the homologic amino acid sequences Dr. Steinman found between contactin and CRM₁₉₇ were “cherry picked,” with Dr. Steinman focusing only on the results that supported his contentions while ignoring the unhelpful results. *Id.* at 34–36.

Ultimately, Dr. Whitton concluded that no evidence beyond the homologies revealed via BLAST searches supported the possibility of a cross-reaction with the conjugate. Dr. Steinman had offered a paper specific to evaluating the parts of a diphtheria protein recognizable by human T helper cells. Whitton Rep. at 40; R. Raju et al., *Epitopes for Human CD4+ Cells on Diphtheria Toxin: Structural Features of Sequence Segments Forming Epitopes Recognized by Most Subjects*, 25 Eur. J. Immunology 3207, 3207 (1995), filed as Ex. 54 (ECF No. 68-14) (“Raju”). But the CRM₁₉₇ protein was distinguishable,³⁹ and Raju did not show that antibodies generated to it *specifically* would cross-react with contactin (let alone via a short amino acid sequence), but instead focused on the generation of T helper cells in reaction to the diphtheria toxin *itself*. Whitton

<https://www.dorlandsonline.com/dorland/definition?id=28385&searchterm=lipid> (last visited Feb. 17, 2023). This is wholly different from a sugar.

³⁸ Dr. Whitton similarly criticized Dr. Steinman’s “filter funnel” framework for how he reasoned his way to the conclusion that the homologies he demonstrated via BLAST searches reliably established a likely cross-reaction. Whitton Rep. at 38-40. He deemed this analytic approach to not only include a number of faulty stages (such as the very effort of looking for homologous sequences), but ultimately reflected circular logic, allowing Dr. Steinman to reach in any case the result most favorable to a petitioner. *Id.* at 40.

³⁹ As I noted in a different decision, CRM₁₉₇ is lab-created, and although it is comparable to diphtheria toxin, it is not the same, reducing the value of analogizing the results of research articles like Raju. *Deshler*, 2020 WL 4593162, at *11.

Rep. at 41. Indeed, the stimulation of helper cells by diphtheria was a foregone conclusion intended by its inclusion in the vaccine—and so Dr. Steinman’s reference to it was not some critical insight showing how the conjugate might aberrantly spark an autoimmune disease. *Id.* at 42–43.

The idea that the vaccine’s alum adjuvant could amplify a presumed autoimmune reaction by enhancing neuroinflammation was similarly dismissed by Dr. Whitton. He noted that Dr. Steinman offered no literature on the contention specific to vaccination and GBS, but rather relied on broader statements about the effect of adjuvants in vaccines generally—to stimulate the kind of pro-inflammatory cytokines that can be seen also in some autoinflammatory diseases like GBS. Whitton Rep. at 43. But these two propositions were not linked with independent persuasive evidence showing a causal relationship. In effect, Dr. Whitton deemed this argument to amount to a contention made in Program cases by other experts that vaccines can be causal of disease via “autoimmune/autoinflammatory syndrome induced by adjuvants,” or “ASIA”—a concept Dr. Whitton proposed possessed “numerous fatal flaws,” and that had been “widely discredited.” *Id.* at 43.

III. Procedural History

As noted, the Petition in this matter was filed nearly seven years ago. Pet. at 1. Respondent filed a Rule 4(c) Report on October 3, 2016, contesting Petitioner’s right to compensation. ECF No. 16. Expert reports were filed through the summer of 2021. After the matter was transferred to me (on March 3, 2021), I held a status conference with the parties and subsequently set a schedule for a ruling on the record. The parties fully briefed the matter and submitted an additional round of expert reports by June 2022, and it is now ripe for resolution. ECF Nos. 72, 76–77, 79–80.

