

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

ROSS DAVENPORT,

Petitioner,

v.

SECRETARY OF HEALTH
AND HUMAN SERVICES,

Respondent.

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No. 19-820V

Special Master Christian J. Moran

Filed: August 24, 2026

Jessica Olins and Leeanne Pedrick, MCT Law, Seattle, WA, for petitioner;
Sara DeStefano and Alyssa Petroff, United States Dep't of Justice, Washington, DC for
respondent.

PUBLISHED DECISION DENYING ENTITLEMENT TO COMPENSATION¹

Ross Davenport received a vaccination against *Streptococcus pneumoniae* called Prevnar and then developed a neurologic disorder called Guillain-Barré syndrome (“GBS”).² Through this claim in the National Childhood Vaccine Injury Compensation Program, Mr. Davenport alleges that Prevnar caused his GBS. He supported his claim with reports from a neurologist whom he retained, Kazim A. Sheikh. His attorneys later advocated through a brief.

The Secretary opposed compensation. The Secretary relied upon two experts he retained. The first is a neurologist, Dara G. Jamieson. The second is a Ph.D. immunologist, who is not a

¹ Because this decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims’ website, and/or at <https://www.govinfo.gov/app/collection/uscourts/national/cofc>, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), the parties have 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. Any changes will appear in the document posted on the website.

² An explanation of some medical terms is found in the appendix. Given that the experts discussed relatively complicated medical subjects, the experts on both sides are encouraged to provide more background information in future cases. Likewise, the attorneys are encouraged to explain medical information in their briefs.

medical doctor, Ross M. Kedl. The Secretary, too, advocated through a brief. A hearing was held during which Mr. Davenport and the three retained experts testified.

A review of the record reveals that Mr. Davenport has not met his burden to show that Prevnar caused his GBS. As discussed below, he has not presented a reliable theory to explain how Prevnar can cause GBS.

I. Medical History

Mr. Davenport was born in 1952. Tr. 17. Until around 2016, Mr. Davenport worked for the city of Spokane, Washington as a supervisor for managing solid waste. Tr. 18. When he was employed, he usually commuted to work by riding his bicycle 12.5 miles each way. Tr. 19.

In retirement, Mr. Davenport continued his active lifestyle. He often rode his bicycle, and he liked to ski. He exercised at a local YMCA. Tr. 18-19. He did not have any limitations on his mobility. Tr. 20. In the litigation, the Secretary has not contended that any health problem from before the vaccination contributed to Mr. Davenport's GBS. See Resp't's Rep., filed Aug. 11, 2020, at 5.

When Mr. Davenport became eligible for Medicare, he had a general wellness examination. The doctor gave him a clean bill of health. Tr. 20; see also Exhibit 1 at 304-08 (Apr. 17, 2018). During this appointment, Mr. Davenport received a dose of Prevnar.³

Prevnar is a vaccine that protects against strains of the streptococcus pneumoniae bacteria. Tr. 60. Prevnar is sometimes referred to as "Prevnar 13" because this vaccine protects against 13 different strains. Tr. 219. Prevnar does not contain any live bacteria. Tr. 162.

A few days after the vaccination, Mr. Davenport was visiting a casino in Idaho. His legs did not feel correct and he could not sit at a slot machine. So, his wife drove home. Tr. 21.

After this, his symptoms progressed. He developed back pain, numbness and weakness in his arms, and intermittent numbness in his legs. He told his primary physician about these problems on May 2, 2018. The doctor obtained an MRI, which revealed spinal stenosis and disc bulging. Exhibit 1 at 351; see also Tr. 22. The doctor sent Mr. Davenport home.

The next day, Mr. Davenport was not any better. His legs were not working and he fell. Weakness prevented him from gripping anything. When his wife communicated these symptoms to the doctor, the doctor advised Mr. Davenport to go to the emergency department. Exhibit 1 at 367; see also Tr. 23.

In the first hospital, Mr. Davenport spent many hours in the emergency department waiting to be seen. Tr. 23. He underwent a lumbar puncture. Exhibit 2 at 48. The doctors again sent Mr. Davenport home. Tr. 24.

When Mr. Davenport arrived home, he fell backwards, going up the steps leading into his house. Mr. Davenport managed to crawl to a couch, where he slept. Tr. 24. The next morning,

³ Mr. Davenport also received the hepatitis B vaccine and a typhoid vaccine. However, these other vaccines are not the basis for his claim for compensation.

Mr. Davenport could not get out of bed and his wife called an ambulance, which brought him to a different hospital. He was admitted to Sacred Heart Hospital, where he stayed for approximately 18 days. Tr. 27.

During this admission, Mr. Davenport underwent an EMG. The results were consistent with acute motor axonal neuropathy (AMAN). Exhibit 4 at 27. The neurologists retained in this litigation agree that AMAN is the appropriate diagnosis for Mr. Davenport. Exhibit A at 12; Exhibit 70.

Acute Motor Axonal Neuropathy

AMAN is a subtype of Guillain-Barré syndrome. GBS is a term that encompasses different types of neurologic disorders. The most common subtype of GBS is known as acute inflammatory demyelinating polyneuropathy, often abbreviated “AIDP.” Because AIDP is much more prevalent in the United States, the term GBS is sometimes used synonymously with AIDP but this is not correct. Tr. 156.

As its name indicates, in AMAN, nerve motor axons are impaired. By way of contrast, in AIDP, the myelin is attacked. Tr. 55-56. As discussed later in this opinion, the testifying experts have some differences of opinion as to whether what is known about AIDP can be transferred to AMAN.

During his stay at Sacred Heart Hospital, Mr. Davenport received treatment for AMAN, including receiving five doses of intravenous immunoglobulin. The details of the treatment for AMAN are generally not relevant to determining whether the Prevnar vaccine caused Mr. Davenport’s AMAN. For more information, see Pet’r’s Br. at 3; Resp’t’s Br. at 3.

While in Sacred Heart, a neurologist, Ruxandra Costa, evaluated Mr. Davenport. Exhibit 4 at 156 (May 4, 2018). Dr. Costa suspected that Mr. Davenport was suffering from the AMAN form of GBS. She also wrote: “this can be related to the pneumonia shot especially if this used any live-attenuated type of strains.” Id. at 158. The doctor who discharged Mr. Davenport from Sacred Heart stated that Mr. Davenport’s AMAN was “[p]ossibly triggered by Prevnar 13—was given to patient 2 w[ee]ks prior [to] admission.” Id. at 13; accord Tr. 55.

After he was discharged from Sacred Heart, Mr. Davenport went to St. Luke’s Rehabilitation Institute. Tr. 28; see also Exhibit 3. With assistance from the rehabilitation specialists, Mr. Davenport improved so that he no longer required a wheelchair. Tr. 29. Mr. Davenport continued to exercise for the next three years.

In the intervening years, Mr. Davenport’s doctors expressed some concern that Prevnar may have caused his AMAN. See, e.g., Exhibit 5 at 3 (Sep. 25, 2018); Exhibit 18 at 11 (Oct. 27, 2020). One doctor exempted Mr. Davenport from receiving the Covid vaccine as a condition for travel. Exhibit 48 at 20 (May 25, 2021). Yet, this same doctor also recommended that Mr. Davenport receive the Covid vaccine. Id. at 8.

When Mr. Davenport testified in 2025, he stated that he was not in much pain. Leg cramps can interfere with his sleeping, but he did not want to take medicine. Tr. 31. He does not ride his bicycle and has not gone skiing since the original episode. Mr. Davenport seemed

determined to maintain a relatively positive attitude. In his words, he was “still kicking, just not as high.” Tr. 33.

II. Procedural History

Mr. Davenport’s case took more twists and turns than a typical case in the Vaccine Program. These various steps are explained in the succeeding sections.

A. Initial Materials

Represented by an attorney at Maglio, Christopher & Toale, Mr. Davenport initiated this case by filing a petition on June 4, 2019. As noted previously, Mr. Davenport alleges that Prevnar caused him to suffer AMAN. He quickly submitted medical records.

The Secretary reviewed this material and advised against an award of compensation. Resp’t’s Rep., filed pursuant to Vaccine Rule 4 on Aug. 11, 2020. The Secretary argued that Mr. Davenport did not meet his burden of proof, noting, in part, that Mr. Davenport had not presented a report from an expert.

Because it appeared likely that the parties would retain reports from people retained for this litigation, a set of Expert Instructions was proposed. After the parties did not object, the instructions became final.

B. Experts and Their Reports

Each party filed reports from two people. Mr. Davenport submitted reports from a neurologist, Dr. Sheikh, and a pediatric immunologist / dermatologist, Marc Serota. However, as discussed below, Mr. Davenport eventually withdrew Dr. Serota’s reports, leaving only reports from Dr. Sheikh. The Secretary retained Dr. Jamieson and Dr. Kedl.

1. Dr. Sheikh and his First Report

Dr. Sheikh attended undergraduate college and medical school in Pakistan. He came to the United States in 1990 to participate in an internship, residency, and then a fellowship in neurology. He became board-certified in neurology in 1998. Exhibit 129 (curriculum vitae).

Dr. Sheikh teaches at the University of Texas, Houston. In that role, he lectures about GBS and how vaccines might cause GBS via molecular mimicry. Tr. 48-49.

Dr. Sheikh directs a GBS / CIDP Center for Excellence. Tr. 48. He obtained this role by applying to the GBS / CIDP Foundation. Tr. 134. For many years, Dr. Sheikh assisted the GBS / CIDP Foundation. He served on the committee that reviews applications for grants to the GBS / CIDP Foundation and acted as chair of that committee. To Dr. Sheikh’s awareness, this committee has not received any proposals to investigate whether Prevnar causes GBS. He is not sure whether the Foundation would fund this research. Tr. 108-12.

