In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 13, 2026

CHRISTINA K. FEE,	*	
Petitioner,	*	No. 19-1979V
v.	*	Special Master Young
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Jeffrey A. Golvash, Golvash & Epstein, LLC, Pittsburgh, PA, for Petitioner
Rachelle Bishop, United States Department of Justice, Washington, DC, for Respondent

RULING ON ENTITLEMENT[1]

On December 30, 2019, Christina Fee ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Pet., ECF No. 1. She alleged that she developed Guillain-Barré Syndrome ("GBS") as the result of a pneumococcal conjugate ("Prevnar 13") vaccine she received on February 2, 2018. *Id.* at 1. Respondent argued against compensation, asserting that Petitioner could not establish a causation-in-fact claim by a preponderance of the evidence. Resp't's Rept. at 7, ECF No. 20.

After carefully analyzing and weighing all the evidence presented in this case in accordance with the applicable legal standards,[3] I find that Petitioner has provided preponderant evidence that

[1] Because this Ruling 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 Ruling will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

[3] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the Ruling will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted);

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the Prevnar 13 vaccine she received on February 2, 2018, caused her to suffer from GBS. Accordingly, Petitioner is entitled to compensation.

I. Procedural History

Petitioner filed her petition and an affidavit on December 30, 2019. Pet., ECF No. 1. Petitioner then filed medical literature and medical records on December 31, 2019, and January 10, 2020. Pet'r's Exs. 1–4(G), ECF No. 5; Pet'r's Exs. 4(H)–4(Q), ECF No. 6; Pet'r's Exs. 4(R)–10, ECF No. 8; Pet'r's Exs. 11–15, ECF No. 10.

Respondent filed his Rule 4(c) report on September 17, 2020, arguing against compensation. Resp't's Rept. On January 28, 2021, Petitioner filed an expert report from Dr. James DeAngelo and supporting medical literature. Pet'r's Ex. 16, ECF No. 23; Pet'r's Exs. 17–26, ECF No. 24; Pet'r's Exs. 27–36, ECF No. 25; Pet'r's Exs. 37–43, ECF No. 26. On April 29, 2021, Respondent filed an expert report and CV from Dr. You-Wen He, and filed supporting medical literature on May 25, 2025. Resp't's Exs. A–B, ECF No. 28; Resp't's Exs. C–U, ECF No. 29.

Petitioner filed a supplemental report from Dr. DeAngelo on October 12, 2021, along with additional medical literature. Pet'r's Exs. 44–54, ECF No. 32. Respondent filed a responsive report and additional literature on January 28, 2022. Resp't's Exs. V–AH, ECF No. 35. Petitioner filed two additional reports from Dr. DeAngelo on June 9, 2022, and January 13, 2023; Respondent filed one additional report from Dr. He on August 18, 2022. Pet'r's Ex. 55, ECF No. 37; Pet'r's Exs. 56–63, ECF No. 38; Resp't's Ex. AI, ECF No. 39; Pet'r's Ex. 64, ECF No. 42.

Petitioner filed a motion for a Ruling on the Record on June 28, 2024. Pet'r's Mot., ECF No. 46. Respondent filed his response on August 11, 2024, and Petitioner filed her reply on August 26, 2024. Resp't's Resp, ECF No. 48; Pet'r's Reply, ECF No. 49. This matter is now ripe for consideration.

II. Medical History

Petitioner was born on December 21, 1960. Pet. at 1. In December 2017, prior to her receipt of the Prevnar 13 vaccine, Petitioner reported she had experienced an upper respiratory infection ("URI"). Pet'r's Ex. 4 at 772. Petitioner's pre-vaccination medical history is otherwise significant for asthma, chronic obstructive pulmonary disease ("COPD"), gastroesophageal reflux disease, irritable bowel syndrome, and breast cancer. *See id.* at 359; Pet'r's Ex. 5 at 2.

On February 2, 2018, Petitioner received a Prevnar 13 vaccination during a preventative health examination with her primary care physician ("PCP") Sharon Dawso. Pet'r's Ex. 2. Twelve days later, on February 14, 2018, Petitioner presented to the emergency department ("ED") at the University of Pittsburgh Medical Center ("UMPC") Cranberry with complaints of pain in her neck and between her shoulder blades, as well as numbness in her fingertips that started on February

see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

11, 2018. Pet'r's Ex. 4 at 358. She noted residual neck pain from a fall approximately three weeks prior and her provider documented that she had previously received the Prevnar 13 vaccine two weeks ago. *Id.* at 363. She was seen by Dr. Richard Wadas, who noted that her numbness and tingling presented in a stocking glove distribution that had not yet reached her forearms or legs. *Id.* at 920. Diagnostic imaging, including a computed tomography ("CT") scan of the head and cervical spine were negative for a spinal cord pathology. *Id.* at 921. Petitioner was given Toradol and advised to follow up with her PCP. *Id.* at 922.

Two days later, on February 16, 2018, Petitioner presented to neurologist Erek Lam with "a [five] day history of progressive bilateral upper and low extremity numbness, weakness, and midthoracic back pain." Pet'r's Ex. 5 at 2. She explained that her symptoms began with pain between her shoulder blades and then progressed to numbness in her fingers the next day. *Id.* She further explained that after her visit to the UMPC Cranberry ED she experienced new leg weakness, tongue numbness, and difficulty sleeping, as well as a new fall due to incoordination. *Id.* She reported that she had received the Prevnar 13 vaccine recently and that she also had a prior cold and URI. *Id.* Dr. Lam opined that her symptoms were consistent with GBS and recommended she present to the ED for hospital admission and intravenous immunoglobulin ("IVIG") therapy. *Id.* at 6.

Petitioner presented to the UMPC Passavant ED later that same day and recounted her history of symptoms. Pet'r's Ex. 4 at 772. The provider noted that her bilateral arm weakness had worsened and that she had been sent there under the instruction of Dr. Lam for presumed GBS. *Id.* She denied any recent illness but acknowledged she had a URI in December 2017. *Id.* She was noted to have ascending weakness and started on a four-day course of IVIG. *Id.* at 761. On her final day of IVIG, a neurological consult from Benjamin Shtrahman noted that Petitioner had been undergoing physical therapy ("PT") and that her increased weakness had made her more uncomfortable while resting due to her inability to turn. *Id.* at 896. Following completion of her IVIG, Petitioner was transferred to the UMPC in-patient rehabilitation unit on February 21, 2018. *Id.* at 767.

Upon admission to the inpatient rehabilitation unit, Petitioner reported no significant change in her symptoms. Pet'r's Ex. 4 at 397. She further reported that the neuropathic pain in her hands and feet prevented her from sleeping. *Id.* Her examination noted a decreased range of motion in her shoulder due to arm weakness and reduced abduction and strength in both arms. *Id.* at 399. Petitioner began PT on February 22, 2018, and her initial examination noted significant weakness, impaired sensation in all extremities, poor balance, and decreased activity tolerance. *Id.* at 3964. She required maximum assistance with sit to stand and bed transfers, and required assistance with her activities of daily living. *Id.* An occupational therapy ("OT") intake conducted the same day also noted Petitioner had difficulty swallowing and lingual numbness. *Id.* at 4274.

A neurology follow-up with Dr. Ojha Ajitesh on February 26, 2018, noted Petitioner's weakness had improved, but "the most disabling symptoms [were] her circumferential numbness/back pain that [was] very bothersome to her." Pet'r's Ex. 4 at 579. Dr. Ajitesh also noted that Petitioner had asymmetric facial involvement and opined her GBS was improving. *Id.* Two days later, Petitioner reported that her paresthesias in her hands and legs was worse when lying down, though she had no new symptoms. *Id.* at 580. Magnetic resonance imaging ("MRI") of her brain was negative. *Id.* Due to frustration with her pain and physical limitations, Petitioner had a

psychiatry consult on March 2, 2018, for anxiety and depression. *Id.* at 402. The psychiatrist recommended amitriptyline and a sleep medication. *Id.* at 409.

On March 19, 2018, Petitioner had a neurology follow-up with Dr. Shtrahman. Pet'r's Ex. 4 at 581. Petitioner complained of "persistent anterior upper abdominal discomfort that radiate[d] in a band like distribution to the thoracic spine," with similar discomfort in both hands. *Id.* Dr. Shtrahman noted that her strength had improved, but still had numbness and parethesias of both the hands and feet. *Id.* He further acknowledged in his patient notes that Petitioner's GBS had been "preceded by pneumonia vaccination by about a week." *Id.* Dr. Shtrahman also noted that Petitioner's symptoms continuing at this level was "somewhat atypical" four-to-five weeks post onset and wrote that he "[n]eed[ed] to consider alternative diagnoses such as vasculitis." *Id.* Brain and spinal MRIs on March 21, 2018, ruled out transverse myelitis. *Id.* at 684.

Petitioner was discharged from PT and OT on March 23, 2018, with partial goals met. Pet'r's Ex. 4 at 2227, 3247. At the time of her discharge she was using a walker, but hoped to graduate to a cane by her return home. *Id.* at 3247. She was also able to complete her usual daily activities independently. *Id.* at 2228. Petitioner was discharged from UMPC on March 24, 2018, with diagnoses of GBS, anxiety, acute adjustment disorder, and depressed mood. *Id.* at 413.