IV. Parties’ Arguments

A. Petitioner

Petitioner maintains that he has met his causation-in-fact burden based on the factors established by the Federal Circuit in *Althen v. Sec’y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005); Mot. at 17–24; Reply at 2–12. Statements from Petitioner’s expert, he purports, support his contention that the pneumococcal vaccine can cause GBS through the theory of molecular mimicry—specifically, that the phosphoglycerol in the vaccine mimics phospholipids within myelin, thereby initiating an autoimmune reaction (a theory that was first introduced in Dr. Steinman’s Third Expert Report). Mot. at 6–9, 17–18; Reply at 4. Petitioner’s expert also identified a second potential mimic between the CRM₁₉₇ protein component used to conjugate the pneumococcal polysaccharides in the vaccine and a human immune response that attack the contactin-1 molecule by employing a “filter funnel” approach that utilizes BLAST searches. Mot. at 9; Reply at 7. He also argues that the aluminum adjuvant present in the vaccine is a known stimulant of cytokines that contribute to GBS. Mot. at 9; Reply at 4–5.

Mr. Trollinger next claims that he has demonstrated a logical sequence of cause and effect that the pneumococcal vaccine “did cause” his injuries. He relied on findings in another case to analogize his own showing as similarly successful. *Maloney v. Sec’y of Health & Hum. Servs.*, No. 19-1713V, 2022 WL 1074087, at *32–35 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (finding three principal reasons to satisfy the second *Althen* prong: 1) petition was appropriately diagnosed with GBS and proffered a sound and reliable mechanism of vaccine causation; 2) statements offered by petitioner’s treating physicians provided supportive evidence; and 3) the evidence did not support an alternative cause for petitioner’s injury); Mot. at 18–21; Reply at 8–9. Finally, the timing of his onset—approximately 10 days after receiving his pneumococcal vaccine—constituted in Petitioner’s view a medically-acceptable timeframe, given filed literature on the timeline consistent with GBS and the 1976 swine flu vaccinations (even though the flu vaccine is not at issue in this case). Mot. at 21; Reply at 9–10. Reply at 14–15. Petitioner relied upon previous pneumococcal/GBS cases, which utilized the evidence presented in Schonberger to support the third *Althen* prong. *Maloney*, 2022 WL 1074087, at *36; *Koller v. Sec’y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947, at *20–23 (Fed. Cl. Spec. Mstr. Oct. 8, 2021) (citing Schonberger with acknowledgement from both experts that there is a lack of other studies that would provide a timeframe for the onset of GBS following vaccination); Mot. at 21–22; Reply at 9–10.

Petitioner also specifically addressed a 2020 GBS/pneumococcal case in which I denied entitlement, in an effort to differentiate this case and the showing he has made herein. *Deshler v. Sec’y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162 (Fed. Cl. Spec. Mstr. July 1, 2020). Petitioner’s expert was not involved in *Deshler*, he notes, and Dr. Steinman’s analysis of phosphoglycerol as the agent of molecular mimicry was not posited. Reply at 11–12. For these two reasons, Petitioner argues that *Deshler* is not relevant to my analysis. *Id.* Instead, he argues that *Koller*—another case decided by a different special master that granted entitlement—should cast guidance on this determination. *Koller*, 2021 WL 5027947. Both cases involved Petitioner’s expert and one of Respondent’s experts in this case (Dr. Whitton), argued the same proposed theory, and used the same literature. Reply at 12.

B. Respondent

In opposing entitlement, Respondent argues that the *Althen* prongs have not been satisfied. Opp. at 11–23. Thus, Petitioner has not preponderantly established a reliable medical theory causally connecting his vaccination to GBS as the theory is too generic to be persuasive in a specific case and the underlying theories have a host of deficiencies. *Id.* at 11. Respondent argues it is insufficient to simply demonstrate some evidence of shared homology and the presence of autoreactive T cells, because while molecular mimicry exists, it rarely leads to disease. *Id.* at 14–15. Additionally, Dr. Steinman’s self-fulfilling BLAST searches are not probative of causation. *Id.*

at 16. In all, Petitioner’s expert provides no scientific support for any association between the pneumococcal vaccine and GBS, but relies on data that exists from the flu vaccine, and he does not present literature discussing the role of alum in GBS specifically even though Respondent’s expert emphasized that alum adjuvants have an excellent safety record. *Id.* at 12–13, 16–17.