Although he sometimes works with immunologists (see Tr. 48, 135), Dr. Sheikh is not an immunologist. As the Secretary brought out on cross-examination, Dr. Sheikh does not have a Ph.D. in immunology. Tr. 94.

Dr. Sheikh disclosed a series of opinions in his first report, which was filed on May 17, 2021. He opined that Mr. Davenport suffered from GBS, without specifying a subtype. Exhibit 19. To explain how Plevnar could cause GBS, Dr. Sheikh relied upon the theory of molecular mimicry. Id. at 6. He presented three variations on the theme of molecular mimicry. In the first, components of Plevnar resemble portions of peripheral nerves known as galactose and gangliosides. In the context of this first theory, Dr. Sheikh cited three articles. Id. at 9-10. In the second variation of molecular mimicry, Dr. Sheikh linked a protein in Plevnar (CRM197) to peripheral nerve protein P0. Id. at 10. In the third variation, Dr. Sheikh discussed adjuvants. Id. at 10.

2. Dr. Jamieson and her First Report

Dr. Jamieson earned her undergraduate degree at George Washington University, majoring in zoology and physics. She then went on to obtain her M.D. from the University of Pennsylvania School of Medicine in 1982. Exhibit J. Afterwards, she completed a neurology residency and a fellowship in cerebrovascular disease at the hospital at the University of Pennsylvania. Tr. 149.

Dr. Jamieson held various academic positions over the following years, and is presently the Clinical Associate Professor of Neurology at Weill Cornell Medicine, New York Presbyterian Hospital. Exhibit J; Tr. 150. She has held this position since 2005. Although she no longer sees patients as of June 2018, she retained her academic position and teaches medical students, residents, fellows, and attendings. Tr. 150.

Dr. Jamieson was formerly the president of the American Society of Neuroimaging and of the Philadelphia Neurological Society. Tr. 150. She is a member of the American Academy of Neurology and teaches courses and gives lectures at their annual meetings. Id. Additionally, Dr. Jamieson participates in editing and peer-reviewing for various journals. Id. at 150-51.

In her first report, Dr. Jamieson discussed GBS and its various subtypes, including AIDP and AMAN. Exhibit A at 7-8. Dr. Jamieson asserted that “Dr. Sheikh’s arguments used to link Mr. Davenport’s acute neuropathic process with his preceding pneumococcal vaccination refer[] to ‘GBS’ and do not distinguishing between AIDP and AMAN. These two distinct entities, with different nerve tissue pathologies and biomarkers, are thus conflated in Dr. Sheikh’s assessment of the reports examining GBS in the context of vaccination.” Id. at 11. Dr. Jamieson stated that Mr. Davenport met the diagnostic criteria for AMAN within about two weeks of the vaccination. Id. at 12.

Dr. Jamieson disagreed with the opinion that Plevnar can cause GBS. She relied upon epidemiologic studies, which are discussed below. Exhibit A at 8-11. She concluded: “More likely than not Mr. Davenport’s development of GBS/AMAN was coincidental to his receipt of Plevnar 13 vaccine, but the two events were not causally related.” Id. at 13.

3. Dr. Kedl and his First Report⁴

After earning a Bachelor of Science in biology, Dr. Kedl obtained a Ph.D. in pathobiology from the University of Minnesota Medical School in 1997. For about three years, he worked for 3M Pharmaceuticals as a senior immunologist. Tr. 282-84.

In 2004, Dr. Kedl started an academic career at the University of Colorado. Currently, he is a full professor in the Department of Immunology and Microbiology. In this role, he teaches at the medical school, which is not proximate to the undergrad campus. Tr. 284. He teaches graduate students and medical students about immunology and vaccination. Tr. 211, 284-85.

Dr. Kedl's research focuses on T cells. A particular interest is whether T cells can be used to fight cancer. Tr. 211. Due to his expertise in how T cells are generated, he has often served on advisory committees studying the effectiveness of adjuvants in the context of vaccines. Tr. 212.

During the hearing, Dr. Kedl was recognized as an expert in immunology. Tr. 215. As pointed out during cross-examination, Dr. Kedl has not worked on neuro-inflammatory conditions specifically. Tr. 257.

Dr. Kedl's first report challenged many opinions from Dr. Sheikh about how Prevnar can cause GBS. A particular point of disagreement was the reliability of molecular mimicry. Dr. Kedl agreed with Dr. Sheikh's assertion that "Molecular mimicry has historically been held as a favored mechanistic explanation for the induction of autoimmunity." Exhibit B at 4. But, in Dr. Kedl's view, "this model no longer maintains scientific plausibility." Id.

Dr. Kedl's first report brought out criticisms of the molecular mimicry theory, which recur throughout the case. Dr. Kedl asserted that molecular mimicry appears to assume that similarities between vaccines and host tissues must be rare. If they were common, then many cross-reactions would occur. However, according to Dr. Kedl, similarities in structures are actually very common. Exhibit B at 11-13 (using CRM197 as an example). Dr. Kedl also opined that any similarity must be biologically relevant, meaning that it can independently contribute to a disease's pathology.

Dr. Kedl followed this reasoning in explaining what (to him) are the deficits in Dr. Sheikh's attempt to connect Prevnar with gangliosides. Dr. Kedl asserted that "no ganglioside-specific antibodies have ever been observed in response to the Prevnar vaccine, either in animal models or clinically." Exhibit B at 16. Dr. Kedl observes that the substances that Dr. Sheikh identified as being critical in the pathogenesis of Prevnar-induced GBS are also present in other human tissues. Yet, these tissues seem not to be the subject of an immune-mediated attack. Id. at 16-17. Dr. Kedl highlighted that vaccines, themselves, demonstrate the lack of cohesion in the theory of molecular mimicry because the components of the flu vaccine change each year. If structural similarity were sufficient, then varying the flu vaccine's strains would be unnecessary. Id. at 18.

⁴ Information about Dr. Kedl's background can be found in his updated curriculum vitae. Exhibit I.

In addition to challenging molecular mimicry as a general proposition and as used to explain how Prevnar could cause GBS, Dr. Kedl also discussed epidemiology. In Dr. Kedl's view, epidemiology weighs against finding an association. While Dr. Sheikh had opined that epidemiological studies may not detect rare events, Dr. Kedl pointed to the discovery that people given the AstraZeneca Covid vaccination had an increased risk of heparin-induced thrombotic thrombocytopenia at a rate of 1:1,000,000. Exhibit B at 5. Thus, it is not sufficient in Dr. Kedl's eyes for Dr. Sheikh to dismiss the epidemiologic studies, such as Baxter and Tseng, which are discussed below.

4. Marc Serota and His Report

In January 2022, Mr. Davenport's attorney of record became Jessica Olins, and since that time, she has represented him. When represented by Attorney Olins, Mr. Davenport obtained a report from Dr. Serota. He is board-certified in "allergy/immunology, dermatology and pediatrics." Exhibit 69 (report) at 1. Dr. Serota generally supported Dr. Sheikh's proposal that molecular mimicry can explain how Prevnar can cause GBS.

5. Dr. Sheikh's Second Report

Via a report filed on May 16, 2022, Dr. Sheikh responded to the opinions presented by Dr. Jamieson and Dr. Kedl. Although Dr. Jamieson noted that Dr. Sheikh had not differentiated AIDP from AMAN, Dr. Sheikh's second report focused on AMAN. In doing so, Dr. Sheikh recognized that his second way of linking Prevnar to GBS, which was through P0, a major protein in myelin, "is less relevant to [the] AMAN form of GBS." Exhibit 70 at 10.

A significant portion of Dr. Sheikh's second report was devoted to addressing Dr. Kedl's question about why the antibody-mediated attack is restricted to nerves when gangliosides are distributed across the human body widely. Dr. Sheikh agreed "this is an important and relevant question." Id. at 3. Dr. Sheikh admitted that "this is a complicated multifaceted issue and not all aspects related to it are completely worked out." Id. Nevertheless, based upon various articles cited below, Dr. Sheikh opined that it is possible for antibodies to attack only certain nerve tissues. Id. at 3-10.

Dr. Sheikh also presented a defense of the theory based on alum in about one page. Id. at 15.

Finally, Dr. Sheikh maintained that epidemiologic studies cannot be applied to any individual. Because of Prevnar's overall record of safety, he advises his patients to get Prevnar. Id. at 16-17.

6. Dr. Jamieson's Second Report

Dr. Jamieson's next report was relatively short, approximately 3.5 double-spaced pages. She maintained her opinion that despite Dr. Serota's first report and Dr. Sheikh's second report, "Mr. Davenport's development of GBS/AMAN was coincidental to his vaccination with Prevnar 13, without a causal relationship." Exhibit E at 4.

7. Dr. Kedl's Second Report

Dr. Kedl continued to challenge the reliability of molecular mimicry as a theory. As he had previously done with flu vaccines, Dr. Kedl pointed out that with Prevnar, “one strain does not produce biologically meaningful cross-reactive immunity to other strains, despite structural similarities that are orders of magnitude better than any they may possess with mammalian gangliosides.” Exhibit F at 3. He went so far as to state: “Molecular mimicry proponents should be open to critique about the perceived supremacy of this outdated model. If science is to move forward, questioning the authority of 30-year-old sacred cows is a necessary step in its pursuit.” Id.⁵

As to Dr. Sheikh's explanation of why antibodies would attack only gangliosides in peripheral nerves, Dr. Kedl stated Dr. Sheikh has shown “what some antibodies against gangliosides can do, but they are fraught with complications when trying to conclude what the normal repertoire of antibodies actually does do.” Exhibit F at 4 (emphasis in original). Dr. Kedl also emphasized “no ganglioside-specific antibodies have been documented in response to the Prevnar vaccine, either in animal models or clinically.” Id. at 2 (emphasis removed).