On April 2, 2018, Petitioner presented to Dr. Rosalinda Raymundo for a follow-up of her prior hospitalization. Pet'r's Ex. 6 at 2. Dr. Raymundo noted in Petitioner's history that her symptoms began "about [five] days after her Prevnar 13" vaccine. *Id.* At the time of the visit Petitioner was able to ambulate using a walker and had complaints of some muscle pain and spasms. *Id.* On examination Dr. Raymundo noted that Petitioner had no focal weakness, but did have diffuse weakness in both arms and legs. *Id.* at 6. Dr. Raymundo further noted that Petitioner would continue her medications and PT and follow-up with a neurologist. *Id.*

Petitioner returned to Dr. Lam on April 18, 2018, with complaints of weakness in both arms and legs, though the right was worse than the left. Pet'r's Ex. 5 at 10. She also reported increased numbness on the left side of her face, jaw clenching, tinnitus, anxiety, and ongoing tongue numbness. *Id.* An electromyography ("EMG") study completed on March 30, 2018, showed evidence of "a demyelinating sensorimotor peripheral neuropathy which would be consistent with [GBS]." *Id.* On examination Dr. Lam noted Petitioner exhibited weakness of the left deltoid, triceps, and finger extensor, but had normal strength of her right arm. *Id.* at 12. She also had minor weakness in her left lower leg with absent deep tendon reflexes throughout. *Id.* Dr. Lam acknowledged that Petitioner had made "slow progress" with regards to her improvement but noted that "[t]here [was] no indication for [chronic inflammatory demyelinating polyneuropathy ("CIDP")] or recurrent [GBS]." *Id.* Dr. Lam advised Petitioner to continue her PT and OT and return if she developed any new symptoms. *Id.*

On May 17, 2018, Petitioner presented to Dr. Lam for another follow-up. Pet'r's Ex. 5 at 17. She reported continued pain and muscle cramping in both arms and legs, with her symptoms worse at night. *Id.* She also reported continued weakness in her left arm and gait instability that required her to continue to use a walker. *Id.* She had stopped at-home PT and was planning to start outpatient PT soon. *Id.* Dr. Lam increased her gabapentin dosage to help with her pain and agreed that she should continue to remain away from work until her symptoms improved. *Id.* at 19.

Petitioner presented to physical medicine and rehabilitation specialist Dr. Shaun Darrah on June 7, 2018. Pet'r's Ex. 7 at 1. She had primary complaints of paresthesias and neuropathic pain, and Dr. Darrah recommended she increase her medication dosage and start outpatient PT. *Id.* at 4. Petitioner began outpatient PT the next day on June 8, 2018. Pet'r's Ex. 8 at 2. Her provider noted that she was using a walker and that Petitioner reported difficulty with balance, transfers, standing, and walking. *Id.* Petitioner also reported for OT and speech therapy later that month, and continued PT until August 2, 2018. *Id.* at 18, 51, 276. She was discharged from OT on September 6, 2018, and she completed speech therapy on September 10, 2018. Pet'r's Ex. 12 at 14, 31.

On September 5, 2018, Petitioner returned to Dr. Lam for her three-month follow-up. Pet'r's Ex. 11 at 36. Dr. Lam noted that Petitioner "ha[d] been making slow and steady [progress] in terms of neurologic symptoms since her last visit." *Id.* He noted that "[d]espite improvement, she [was] still frustrated with her situation as she continue[d] to have gait instability and difficulties with mobility as well as coordination." *Id.* She also continued to complain of weakness in her left arm and numbness on the left side of her face. *Id.* She reported no new neurologic symptoms, but she did report the development of oral and tongue ulcers. *Id.* On examination Dr. Lam noted that her left arm weakness had improved and that she continued to have mild weakness in her left lower leg. *Id.* at 37. He also observed that Petitioner's deep tendon reflexes were now present at 1+ in the upper extremities, but still absent in the lower extremities. *Id.* Overall, Dr. Lam opined that Petitioner's improvement in symptoms was consistent with severe GBS and that she was slowly improving. *Id.*

Petitioner returned for a routine follow-up with Dr. Lam on March 5, 2019. Pet'r's Ex. 11 at 24. Dr. Lam noted that Petitioner's symptoms continued to improve and that she had begun to participate in physical activity on her own. *Id.* She no longer used a walker or cane but still had some mild balance trouble and continued to experience left sided weakness and numbness. *Id.* Dr. Lam opined that Petitioner's condition was improving as expected and advised her to follow-up in six months. *Id.* at 25.

On April 3, 2019, Petitioner presented to Dr. Raymundo with complaints of a "weird feeling in her head," a feeling of tremors, and a pinching sensation in her chest. Pet'r's Ex. 6 at 15, 21. Petitioner's examination was unremarkable and she was diagnosed with stable COPD, impaired fasting glucose, and GBS. *Id.* at 21.

Petitioner followed up with Dr. Lam on September 5, 2019, and noted that her symptoms had remained largely the same since her visit in March. Pet'r's Ex. 11 at 15. She reported that her medication was working at its current level and that she intended to travel to Europe with her daughter in the near future. *Id.* Dr. Lam noted that although her pain and sleep were problematic, they were controlled by her medication and advised her to return in six months. *Id.* at 16.

No other relevant medical records were filed in this case.

III. Petitioner's Affidavit

On December 30, 2019, Petitioner filed a brief affidavit. Pet'r's Aff.[4] Her affidavit did not contain any specific factual assertions, but stated that "the facts and/or averments contained in the foregoing Petition are true and correct to the best of my knowledge, information, and belief." *Id.*

IV. Experts

A. Expert Qualifications

1. Petitioner's Expert, Dr. James DeAngelo, D.O.

Dr. DeAngelo is an allergist and immunologist and is the managing partner, owner, and director of clinical research at Allergy and Clinical Immunology Associates of Pittsburgh. Pet'r's Ex. 16 at 1. He also serves as a Clinical Assistant Professor for the University of Pittsburgh School of Medicine. *Id.* Dr. DeAngelo received his doctorate of osteopathy ("D.O.") from the Philadelphia College of Osteopathic Medicine in 1991. Pet'r's Ex. 65 at 1. He then completed an internship in Family medicine at Millcreek Community Hospital, a residency in internal medicine at West Virginia University, and a fellowship in Allergy and Immunology at the Cleveland Clinic Foundation. *Id.* He is board certified in internal medicine and allergy and immunology, and has served as the principle investigator on "dozens of clinical trials." Pet'r's Ex. 16 at 1. His "expertise is managing allergic and immunologic adverse reactions to allergens, toxins, medications, and immunizations." *Id.*

2. Respondent's Expert, Dr. You-Wen He, M.D., Ph.D.

Dr. He is a Professor of Immunology at Duke University Medical Center and has been conducting immunology research since 1986. Resp't's Ex. A at 2. He received his medical degree from The Fourth Military Medical University in Xian, China, in 1986, and his Ph.D. in Microbiology and Immunology from the University of Miami School of Medicine in 1996. Resp't's Ex. B at 2. He completed his residency at Qindu Hospital in Xian, China, and completed an immunology fellowship at the Howard Hughes Medical Institute at the University of Washington. *Id.* Dr. He's research areas "include both innate and adaptive immunity against viral and bacterial infections." Resp't's Ex. A at 2. He has also directed research on human responses to several viral infections and is the co-principal investigator for four clinical trials focusing on cancer immunotherapy. *Id.*

B. Expert Reports

1. Dr. DeAngelo's Initial Report

Dr. DeAngelo began by first describing GBS, and opined that Petitioner's clinical presentation, "including tingling/numbing in her bilateral extremities, ataxic gait, and reduced reflexes together with EMG evidence of demyelinating sensorimotor peripheral neuropathy, support the diagnosis of GBS." Pet'r's Ex. 16 at 8. He argued that Petitioner presented with "a classic case of post-vaccination GBS with her disease onset manifesting approximately eight [] days following receipt of her Prevnar 13 vaccine and her GBS [] progressing to the point of hospitalization [14] days post-vaccination." *Id.*

[4] Petitioner did not assign an exhibit number to her affidavit.

Dr. DeAngelo explained that "in most cases," GBS is caused by an immune stimulus such as influenza, Epstein-Barr Virus ("EBV"), *Haemophilus influenzae*, or *Campylobacter jejuni*, but it has also been "associated and reported with vaccination." Pet'r's Ex. 16 at 8 (citing Pet'r's Ex. 19;[5] Pet'r's Ex. 26;[6] Pet'r's Ex. 28;[7] Pet'r's Ex. 34;[8] Pet'r's Ex. 36;[9] Pet'r's Ex. 39).[10] He opined that "[o]ne proposed mechanism by which this immune stimulus from an infectious agent causes GBS is molecular mimicry." *Id.* (citing Pet'r's Ex. 42). "[M]olecular mimicry is defined as the dual recognition, by a single B- or T-cell receptor, of a microbe's structure and an antigen of the host," and is the mechanism by which infections can trigger autoimmune diseases via cross-reactive antibodies or T-cells. *Id.* (citing Pet'r's Ex. 42).[11]

With regard to the Prevnar 13 vaccine, Dr. DeAngelo opined that "[t]he relationship of GBS to the Prevnar 13 vaccine is essentially one of an autoimmune reaction to the vaccine utilizing molecular mimicry." Pet'r's Ex. 16 at 10. He analogized *Streptococcus pneumoniae* to *Haemophilus influenzae*, as both are "an encapsulated pathogenic bacterial organism," and cited to two case reports identifying cases of GBS following *Pneumococcus pneumoniae* infection. *Id.* (citing Pet'r's Ex. 20;[12] Pet'r's Ex. 27).[13] Dr. DeAngelo specifically pointed to Khatib et al., which wrote

> It is possible therefore that pneumococcus has antigens which triggered an immune response and cross-reacted with peripheral nervous system surface components due to molecular mimicry. This is due to natural transformation of *Streptococcus pneumoniae*'s capsular polysaccharide. This highlights the role of host immunity in determining clinical manifestations of disease; it is not solely due to bacterial virulence.