Under *Althen* prong two, Respondent argues, Petitioner puts forth no independent evidence, but instead relies on evidence for prongs one and three. *Opp.* at 20. Although some treaters have referred to the temporal association between receipt of vaccination and development of GBS, these notations are general and do not identify the precise vaccine or date received. *Id.* Similar to *Deshler*, Respondent argues that “although the treater views in this case do aid Petitioner’s showing, they ultimately relied too much on the obvious temporal relationship between vaccination and injury to carry Petitioner’s ‘did cause’ burden.” *Deshler*, 2020 WL 4593162, at *21. And Petitioner’s showing under *Althen* prong three also fails, because it relies on Schonberger, which links GBS and the 1976 swine flu vaccine—a totally different vaccine that does not contain any aluminum adjuvants—like the vaccine herein. *Id.* at 21. Respondent further contends that the Petitioner here relied on a similar theory used in *Deshler*, and that Petitioner has not offered any compelling reasons for the Court to reach a different conclusion in this case. *Reply* at 16.

V. Applicable Law

A. Standards for Vaccine Claims

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a “Table Injury”—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a “Non-Table Injury”). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec’y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).⁴⁰ In this case, Petitioner cannot assert a Table claim (as there is no such claim with respect to the pneumococcal vaccine).

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s

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

existence.” *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec’y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec’y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 418 F.3d at 1278: “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.”

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

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

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory’s scientific or medical *plausibility*. *See Boatmon v. Sec’y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec’y of Health & Hum. Servs.*,

746 F.3d 1334, 1339 (Fed. Cir. 2014) (“[h]owever, in the past we have made clear that simply identifying a ‘plausible’ theory of causation is insufficient for a petitioner to meet her burden of proof” (citing *Moberly*, 592 F.3d at 1322)). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec’y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard).

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

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff’d*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec’y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den’d*, 100 Fed. Cl. 344, 356 (2011), *aff’d without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical

understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec’y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec’y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff’d mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec’y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den’d*, (Fed. Cl. Dec. 3, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

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

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

Accordingly, if the medical records are clear, consistent, and complete, then they should

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

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

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

C. *Analysis of Expert Testimony*

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

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

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

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec’y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. See, e.g., *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner’s case. Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen v. Sec’y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); see also *Isaac v. Sec’y of Health & Hum. Servs.*, No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for*

review denied, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

E. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record, and the parties have not challenged my determination to do so. Mot. at 1; Opp. at 1. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Overview of Relevant Medical Terms and Prior Decisions

It is well understood that GBS is an acute, monophasic peripheral neuropathy involving rapidly progressive and ascending motor neuron paralysis, which is thought to have an autoimmune mechanism. *Yuki* at 691. Although its etiology is mostly unknown, two-thirds of GBS cases follow an antecedent infection (typically an upper respiratory tract or gastrointestinal infection) beginning a few weeks prior to symptoms onset. *Id.* at 691–92. A GBS diagnosis requires a thorough medical assessment involving the patient’s clinical presentation, plus other kinds of diagnostic testing, such as nerve conduction studies or CSF analysis. *Id.* at 692.

Much is known about not only GBS’s likely pathogenesis (albeit in defined circumstances pertinent to certain variants), but also its association with one particular vaccine covered by the Program: the flu vaccine. There are several evidentiary components supporting this association, as I have discussed in other cases. *Bielak v. Sec’y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *34 (Fed. Cl. Spec. Mstr. Dec. 9, 2022). And these evidentiary components have been deemed sufficient to preponderantly demonstrate the flu vaccine can likely cause GBS.⁴¹ *Mason v. Sec’y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (noting that the flu vaccine-GBS association is supported by a “mix of (a) knowledge about how molecular mimicry “works” in GBS's pathogenesis, (b) trustworthy animal experiments that model demyelinating injuries in the context of the molecular mimicry mechanism, and (c) solid (if somewhat old) epidemiologic evidence . . . establishing a higher incidence of GBS after vaccination when compared to an unvaccinated population”).