This report was the final report. See Pet'r's Status Rep., filed Dec. 18, 2022 (declining to present additional reports from either Dr. Serota or Dr. Sheikh). Accordingly, the parties were directed to argue their positions via briefs. Order, issued Feb. 24, 2023.

C. Briefs

Mr. Davenport claimed that he was entitled to compensation. Pet'r's Br., filed Aug. 4, 2023. The brief discussed the qualifications of both people he had retained, Dr. Serota and Dr. Sheikh. Id. at 9-12. Otherwise, the brief did not rely upon opinions from Dr. Serota.

In terms of theories to explain how Prevnar can cause AMAN, Mr. Davenport advanced the three theories from Dr. Sheikh. These are (1) a ganglioside / galactose theory, (2) a CRM197-P0 theory, and (3) a theory involving adjuvants. Pet'r's Br. at 70-91. Mr. Davenport also argued against giving much weight to the epidemiologic studies. Id. at 91-94. In closing, Mr. Davenport stated that he was “amenable” to a ruling on the papers without oral testimony. Id. at 103.

The Secretary contended that Mr. Davenport was not entitled to compensation. Resp't's Br., filed Oct. 13, 2023. The Secretary, too, did not oppose a ruling on the record.

The Secretary argued that his experts were better qualified to opine on the topics relevant to adjudicating Mr. Davenport's claim for compensation. Resp't's Br. at 15-18. In doing so, the Secretary described Dr. Serota as an “attending dermatologist.” Id. at 16 (citing Dr. Serota's updated curriculum vitae). The Secretary further asserted that for Dr. Serota, “respondent was unable to find any education, training, or experience meaningfully relevant to the medical issues in this case.” Id. at 17. Thus, the Secretary largely did not respond to Dr. Serota and, instead, addressed the theories offered by Dr. Sheikh.

⁵ In his oral testimony, Dr. Kedl characterized this statement as “hyperbole.” Tr. 294.

In opposing the theories from Dr. Sheikh, the Secretary began with a discussion of epidemiology. Id. at 19-22. To the Secretary, epidemiology was more valuable than a single case report Dr. Sheikh cited.

The Secretary also addressed the three theories that Mr. Davenport had advanced. Id. at 22-28. With respect to the theory based on alum, the Secretary noted that this portion of Dr. Sheikh's second report was extremely similar to a portion of report submitted by a different expert in another case, Lawrence Steinman. The Secretary recommended clarification. Id. at 27 n.12.

After the Secretary argued against compensation, the time for submitting a reply surprisingly lapsed without the submission of any brief from Mr. Davenport.

D. Preparation for a Hearing

A review of the materials suggested that oral testimony was appropriate. Order, issued May 24, 2024. One reason was that the undersigned had not adjudicated any cases in which petitioners had asserted that Plevnar can cause GBS. In preparation for the hearing, Mr. Davenport was required to obtain an affidavit from Dr. Sheikh regarding the alum portion of his second report. Mr. Davenport was also required to explain whether he was relying upon reports from Dr. Serota.⁶

For the question about the authorship of the portion of the report on alum, Dr. Sheikh declared that he had spoken with Dr. Steinman about this topic. Exhibit 120 (Dr. Sheikh's affidavit). Likewise, Mr. Davenport procured an affidavit from Dr. Steinman in which Dr. Steinman averred that he had spoken to Dr. Sheikh about alum. Exhibit 119 (Dr. Steinman's affidavit).

For Dr. Serota, Mr. Davenport stated that "Petitioner revised his litigation strategies and will now primarily rely on Dr. Sheikh's reports." Pet'r's Status Rep., filed June 25, 2024.

A status conference was held to discuss these issues. With respect to the controversy over the authorship of the alum portion, the Secretary was not satisfied with the disclosures from Dr. Sheikh and Dr. Steinman. The Secretary proposed that because it appeared that Dr. Sheikh lacked sufficient expertise in this area, the alum-based theory be removed from the case. In response, Mr. Davenport agreed to remove the alum-based theory. See Morrison v. Sec'y of Health & Hum. Servs., No. 18-386V, 2024 WL 3738934, at *8 n.8 (Fed. Cl. Spec. Mstr. July 18, 2024) (noting that in a case in which an attorney from the same law firm as representing Mr. Davenport declined to pursue a theory based upon alum).

On the topic of Dr. Serota's continued participation, Mr. Davenport's position was unclear. Counsel for Mr. Davenport reported that communicating with Dr. Serota was difficult. Counsel seemed interested in keeping the literature Dr. Serota cited in the record regardless of whether Dr. Serota testified. The undersigned explained that ordinarily, experts should be

⁶ The Secretary also was directed to submit a summary of the articles on which he was relying, which the Secretary's October 13, 2023 brief had neglected to do. The Secretary did so on July 8, 2024.

prepared to answer questions orally about the opinions they have expressed in writing. To the extent that Dr. Sheikh wished to rely upon articles that Dr. Serota had cited, Dr. Sheikh must disclose his own opinion about the articles. Order, issued July 9, 2024.

Mr. Davenport presented his positions in a status report filed on August 8, 2024. He requested that his case be adjudicated on the papers. This request was surprising because it would seem that petitioners would prefer an opportunity to demonstrate the persuasiveness of their expert's opinions through oral testimony. Regardless, the parties were given an opportunity to discuss whether the hearing should be held in-person at a remote location or via videoconferencing. Order, issued Aug. 20, 2024.

With respect to Dr. Serota, Mr. Davenport proposed striking his report. Pet'r's Status Rep., filed Aug. 8, 2024. He was afforded an opportunity to discuss with Dr. Sheikh whether Dr. Sheikh wanted to rely upon articles that Dr. Serota had cited. Order, issued Aug. 20, 2024. Dr. Sheikh identified the articles on which he wanted to rely. Exhibit 121.

After receiving information from the parties, a hearing was scheduled to take place in Denver, Colorado from August 28-29, 2025. Order, issued Nov. 6, 2024. The parties were given an opportunity to submit any additional medical articles before the hearing. However, due to budget limitations at the Office of Special Masters, travel to Denver was not possible. Thus, the parties were directed to express whether they preferred bringing their witnesses to Washington, DC or conducting the hearing via videoconferencing. Order, issued June 12, 2025.

In preparation for the hearing, the undersigned asked the parties a series of questions. The undersigned stated that Mr. Davenport was advancing a theory based upon gangliosides and glycolipids, which the undersigned labeled as theory 1(a) and theory 1(b). Mr. Davenport was also advancing a theory based upon a component of myelin, P0, although Mr. Davenport suffered from a disorder of his axons. Thus, the parties were expected to comment. Order, issued July 3, 2025.

On the day this order issued, Mr. Davenport announced that Dr. Sheikh was not available for the hearing, which the November 6, 2024 order had scheduled for August 28-29, 2025. Dr. Sheikh had "an unforeseen and pressing family matter." Pet'r's Mot., filed July 3, 2025. As remedy, Mr. Davenport proposed transferring the case to another special master. The basis for this request was not evident. In any event, after conferring with the chief special master, this request was denied. Order, issued July 7, 2025.

Another status conference was held on July 29, 2025. The parties agreed to present their witnesses through videoconferencing at a hearing on November 13-14, 2025. Mr. Davenport withdrew his reliance on a theory involving P0. Thus, Mr. Davenport was proceeding only on the theory involving gangliosides and glycolipids. Order, issued July 30, 2025. For this theory, the parties agreed that although this theory had been presented to other special masters, special masters had not evaluated the merit of this theory. See Brown v. Sec'y of Health & Hum. Servs., No. 18-1074V, 2025 WL 2017882, at *25, *27 (Fed. Cl. Spec. Mstr. June 23, 2025); Bartoszek v. Sec'y of Health & Hum. Servs., No. 17-1254V, 2024 WL 4263604, at *9 (Fed. Cl. Spec. Mstr. Aug. 27, 2024).

With the removal of the theories involving alum and P0, the case was narrowed to an assessment of the ganglioside and glycolipid theory. The undersigned attempted to identify components of the theory on which the parties agreed and on which the parties differed. Order, issued Nov. 5, 2025. This attempt was largely successful. See Jt. Status Rep't, filed Nov. 10, 2025. With the clarifications from the parties, the ganglioside / glycolipid theory can be divided into the following components:

Part	Aspect	Pet'r's Assertion	Resp't's Response
1	Structural	Prevnar contains some strains that contain galactose residues that are terminal.	Not disputed
2	Structural	Myelin contains gangliosides.	Not disputed
3	Structural	Some gangliosides in myelin and axons contain a terminal galactose residue.	Not disputed
4	Immunologic	In responding to Prevnar, the human immune system sees galactose residues and develops an immune response to the galactose residues.	Disputed
5	Immunologic / Neurologic	Anti-galactose antibodies (or anti-ganglioside antibodies) produced in response to Prevnar go to the peripheral nervous system to attack gangliosides in myelin and axons, causing AMAN.	Disputed

E. Hearing

The parties presented oral testimony at a hearing on November 13-14, 2025, which was conducted by videoconferencing. Mr. Davenport testified. He made a favorable impression as a person who was healthy before receiving Prevnar. He movingly explained how AMAN has affected his life. For someone who had suffered greatly, his outlook was refreshingly positive.

Dr. Sheikh testified. Although Dr. Sheikh had written reports in the Vaccine Program since approximately 2014 (Tr. 114), this occasion was his first time testifying orally. Tr. 95. Before testifying, Dr. Sheikh met with Mr. Davenport's attorneys four times. Each session to prepare Dr. Sheikh's testimony lasted approximately two or two and a half hours. Tr. 116.