Id. (citing Pet'r's Ex. 27 at 2). Yu et al. also identified that the four most common infectious triggers of GBS (*Campylobacter jejuni*, cytomegalovirus, Epstein-Barr virus, and *Mycoplasma pneumoniae*) possess "carbohydrate sequences (antigens) in common with peripheral nerve

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[5] C. W. Ang et al., *Cross-Reactive Anti-Galactocerebroside Antibodies and Mycoplasma Pneumoniae Infections in Guillain-Barré Syndrome*, 130 J. NEUROIMMUNOLOGY 179 (2002).
[6] Y. Y. Ju et al., *Haemophilus Influenzae as a Possible Cause of Guillain-Barré Syndrome*, 149 J. NEUROIMMUNOLOGY 160 (2004).
[7] *Guillain-Barre Syndrome*, THE MAYO CLINIC (last accessed Jan. 26, 2026) https://www.mayoclinic.org/diseases-conditions/guillain-barre-syndrome/symptoms-causes/syc-20362793.
[8] J. Stowe et al., *Investigation of the Temporal Association of Guillain-Barre Syndrome With Influenza Vaccine and Influenza-Like Illnesses Using the United Kingdom General Practice Research Database*, 169 AM. J. EPIDEMIOLOGY 382 (2009).
[9] C. C. Tam et al., *Guillain-Barré Syndrome and Preceding Infection With Campylobacter, Influenza and Epstein-Barr Virus in the General Practice Research Database*, 2 PLOS ONE 344 (2007).
[10] B. Van den Berg et al., *Guillain-Barré Syndrome: Pathogenesis, Diagnosis, Treatment and Prognosis*, 10 NATURE REV. NEUROLOGY 469 (2014).
[11] R. K. Yu et al., *Ganglioside Molecular Mimicry and Its Pathological Roles in Guillain-Barré Syndrome and Related Diseases*, 74 INFECTION & IMMUNITY 6517 (2006).
[12] G. Bianchi & G. Domenighetti, *Pneumococcus Pneumoniae Infection and Guillain-Barré Syndrome: Fortuitous or Specific Association?*, 32 INTENSIVE CARE MED. 338 (2006).
[13] H. E. Khatib et al., *Case Report: Guillain-Barré Syndrome With Pneumococcus – A New Association in Pediatrics*, 11 IDCASES 36 (2017).

tissue." Pet'r's Ex. 42 at 1. Dr. DeAngelo then cited to Sadarangani[14] and stated that "a polysaccharide capsule surrounds such organisms. This capsule serves as both a virulence factor and the key to the heightened protection of protein-polysaccharide conjugate vaccines, such as the Prevnar 13." Pet'r's Ex. 16 at 10–11 (citing Pet'r's Ex. 32). He stated "this resemblance results in molecular mimicry" as described by Susuki et al.,[15] Yu et al., and Yuki et al.[16] *Id.* at 11 (citing Pet'r's Ex. 35; Pet'r's Ex. 42; Pet'r's Ex. 43).

Dr. DeAngelo then argued that because of the "strong support in the scientific literature for GBS's infectious origins via molecular mimicry, it follows that vaccination with antigens specific to these same disease-causing organism[s] can trigger . . . GBS in a small subset of patients after routine immunization." Pet'r's Ex. 16 at 11 (citing Pet'r's Ex. 36; Pet'r's Ex. 39). Dr. DeAngelo argued that studies showing a connection between the 1976 and 2009 H1N1 vaccine and GBS support his argument that a similar connection could be made between Prevnar 13 and GBS. *Id.* He further cited to three case reports identifying cases of GBS possibly related to Prevnar 13 vaccination. *Id.* (citing Pet'r's Ex. 22;[17] Pet'r's Ex. 31;[18] Pet'r's Ex. 38).[19]

According to Dr. DeAngelo, a connection between Prevnar 13 and GBS could be made due to protein-conjugated nature of the Prevnar 13 vaccine. Pet'r's Ex. 16 at 11. "Adding a carrier protein to the polysaccharide antigen [of Prevnar 13] activates the B-Cell, which internalizes the newly created neoantigen by the B-cell processing." *Id.* (citing Pet'r's Ex. 37).[20] "Such processing permits the presentation of the antigenic molecules to T-cells in the context of MHC II, with resultant full activation of T-helper cells and a more complete, vigorous, and long-lasting immune response than the T-independent response associated with the pure polysaccharide antigen." *Id.*

Dr. DeAngelo next turned to Heidenreich et al.[21] to explain the role of T-cells in "acute" GBS. Pet'r's Ex. 16 at 11 (citing Pet'r's Ex. 25). The article argued that T-cells controlled ganglioside GM1-specific B cells, and that cytokines produced by the mitogen activated T-cells "may stimulate GM1-specific B cells as a bystander effect." Pet'r's Ex. 25 at 10. The article also acknowledged that "[t]he antigenic specificity of the T cells that could interact with GM1-specific B cells in GBS . . . is still unknown." *Id.* However, the authors speculated that "an immune response to glycoproteins carrying glycoconjugates as a hapten could induce a B cell response to the GM1 ganglioside-dependent on a T cell-response directed to the carrier protein." *Id.* "Alternatively,

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[14] Manish Sadarangani, *Protection Against Invasive Infections in Children Caused by Encapsulated Bacteria*, 9 FRONTIERS IN IMMUNOLOGY 2674 (2018).
[15] K. Susuki, *Acute Motor Axonal Neuropathy After Mycoplasma Infection: Evidence of Molecular Mimicry*, 62 NEUROLOGY 949 (2004).
[16] N. Yuki et al., *A Bacterium Lipopolysaccharide that Elicits Guillain-Barré Syndrome Has a GM1 Ganglioside-like Structure*, 178 J. EXPERIMENTAL MED. 1771 (1993).
[17] Chad Conner et al., *13-Valent Pneumococcal Conjugate Vaccine-Induced Guillain-Barré Syndrome*, 158 CHEST 59 (2020).
[18] Nidhi Ravishankar, *Guillain-Barre Syndrome Following PCV Vaccine*, 2 CLINICS IN SURGERY 1413 (2017).
[19] Hung Fu Tseng et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults*, 5 OPEN FORUM INFECTIOUS DISEASES 1 (2018). This is also cited as Resp't's Ex. P.
[20] A. Torres et al., *Pneumococcal Vaccination: What Have We Learnt So Far and What Can We Expect in the Future?*, 34 J. CLINICAL MICROBIOLOGY & INFECTIOUS DISEASES 19 (2014).
[21] Fedor Heidenreich et al., *T Cell-Dependent Activity of Ganglioside GM1-Specific B Cells in Guillain-Barré Syndrome and Multifocal Motor Neuropathy In Vitro*, 49 J. NEUROLOGY 97 (1994).

activation of GM1-specific B cells by bystander cytokines could apply to a T cell response with specificity for antigens like myelin proteins that are not known to share glycoconjugates with the ganglioside GM1 but are recognized by T cells from some GBS patients." *Id.* Similarly, Ravishankar argued that due to the T-cell dependent response of the conjugated *Streptococcus pneumonia* vaccines, "it can be inferred that the conjugated vaccine produces the enhanced B-cell immune response leading to autoimmune reaction to the peripheral nerves." Pet'r's Ex. 31 at 2.

Dr. DeAngelo argued that somatically mutated diverse V genes "encode the production of high-affinity anti-GM1 IgM antibodies," which could "suggest that GBS patients possess or develop a somatic mutation that predisposes susceptible individuals to the production of such high-affinity anti-GM1 IgM antibodies." Pet'r's Ex. 16 at 13 (citing Pet'r's Ex. 17;[22] Pet'r's Ex. 30).[23] He theorized that "individuals with such somatic mutations have a population of preexistent GM1-specific B cells that . . . initiate via molecular mimicry the high-affinity anti-GM1 IgM production, and later IgG autoantibodies, characteristic of GBS." *Id.*

To explain how the Prevnar 13 vaccine has sufficient homology to induce molecular mimicry in Petitioner's case, Dr. DeAngelo asserted that such homology could be "created through intentional haptenization of vaccine antigens with conjugates in vitro or unintentionally with host proteins in vivo following vaccination." Pet'r's Ex. 16 at 13. In the case of Petitioner, Dr. DeAngelo argued that

> [T]he conjugation of a synthesized protein carrier molecule to the polysaccharide component of one or more of the 13 serotypes contained in the Prevnar-13® vaccine haptenated with her own host proteins, lipids, and carbohydrates. This process generated a neoantigen of sufficient homology to a GM1 epitope to transform her preexistent GM1-specific B cells from their nascent low-affinity anti-GM1 IgM antibody production to a new high-affinity anti-GM1 IgM and G antibody production capable of T-dependent isotype switching and the evolution of her GBS.

Id.

With respect to the logical sequence of cause and effect, Dr. DeAngelo opined that "the previously described process of molecular mimicry brought about by polysaccharide-protein conjugation and hapten formation is what led to [Petitioner's] post-vaccination GBS." Pet'r's Ex. 16 at 13. He noted that Petitioner's symptoms began approximately eight days after vaccination and reached nadir approximately 14 days after vaccination. *Id.* "It is well-recognized and accepted in the medical literature that a [one-to-two-week] post-vaccine interval would be an appropriate amount of time to permit the . . . development of GBS." *Id.* at 13–14. He further noted that while such immunogenicity was uncommon with vaccines, "it does occur and that genetic factors play a

[22] Maria E. Alaniz et al., *Normally Occurring Human Anti-GM Immunoglobulin M Antibodies and the Immune Response to Bacteria*, INFECTION & IMMUNITY 2148 (2004).

[23] Gary Paterson et al., *Analysis of Anti-GM1 Ganglioside IgM Antibodies Cloned From Motor Neuropathy Patients Demonstrates Diverse V Region Gene Usage With Extensive Somatic Mutation*, 155 J. IMMUNOLOGY 3049 (1995).

prominent role in this process." *Id.* at 14 (citing Pet'r's Ex. 21).[24] Dr. DeAngelo also argued that there "is no other logical inciting cause for her GBS symptomology" and no "evidence of recent illness or infection in the weeks preceding GBS onset." *Id.*

In addressing the timing of Petitioner's symptom onset, Dr. DeAngelo noted that Petitioner began to experience numbness in her fingertips and pain between her shoulders approximately eight days after vaccination, with hospitalization 14 days after vaccination. Pet'r's Ex. 16 at 14. He argued this was consistent with Fokke et al. and Willison et al., which note GBS symptoms begin approximately one to two weeks after immune stimulation and peak in approximately two to four weeks. *Id.* (citing Pet'r's Ex. 24;[25] Pet'r's Ex. 41).[26] He similarly argued that his prior case report citations supported a one-to-two week interval between vaccination and onset. *Id.* (citing Pet'r's Ex. 22; Pet'r's Ex. 31; Pet'r's Ex. 38).