As a result, too many well-reasoned decisions to count have found the issue resolved (at least for purposes of deciding Program cases). *See, e.g., Chinea v. Sec’y of Health & Human Servs.*, No. 15-095V, 2019 WL 1873322 (Fed. Cl. Spec. Mstr. Mar. 15, 2019); *Strong v. Sec’y of Health & Human Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018); *Stitt v. Sec’y of Health & Human Servs.*, No. 09-653V, 2013 WL 3356791 (Fed. Cl. Spec. Mstr. May 31, 2013); *Stewart v. Sec’y of Health & Human Servs.*, No. 06-777V, 2011 WL 3241585, at *16 (Fed. Cl. Spec. Mstr. July 8, 2011). Such cases often also rely on the theory of molecular mimicry, proposing that antibodies produced by B cells in response to a vaccine’s viral antigen components can cross-attack the myelin sheath (because the target antigen and gangliosides of the myelin sheath share structural homology), thereby causing demyelination of peripheral nerves. *Chinea*, 2019 WL 1873322, at *15.⁴²

⁴¹ Of course, it is axiomatic that prior decisions do not bind my determination herein. Rather, I decide this case based on the evidence before me. However, it is not only useful, but prudent, to take into account prior determinations—both for guidance and to avoid “reinventing the wheel” when deciding like Program cases.

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

The same is not true for the pneumococcal vaccine, however (which as noted above does not also have a counterpart Table claim for GBS). I recently discussed at length the reasons why. See generally *Bielak*, 2022 WL 18058244, at *33–37 (pneumococcal vaccine not shown to cause GBS). In short, the flu and pneumococcal vaccines are wholly distinguishable in composition, provoke the intended immune response in completely different ways, and evidence relevant to flu vaccine causality is missing for the pneumococcal vaccine. *Id.* at *36. I also observed in *Bielak* that there are only a limited number of decisions in which special masters have considered causation in this context—and while some decisions have unquestionably gone the petitioner’s way, their reasoning is unpersuasive, giving excessive weight to molecular mimicry’s explanatory power based on a distinguishable context, or not fully grappling with Respondent’s counter-arguments. See, e.g., *Gross v. Sec’y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); *Maloney*, 2022 WL 1074087; *Pierson v. Sec’y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Koller v. Sec’y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). I also observed that these favorable cases cited the same items of literature offered in *Bielak* (and this case as well), but that they were far less supportive of causation than assumed. *Bielak*, 2022 WL 18058244, at *15–17, 32 (discussing Chang, Gilburd, Ho, Kanter, and Nakos).

Bielak no more controls the outcome of this case than determinations like *Koller* favorable to the Petitioner do. But I reference it because the similarities in theory offered therein are too reminiscent of what is proposed in this case to ignore. There is ample reason to doubt the persuasiveness of the general theory that the pneumococcal vaccine “can cause” GBS, especially where that theory over-relies on a “connect the dots” approach that tries to demonstrate a link between indirectly-present components⁴³ of the vaccine and myelin structures (in this case, lipids), but without additional evidence making pathogenicity in the wake of vaccination more likely. All vaccines do not cause GBS simply because one vaccine likely does—but decisions implicating the pneumococcal vaccine as causal seem too eager to apply what is known about the flu vaccine as a template to any covered vaccine.

My determination in *Deshler*, 2020 WL 4593162, also has some relevance to the current case, although it is less on point. The *Deshler* petitioner relied on a theory that the conjugate component of the Prevnar-13 vaccine *itself* had caused an aberrant, mimicry-driven autoimmune process relating to the vaccine’s antigens. *Deshler*, 2020 WL 4593162, at *27. Petitioner’s expert in *Deshler* opined, as here, that the subsequent B cell reaction (the primary goal of the vaccine) was driven by the polysaccharide component of the vaccination, although (unlike this case) he conceded that he could not demonstrate mimicry between the *S. pneumoniae* polysaccharides and

⁴³ By indirect, I mean only that the vaccine is not manufactured or engineered to specifically include phospholipids or phosphoglycerol—but that the polysaccharide antigens in the vaccine *themselves* happen to contain these sub-structures.

self-structures. *Id.* Respondent’s expert argued in reaction that the polysaccharides contained in the vaccine did not share structural homology with self-structures of the peripheral nervous system, and thus could not contribute to the pathogenesis of GBS via a molecular mimicry-driven cross-reaction to the vaccine’s antigens. *Id.* at *27. I concurred with Respondent, while also finding that the petitioner relied too heavily on the temporal association between vaccination and onset as evidence of causation (and that there was another potential explanation for the claimant’s GBS that had not been rebutted). *Id.* at *22, 27.