Dr. Sheikh's demeanor while testifying may have reflected his lack of experience in testifying. His testimony, particularly on direct examination, came across as overly regimented / scripted. By way of contrast, his relatively short rebuttal testimony appeared much more spontaneous. The undersigned anticipates that as Dr. Sheikh takes the witness stand more frequently, his ability to present his opinions persuasively will increase. In this regard, Dr. Sheikh should also learn from the experience regarding the alum theory. Dr. Sheikh should not have taken about a page from another expert's report without crediting the original expert. See Raymo v. Sec'y of Health & Human Servs., No. 11-0654V, 2014 WL 1092274, at *13-14 (Fed. Cl. Spec. Mstr. Feb. 24, 2014) (discussing plagiarism). Dr. Sheikh would have made a better impression if this controversy had not developed.

After Dr. Sheikh finished his oral testimony, the Secretary called both his experts. Dr. Jamieson was generally fine. During cross-examination on two occasions, she was asked her opinions about immunologic topics. Tr. 179-80. She answered that she was deferring to Dr.

Kedl. This deferral seemed inappropriate as Mr. Davenport’s attorney was probing the limits of Dr. Jamieson’s opinions.⁷

Of the three testifying professionals, Dr. Kedl made the most favorable impression. He explained complicated topics in a way that made them understandable.⁸

Following the hearing, the parties were not directed to argue their cases through post-hearing briefs. Thus, with the completion of testimony, the case is ready for adjudication.

III. Standards for Adjudication

A petitioner is required to establish his case by a preponderance of the evidence. 42 U.S.C. § 300aa–13(1)(a). The preponderance of the evidence standard requires a “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence.” Moberly v. Sec’y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010) (citations omitted). Proof of medical certainty is not required. Bunting v. Sec’y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991).

Distinguishing between “preponderant evidence” and “medical certainty” is important because a special master should not impose an evidentiary burden that is too high. Andreu v. Sec’y of Health & Hum. Servs., 569 F.3d 1367, 1379-80 (Fed. Cir. 2009) (reversing special master's decision that petitioners were not entitled to compensation); see also Lampe v. Sec’y of Health & Hum. Servs., 219 F.3d 1357 (Fed. Cir. 2000); Hodges v. Sec’y of Health & Hum. Servs., 9 F.3d 958, 961 (Fed. Cir. 1993) (disagreeing with dissenting judge's contention that the special master confused preponderance of the evidence with medical certainty).

IV. Elements of a Petitioner’s Claim

To receive compensation, a petitioner must establish five elements. 42 U.S.C. § 300aa–11(c)(1)(A) through (E); 42 U.S.C. § 300aa–13(a)(1)(A) (authorizing special masters to award compensation when petitioners establish items listed in section 11(c)(1)). Often a critical element is causation, which is found in paragraph (C) of 42 U.S.C. § 300aa–11(c)(1).

When a petitioner seeks compensation for a claim not based upon the Vaccine Injury Table (like Mr. Davenport here), a petitioner is not entitled to a presumption of causation. Instead, a petitioner bears the burden of showing that the vaccine was the cause-in-fact for his injury. For causation-in-fact cases, the Federal Circuit has defined components of a petitioner’s burden. Petitioners bear a burden “to show by preponderant evidence that the vaccination

⁷ Mr. Davenport’s attorney appeared to accept Dr. Jamieson’s deferral to Dr. Kedl. The attorney did not seek an order requiring Dr. Jamieson to answer the questions.

⁸ Between the pre-trial conference and the hearing, Dr. Kedl created a demonstrative exhibit with information about enzymes from the Human Atlas Protein website and Mr. Davenport objected to its use. Tr. 9-12. A problem with the proposed demonstrative was that Dr. Kedl had not disclosed any opinions with respect to enzymes. See Tr. 199-202. The Secretary withdrew the demonstrative exhibit. Tr. 209.

brought about [the vaccinee's] injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.” Althen v. Sec’y of Health & Hum. Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005).

V. Analysis—Althen Prong One

Relevant evidence concerning the general topic of whether Prevnar can cause GBS falls into two broad categories. The first category (discussed in section A, below) concerns the theory that Dr. Sheikh advances, molecular mimicry. The second category (discussed in section B, below) concerns literature, such as epidemiologic studies, that discuss the topic. Another type of literature is case reports. Section C contains a short conclusion. Section D touches upon the remaining Althen prongs.

A. Molecular Mimicry

1. Introduction

Molecular mimicry is often advanced by petitioners in the Vaccine Program. Hoffman v. Sec’y of Health & Human Servs., No. 19-111V, 2024 WL 4444773 (Fed. Cl. Spec. Mstr. Sep. 13, 2024) (listing cases in appendices). At a basic level, molecular mimicry is a theory that can be reduced to two parts: (a) a similarity between components of an antigen, such as an infectious agent or a vaccine, and host tissue, and (b) a pathologic cross-reaction. Tr. 63, 222; see also Pet’r’s Br. at 45; Exhibit 19 at 9; Exhibit B at 9.⁹

By way of background, molecular mimicry was first proposed in the early 1980’s. Tr. 66. Dr. Sheikh’s first report cited three articles published in 1998-2005. Exhibit 19 at 6; see also Tr. 96-97; Romine v. Sec’y of Health & Human Servs., No. 19-468V, 2026 WL 937898, at *9 (Fed. Cl. Spec. Mstr. Mar. 13, 2026). As discussed below, in light of scientific advances since then, Dr. Sheikh and Dr. Kedl dispute the usefulness of molecular mimicry. Tr. 143-44, 247, 251; c.f. Tr. 196 (Dr. Jamieson does not teach students about “the immunological aspects of” molecular mimicry).

A significant discovery was the sequencing of the whole human genome. The technology led researchers to discover that infectious agents share homology with humans abundantly. Exhibit B at 12; Tr. 223-24, 306-07; see also Romine, 2026 WL 937898, at *9. Dr. Sheikh agrees that homology is common. Tr. 86, 129.

The commonness of homology undermines molecular mimicry as a theory. If homology occurs often, why do autoimmune diseases arise so rarely? See Exhibit B at 11-12. One possibility is host susceptibility. Tr. 86. But, any susceptibility in Mr. Davenport has not been shown here. Exhibit 19 (Dr. Sheikh’s first report) at 7-8; Tr. 88, 175.

⁹ The term “homology” is used when discussing molecular mimicry. “Homology” is defined as “the quality of being homologous; the morphological identity of corresponding parts; structural similarity due to descent from a common form.” *Dorland’s* at 868.

Outside the context of litigation, Dr. Sheikh described molecular mimicry in an article he prepared as “unproven.” Sheikh;¹⁰ Tr. 97-98. During his rebuttal testimony, Dr. Sheikh noted that this article was published in 1998. Tr. 313.

As support for his current opinion that molecular mimicry explains how Prevnar can cause GBS, Dr. Sheikh analogizes to an instance in which molecular mimicry is more supported. Medical researchers including Nobuhiro Yuki have linked a bacterium, *C. jejuni*, to GBS via molecular mimicry.¹¹ Exhibit 19 at 6; Tr. 98, 128, 315. Although Dr. Kedl recognized this connection, he pointed out that showing a living organism can be the basis for molecular mimicry is not the same as showing a non-replicating vaccine can be. Tr. 269-71.

Dr. Kedl illustrated his disagreement with molecular mimicry as a theory by discussing an example from medical history. An early vaccine against rabies, the Semple vaccine, was made from the brain and spinal cords of sheep that had been infected with rabies. People at risk of developing rabies were given 20-30 doses of the Semple vaccine. Under those conditions, about one in one thousand people developed neurologic diseases. But, the model of the Semple vaccine does not resemble what happens with modern vaccines. Tr. 225-27; see also Tompkins v. Sec’y of Health & Hum. Servs., No. 10-261V, 2013 WL 3498652, at *21 (Fed. Cl. Spec. Mstr. June 21, 2013), mot. for rev. denied, 117 Fed. Cl. 713 (2014); D.G. v. Sec’y of Health & Hum. Servs., No. 11-577V, 2019 WL 2511769, at *173 (Fed. Cl. Spec. Mstr. May 24, 2019).

Dr. Kedl opined homology, by itself, does not predict cross-reactivity. In his first report, Dr. Kedl demonstrated at least an inconsistency in the theory of molecular mimicry. Dr. Kedl explained that flu vaccines change each year because flu viruses are slightly different, but very homologous. If molecular mimicry functioned in real life, then there would be a “universal” flu vaccine because one dose would supplant the need for multiple flu vaccines. But, this is not how real life functions. Exhibit B at 18. Similarly, in his oral testimony, Dr. Kedl emphasized that each strain of *Streptococcus pneumoniae* is very similar to each other. However, the exposure to one strain does not provide immunity to other strains. The specificity of the immune system’s response explains why the vaccine is Prevnar “thirteen.” Tr. 255; accord Exhibit F at 3. Dr. Sheikh did not directly rebut this point about Prevnar-13. When Dr. Sheikh was asked a similar question about the flu vaccine, which contains three strains, he did not respond persuasively. Tr. 131.

Another example demonstrating the difference between homology and cross-reactivity is found in the article by Susumu Kusunoki and colleagues.¹² In this experiment, researchers found that *Mycoplasma pneumoniae* share a chemical structure resembling a glycolipid, galactocerebroside. Although the authors proposed molecular mimicry as a way that

¹⁰ K.A. Sheikh et al., Molecular Mimicry in Guillain-Barré Syndrome, 845 ANN. N.Y. ACAD. SCI. 307 (1998). Filed as Exhibit 21.