Dr. DeAngelo also addressed Petitioner's reported URI, which he noted was "approximately two [] months prior to the onset of her GBS symptoms." Pet'r's Ex. 16 at 15. "The chronology of this illness is inconsistent with this illness being the cause of her GBS." *Id.* Dr. DeAngelo also noted that Petitioner's hospital workup "did not note any identifiable infectious agent." *Id.*

2. Dr. He's Initial Report

Dr. He began his report by providing an overview of GBS, and noting that "two-thirds of GBS patients report symptoms of a respiratory or gastrointestinal tract infection before onset of GBS." Resp't's Ex. A at 3 (citing Resp't's Ex. I at 1).[27] In the case of *Campylobacter jejuni*, Dr. He noted that 70% of patients reported having a diarrheal illness within 12 weeks of GBS onset, and that 47% of GBS patients had a respiratory infection within 13 of GBS onset. *Id.* (citing Resp't's Ex. I at 4). Dr. He acknowledged that although the pathological mechanism for GBS is not firmly established, "cross-reactive autoantibodies may play some roles in its immunopathogenesis. The cross-reactive antibodies may attack peripheral nerves and roots and cause demyelination and axonal damage." *Id.*

Dr. He continued by asserting that there was no evidence to associate Prevnar 13 with GBS, stating that no systemic study "even listed [Prevnar 13] as a potential cause for GBS development due to a complete lack of evidence." Resp't's Ex. A at 4. Via a PubMed search, Dr. He identified two peer-reviewed reports assessing adverse events following Prevnar 13 vaccination. *Id.* The first, Haber et al.,[28] examined adverse events catalogued in the Vaccine Adverse Event Reporting System ("VAERS") following Prevnar 13 in adults older than 19 and found 11 potential cases of GBS following Prevnar 13 vaccination. Resp't's Ex. O at 4–5. However, Dr. He argued Haber et

[24] Peter J. Bugelski, *Genetic Aspects of Immune-Mediated Adverse Drug Effects*, 4 NATURE REVIEWS 59 (2005).
[25] Christiaan Fokke et al., *Diagnosis of Guillain-Barré Syndrome and Validation of Brighton Criteria*, 137 BRAIN 33 (2014).
[26] Hugh J. Wilson et al., *Guillain-Barré Syndrome,* 388 LANCET 717 (2016).
[27] J. B. Winer et al., *A Prospective Study of Acute Idiopathic Neuropathy*, 51 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 613 (1988).
[28] Penina Haber et al., *Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PVC13) in Adults Aged >19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012 – December 31, 2025*, 34 VACCINE 6330 (2016).

al. could not be used as "reliable epidemiologic [or] medical evidence" because VAERS allows for reports to "be filed when the diagnosis is 'presumptive' rather than confirmed, and reports may be filed prior to the discovery of alternative causes." Resp't's Ex. A at 4. The second article, Tseng et al., analyzed adverse events in elderly adults receiving the Prevnar 13 vaccine and the 23-valent pneumococcal polysaccharide vaccine ("PPSV23"). Pet'r's Ex. 38 at 1. The authors concluded that "the data do not support an increased rate of adverse events following [Prevnar 13] administration in elders compared to PPSV23 and should provide reassurance regarding continued use of [Prevnar 13]. *Id.* Accordingly, Dr. He opined that "there is a complete lack of evidence to associate [Prevnar 13] vaccination to GBS development." Resp't's Ex. A at 4.

Turning to Dr. DeAngelo's report, Dr. He first agreed that GBS was a post-infectious, disease, but asserted that "[c]iting infection-related GBS cases to suggest a potential link between GBS and [Prevnar 13] vaccination is farfetched." Resp't's Ex. A at 5. He argued that molecular mimicry is an "old theory" that has been "strongly challenged by recent scientific evidence from large sequencing of proteomes of microbial pathogens," which showed "massive epitope sharing" between viruses and humans. *Id.* Kanduc et al.[29] found that of 30 viral proteomes examined, 90% of the pentamer peptides were "widely, intensively[,] and repeatedly scattered throughout the human proteome." Resp't's Ex. Q at 3. Dr. He argued that the existence of these sequence similarities "would suggest a 100% autoimmune disease rate in the general population after either infection or vaccination according to the molecular mimicry theory, which is obviously not the case." Resp't's Ex. A at 6. Citing to another report by Kanduc,[30] Dr. He asserted that "following three decades of intensive research in the field, a causative link between molecular mimicry and human autoimmune disease is still sub judice, and the molecular mimicry hypothesis has received no validation." *Id.* (citing Resp't's Ex. T at 3). Dr. He argued that the cross-reactivity of T-cells and antibodies between self-proteins and infectious agents is "widely detectable," and cited to the Institute of Medicine ("IOM") Committee's statement of evidence needed to show the existence of molecular mimicry:

> (1) a susceptible host whose genetic background and adaptive immune responses allows emergence of self-reactive immunity, (2) exposure to an exogenous agent which expresses antigens that are immunologically similar to self-antigen(s), and (3) a host immune response to the exogenous agent that cross-reacts with biologically relevant host tissue structures and causes tissue damage and clinical disease.

Resp't's Ex. K at 13.[31] The IOM Committee also noted that "linear amino acid sequence homology . . . [is] not sufficient to prove that molecular mimicry is the pathogenic mechanism for a disease." *Id.* Dr. He thus concluded that "there is no evidence to satisfy the above-stated three essential criteria" in Petitioner's case, and argued that "[i]f Dr. DeAngelo's theory were accepted as valid . . . then presumably all vaccines can cause autoimmune conditions in all vaccinated people." Resp't's Ex. A at 6.

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[29] Darja Kanduc et al., *Massive Peptide Sharing Between Viral and Human Proteomes*, 29 PEPTIDES 1755 (2008).
[30] Darja Kanduc, *Peptide Cross-Reactivity: The Original Sin of Vaccines*, 4 FRONT BIOSCIENCE 1393 (2012).
[31] Inst. of Med., *Adverse Events Associated with Childhood Vaccines: Evidence Causality* (Kathleen Stratton et al. eds., 2012).

With regard to Dr. DeAngelo's reliance on studies showing a post-infectious link between GBS and other diseases, Dr. He argued that those "pathogens have likely created a host environment [where] multiple immune tolerance mechanisms are broken." Resp't's Ex. A at 7. "However, there is no evidence that [Prevnar 13] vaccination can break down these immune tolerance mechanisms to cause autoimmune disease." *Id.*

Turning to the specifics of Dr. DeAngelo's theory, Dr. He stated "it is unclear what specific antigenic polysaccharide carbohydrate sequences [Dr. DeAngelo] referred to and what similarly exists between those on [Prevnar 13] and self-tissues." Resp't's Ex. A at 8. Regardless, Dr. He again quoted Kanduc's report and argued that "the distinction between infection- and vaccination-induced immune responses should be emphasized as they are not the same. Citing infection-related GBS cases to suggest a potential link between [Prevnar 13] vaccination and GBS is unfounded." *Id.* at 8–9.

Dr. He also asserted Dr. DeAngelo was "incorrect" to claim that there was strong support in the medical literature for GBS's infectious origin via molecular mimicry. Resp't's Ex. A at 9. "There is strong evidence to link GBS development to various types of infections, however, there is no in vivo data to support that infections cause GBS via molecular mimicry." *Id.* Accordingly, Dr. He challenged Dr. DeAngelo's overall theory that GBS could be caused by vaccine-mediated molecular mimicry as "flawed as the premise for infection-molecular mimicry-GBS does not exist in the first place." *Id.* Dr. He also reiterated the difference in immune responses of a wild-type infection compared to a vaccination, where "the immune stimulation capacity is very limited." *Id.*

Dr. He specifically challenged the credibility of the literature relied on by Dr. DeAngelo, starting with Tam et al. He noted that although the authors found positive associations between GBS and various infections, they also found "evidence of a protective effect of influenza vaccination." Resp't's Ex. A at 9 (citing Pet'r's Ex. 36). He also challenged the Van den Berg et al. and van Doorn[32] articles, which he characterized as "review[s], not [] original report[s] and did not have any cited references for [Prevnar 13] vaccination and GBS." *Id.* (citing Pet'r's Ex. 39; Pet'r's Ex. 40). He contested the Mayo Clinic article as being "a webpage for patient information" and failing to provide supporting evidence. *Id.* (citing Pet'r's Ex. 28). In Tseng et al., Dr. He highlighted the authors' conclusion that Prevnar 13 did not have an increased rate of adverse effects when compared to PPSV23. *Id.* at 10 (citing Pet'r's Ex. 38). He also criticized Ravishankar's case report, as it was not indexed in PubMed and "provided a temporal relation between [Prevnar 13] and GBS development at [nine] months apart." *Id.* (citing Pet'r's Ex. 31). He also challenged Conner et al., as it "is a meeting post without peer review." *Id.* (citing Pet'r's Ex. 22). "Taken together, it is clear none of the cited reference[s] provided solid evidence to support Dr. DeAngelo's claim on a potential link between [Prevnar 13] vaccination and GBS." *Id.*

Dr. He next stated his agreement with Dr. DeAngelo's description of the development process of Prevnar 13 and the role of carrier proteins in inducing T-cell dependent responses. Resp't's Ex. A at 11. However, he stated that there was no evidence to support the "conjugation of a synthesized protein carrier molecule [which] generated a neoantigen of sufficient homology to a GM1 epitope to transform [Petitioner's] preexistent GM1-specific B cells from low-affinity to high-affinity antibody." *Id.* at 11–12. He also reiterated that there was no evidence that Prevnar 13 could cause GBS. *Id.*

[32] Pieter A. van Doorn, *Diagnosis, Treatment and Prognosis of Guillain-Barré Syndrome (GBS)*, 42 PRESSE MED. 193 (2013).

Dr. He did not address the validity of the eight-day window between vaccination and symptom onset, but instead pointed to Petitioner's prior URI four-to-eight weeks before onset and argued this was the more likely cause of her GBS. *Id.* at 13. He again referenced the 12-week timeline for 70% of *Campylobacter jejuni* infections and the 13-week timeline experienced by 47% of patients suffering from a respiratory infection prior to GBS as supportive of his argument. *Id.* Accordingly, "[g]iven the complete lack of evidence" connecting Prevnar 13 to GBS and the "extensive clinical evidence associating [URIs] with GBS," Dr. He concluded Petitioner's prior URI was the more likely cause of her GBS. *Id.*

3. Dr. DeAngelo's First Supplemental Report

Dr. DeAngelo acknowledged that while the immune response to an infection and vaccine were different, the latter was "still sufficient to induce protection to the pneumococcal pathogen and therefore also has the potential to produce autoreactivity in the susceptible host." Pet'r's Ex. 44 at 1. He also disputed Dr. He's criticisms of molecular mimicry as a scientific theory, explaining that "it is widely scientifically accepted that the development of autoimmunity following infection or vaccination is dependent on unique genetically and environmentally determined susceptibilities." *Id.* He also cited to Segal & Shoenfeld[33] as another example of that implicates molecular mimicry in the development of GBS via vaccines, and asserted that Dr. He's argument regarding the lack of supporting literature was "overstated." *Id.* at 2 (citing Pet'r's Ex. 54).