II. Petitioner Has not Carried His Burden of Proof to Show That the Pneumococcal Vaccine “Can Cause” GBS

Petitioner’s GBS diagnosis is largely uncontested,⁴⁴ leaving me to determine whether the three *Althen* prongs are met. This case turns wholly on the first prong⁴⁵—for I find (consistent with my analysis in *Bielak* (2022 WL 18058244, at *33–37)) that Petitioner has not preponderantly demonstrated that the pneumococcal vaccine can cause GBS.

The pathologic mechanism Dr. Steinman embraces⁴⁶—molecular mimicry—is well-established in the Vaccine Program as providing a reliable scientific explanation for how GBS may *often* occur after receipt of the flu vaccine specifically. *See Chinae*, 2019 WL 1873322, at

⁴⁴ Dr. Vartanian raised some questions about aspects of the EMG testing that were inconsistent with a demyelinating polyneuropathy, but he never clearly or centrally advocated that the diagnosis was incorrect—and his other comments on the medical record seemed to relate more to potential alternative causes than to the diagnosis. Respondent also does not contend in his brief that Mr. Trollinger’s illness is not properly understood to be GBS. In any event, my determination that entitlement was not established primarily lies in my finding that the pneumococcal vaccine has not been shown to be likely causal of GBS.

⁴⁵ My determination on the first prong is dispositive (since claimants must satisfy all three prongs to prevail), and therefore I do not include discussion of the other two prongs. I do, however, acknowledge treater support in Petitioner’s medical records for a vaccine causal relationship. Ex. 4 at 36–37; Ex. 9 at 6. Petitioner can point to a number of instances in the record where a treating physician offered the view that the pneumococcal vaccine had a connection to his neurologic injury—as well as the admonition to avoid vaccination in the future. Ex. 4 at 80 (July 27, 2015 visit); Ex. 9 at 6 (August 31, 2015 visit); Ex. 11 at 15 (April 2016 visit). Such evidence supports the second *Althen* prong. But I am not bound to accept a treater’s opinion. *Snyder*, 88 Fed. Cl. at 746 n.67. And it appears in several instances that treaters deemed the *temporal* association significant in proposing a vaccine association. *See, e.g.*, Ex. 4 at 36–37, 80 (Dr. Smith’s impression that Petitioner’s GBS was “likely due to pneumonia shot that [Petitioner] got one week before presentation” is juxtaposed with his note that Petitioner “apparently had a Pneumovax shot recently”). There is also evidence suggesting that before the formal onset of Petitioner’s neurologic symptoms, he experienced some form of gastrointestinal distress severe enough to send him to the ER (arguably allowing for the possibility that an infection was causal). Ex. 3 at 2. But I cannot on this record find a likely alternative explanation.

Petitioner’s *Althen* prong three showing was limited to the argument that his onset/vaccination timeframe was consistent with Schonberger—a study involving the flu vaccine (and in fact a version of that vaccine not generally administered since the 1970s). Schonberger at 105. This was far weaker than his prong two evidence, although the timing of his onset ten days after vaccination is not inconsistent with Dr. Steinman’s theory.

⁴⁶ Although Program claimants are not *required* to propose a mechanism, they often attempt to do so—and thus invite scrutiny into whether the proposed mechanism has preponderant support. *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano*, 440 F.3d at 1325–26).

*29. But as I have now observed too many times in other cases, a claimant cannot simply invoke the concept and deem the “can cause” prong satisfied. *McKown v. Sec’y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (“merely chanting the magic words ‘molecular mimicry’ in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the mechanism to the injury and/or vaccine in question”) (emphasis in original), *mot. for review denied*, 76 Fed. Cl. 452 (2007)). This is because, as Dr. Whitton noted, homologic similarity (whether due to sequential chains of amino acids (relevant to proteins), structural similarities of molecules, or both) is commonplace in nature, but without pathologic outcomes. Whitton Rep. at 36. This, plus the fact that a number of distinguishable insults (viral vs. bacterial infections) can result in different forms of GBS, means that Program claimants do not satisfy the first prong simply by offering molecular mimicry to explain pathologic mechanism.