¹¹ Nobuhiro Yuki, Infectious origins of, and molecular mimicry in, Guillain-Barré and Fisher syndromes, 1 LANCET INFECT. DIS. 29 (2001). Filed as Exhibit 73.

¹² Susumu Kusunoki et al., Anti-Gal-C antibodies in GBS subsequent to mycoplasma infection: evidence of molecular mimicry, 57 NEUROLOGY 736 (2001). Filed as Exhibit 26.

Mycoplasma pneumoniae could cause demyelinating GBS, Dr. Sheikh stated that Kusunoki did not show cross-reactivity. Tr. 121 (Dr. Sheikh: Kusunoki “shows molecular mimicry, but it doesn’t show disease induction”); see also Tr. 293-94 (Dr. Kedl’s testimony about Kusunoki).

Based, in part, on this evidence, Dr. Kedl compared molecular to a sacred cow that has outlived its usefulness. Exhibit F at 3. In his oral testimony, Dr. Kedl acknowledged he engaged in a bit of hyperbole. Tr. 294. Even so, Dr. Kedl asserted that the perpetuation of molecular mimicry as a “failure of the field.” Tr. 255. A problem, according to Dr. Kedl, is that the neurologic community has not remained consistent with immunology. Tr. 247, 310. Dr. Kedl asserted that there is a “moral obligation” to stop looking for a cause of GBS (Prevnar vaccine) when we know that it is not the cause of GBS. Tr. 256. Instead, we should think more creatively. Tr. 295.

2. Appellate Precedents on Molecular Mimicry

A slightly different question concerns the level of proof when petitioners rely upon molecular mimicry. Because molecular mimicry is advanced so frequently, appellate authorities have had opportunities to review how special masters have considered evidence about molecular mimicry. In December 2019, the undersigned identified the leading precedents as W.C. v. Sec’y of Health & Hum. Servs., 704 F.3d 1352 (Fed. Cir. 2013), and Caves v. Sec’y of Dep’t. of Health & Hum. Servs., 100 Fed. Cl. 119 (2011), aff’d sub nom., 463 F. App’x 932 (Fed. Cir. 2012). Tullio v. Sec’y of Health & Hum. Servs., No. 15-51V, 2019 WL 7580149, at *12-14 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), mot. for rev. denied, 149 Fed. Cl. 448 (2020). While Tullio describes those cases in more detail, their essence appears to be that although molecular mimicry is accepted in some contexts, special masters may properly require some empirical evidence to show that a particular vaccine can cause a particular disease.

In the next approximately six years, appellate authorities reviewing decisions involving molecular mimicry have generally endorsed the approach of looking for some evidence that persuasively shows that a portion of a vaccine resembles a portion of human tissue, which contributes to causing the disease, and that the immune system will respond to the relevant amino acid sequence. Chronologically, the list of more recent appellate cases begins with the opinion in Tullio, which denied the motion for review. 149 Fed. Cl. 448, 467-68 (2020).

Another example in which the Court of Federal Claims held that the special master did not elevate the petitioner’s burden of proof in the context of evaluating the theory of molecular mimicry is Morgan v. Sec’y of Health & Hum. Servs., 148 Fed. Cl. 454, 476-77 (2020), aff’d in non-precedential opinion, 850 F. App’x 775 (Fed. Cir. 2021). In Morgan, the Chief Special Master found that petitioner had not presented persuasive evidence about a relevant antibody. Id. at 477. The Chief Special Master also noted that the articles about the relevant disease do not list the wild flu virus as potentially causing the disease. Id. When examining this analysis, the Court of Federal Claims concluded: “the Chief Special Master did not raise the burden of causation in this case; petitioner simply failed to meet it.” Id.

The Federal Circuit also evaluated the Chief Special Master’s approach in Morgan. The Federal Circuit concluded: “We discern no error in the special master’s causation analysis.” 850 F. App’x 775, 784 (Fed. Cir. 2021).

Most other recent appellate cases follow this path. See, e.g., Stricker v. Sec’y of Health & Hum. Servs., 170 Fed. Cl. 701, 720-21 (2024); Duncan v. Sec’y of Health & Hum. Servs., 153 Fed. Cl. 642, 661 (2021) (finding the special master did not err in rejecting a bare assertion of molecular mimicry and stating “there is an important difference between the general theory of molecular mimicry and the more specific theory that the vaccine at issue is capable of triggering an autoimmune response that culminates in the petitioner’s injury”); Caredio v. Sec’y of Health & Hum. Servs., No. 17-79V, 2021 WL 6058835, at *11 (Fed. Cl. Dec. 3, 2021) (indicating that a special master did not err in requiring more than homology and citing Tullio); Yalacki v. Sec’y of Health & Hum. Servs., 146 Fed. Cl. 80, 91-92 (2019) (ruling that special master did not err in looking for reliable evidence to support molecular mimicry as a theory); but see Patton v. Sec’y of Health & Hum. Servs., 157 Fed. Cl. 159, 169 (2021) (finding that a special master erred in requiring petitioner submit a study to establish medical theory causally connecting flu vaccine to brachial neuritis).

The Court of Federal Claims has explained why petitioners must present some evidence to show the persuasiveness of molecular mimicry as a theory in their cases. Dennington v. Sec’y of Health & Hum. Servs., 167 Fed. Cl. 640 (2023), appeal withdrawn, No. 2024-1214 (Fed. Cir. Mar. 25, 2024). There, Ms. Dennington alleged that a tetanus-diphtheria-acellular pertussis (“Tdap”) vaccine caused her to develop GBS. Id. at 644. She supported her claim with two reports from a neurologist, Carlo Tornatore, who put forward molecular mimicry. Id. at 647-49. The chief special master denied entitlement. Id. at 656.

The Court of Federal Claims denied a motion for review because the Chief Special Master did not commit any error in evaluating Ms. Dennington’s prong one evidence. The Court emphasized the lack of evidence supporting Dr. Tornatore’s opinion:

- “While Petitioner and Dr. Tornatore put forth the well-established medical theory of molecular mimicry as the mechanism through which the Tdap vaccine could cause GBS, nowhere in Dr. Tornatore’s expert reports, nor in Petitioner’s briefs, do they specifically tie the Tdap vaccine to GBS through molecular mimicry.” Id. at 653.
- “Dr. Tornatore never actually explains how molecular mimicry might occur from the Tdap vaccine specifically, nor does he elaborate on how molecular mimicry could cause the specific autoimmune system reaction that could cause GBS.” Id.
- “There is nothing in Dr. Tornatore’s report that explains or even alludes to what antigens or structures in the Tdap vaccine could share homology with possible host antigens and how these antigens could react in the manner GBS is believed to progress.” Id. at 654.
- “The literature upon which he relies make no mention of any causal connection between GBS and the Tdap vaccine.” Id.

Based upon these observations, the Court criticized the lack of specificity in Dr. Tornatore’s opinions:

In fact, because Dr. Tornatore does not offer any specific explanation as to the distinct connection between Tdap, molecular mimicry, and GBS, one could take Dr. Tornatore's causation theory and substitute any table vaccine (e.g., the measles vaccine) and any autoimmune disorder (e.g., autoimmune encephalitis) and Dr. Tornatore's expert report's discussion of molecular mimicry would require absolutely no changes. That is how general her molecular mimicry theory is—it does not matter which vaccine and which autoimmune disorder are plugged in. But Althen prong one requires more.

Id.

In accordance with precedents such as W.C., Caves, Tulio, Yalacki, Stricker, Duncan, and Dennington, the undersigned will look to see whether any evidence supports the theory in this case.

3. Specific Proposal

The background about molecular mimicry and legal precedents are foundations for evaluating Dr. Sheikh's opinion that molecular mimicry can explain how Plevnar can cause GBS (or the more specific subtype, AMAN). To recap, Dr. Sheikh proposes that in responding to Plevnar, the human immune system sees galactose residues, the immune system produces anti-galactose antibodies, and the anti-galactose antibodies go to the peripheral nervous system, where they attack gangliosides in myelin and axons causing AMAN. See Section II.D. (presenting Joint Status Report, filed Nov. 10, 2025).

Dr. Sheikh's molecular mimicry lacks persuasiveness for two independent reasons. First, Mr. Davenport has not demonstrated the reliability of the assertion that Plevnar leads to the creation of anti-galactose antibodies. As described below, this point is relatively simple. The simplicity derives from the overall lack of dispute about this point. On the other hand, the second flaw with Dr. Sheikh's molecular mimicry theory is more complicated and disputed. This point is whether anti-galactose antibodies cause AMAN.

a) Lack of Support for Plevnar Leading to Anti-Galactose Antibodies

A fundamental step required by Dr. Sheikh's theory is that Plevnar instigates the production of antibodies that attack galactose. This step is foundational in the sense that if Plevnar does not prompt the production of anti-galactose antibodies, then any consideration of whether anti-galactose antibodies are pathologic is hypothetical. Mr. Davenport has not established the persuasiveness of this assertion.

A review of the evidence shows that the topic of whether Plevnar induces anti-galactose antibodies has recurred throughout this case. Dr. Sheikh's first report stated: "In *theory*, Plevnar 13 CPS can induce anti-glycan antibodies." Exhibit 19 at 10 (emphasis added). Dr. Kedl challenged this assertion in his first report. Dr. Kedl wrote: "It is first worth noting that no ganglioside-specific antibodies have ever been observed in response to the Plevnar vaccine, either in animal models or clinically." Exhibit B at 16. Despite this challenge, it appears that Dr. Sheikh did not reinforce the basis for his theoretical assertion that Plevnar can induce anti-glycan antibodies. See Exhibit 70. But, Dr. Kedl returned to this point in his second report: "what needs to be emphasized is that no ganglioside-specific antibodies have been documented in

response to the Prevnar vaccine, either in animal models or clinically.” Exhibit F at 2. The Secretary repeated this argument, maintaining that Dr. Sheikh’s “proposed theory is not scientifically sound because ‘no ganglioside-specific antibodies have been documented in response to the Prevnar vaccine.’” Resp’t’s Br. at 24, quoting Exhibit F at 2.