Further, Dr. DeAngelo cited to Cutillo et al.[34] to "highlight the possibility that antiganglioside antibodies ("AGAs") could have a pathophysiological role." Pet'r's Ex. 44 at 2 (citing Pet'r's Ex.47). Specifically, in linking AGAs to the development of GBS, the authors noted that "direct axonal damage is mediated by the attack of AGAs against gangliosides, located in the nodes of Ranvier." Pet'r's Ex. 47 at 5. The authors also proposed "a model whereby antiganglioside antibodies arose through a T-cell dependent process following vaccination in genetically susceptible patients" by examining cases of narcolepsy following both infection and vaccination with H1N1. Pet'r's Ex. 44 at 2 (citing Pet'r's Ex. 47). Dr. DeAngelo thus asserted that Cutillo et al. provides "a strong argument that molecular mimicry in a genetically susceptible host is the likely mechanism behind not only vaccine-associated narcolepsy, but also other antiganglioside antibody-mediated neurologic diseases, most notably GBS." *Id.* at 3.

Dr. DeAngelo next cited to Bianchi et al. and Khatib et al. to argue "the association between pneumococcal antigens contained in vaccines and GBS is also supported by cases of GBS following natural infection with encapsulated organisms such as pneumococcus." Pet'r's Ex. 44 at 3 (citing Pet'r's Ex. 20; Pet'r's Ex. 27). Accordingly, Dr. DeAngelo posited that Petitioner's "vaccine components formed hapten to host molecular coagulates that lead to T cell-dependent stimulation of preexistent GM1-specific B cells via molecular mimicry enhanced by epitope spreading, antigen non-specific bystander activation, and polyclonal activation." *Id.* Dr. DeAngelo stated that haptenation "is a well-established mechanism whereby a non-immunogenic drug or

[33] Yahel Segal & Yehuda Shoenfeld, *Vaccine-Induced Autoimmunity: The Role of Molecular Mimicry and Immune Crossreaction*, 15 CELLULAR & MOLECULAR IMMUNOLOGY 586 (2018).
[34] Gianni Cutillo et al., *Physiology of Gangliosides and the Role of Antiganglioside Antibodies in Human Disease*, 17 CELLULAR & MOLECULAR IMMUNOLOGY 313 (2020).

vaccine is rendered immunogenic via molecular mimicry. *Id.* at 5. He described the haptenization process of Prevnar 13 as follows:

> The manufacturing process links the capsular antigens of *Streptococcus pneumoniae* serotypes . . . to the Diphtheria CRM197 Protein to enhance its immunogenicity. . . . Conjugating the polysaccharide capsular antigens to the CRM197 carrier protein to form a hapten-carrier complex . . . dramatically enhances the immunogencity of polysaccharide antigens by enhancing immune recognition of the pathogen's polysaccharide capsule. . . . [T]he CRM197 carrier protein permits the presentation of the antigen to carrier-peptide-specific helper T cells in the context of an MHC class-II dependent response. This T-cell-dependent presentation enhances the B-cell immune response compared to the T-independent processes. . . . Thus, this conjugation is the key to the heightened protection of protein-polysaccharide conjugate vaccines, such as [Prevnar 13]. . . . [H]owever, the introduction of any foreign material, such as the CRM197 protein . . . raises the possibility of host protein haptenation of the CRM197 protein or its conjugated polysaccharide side chains.

Id. at 3–5 (citing Pet'r's Ex 23; Pet'r's Ex. 45). Cutillo et al. suggested that AGAs arise upon hapten complex formation between H1N1 proteins and gangliosides, "T cells specific for the H1N1 proteins provide the impetus for antiganglioside antibody expression by B cells and subsequent differentiation of these B cells to antibody-secreting plasma cells." *Id.* at 5. Dr. DeAngelo then cited to Saariaho et al.[35] to show that "the same process is likely responsible for post-vaccination GBS." *Id.* (citing Pet'r's Ex. 53). Dr. DeAngelo also noted that haptenated proteins can induce different immune responses than native proteins, and thus theorized that "such an enhancement is . . . potentially detrimental in a host with preexistent GM1-specific B cells." *Id.* at 7 (citing Pet'r's Ex. 51). Dr. DeAngelo continued by speculating that T cell dependent stimulation through haptenation may result in "haptenated glycoconjugates of sufficient homology to the GMI ganglioside as to activate preexistent GM1-specific B cells via the process of molecular mimicry." *Id.* He concluded that in the case of Petitioner, this process in her Prevnar 13 vaccine "transformed her preexistent GM1-specific B cells from their nascent low-affinity anti-GM1 IgM antibody production to a new high-affinity anti-GM1 IgM and G antibody production capable of T-dependent isotype switching and evolution of her GBS." *Id.*

Turning to Dr. He's claim that Petitioner's prior URI was the more likely cause of her GBS, Dr. DeAngelo contended that the 13-week timeframe provided by Dr. He was inaccurate, and instead his proposed a one-to-two-week timeline following immune stimulation was proper. Pet'r's Ex. 44 at 7. He also again cited to the case reports of Tseng et al., Connor et al., and Ravishankar to show support for this timeframe, and concluded that the Prevnar 13 vaccine being the cause of her GBS is "very much in accord with the subject literature." *Id.* at 8. He also theorized that Petitioner's prior URI may have been a "first hit" to her immune system, "initiating an otherwise uneventful initial immunological response," with the Prevnar 13 vaccine serving as a "second hit, resulting in a sufficient reactivation" of her T and B cells to cause her GBS. *Id.*

[35] Anna-Helena Saariaho et al., *Autoantibodies Against Ganglioside GM3 Are Associated with Narcolepsy-Cataplexy Developing After Pandemrix Vaccination Against 2009 Pandemic H1N1 Type Influenza Virus*, 63 J. AUTOIMMUNITY 68 (2015).

4. Dr. He's First Supplemental Report

Dr. He first explained the significance of an immune response resulting from vaccination compared to that resulting from an active infection. Resp't's Ex. V at 1. He explained that the host immune system looks for pattern recognition receptors ("PRRs") present on pathogen-associated molecular patterns ("PAMPs"), and the kind of PRRs and PAMPs engaged by the immune system determine the kind of immune response generated. *Id.* at 1–2. "Thus, the more PRRs that are engaged by PAMPs, the stronger the immune activation becomes due to the expression of many more inflammatory cytokines." *Id.* at 2. Dr. He argued this was significant because microbial pathogens contain more PAMPs, allowing for a much broader immune response via PRR stimulation than a vaccine. *Id.* Using *Streptococcus pneumoniae* as an example, Dr. He argued that the wild-type bacterium could stimulate at least eight different PRRs, while the Prevnar 13 vaccine could only stimulate one PRR. *Id.* Dr. He also argued that the immune response between the two was altered because wild type viruses and bacteria have natural infection routes, while a vaccine is administered via intramuscular, intradermal, or subcutaneous injections. *Id.* "During the infection process, a large number of non-immune cells . . . undergo cell death and release [a] large quantity of immune stimuli called danger-associated molecular patterns," which he argued further exacerbated the immune response and led to patient mortality. *Id.* at 3. He referenced his own review of COVID-19 pathogenesis and stated that there were "three stages of immunopathological pathway leading to COVID-19 mortality:" initiation, amplification, and consummation. *Id.* (citing Resp't's Ex. AD).[36] "In stark contrast, vaccines such as [Prevnar 13], with only one type PAMP, are taken up at the injection sites by antigen-presentation cells to prime lymphocytes at regional lymph nodes." *Id.* (citing Resp't's Ex. AE).[37] Dr. He thus asserted that "it is clear that wild type bacterium or virus has a very different process to induce immune responses from its corresponding vaccine," and concluded that this difference "in breadth and depth of immune responses" is why GBS is associated with infections, but not the Prevnar 13 vaccine. *Id.*

Dr. He next addressed Dr. DeAngelo's overall theory of molecular mimicry and argued that infections that cause GBS did so through a breakthrough of the immune system, not through molecular mimicry. Resp't's Ex. V at 3–4. He explained that immune checkpoint receptors act as "counter-balance controls" to the immune response, and that only "rare uncontrolled activations of the immune system can . . . develop autoimmune diseases in susceptible hosts." *Id.* at 4. "Therefore, it is not surprising that some infections are linked to the development of various autoimmune diseases." *Id.* Dr. He also argued that Dr. DeAngelo did not provide evidence to show that (1) a synthesized protein carrier molecule generated a neoantigen of sufficient homology to a GM1 epitope, (2) that Petitioner had preexisting GM1-specific B cells that were low affinity, and (3) that the neoantigen transformed Petitioner's preexisting low affinity anti-GM1 antibody to a high-affinity antibody, causing her GBS. *Id.* at 4–5. Further, Dr. He criticized Cutillo et al., as "there is no evidence presented in this review to support that [Prevnar 13] can induce AGAs," and again criticized Dr. DeAngelo's reliance on case reports to show an association between Prevnar 13 and GBS. *Id.* at 5–6.

[36] Ligong Lu et al., *Preventing Mortality in COVID-19 Patients: Which Cytokine to Target in a Raging Storm?*, 8 FRONTIERS IN CELL AND DEVELOPMENTAL BIOLOGY 677 (2020).
[37] Bali Pulendran & Rafi Ahmed, *Immunological Mechanisms of Vaccination*, 12 NATURE IMMUNOLOGY 509 (2011).

Turning to the specifics of Dr. DeAngelo's theory, Dr. He stated that the number of polysaccharides from different serotypes did not matter, as they still only stimulated one PRR. Resp't's Ex. V at 7. He also critiqued the majority of Dr. DeAngelo's theory as "speculation," including Dr. DeAngelo's theory of the development of a potential neoantigen. *Id.* at 8. He furthered this critique by noting that Cutillo et al. acknowledged the mechanisms by which H1N1 caused narcolepsy "remained elusive," and "[t]hus, Dr. DeAngelo based his speculation on another speculation." *Id.* (citing Pet'r's Ex. 47 at 6). Further, Dr. He noted that there was "no evidence that hapten complex has actually formed between H1N1 proteins and gangliosides." *Id.* He also argued that there was no evidence that a link between *Streptococcus pneumoniae* to the CRM197 carrier protein would be detrimental with or without GM1-specici B cells, or that such a linkage would "form a neoantigen with sufficient homology to the GM1 ganglioside." *Id.* Dr. He further contended that "[n]othing in the record supports that [Petitioner had] preexistent GM1-specific B cells." *Id.* at 9. Dr. He also reiterated his belief that Petitioner's December 2017 URI was the cause of her GBS and that there was no evidence to support a link between Prevnar 13 and GBS. *Id.* at 10.