Rather, a petitioner’s expert(s) must offer evidence specific to the context of the case—the injury as well as the vaccine in question. And even in the context of the flu vaccine, where molecular mimicry is a more reliably offered mechanism, Program cases have often involved successful demonstrations by petitioners that the wild flu viral components of the vaccine contain amino acid sequences that share sequential and structural homology to self-structures (gangliosides) that would be the putative target of autoimmune attack. *See generally Pierson*, 2022 WL 322836, at *24 (citing *Chinea*, 2019 WL 1873322, at *15). But a bare demonstration of a cross-reactive *potential* would not be enough to prevail *even* in the case of a flu-GBS claim. Rather (as discussed in *Bielak*) a number of factors *other* than amino acid homology have supported the vaccine-injury association. *Bielak*, 2022 WL 18058244, at *33, 36. It is this kind of showing in the totality that crosses the preponderant “line.”

Has Petitioner offered sufficient proof, through the efforts of Dr. Steinman? At the outset, I note that there is no baseline association between the wild *S. pneumoniae* infection and GBS (as compared to a wild flu infection), heightening the need for corroborative evidence. In addition, Dr. Whitton noted that the medical community has not even entertained the concept at the heart of Dr. Steinman’s theory: that a target antigen for GBS *could* be phospholipids, rather than gangliosides. Whitton Rep. at 8-9, 30.⁴⁷ Proving that a vaccine could cause an injury, and in a specific way, that the existing scientific consensus does not support for the vaccine’s antigenic source, would be an uphill battle.

This still left room for Dr. Steinman to propose a theory that could pass the preponderant test, *if* supported by sufficient and reliable medical or scientific evidence. Any number of kinds of evidence in combination could meet the standard. What Dr. Steinman has done, however, is simply

⁴⁷ Indeed—if correct, Dr. Steinman has effectively identified a *new/alternative pathogenesis* for GBS not previously recognized by the peripheral neuropathy experts who study GBS far more intently and consistently than Dr. Steinman. *See generally* Hughes II and Kwan. The lack of widely-accepted support for this novel theory only gives credence to the inference that Dr. Steinman’s theory has been crafted to meet the needs of the case.

couple his usual BLAST search molecular biology showing, in which he attempts to make associations between molecular sub-components of a vaccine's antigens and some self/host tissue components found in the general vicinity of the injury (here, peripheral nerve myelin), with conclusory reasoning that ultimately places undue weight on the temporal relationship between the claimant's vaccination and onset manifestation.

Dr. Whitton, however, persuasively and comprehensively rebutted his showing. First, Dr. Whitton demonstrated that the first iteration of Dr. Steinman's causation theory erroneously relied on the presence of "phospholipids" in the pneumococcal vaccine for the mimicking cross-reaction he posited. This could be ignored as a minor mistake of scientific imprecision, especially since (based on Dr. Steinman's third report) Dr. Whitton conceded that phosphoglycerol (or more accurately glycerophosphate) *was* found in at least some of the vaccine's 13 polysaccharide antigens. But the outset error reasonably calls into question what followed, including Dr. Steinman's explanation about the evolution of his thinking on the topic—an evolution that relied less on new discoveries or scientific findings, and more on the fact that unfortunate delay in the case's progress allowed him to sand off the rough edges of his earlier, inaccurate theory.

Second, Dr. Whitton made numerous insightful critiques of the specific components of the core causation theory set forth in Dr. Steinman's third report. That report, it should be noted, *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. But this showing left out too much to find it was enough to meet the Petitioner's preponderance burden of proof. For, as Dr. Whitton demonstrated, it is not likely that antibodies generated to the phosphoglycerol-specific vaccine antigens can *in turn* react with host tissue phospholipids, or that GBS is *primarily or initially* mediated by such a cross-reaction, with articles like Nakos and Gilburd admitting that the existence of antibodies (which could be the *product* of an ongoing disease started by something distinguishable) does not in turn imply pathogenic primacy. It may not even be the case, as Dr. Whitton maintained, that phosphoglycerol is found in phospholipids in a chemical state recognizable by a pneumococcal antigen-produced antibody. While both experts were well-qualified to offer the opinions they did, I give more weight overall to Dr. Whitton when opining on the biochemistry and immunologic topics relevant to the causation theory; his expert report covering them overall demonstrated far more precision and care in terminology than Dr. Steinman's efforts.