The testimony during the hearing confirmed the lack of reliability for Dr. Sheikh’s proposal that Prevnar can lead to the production of anti-galactose antibodies. When asked, Dr. Sheikh stated that he has no proof for this point. Tr. 122. Further, Dr. Sheikh recognized that this aspect of his opinion is easily tested but has not been tested. *Id.* In accordance with his reports, Dr. Kedl testified that there is no evidence that Prevnar elicits a response specific for gangliosides. Tr. 216-17, 249-50. He later explained that no one has looked probably because there is no epidemiological basis for associating *Streptococcus pneumoniae* or Prevnar vaccine with GBS. Tr. 307.

When a great analytical gap separates the data and the opinion, a special master may reject the expert’s opinion. *Cedillo v. Sec’y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010). Here, Mr. Davenport has presented no reliable data to support Dr. Sheikh’s theoretical opinion that Prevnar can induce anti-galactose antibodies. Thus, Dr. Sheikh’s opinion is not credited. The lack of support for this step in Dr. Sheikh’s opinion is fatal.

b) Lack of Support for Assertion that Anti-Galactose Antibodies Cause AMAN

Dr. Kedl reasoned that because the assertion that Prevnar leads to the production of anti-galactose antibodies is “pure speculation,” whether the anti-galactose antibodies cause AMAN is irrelevant. Exhibit F at 2. This reasoning is sensible. Nevertheless, this complicated issue is addressed, beginning with a re-review of the disclosure of opinions in the experts’ reports.

Dr. Sheikh’s first report states: “Experimental studies demonstrate that the anti-ganglioside antibodies can produce inflammatory nerve injury mimicking axonal GBS in experimental models.” Exhibit 19 at 6 (citing He).¹³ Similarly, Dr. Sheikh wrote: “experimental animals studies demonstrate that anti-GalC antibodies produce inflammatory nerve injury resembling demyelination seen in AIDP patients.” *Id.* (citing Saida).¹⁴ Dr. Kedl disagreed. Dr. Kedl was skeptical of anti-galactose antibodies causing AMAN because anti-galactose antibodies should also attack other structures containing galactose. Exhibit B at 16. Dr. Kedl provided a specific example. He pointed out that keratan sulfate is another molecule that contains galactose and is present in the eye, cartilage, and bone. By Dr. Sheikh’s theory, the anti-galactose antibodies should also cause joint disfunction and/or blindness. But, they do not. *Id.* at 17.

Unlike Dr. Kedl’s critique of Dr. Sheikh’s assertion that Prevnar can induce anti-galactose antibodies to which Dr. Sheikh did not respond, Dr. Sheikh addressed Dr. Kedl’s opinion. Dr. Sheikh recognized that the question of why anti-galactose antibodies would predominantly injure

¹³ Lan He et al., Anti-Ganglioside Antibodies Induce Nodal and Axonal Injury via Fcγ Receptor-Mediated Inflammation, 35 J. NEUROSCI. 6770 (2015). Filed as Exhibit 25.

¹⁴ Kyoko Saida et al., In Vivo Demyelination Induced by Intraneural Injection of Anti-Galactocerebroside Serum, 95 AM. J. PATHOL. 99 (1979). Filed as Exhibit 28.

the nerves of the peripheral nervous system as opposed to other tissues in the human body is an “important and relevant question.” Exhibit 70 at 3. Dr. Sheikh also “admit[ted] that this is a complicated multifaceted issue and not all aspects related to it are completely worked out.” Id. Dr. Sheikh wrote multiple pages about this topic. Unfortunately, however, his opinion is neither easily understood nor easily summarized. See id. at 3-10. Dr. Sheikh did not respond to keratan sulfate.

Dr. Kedl challenged the persuasiveness of Dr. Sheikh’s explanation. Dr. Kedl put forward three reasons for why Dr. Sheikh has not persuasively explained why anti-galactose antibodies would attack only nerve cells. These are: (1) the underlying experiments do not involve tissues other than tissues from the nervous system; (2) if Prevnar can induce anti-galactose antibodies with fine specificity for nervous system tissue, then Prevnar can induce anti-galactose antibodies that are cross-reactive more broadly; and (3) Dr. Sheikh’s complicated explanation “violates the ‘law of parsimony,’ colloquially known as ‘Occam’s Razor.’” Exhibit F at 4-5. In this context, Dr. Kedl suggests that the anti-galactose antibodies may indicate (not cause) neuronal disease. Id. at 5.

The parties’ briefs did not flesh out this issue effectively. To Mr. Davenport’s credit, he devoted about two pages to the topic of whether anti-galactose antibodies can cause GBS. See Pet’r’s Br. at 50-51. The Secretary did not discuss this topic. See Resp’t’s Br.

Although not a point of emphasis in the pre-hearing briefs, the parties solicited testimony from their experts during the hearing. Similar to the opinion Dr. Sheikh expressed in his second report, he acknowledged that why axons (but not myelin) are attacked in AMAN is not well understood. Tr. 76-79; see also Tr. 137. But, Dr. Sheikh stated that the Lopez¹⁵ article proposes the “fine specificity” that he was proposing. Likewise, the He article also shows that the antibodies are pathogenic. Tr. 65-67, 137. The He article was a basis for revising Dr. Sheikh’s statement in a 1999 article that whether anti-GM1 antibodies were pathogenic was unknown. Tr. 101-02.

In contrast, Dr. Kedl questioned whether the anti-galactose antibodies were causative. Dr. Kedl stated that about 30-50% of the patients with AMAN have anti-ganglioside antibodies. Tr. 216, 305-06. Thus, in the remaining patients, something other than anti-ganglioside antibodies is driving the disease.

Dr. Kedl also returned to a topic discussed in his expert reports—the commonness of gangliosides. Tr. 231-32. He reiterated that keratan sulfate is a carbohydrate containing galactose and, therefore, would potentially be vulnerable to the anti-galactose antibodies. Tr. 238. The lack of attack on keratan sulfate suggests that the anti-galactose antibodies are not causative. Dr. Kedl added that infectious agents that are at least associated with GBS, such as cytomegalovirus, do not express gangliosides. Tr. 247, 293. For these reasons, Dr. Kedl suggested that anti-ganglioside antibodies may simply indicate that damage has been done by something else. Tr. 242, 247, 293-94.

¹⁵ Pablo H. H. Lopez et al., Structural requirements of anti-GD1a antibodies determine their target specificity, 131 BRAIN 1926 (2008). Filed as Exhibit 77.

Overall, an assessment of the reports, articles, briefs, and testimony suggests that Mr. Davenport has not supported this aspect of the case with preponderant evidence. One reason is that Dr. Sheikh's oral presentation on this topic was not very helpful. See Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1325-26 (Fed. Cir. 2010).¹⁶ Admittedly, the topic is, to use Dr. Sheikh's terminology, "a complicated multifaceted issue." Exhibit 70 at 3. But, the complicated nature of the topic calls for a better presentation. Instead, Mr. Davenport's attorneys showed a series of tables and figures from various articles without really explaining the background principles. Although this testimony and associated images have been reviewed and re-reviewed, it is not readily apparent that this presentation makes it likely that galactose is a target for AMAN. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1330 (Fed. Cir. 2016) (recognizing that "if the technical complexity of a particular study is such that the relevance of the medical study or its particular findings cannot be understood by the special master without expert assistance that was not provided, then the special master may conclude that this evidence or portion of the evidence is entitled to little or no weight"). At a basic level, opinions that are poorly explained (and therefore poorly understood) are not persuasive.

Another reason for not finding Dr. Sheikh's proposal that anti-ganglioside antibodies cause AMAN is that Dr. Sheikh did not persuasively rebut Dr. Kedl's point that gangliosides are found throughout the body and, therefore, should be attacked by the anti-ganglioside antibodies that are associated with GBS. For example, Dr. Sheikh did not respond effectively to Dr. Kedl's example of keratan sulfate.

Overall, Mr. Davenport has not met his burden of demonstrating the reliability of Dr. Sheikh's opinion that Prevnar can cause AMAN through molecular mimicry.

B. Epidemiology and Case Reports

For a lengthy discussion of the value of epidemiologic studies in the Vaccine Program, see Tullio v. Sec'y of Health & Hum. Servs., No. 15-51V, 2019 WL 7580149, at *5-8 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), mot. for rev. denied, 149 Fed. Cl. 448, 475 (2020). A purpose of epidemiology is an attempt to separate chaos in the universe from causal relationships. Tr. 274.

This section evaluates three types of studies. In the first type, which is discussed in section 1, researchers looked to see whether people who received *vaccines* against pneumococcus developed GBS. In the second type, which is discussed in section 2, the question is whether people who are *infected* with the pneumococcus bacteria develop GBS. Section 3 discusses case reports.

¹⁶ In this passage, the Federal Circuit states that a special master may consider the "credibility" of an expert. As explained in the text, Dr. Sheikh was not persuasive. This finding that Dr. Sheikh lacked persuasiveness should not be interpreted as implying that Dr. Sheikh lacked sincerity or that Dr. Sheikh was dishonest.