5. Dr. DeAngelo's Second Supplemental Report

In his second supplemental report Dr. DeAngelo proposed a new theory: that molecular mimicry caused "a homologous interplay between the phosphoglycerol/phosphorylcholine within the Prevnar 13 vaccine and the phospholipid components in the human myelin sheath." Pet'r's Ex. 55 at 2. He cited to Gilburd et al.,[38] which studied "the reactivity of GBS sera with various phospholipids," and found that "[s]ix of the 16 GBS sera had autoantibodies to one or more of the antigens studied." Pet'r's Ex. 57 at 1. However, the study noted that there was "no significant association between the presence of specific antiphospholipid antibodies . . . and GBS when compared to controls, whereas there was a significant increase in the level of all anti-phospholipid antibodies in the sera of [] patients with cerebritis." *Id.* at 4. The report further concluded that the presence of these antibodies was more likely "a result of myelin damage, rather than [the] cause [of] the inflammatory demyelination." *Id.* at 5. Ultimately, the report did "not show a significant increase in any specific antiphospholipid antibody in patients with [GBS]," and attributed the increase found in some patients to "a result of myelin damage or . . . of cross reaction with other anti-myelin antibodies." *Id.* Dr. DeAngelo also referenced Nakos et al.,[39] which found that anti-phospholipid antibodies were found in all nine GBS patients studied, but none of the controls. Pet'r's Ex. 61 at 1. Dr. DeAngelo asserted that these two studies showed "many of the antibodies in GBS target phospholipids." Pet'r's Ex. 55 at 2. He also cited to Ho et al.,[40] which proposed that "autoimmune responses in [multiple sclerosis ("MS")] target the myelin phospholipids" as another example of a demyelinating disease targeting myelin phospholipids. *Id.* (citing Pet'r's Ex. 59).

Connecting this to the Prevnar 13 vaccine, Dr. DeAngelo noted that the polysaccharide of one of the 13 serotypes contained in the vaccine was comprised partially of glycerol-phosphate, which played a role in the antigenicity and immunogenicity of the vaccine. Pet'r's Ex. 55 at 2

[38] B. Gilburd et al., *Autoantbodies to Phospholipids and Brain Extract in Patients with the Guillain-Barre Syndrome: Cross-Reactive or Pathogenic?*, 16 AUTOIMMUNITY 23 (1993).
[39] G. Nakos et al., *Anti-Phospholipid Antibodies in Serum From Patients With Guillain-Barré Syndrome*, 31 INTENSIVE CARE MED. 1401 (2005).
[40] Peggy P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 4 SCI. TRANSLATION MED. 137 (2012).

(citing Pet'r's Ex. 56).[41] Ohori et al.[42] also found that Prevnar 13 contains phosphorylcholine, and that the vaccine caused an immune response against phosphorylcholine. Pet'r's Ex. 62 "Therefore, another plausible mechanism whereby molecular mimicry can trigger GBS following Prevnar 13 vaccination would involve the homologous interplay between the phosphoglycerol/phosphorylcholine within the Prevnar 13 vaccine and the phospholipid components in the human myelin sheath. Pet'r's Ex. 55 at 2. He then cited several studies showing the presence of phospholipid antibodies in MS patients and argued this was part of the pathogenesis of demyelinating disease, including GBS. *Id.* at 3.

Dr. DeAngelo next addressed Dr. He's proposed timeline of Petitioner's infection and critiqued his reliance on Winer et al. as the 13-week period referenced in the article "was the furthest point out in weeks where a patient actually developed GBS." Pet'r's Ex. 55 at 4. He argued that "the [13] week interval is merely the end of the distribution identified by the authors, and they could have just as easily chosen four [] weeks to [13] months and arrived at a similar percentage." *Id.* He then provided a chart from the article to demonstrate the range of GBS onset referenced in the article.

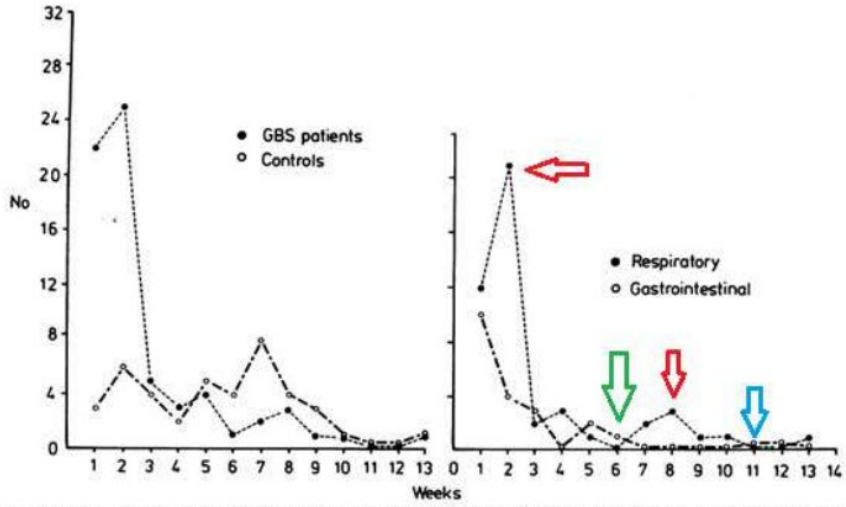

Fig. *The number of patients or controls giving a history of antecedent infections at intervals before the onset of neuropathic symptoms is shown. The panel on the left compares patients with controls while that on the right gives the relative incidence of respiratory and gastrointestinal symptoms within the patient group alone.*

> As can be seen from the figure in question, GBS onset peaks at week two [] (red arrow), declines to near zero by week six [] (green arrow), shows another much smaller peak in week eight [] (2nd red arrow), and remains near zero with only a tiny blip of an increase at week [13].

Id. at 4 (citing Resp't's Ex. I at 4). He explained that the Prevnar 13 vaccine, which was administered approximately eight days before the beginning of her GBS symptoms, would be more in line with the data provided in Winer et al., as her December 2017 URI would have been "outside

[41] Janoi Chang et al., *Relevance of O-Acetyl and Phosphoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysaccharide*, 30 VACCINE 7090 (2012).
[42] Junichiro Ohori et al., *Phosphorylcholine Intranasal Immunization With a 13-Valent Pneumococcal Conjugate Vaccine Can Boost Immune Response Against Streptococcus Pneumoniae*, 38 VACCINE 699 (2020).

the expected post-infectious disease course." *Id.* He also reiterated that Petitioner's hospital workups did not identify "any identifiable infectious or noninfectious etiology" to Petitioner's symptoms, while noting that her neurology notes identified that she had previously received the Prevnar 13 vaccine. *Id.* at 5.

6. Dr. He's Second Supplemental Report

In Dr. He's second supplemental report, he first noted that Gilburd et al. did not support Dr. DeAngelo's proposition, and in fact noted that the antibodies were instead produced "as a result of the myelin damage rather than cause the demyelination." Resp't's Ex. AI at 2. Regarding Dr. DeAngelo's use of MS to analogize a similar demyelination process in GBS, Dr. He first stated that "it is unclear whether GBS patients have similar types of autoantibodies targeting a phosphate group," and further noted that there was "no evidence that these autoantibodies are the initiators of MS, let alone GBS." *Id.* He further noted that there was no evidence that Prevnar 13 "vaccination can induce autoantibodies targeting the phosphate group in phosphatidylserine and oxidized phosphatidylcholine." *Id.*

Dr. He also criticized Dr. DeAngelo's new theory as it "assume[s] that the antibodies against the glycerol-phosphate group . . . or antibodies against phosphorylcholine . . . would cross-react with the phospholipid components in the human myelin sheath. There is no evidence for this assumption." Resp't's Ex. AI at 3. He cited to the IOM to show that "naturally occurring and postinfectious cross-reactive antibodies and T cells are relatively common and most frequently not pathogenic," and can also be secondary to a nonspecific tissue injury. *Id.* at 4. (citing Resp't's Ex. K at 4). Dr. He further critiqued Dr. DeAngelo's use of Grievink et al.,[43] as the study attempted to induce IgM antibodies against oxidized low density lipoprotein through Prevnar 13 vaccination for atherosclerosis prevention. *Id.* (citing Pet'r's Ex. 58 at 6). However, the study found "no increase in protective anti-phosphorylcholine . . . antibodies" and concluded that "this work shows that vaccination against pneumococcal does not seem to be a suitable treatment option to help prevent atherosclerosis development." *Id.* (citing Pet'r's Ex. 58 at 6).

Dr. He also contested Dr. DeAngelo's assertion that CRM197 haptenation could result in molecular mimicry-induced autoimmunity as "completely unfounded." Resp't's Ex. AI at 5. He cited to Vingtdeux et al.[44] to show that CRM197 haptenization with AβpE3 produced specific antibodies that did not cross-react. *Id.* (citing Pet'r's Ex. 63). The authors ultimately concluded that "CRM197 has potential as a safe and suitable vaccine carrier for active and selective immunization against specific protein sequence modifications or conformations," and Dr. He noted that there was no evidence that CRM197 could "induce molecular mimicry-induced autoimmunity." Pet'r's Ex. 63 at 1; Resp't's Ex. AI at 6.

Regarding the timing of Petitioner's symptoms, Dr. He pointed out that Winer et al. showed a "significant level" of GBS cases around eight weeks after respiratory infections. Resp't's Ex. AI

[43] Hendrika W. Grievink et al., *The Effect of a 13-Valent Conjugate Pneumococcal Vaccine on Circulating Antibodies Against Oxidized LDL and Phosphorylcholine in Man, A Randomized Placebo-Controlled Clinical Trial*, 9 BIOLOGY 345 (2020).