Dr. Whitton also showed that Dr. Steinman's practice of employing BLAST searches may to some degree make less than optimal use of the database (although I do not dispute the baseline utility it provides him in establishing potential homologous sequences when comparing a variety of compounds, protein or otherwise, common to vaccines and human tissue). But it is not even evident

that the considerable effort Dr. Steinman put into this aspect of his opinion (which took up pages upon pages of his reports) had utility to his theory, given the holes in his reasoning on other, equally-important issues. Rather, the science-heavy showing⁴⁸ he made on this front did more to obscure and confuse than to support the theory that the vaccine can cause GBS. I need not decide if Dr. Steinman offered sufficient homology between phosphoglycerols in two or three of the Prevnar vaccine's 13 antigens and host tissue to conclude, based on the overall evidence, that causation was not established.

Dr. Steinman's reports also overstated points that were more prosaic in nature, attempting to "flip" a fact about the expected vaccine response into evidence supporting his theory. For example, he made emphatic reference to Bryson, as if it were tailor-made to support his causation opinion. *See* Steinman Third Rep. at 13 (citing Bryson) ("23F is in Prevnar 13!!"). But the study itself had no such focus; Bryson's authors mainly appear to have selected a pneumococcal antibody generated in response to a vaccine-containing strain because they were aware that a related gene pair also played a role in causing production of a different antibody—thus allowing them to study that issue, as opposed to GBS pathogenesis of the immune reaction to the pneumococcal vaccine. Bryson thus provides only cursory, secondary support for Dr. Steinman's contention that a vaccine strain specifically-containing a phosphoglycerol group prompts an immune response—and ultimately stands mostly for the conclusion that the pneumococcal vaccine *reliably functions as intended*. Bryson's authors were not looking to see if the antibody had a potential *cross-reactive* response—a far more important aspect of Dr. Steinman's opinion.

This latter point was an especially important omission in Dr. Steinman's theory. The evidence that antibodies produced in response to the pneumococcal vaccine can attack nerve structures and instigate GBS in the process was mostly derived from the articles I discussed at length but rejected in *Bielak*. *See Bielak*, 2022 WL 18058244, at *15–17, 32 (discussing Chang, Gilburd, Ho, Kanter, and Nakos). Such items are either specific to MS or openly acknowledge that they cannot shed light on whether antibodies oriented against myelin phospholipids *initiate* an autoimmune attack or are created in the *midst* of an attack started by some other factor. Thus, it has not been preponderantly shown these autoantibodies "cause" GBS—only that they are *present* in GBS, or MS (a distinguishable condition even if it too involves demyelination). And Dr. Whitton convincingly explained why it could not be assumed, and had not been shown, that an antibody produced in response to one of the vaccine's phosphoglycerol-containing polysaccharides would be recognized by a myelin phospholipid structure.

⁴⁸ The theory proposed in this case was in fact on the *more* science-heavy end of the spectrum, even for Program cases. Its inordinate scientific complexity and repeated reference to biochemistry and microbiology topics was only magnified in the back-and-forth between Drs. Whitton and Steinman in their final reports. (Indeed, these two reports together constitute 75 pages of work).

The other two components of Petitioner’s causation theory were even less well substantiated. The contentions about a cross reaction between the vaccine conjugate and contactin, for example, relied on another painstaking effort to prove homology, but without hardly any evidence that (a) vaccine recipients mount a response to the conjugate that would *produce* anti-contactin antibodies, and (b) that contactin is a likely GBS pathogenic target. Dr. Whitton persuasively rebutted these points. Whitton Rep. at 40–41. The arguments Dr. Steinman raised about the role alum would play in the process of pathogenicity was particularly bare-bones, and (as Dr. Whitton noted) was hard to distinguish from the routinely-rejected causation theory “ASIA,” in which the alum-based adjuvant is deemed central to autoimmune disease.