1. Pneumococcal vaccines and GBS

Here, four articles explored whether people who received a vaccine against pneumococcus developed GBS at an increased incidence. These are Baxter, Haber, Tseng, and Cordonnier.

a) *Baxter*¹⁷

This group of researchers investigated “the possible relationship between GBS and vaccinations.” Baxter at 198. To do so, they consulted electronic medical records from Kaiser Permanente Northern California. Their study spanned 13 years and more than 30 million person-years. The researchers conducted two different types of analysis, a case-centered study and a cohort analysis. *Id.* at 198-99. In short, the researchers looked to see if people receiving various vaccines developed GBS more frequently. One of the vaccines was pneumovax 23 (labeled as PPV-23 in Baxter). The Baxter researchers did not study Prevnar because Prevnar was not available. The researchers “found no evidence of an increased risk of GBS following any vaccination, as well as vaccinations combined.” Baxter at 201; *accord* Tr. 166. The researchers acknowledged that they “had limited power to fully assess the risk of GBS following vaccination due to the rarity of the outcome.” However, they concluded that “the low numbers of GBS cases that were temporally associated with vaccination, coupled with our results, provide reassurance that the risk of GBS following any vaccine, including influenza vaccines, is extremely low.” *Id.* at 203.

b) *Haber*¹⁸

A group of researchers from the CDC consulted the VAERS database for adverse events following Prevnar 13. Special masters are familiar with the VAERS system. As explained in Haber, “VAERS is a national vaccine safety surveillance program run by CDC and the Food and Drug Administration (FDA). This early warning system is designed to detect possible safety issues with U. S.-licensed vaccines.” Haber at 6631. A basic purpose of the vaccine adverse event reporting system is to surveil for potential problems that warrant further investigation.

Often, an analysis of VAERS data can be unreliable because researchers do not know how many doses of a vaccine were given. *Puckett v. Sec’y of Health & Hum. Servs.*, No. 21-1125V, 2024 WL 3160589, at *4 (Fed. Cl. Spec. Mstr. June 3, 2024) (citing cases); *Ferguson v. Sec’y of Health & Hum. Servs.*, No. 17-1737V, 2021 WL 6276204, at *18 n.48 (Fed. Cl. Spec. Mstr. Dec. 10, 2021). The Federal Circuit has recognized difficulty in drawing conclusions from data when the denominator is not known. *GHS Health Maintenance Organization, Inc. v. United States*, 536 F.3d 1293, 1302 n.6 (Fed. Cir. 2008). In Haber, however, there is datum to fill the

¹⁷ Roger Baxter et al., Lack of Association of Guillain-Barré Syndrome With Vaccinations, 57 CLIN. INFECT. DIS. 197 (2013). Filed as Exhibit A-2.

¹⁸ Penina Haber et al., Post-licensure surveillance of 13-valent pneumococcal conjugate vaccine (PCV13) in adults aged ≥19years old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012-December 31, 2015, 34 VACCINE 6330 (2016). Filed as Exhibit A-5.

denominator. Haber researchers stated that in the study, approximately 16,000,000 doses were distributed. Haber at 6333; see also Tr. 164.¹⁹

In the relevant time, the VAERS database contained 10 reports of people age greater than 65 years old developing GBS. Tr. 181. The question then becomes: do 10 reports of GBS raise concern? The usual annual background incidence of GBS is roughly 1 to 2 cases per 100,000 people. Allen & Parry at 398.²⁰

For adverse events generally, the Haber researchers stated: “We . . . did not identify any new safety concerns or unexpected [adverse events].” Haber at 6333. For the question of GBS specifically, the Haber researchers reported: “Our data mining analysis noted no disproportionate reporting for GBS.” Haber at 6334. The Secretary’s expert, Dr. Jamieson emphasizes this aspect of the Haber article. Tr. 165.

Through cross-examination, Mr. Davenport brought forward the numerator, meaning the number of cases reported. Tr. 276 (Dr. Kedl). But, when considered in the context of the denominator (the population exposed to the vaccination), these reports in Haber resemble a case series. Case series are not a persuasive basis for finding causation. Virissimo v. Sec’y of Health & Hum. Servs., No. 24-1168V, 2025 WL 2439181, at *3 (Fed. Cl. Spec. Mstr. July 30, 2025); Cerrone v. Sec’y of Health & Hum. Servs., No. 17-1158V, 2023 WL 3816718, at *19 (Fed. Cl. June 1, 2023) (summarizing opinion of the Secretary’s expert), mot. for rev. denied, 168 Fed. Cl. 745 (2023), aff’d, 146 F.4th 1113 (Fed. Cir. 2025); see also K.O. v. Sec’y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *11-12 (Fed. Cl. Spec. Mstr. July 7, 2016) (discussing appellate precedent on case reports). Dr. Kedl was persuasive when, in the context of discussing Haber, he stated that the identification of some cases of GBS occurring after Prevnar is not the same as showing a causal association. Tr. 276.

c) *Tseng*²¹

This group explored how the safety of the two pneumococcal vaccinations compared. Tr. 165. The older vaccine was pneumovax (labeled as PPSV 23). The newer vaccine was Prevnar 13 (labeled as PCV 13). To determine whether one vaccine had more adverse events, the researchers consulted the Vaccine Safety Database (VSD). Tseng at 2. The VSD contains medical information about millions of people. Sparrow v. Sec’y of Health & Hum. Servs., No. 18-295V, 2024 WL 1599165, at *19 (Fed. Cl. Spec. Mstr. Mar. 19, 2024) (the “VSD has data on >9 million subjects annually”), mot. for rev. denied, 173 Fed. Cl. 177 (2024), appeal docketed, No. 2025-1161 (Fed. Cir. Nov. 8, 2024). For more information about the VSD, see Dwyer v. Sec’y of Health & Hum. Servs., No. 03-1202V, 2010 WL 892250, at *67 n.284 (Fed. Cl. Spec.

¹⁹ Technically, the doses distributed could exceed the doses administered. But, the number of doses distributed can serve as a rough proxy for the number of doses administered. Likewise, the numerator (adverse events) may be affected by underreporting.

²⁰ Jeffrey A. Allen & Gareth J. Parry, Acquired Immunologic Neuropathies, 35 SEMIN. NEUROL. 398 (2015). Filed as Exhibit A-1.

²¹ Hung Fu Tseng et al., Pneumococcal Conjugate Vaccine Safety in Elderly Adults, 5 OPEN FORUM INFECT. DIS. 1 (2018). Filed as Exhibit B-26.

Mstr. Mar. 12, 2010); Werderitsh v. Sec'y of Health & Hum. Servs., No. 99-319V, 2005 WL 3320041, at *15 (Fed. Cl. Spec. Mstr. Nov. 10, 2005). The researchers searched specifically for GBS. Tseng at 3; Tr. 100. The incidence of GBS was not meaningfully different. Id. at 6 (Table 3); Tr. 165.

d) *Cordonnier*²²

This group of researchers explored the efficacy and safety of pneumococcal vaccines in a group (approximately 250 people) who received hematopoietic stem cells transplants. Six months after the last PCV 13 vaccination, participants provided researchers information about serious adverse events through a telephone survey. Cordonnier at 314. Of the group that responded, one person reported Guillain-Barré syndrome that developed 29 days after PCV13 dose 4 and 1 day after PPSV23. Id. at 319. The researchers refrained from concluding that there was a causal relationship because the person “had a complex constellation of comorbid conditions, received concomitant medications, and [was] exposed to multiple infections and [graft-vs-host disease].” Id. at 321; accord Tr. 167.

e) *Summary*

Collectively, these studies tend to reinforce each other in suggesting that an association between a pneumococcal vaccine and GBS has not been found, despite attempts to find such an association. A summary of these studies is:

Epidemiological Studies on Pneumococcal Vaccines and GBS			
<u>Study & Year</u>	<u>Vaccine</u>	<u>Population</u>	<u>Finding</u>
Baxter, 2013	Pneumovax	13 years and 30 million-person years	No increased risk
Cordonnier, 2015	Prevnar and Pneumovax	241 Recipients of Stem Cell Transplant	Causation not ascribed to one instance of GBS
Haber, 2016	Prevnar	16 million doses distributed	No disproportionate reporting
Tseng, 2018	Prevnar and Pneumovax	Millions in Vaccine Safety Datalink	No difference in safety

While it is true that these epidemiological studies have other limitations, all studies have some limitations. Martinez v. Sec'y of Health & Hum. Servs., No. 16-738V, 2022 WL 4884923,

²² Catherine Cordonnier et al., Immunogenicity, Safety, and Tolerability of 13-Valent Pneumococcal Conjugate Vaccine Followed by 23-Valent Pneumococcal Polysaccharide Vaccine in Recipients of Allogeneic Hematopoietic Stem Cell Transplant Aged >2 Years: An Open-Label Study, 61 CLIN. INFECT. DIS. 313 (2015). Filed as Exhibit 37 and Exhibit A-3.

at *30 (Fed. Cl. Spec. Mstr. Sep. 9, 2022), mot. for rev. denied, 165 Fed. Cl. 76 (2023). It would be unusual for one study to be decisive. The Secretary is not burdened with an obligation to prove beyond a reasonable doubt that Prevnar cannot cause GBS. Instead, Mr. Davenport bears a burden to present reliable evidence showing, more likely than not, that Prevnar can cause GBS. Although the burden rests with the petitioner, the Secretary can introduce evidence undermining a petitioner's case. Bazan v. Sec'y of Health and Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008). Here, the epidemiological studies on pneumococcal vaccines make the claim less likely, but not impossible.

2. Pneumococcus bacteria and GBS

The previous section addresses studies on pneumococcal vaccines and GBS. A different way of looking at the question is whether the *Streptococcus pneumoniae* bacteria is associated with GBS.