[44] Valérie Vingtdeux et al., *A Modification-Specific Peptide-Based Immunization Approach Using CRM197 Carrier Protein: Development of a Selective Vaccine Against Pryoglutamate Aβ Peptides*, 22 MOLECULAR MED. 841 (2016).

at 7. Accordingly, he opined Petitioner's URI "clearly falls within the window to cause her GBS." *Id.*

7. Dr. DeAngelo's Third Supplemental Report

Dr. DeAngelo first addressed Dr. He's assertion that his theory lacked any evidence by stating that "I am being asked to present a sound and reliable medical theory on plausible vaccine causation and not direct evidence or definitive proof of its existence in the medical literature." Pet'r's Ex. 64 at 1. He then repeated his theory of phospholipid targeting via molecular mimicry that he proposed in his second supplemental report. *Id.* at 2. He also argued that while some cross-reactions are not always harmful, he cited to his prior filed literature and stated that "it is equally well-understood in the medical community that such cross-reactivities between self-proteins and/or other self-antigens have at the same time been linked to different immune-mediated demyelinating diseases such as GBS." *Id.* at 3. Dr. DeAngelo next turned to Dr. He's critique of the Grievink report, and acknowledged that Dr. He's conclusion regarding a lack of increase in protective anti-phosphorylcholine was correct. *Id.* However, he argued that "subgroup analysis by the authors suggested an induction of [phosphorylcholine]-specific . . . antibodies in individuals receiving two doses six months apart . . . leav[ing] the door open for future research in the area." *Id.*

Turning to the issue of Petitioner's prior URI, Dr. DeAngelo took issue with Dr. He's theory, as "Dr. He suggest[ed] that just because there is 'extensive clinical evidence linking [URI] to GBS and that an infection may have occurred [five-to-seven] weeks before GBS symptom onset, which is within the [one-to-13] weeks window of the Winer et al. article, that a [URI] is the cause of [P]etitioner's GBS.'" Pet'r's Ex. 64 at 4 (quoting Resp't's Ex. AI at 7). Dr. DeAngelo then reiterated his prior argument that Petitioner's Prevnar 13 vaccine, being administered eight days before onset, was more in line with the Winer et al. data than Dr. He's theory. *Id.* at 5. Dr. DeAngelo also acknowledged that infections are the most common trigger for GBS but cited to the National Institute of Health ("NIH") GBS fact sheet, which stated that onset occurred after "a few days or weeks" and noted that "[i]n rare cases, vaccinations may increase the risk of GBS." *Id.*[45] The remainder of Dr. DeAngelo's report was a recitation of his prior-stated theory of causation. *Id.* at 5–6.

V. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) that the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of her receiving a covered vaccine. *See* § 300aa-11(c)(1)(C); *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, 1319–20 (Fed. Cir. 2006). In this case, GBS is not a Table injury associated with the Prevnar 13 vaccine, and thus Petitioner must prove by preponderant evidence that her injury was caused-in-fact by a Table vaccine.

[45] Petitioner did not file the cited NIH reference as an exhibit.

A. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993); *but see Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejection the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 538 (2001) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance").

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting *Murphy v. Sec'y of the Dep't of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (Fed. Cl. 1991)). Ultimately, a determination regarding a witness' credibility is needed when determining the weight that such testimony should be afforded. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009); *Bradley v. Sec'y of Dep't of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. *Valenzuela v. Sec'y of Health & Hum. Servs.*, No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); *see also Eng. v. Sec'y of Health & Hum. Servs.*, No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

B. Causation-In-Fact

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999).

In the seminal case of *Althen v. Sec'y of Health & Hum. Servs.*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). The *Althen* test requires petitioners to set forth: "(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." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano*, 440 F.3d at 1326.

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. den'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). 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 theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278 – 81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1360 (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322; *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.* requires that courts determine the reliability of an expert opinion before it may be considered as evidence. 509 U.S. 579 (1993). However, in the Vaccine Program, the *Daubert* 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) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to the persuasiveness of expert testimony already admitted."); *see also Cedillo v. Sec'y of health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). Under *Daubert*, the

> Factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or

> potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

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

The *Daubert* factors are "meant to be helpful, not definitive." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 151 (1999). The factors do not "constitute a 'definitive checklist or test'" and may be applied differently depending on the facts of a particular case. *Id.* at 150 (quoting *Daubert*, 509 U.S. at 593).

"In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establish a standard of evidentiary reliability." *Daubert*, 509 U.S. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. 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." *Synder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *24 (Fed. Cl. Spec. Mstr. Nov. 28, 2016) (stating that the Vaccine Act "require[s] a chain of reliable propositions supporting [a] petitioner's theory[]").

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. 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; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed. Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen* 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. This is because "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.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally

contemporaneous to the medical events." *Cucuras*, 993 F.2d at 1528. However, there is no "presumption that medical records are accurate and complete as to all of the patient's physical conditions." *Kirby*, 997 F.3d at 1383 (finding that a special master must consider the context of a medical encounter before concluding that it constitutes evidence regarding the absence of a condition). While a special master must consider these opinions and records, they are not "binding on the special master or court." § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

In determining the accuracy and completeness of medical records, special masters will consider various explanations for inconsistencies between contemporaneously created medical records and later given testimony. The Court of Federal Claims has identified four such explanations for explaining inconsistencies: (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 with testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of health & Hum. Servs.*, 110 Fed. Cl. 184, 203 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014).

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to finger causation-in-fact." *de Bazan v. Sec'y of health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of health & Hum. Servs.*, 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that she suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen*, 35 F.3d at 547. In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Kundsen*, 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden the shifts to the government to prove that an alternative cause, unrelated to the

administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

VI. Analysis

A. *Althen* Prong One

Under *Althen* prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused or sustained injury. *Andreu*, 569 F.3d at 1375; *Pafford*, 451 F.3d at 1355–56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. *Boatmon*, 941 F.3d at 1359; *see also Knudsen*, 35 F.3c at 548; *Veryzer v. Sec'y of Health & Hum. Servs.*, 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"), *aff'd* 475 F. App'x 765 (Fed. Cir. 2012). If Petitioner relies upon a medical opinion to support her theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. *See Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision oftentimes is based on the credibility of the experts and the relative persuasiveness of their competing theories"); *Perriera v. Sec'y of Health & Hum. Servs.*, 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing *Fehrs v. United States*, 620 F.2d 255 (Ct. Cl. 1980))). Importantly, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde*, 746 F.3d at 1339. Instead, Petitioner must show it was more likely than not that the vaccine caused the condition alleged. *Id.*

First, Petitioner misstates her burden under the first *Althen* prong. Petitioner's brief states that she need only provide "a plausible medical theory causally connecting her Prevnar 13 vaccine with the onset of her GBS." Pet'r's Mot. at 9. However, as noted above, the Federal Circuit has explained that mere plausibility is insufficient to satisfy Petitioner's burden. *LaLonde*, 746 F.3d at 1339. Despite this error, it is not fatal to Petitioner's claim, as her theory still satisfies the preponderant standard for *Althen* prong one. Dr. DeAngelo presented two theories in support of Petitioner's claim throughout his reports. However, Petitioner's brief indicates that Dr. DeAngelo believed his second theory of a cross-reaction between the phosphoglycerol components of the Prevnar 13 vaccine and the phospholipid components of the myelin sheath was more likely than his initial theory. *See* Pet'r's Mot. at 11. Because this second theory is the primary focus of Petitioner's argument in her Motion for a Ruling on the Record, and I find that this theory satisfies the first *Althen* prong, I will not discuss Dr. DeAngelo's other theory regarding the vaccine's polysaccharide carbohydrate sequence homology with peripheral nerve tissue.

Dr. DeAngelo based his theory on molecular mimicry, which has been accepted as a sound and reliable theory in many demyelinating conditions in the Vaccine Program, including GBS. *See*, *e.g.*, *Conte v. Sec'y of Health & Hum. Servs.*, No. 17-403V, 2020 WL 5743696 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established

and well-settled in the Vaccine Program"); *Barone v. Sec'y of Health & Hum. Servs.*, No. 11-707V, 2014 WL 6834557, at *8–9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting molecular mimicry "has been accepted in other Program cases as a reliable medical explanation for how various autoimmune conditions could develop after the receipt of different kinds of vaccinations"); *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947, at *18 (Fed. Cl. Spec. Mstr. Oct. 8, 2021); *Maloney v. Sec'y of Health & Hum. Servs.*, No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); *Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); *Sprenger v. Sec'y of Health & Hum. Servs.*, No. 18-279V, 2023 WL 8543435, at *18-20 (Fed. Cl. Spec. Mstr. Nov. 14, 2023); *Parker v. Sec'y of Health & Hum. Servs.*, No. 20-411V, 2023 WL 9261248, at *21-22 (Fed. Cl. Spec. Mstr. Dec. 20, 2023).

Dr. He denounced molecular mimicry as a theory outright, contending that if it were true, all vaccines could cross-react to induce all kinds of autoimmune conditions. However, as noted by Dr. DeAngelo, it is well accepted in the scientific community that molecular mimicry is also largely based off an individual's unique genetic susceptibility in addition to the existence of cross-reactive antigens. Nonetheless, Dr. He argued that the traditional criteria provided by the IOM must be met to establish whether a vaccine can cause GBS via molecular mimicry. *See* Resp't's Ex. A at 7. The first criterion is a susceptible host with a similarity between a host epitope and an epitope of the vaccine. Dr. DeAngelo provided an example of this homology by identifying a phospholipid in myelin the host epitope and a serotype in the vaccine (18C) as the vaccine epitope. The second criterion is exposure to an epitope which expresses antigens that are immunologically similar to self-antigens. Dr. DeAngelo supported his assertion that GBS patients have phospholipid antibodies by citing Gilburd et al.

Dr. DeAngelo acknowledged the lack of epidemiological evidence (other than the case reports), and he does not cite any animal models to support his theory. Therefore, and as conceded by Dr. DeAngelo, the remaining traditional criteria used to prove molecular mimicry are not fulfilled. However, given the state of current scientific knowledge, it may be impossible or impractical in many cases for a petitioner to satisfy these criteria for his claim to be successful. Indeed, a careful review of the studies cited by Dr. He show that Prevnar 13 was either not studied or there was insufficient data to draw a conclusion. *See* Resp't's Ex. K (not analyzing Prevnar 13); *see also* Resp't's Ex. AD (analysis of the mechanisms of COVID-19); Resp't's Ex. P (comparing Prevnar 13 to PPSV23). Haber et al. appears to be the only study specifically identifying Prevnar 13 and examining its effects on the population writ large. However, the data and methodology used by the authors to reach their conclusions were not provided. Accordingly, the lack of epidemiological information does not support arguments advanced by either side regarding a causal association between Prevnar 13 and GBS.