Dr. Steinman unpersuasively defended his consistent reliance on molecular biology-heavy showings in this report (as he does in nearly every Vaccine Act case) as justifying his lack of other supportive evidence. *See, e.g.*, Steinman Fourth Rep. at 15 (“[t]he best that can be done”). Indeed, he seemed at times to employ the many molecule images strewn throughout his report as a smoke screen, to hide his theory’s weakness in a haze of difficult science. His primary argument, however—that he could not do better—amounts to a demand to lower the *Althen* standard, primarily because proving causation is difficult. In fact, the absence of sufficient reliable science in support of a theory is *not* an occasion to accept a non-preponderant showing. *Caves v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 143 (2011), *aff’d*, 463 F. App’x 932 (Fed. Cir. 2012) (“the standard of proof does not operate as a sliding scale that varies depending on the quantity and quality of the scientific evidence that is available”). And my determination that the overall amount of evidence offered by Petitioner did not tip the scales, so to speak, in his favor, is not equivalent to a demand for certainty on causation. *Hodges v. Sec’y of Health & Hum. Servs.*, 9 F.3d 958, 962 (Fed. Cir. 1993) (noting rejection of medical opinion on causation did not amount to requiring certainty, but instead reflected special master’s inquiry into whether “some degree of acceptable scientific support” existed to conclude preponderance standard had been met).

Worse is the fact that Dr. Steinman’s evolved theory seems to have been the product of the case’s slow progress rather than any new scientific or medical understanding of GBS and its association with the pneumococcal vaccine. What new scientific or medical studies on the subject were published between 2017 (when Dr. Steinman’s first round of reports were filed) and his last report, in May 2022?⁴⁹ Why could the thinking on the theory in this case not have been performed when an expert opinion was first sought? As my analysis makes clear, I have taken the revised theory presented on its own terms, rejecting it for the reasons stated, rather than because it looks like an unmerited attempt at a “do-over.” But I cannot help but conclude that Dr. Steinman refined his theory *solely* because the case’s slow course permitted him the time to do so, rather than because compelling independent reasons existed.

⁴⁹ Only two items filed after the third report were published in 2021-2022 (with Dr. Steinman as a coauthor on one). *See* Robinson; Wekerle.

In the end, 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. If 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. In such a world, all vaccines "can cause" GBS, simply because of the intersection between the immune stimulation vaccines cause and autoimmune diseases—and causation claims become indistinguishable from Table claims (since the very question of causation is no longer subject to weighing of evidence but a fact issue easily satisfied by this kind of expert opinion).

III. This Case Was Appropriately Decided on the Papers

In ruling on the record, I am choosing not to hold a hearing. Determining how best to resolve a case is a matter that lies generally within my discretion, and although the parties have not contested my choice of adjudicatory method, I shall explain why a hearing was not required.

Prior decisions have recognized that a special master's discretion in deciding whether to conduct an evidentiary hearing "is tempered by Vaccine Rule 3(b)," or the duty to "afford[] each party a full and fair opportunity to present its case." *Hovey*, 38 Fed. Cl. at 400–01 (citing Rule 3(b)). But that rule also includes the obligation of creation of a record "sufficient to allow review of the special master's decision." *Id.* Thus, the fact that a claim is legitimately disputed, such that the special master must exercise his intellectual faculties to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes without a hearing—although they should only so act if a party has been given the proper "full and fair" chance to prove their claim.

The present claim could be, and was, fairly resolved without the need for live testimony from the experts. Respondent's questions regarding Mr. Trollinger's diagnosis were not followed through, making this case primarily a dispute regarding causation. That issue was something that could be decided by careful consideration of the expert reports and associated literature. Moreover, questions about the pneumococcal vaccine-GBS association raised issues I have addressed recently, relying on the same arguments and items of literature. And despite how complex the science on the issue became, the "topline" deficiencies in the theory were not only quite evident from the reports but were dispositive no matter how the more esoteric disputes between the experts were resolved. Otherwise, evidence that a vaccine "did cause" the alleged injury, or did not, could be adduced from the record, without the need for witness assertions.

Overall, careful review of briefs, expert reports, articles/literature, and the record were sufficient to reach a just and defensible conclusion. This case is nearly seven years old, and thus there was value to selecting the most expeditious form of resolution for such a claim.

CONCLUSION

Claimants must carry their burden of proof—here, by preponderantly establishing, via an offering of sufficient evidence *specific to the pneumococcal vaccine in question*, how it could cause GBS. This has not been accomplished in this case. Accordingly, I deny entitlement.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.⁵⁰

IT IS SO ORDERED.

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

⁵⁰ Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.