The experts agreed that an infection with *Streptococcus pneumoniae* has been reported to precede the development of Guillain-Barré syndrome rarely. Tr. 102-03 (Dr. Sheikh), Tr. 160 (Dr. Jamieson), Tr. 216-18 (Dr. Kedl), Tr. 247 (Dr. Kedl), Tr. 255-56 (Dr. Kedl). To illustrate that an association has been noted, Dr. Sheikh relied upon a case report by Bianchi.²³ A case report, however, provides relatively little (if any) support for the assertion that the antecedent event caused the subsequent event. See Tr. 258-60. When bacteria do not cause a disease, then there is a decreased likelihood that a vaccine against those bacteria causes the disease. See Gamboa-Avila v. Sec'y of Health & Hum. Servs., 166 F.4th 1318, 1322 (Fed. Cir. 2026) (ruling that special master was not arbitrary in denying entitlement because, in part, the special master noted that the wild bacteria is not associated with GBS).

3. Case Reports regarding Pneumococcal Vaccines and GBS

In support of his opinion that Prevnar can cause GBS, Dr. Sheikh cited a case report by Nidhi Ravishankar.²⁴ This evidence carries negligible weight for three reasons.

First, as a general proposition, case reports are not a reliable basis for inferring causation because case reports present a sequence of events. Tr. 217, 274-75. For a case discussing appellate precedents regarding case reports in the Vaccine Program, see K.O., 2016 WL 7634491.

Second, Dr. Jamieson proposed that the subject of the case report did not suffer from Guillain-Barré syndrome. Tr. 168-69. Dr. Jamieson's opinion appears persuasive because, in part, Dr. Sheikh did not contradict it during his rebuttal testimony.

²³ Giorgia Bianchi & Guido Domenighetti, Pneumococcus pneumoniae infection and Guillain-Barré syndrome: fortuitous or specific association?, 32 INTENSIVE CARE MED. 338 (2006). Filed as Exhibit 41 and Exhibit 66.

²⁴ Nidhi Ravishankar, Guillain-Barre Syndrome Following PCV Vaccine, 4 J. NEUROL. NEUROSURG. 1 (2017). Filed as Exhibit 39.

Third, as noted in several opinions from special masters, the provenance of this case report appears questionable as the author, a medical student, published in two different journals. See Anderson v. Sec’y of Health & Human Servs., No. 18-484V, 2024 WL 557052, at * 28 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); Bielak v. Sec’y of Health & Human Servs., No. 18-761V, 2023 WL 35509, at *35 (Fed. Cl. Spec. Mstr. Jan. 3, 2023); Pierson v. Sec’y of Health & Human Servs., No. 17-1136V, 2022 WL 322836, at *29 n.34 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); see also Tr. 167-68.

4. Synopsis on pneumococcal bacteria, pneumococcal vaccines and GBS

In sum, the Secretary has presented two similar, but not identical, points. First, three large-scale epidemiological studies have looked for an increased risk of GBS after vaccines against *Streptococcus pneumoniae*. They did not find an increased risk. Second, there appears to be a very small number of reports of a wild *Streptococcus pneumoniae* infection preceding GBS. See Tr. 103 (Dr. Sheikh affirming that “the best evidence [he has] providing a link between pneumococcal infection and GBS are the handful of case reports that [he] cited”).

Together, these lines of evidence weigh against finding that Prevnar can cause GBS. Thus, they tend to reinforce the previous finding that the molecular mimicry theory discussed above is not persuasive.

C. Summary on *Althen* Prong One

Differentiating the finding that Mr. Davenport did not meet his burden of proof regarding the potential causative role of anti-ganglioside antibodies from the previous finding regarding whether Prevnar can induce anti-ganglioside antibodies is important. As explained in section V.A.3. above, the present case contains almost no evidence that Prevnar induces anti-ganglioside antibodies. The evidence is not close.

By way of contrast, some evidence, such as the He article, arguably supports the proposition that anti-galactose antibodies cause at least some cases of GBS. Thus, the issue is not as easily open and shut as the question about Prevnar inducing anti-galactose antibodies. Conceivably, a better evidentiary presentation, starting with the disclosure of opinions in reports, could lead to a different outcome on this point. But, that hypothetical case was not presented for Mr. Davenport. See Sword v. United States, 44 Fed. Cl. 183, 190 (1999) (denying a motion for review and stating “counsel must live with their witnesses’ testimony; they do not usually get a chance to do it better a second time”).

Moreover, whether anti-galactose antibodies cause AMAN is no more than a third-level point. The more important factors include the lack of epidemiology associating vaccines against *Streptococcus pneumoniae* with GBS and the lack of association between *Streptococcus pneumoniae* infections and GBS. See Gamboa-Avila, 166 F.4th at 1322 (stating that a denial of compensation based, in part, on these points was not arbitrary or capricious). Another more important factor is the lack of persuasive proof for the assertion that Prevnar induces anti-galactose antibodies. In other words, even if Mr. Davenport had established that anti-ganglioside antibodies can cause AMAN, the result would have been the same. Other weaknesses would prevent a finding favorable to Mr. Davenport on Althen’s first prong.

D. Remaining *Althen* Prongs

A finding that a petitioner did not succeed on one *Althen* prong is dispositive. *W.C. v. Sec’y of Health & Hum. Servs.*, 704 F.3d 1352, 1358 (Fed. Cir. 2013) (“a petitioner must establish all three prongs of the *Althen* test”); *Hibbard v. Sec’y of Health & Hum. Servs.*, 698 F.3d 1355, 1365 (Fed. Cir. 2012). Thus, the remaining *Althen* prongs can be discussed only briefly.

To start with the third *Althen* prong, Mr. Davenport asserted that his neurologic problem started within two weeks of the vaccination. Pet’r’s Brief, filed Aug. 4, 2023, at 98. Dr. Jamieson agreed that this period “falls within the accepted time period for a triggering linkage.” Exhibit A at 12. Thus, Mr. Davenport prevails upon prong 3. However, a “[t]emporal association is not sufficient, however, to establish causation in fact.” *Grant v. Sec’y of Health & Hum. Servs.*, 956 F.2d 1144 (Fed. Cir. 1992).

As for the second *Althen* prong, the Federal Circuit has instructed special masters to consider carefully the views of a treating doctor. *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1326 (Fed. Cir. 2006). Here, Mr. Davenport has identified some statements from treating medical professionals that could be interpreted as supporting his claim. See Pet’r’s Brief, at 95-98; But see Resp’t’s Br., filed Oct. 13, 2023, at 30 (challenging petitioner’s prong two arguments). If Mr. Davenport had met his burden regarding to present a reliable theory, then exploring these statements in detail would be worthwhile. But, in light of the lack persuasive proof on one element, the analysis of a second element need not be undertaken. *Langland v. Sec’y of Health & Hum. Servs.*, 109 Fed. Cl. 421, 438 (2013) (“prong one of *Althen* must be demonstrated, for prongs two and three to matter”). Therefore, no finding is made regarding the second *Althen* prong.

VI. Conclusion

Mr. Davenport presented emotionally compelling testimony about how his neurological disease affected him. He merits sympathy for this suffering. His resilient spirit is admirable.

However, petitioners receive compensation through the Vaccine Program only by presenting persuasive proof. As explained above, the record when considered as a whole does not weigh in Mr. Davenport’s favor.

The Clerk's Office is instructed to enter judgment in accord with this decision unless a motion for review is filed. Information about filing a motion for review, including the deadline, can be found in the Vaccine Rules, which are available on the website for the Court of Federal Claims.

IT IS SO ORDERED.

s/Christian J. Moran
Christian J. Moran
Special Master

Appendix: Explanation of Terms

A. ***Streptococcus pneumoniae***. “Streptococcus” is a genus of bacteria. “Pneumoniae” is a species, which causes pneumonia and other disorders. There are “many serovars, distinguished on the basis of the specific capsular polysaccharide.” Dorland’s at 1753. A “serovar,” in turn, is a “taxonomic subdivision of bacteria based on the kinds and combinations of constituent antigens present in the cell” and is also called a serotype. Id. at 1670.

B. **Prevnar**. Prevnar is a vaccine against different serotypes of *Streptococcus pneumoniae*. Prevnar contains capsular polysaccharides. Exhibit 124 at 1, cited in Pet’r’s Br., filed July 22, 2025, at 8.

C. **Axons**. The peripheral nervous system contains axons, which are similar to wires in that they carry electrical charges. Like wires, axons are generally insulated and the insulating material for axons is known as myelin.¹

Axons communicate with other axons at the nodes of Ranvier. Dorland’s at 1263; Tr. 67; see also Dorland’s at 1234 (illustration). At the node of Ranvier, the axon is not myelinated. Id.; Tr. 123. This relative lack of insulation exposes the axon and its constituent parts to the immune system.

D. **Gangliosides**. Axons contain substances called “gangliosides.” See Dorland’s at 752. One type of ganglioside is called GM1. Dorland’s at 752. Another ganglioside is called GD-1a.

Gangliosides are mostly carbohydrates and lipids. Tr. 230. Gangliosides are not proteins. The particular ganglioside of interest in Dr. Sheikh’s theory, GM-1, is formed through a process involving five enzymes. GM-1 is highly expressed in nerve cells, cells in the gastrointestinal tract, and in immune cells. Tr. 231; see also Tr. 237 (describing gangliosides as very common).

E. **Galactose**. Gangliosides are composed in part with galactose. Dorland’s at 746. Galactose is a component of galactocerebroside. Dorland’s at 741; id. at 328 (defining “cerebroside”). “Galactocerebroside” is often abbreviated “Gal-C.” Galactose can also be found in glycolipids. Dorland’s at 785.

¹ Pathogenic attacks on myelin are known as “demyelination.” Dorland’s at 480. However, Mr. Davenport’s type of GBS spared his myelin. Exhibit A (Dr. Jamieson) at 7, 13; Exhibit 19 (Dr. Sheikh) at 5.