Dr. He's insistence that Petitioner provide epidemiologic evidence to support his theory is also inconsistent with the nature of the Vaccine Program. Indeed, Dr. He requires scientific certainty, which is a bar too high. *See Knudsen*, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program"). Regardless, Petitioner does not need to make a specific type of evidentiary showing (i.e., epidemiologic studies) to satisfy his burden, and Respondent's burden to present any rebuttal evidence only arises after Petitioner has presented a prima facie showing of causation. *See Capizzano*, 440 F.3d at 1325–26; *see also Hammitt*, 98 Fed. Cl. at 726.

Dr. He also did not refute the scientific data or foundational evidence used by Dr. DeAngelo to support his theory. Instead, Dr. He again claimed that Dr. DeAngelo's theories are "unfounded" and based off the assumption that a cross-reaction would occur. However, as explained above, these criticisms lay too high a bar than what is required from petitioners in the program. Dr. DeAngelo identified the serotype 18C component of the vaccine as containing phosphoglycerol, which could initiate development of anti-phospholipid antibodies that could cross-react with epitopes on peripheral nerve myelin or axonal glycoproteins. He supported this as a possible pathological mechanism by citing to Ho et al., which found that myelin phospholipids are targeted by an immune response to MS, resulting in demyelination. In analyzing their findings, the authors applied this phenomenon to autoimmune disease generally, explaining that that "[t]he adaptive immune system may target molecules that are aberrantly modified or expressed and contribute to the autoimmune pathology." Pet'r's Ex. 59 at 9. Dr. DeAngelo also cited Ohori et al., which found that Prevnar 13 contains phosphorylcholine, and that the human body initiates an immune response to phosphorylcholine. He further showed that patients with GBS have autoantibodies to phospholipids. While Gilburd et al. speculated that the presence of these antibodies may be the result of demyelination and not the cause, Nakos et al. later found that phosphatidylinositol, phosphatidylcholine, and phosphatidic acid to be the main antigens of anti-phospholipid antibodies in GBS patients. *See* Pet'r's Ex. 61 at 1. The authors explained that while prior studies considered anti-phospholipid antibodies would only target some phospholipids, their study "confirm[ed] the results of a previous study [] which found that these antibodies are directed against all the phospholipids, including . . . [phosphatidylcholine]." Resp't's Ex. A at 3. Further, Dr. He himself admitted in his reports that "cross-reactive autoantibodies may play some roles in [the] immunopathogenesis [of GBS]. The cross-reactive antibodies may attack peripheral nerves and roots and cause demyelination and axonal damage." *Id.*

Additionally, the causal theory proffered by Petitioner has previously been accepted as sound and reliable in other cases, decided by various special masters. *See*, *Cooper v. Sec'y of Health & Hum. Servs.*, 2024 WL 1522331 (Fed. Cl. Spec. Mstr. Mar. 12, 2024); *Anderson v. Sec'y of Health & Hum. Servs.*, 2024 WL 557052 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); *Parker, v. Sec'y of Health & Hum. Servs.*, 2023 WL 9261248 (Fed. Cl. Spec. Mstr. Dec. 20, 2023); *Koller*, 2021 WL 5027947. While these prior decisions are not binding on myself, I can consider them as I form my opinion. *See Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998), *aff'd*, 191 F.3d 1344 (Fed. Cir. 1999); *Boatmon*, 941 F.3d at 1358. I agree with the reasoning offered by my colleagues in these other cases, and for many of the same reasons find Petitioner's theory here sound and reliable and proven by preponderant evidence.

I recognize that there is not uniformity between the special masters in decisions addressing the Prevnar 13 vaccine and GBS. *See*, *e.g.*, *Deshler v. Sec'y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *19 (Fed. Cl. Spec. Mstr. July 1, 2020); *Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at *26 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review denied*, 167 Fed. Cl. 127; *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at *31-32 (Fed. Cl. Spec. Mstr. Jan. 3, 2023); *Gamboa-Avila v. Sec'y of Health & Hum. Servs.*, No. 18-925V, 2023 WL 6536207, at *25 (Fed. Cl. Spec. Mstr. Sept. 11, 2023); *McConnell v. Sec'y of Health & Hum. Servs.*, No. 18-1051V, 2022 WL 4008238, at *9 (Fed. Cl. Spec. Mstr. Aug. 19, 2022). I acknowledge these cases but also note that the decisions of other special masters or Court of Federal Claims' judges are not binding on special masters. *Boatmon*, 941 F.3d at 1358; *Hanlon*, 40 Fed. Cl. at 630.

Accordingly, I find that Petitioner has proven by preponderant evidence a sound and reliable causal theory establishing that the Prevnar 13 vaccine can cause GBS, satisfying *Althen* prong one.

B. *Althen* Prong Two

Under *Althen* prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Capizzano*, 440 F.3d at 1324 (quoting *Althen*, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" *Pafford*, 451 F.3d at 1356 (internal citations omitted). This includes instances where an action is "a substantial factor in bringing about the harm, and that the harm would not have occurred but for that action." *Shyface*, 165 F.3d at 1365 (internal citations omitted). The Federal Circuit has explained that in such cases, "while the vaccination must be a substantial factor in the [injury], it need not be the sole factor or even the predominate factor." *Pafford*, 451 F.3d at 1357.

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause-and-effect show[s] that the vaccination was the reason for the injury.'" (quoting *Althen*, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. *Curcuras*, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Capizzano*, 400 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. *Id.* at 1325–26.

There is preponderant evidence in the record to support a logical sequence of cause and effect showing the February 2, 2018 Prevnar 13 vaccine caused Petitioner's GBS. Following vaccination, Petitioner began to experience symptoms of numbness, tingling, ataxia, and weakness in her arms and legs, eventually leading to her hospitalization on February 16, 2018. The period of eight days between vaccination and onset, and 14 days between vaccination and nadir, is consistent with the literature. *See* Pet'r's Ex. 24; *see also* Pet'r's Ex. 41. Additionally, Petitioner's providers, including her neurologist, continuously noted that Petitioner had previously received the Prevnar 13 vaccine prior to her symptom onset. *See* Pet'r's Ex. 4 at 363, 413, 581, 759; Pet'r's Ex. 5 at 2, 10, 17; Pet'r's Ex. 6 at 2; Pet'r's Ex. 11 at 36. It is also notable that while Petitioner reported her December 2017 URI to her providers when asked, it was not repeatedly included in Petitioner's chart in the same way her Prevnar 13 vaccination was continuously mentioned. Rather, it appears from Petitioner's medical record that her providers considered the Prevnar 13 vaccine in the development of her GBS while disregarding her prior URI.

Accordingly, Petitioner has presented preponderant evidence consistent with her proposed mechanism to show a logical sequence of cause and effect that the Prevnar 13 vaccine was the actual cause of her GBS and has satisfied the second *Althen* prong.

C. *Althen* Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationships." *Id.* Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under *Althen* prong one). *Id.*; *Koehn v. Sec'y of Health & Hum. Servs.*, 773 F.3d at 1239, 1243 (Fed. Cir. 2014); *Shapiro*, 101 Fed. Cl. at 542; *see Pafford*, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does constitute preponderant evidence of vaccine causation. *See, e.g., Veryzer*, 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that [P]etitioner must posit a medical theory causally connecting the vaccine and injury"). As stated above, I find that Petitioner's symptom onset eight days after vaccination is consistent with the medical literature regarding GBS onset. *See* Pet'r's Ex. 24; *see also* Pet'r's Ex. 41. Dr. He does not contest whether Petitioner's eight-day onset is appropriate, and Respondent poses no objection to the proposed timing of Petitioner's theory. Accordingly, I find by preponderant evidence that this timing is consistent with a proximate temporal relationship between vaccination and injury. *See Althen*, 418 F.3d at 1278. Thus, Petitioner has satisfied the third *Althen* prong.

D. Alternative Causation

Because Petitioner has established a prima facie case for causation, Petitioner is entitled to compensation unless Respondent can put forth preponderant evidence "that [Petitioner's] injury was in fact caused by factors unrelated to the vaccine." *Whitecotton v. Sec'y of Health & Hum. Servs*., 17 F.3d 374, 376 (Fed. Cir. 1994), *rev'd on other grounds sub nom.*, *Shalala v. Whitecotton*, 514 U.S. 268 (1995); *see also Walther v. Sec'y of Health & Hum. Servs.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007). Here, Respondent contends that Petitioner's GBS is the result of her December 2017 URI rather than her Prevnar 13 vaccination. Respondent's expert, Dr. He, relied significantly on Winer et al. to show that 47% of GBS patients experienced some form of respiratory infection in the 13 weeks prior to onset, and thus concluded that Petitioner's URI must be the cause of her GBS. Resp't's Ex. I at 4. However, the authors noted that a significant association between respiratory infections and GBS "was present for an interval of [one] month but not [three] months before onset of GBS." *Id.* The study concluded that "the greatest relative risk of developing GBS is seen in the first [two] weeks following infection." *Id.* at 5. This would be more in line with Petitioner's theory that the Prevnar 13 vaccine caused her GBS, as well as with the literature filed by Petitioner which largely shows a one-to-two-week gap between immune stimulation and onset in cases following vaccination. *See* Pet'r's Ex. 22 (two weeks between Prevnar 13 vaccination and GBS onset); *see also* Pet'r's Ex. 31 (observing a one-to-four-week onset period following PPSV23 vaccination). Accordingly, I find Respondent has failed to show that it is more likely than not that an alternative cause was the sole cause of Petitioner's injury. Thus, Respondent did not prove by a preponderance of the evidence that Petitioner's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B).

VII. Conclusion

For the reasons discussed above, I find that Petitioner has established by preponderant evidence that her Prevnar 13 vaccine caused her GBS. Therefore, Petitioner is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Herbrina D. S. Young
Herbrina D. S. Young
Special Master