In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 19-729V

Filed: January 15, 2026

RICARDO RODRIGUEZ,	*
Petitioner,	*
v.	*
SECRETARY OF HEALTH AND HUMAN SERVICES,	*
Respondent.	*

Christina Ciampolillo, Conway, Homer, P.C., Boston, MA, for Petitioner.
Camille Jordan Webster, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

Shah, Special Master:

On May 17, 2019, Ricardo Rodriguez ("Petitioner" or "Mr. Rodriguez") filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. §§ 300aa-10 *et seq*.[2] (the "Vaccine Act" or "Program"). ECF No. 1 ("Pet."). The petition alleges that Petitioner developed Guillain-Barré syndrome ("GBS") caused by the meningococcal and human papillomavirus ("HPV") vaccines he received on July 3 and August 4, 2017. *Id*. at 1.

[1] Because this Decision contains a reasoned explanation for the action in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), 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. For ease of citation, all "§" references to the Vaccine Act in this Decision will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

For the reasons discussed in this decision, I find that Petitioner has proven that the HPV vaccine caused him to develop GBS. Briefly, he has preponderantly established he developed GBS, that the HPV vaccine can cause GBS, that there was a logical sequence of cause and effect between the vaccination and the development of his GBS, and that the timeframe between vaccination and onset of GBS symptoms was appropriate. The petition is accordingly granted.

I. PROCEDURAL HISTORY

After filing the petition, Petitioner filed medical records and an affidavit. Exs. 1-5. On January 17, 2020, Respondent filed a Rule 4(c) Report contending that entitlement should be denied. ECF No. 14 ("Report") at 1, 6.

Petitioner filed an expert report from David M. Simpson, M.D., along with Dr. Simpson's curriculum vitae and medical literature, on April 29, 2020. Exs. 6-18. On October 26, 2020, Respondent filed an expert report from Peter D. Donofrio, M.D., along with Dr. Donofrio's curriculum vitae and medical literature. Ex. A & Tabs 1-6. On November 29, 2020, Respondent filed an expert report from S. Mark Tompkins, Ph.D., along with Dr. Tompkins's curriculum vitae. Ex. B & Tab 1. On January 11, 2021, Respondent filed medical literature relating to Dr. Tompkins's report. Ex. B, Tabs 2-24.

On May 3, 2021, Petitioner filed a supplemental expert report and medical literature from Dr. Simpson. Exs. 19-29. Respondent filed a supplemental expert report by Dr. Donofrio on October 12, 2021, and medical literature on October 14, 2021. Ex. C & Tabs 1-4.

On October 26, 2021, former Special Master Katherine E. Oler held a status conference at which she noted that "the two main issues in this case are diagnosis and causation." ECF No. 39 at 1. Special Master Oler explained that after reviewing the medical records and expert reports, she believed "that a diagnosis of GBS is supportable in this case." *Id.*; *see* Ex. 2 at 195, 884-96. Moreover, although she did not make a finding on causation during the status conference, Special Master Oler noted that the onset of Petitioner's symptoms following vaccination was within a medically appropriate timeframe. ECF No. 39 at 1. Special Master Oler indicated that it would be beneficial for the parties to engage in settlement negotiations. *Id*.

On November 29, 2021, Respondent indicated he was "not interested in settlement on the current record." ECF No. 40. The parties indicated their intent to proceed with an entitlement hearing. ECF No. 42.

Petitioner filed additional medical records on February 16, 2023, and additional medical literature on April 20, 2023. Exs. 30-33. The parties filed pre-hearing briefs on April 19, May 3, and May 5, 2023. ECF Nos. 54, 63, 65.

Special Master Oler conducted an entitlement hearing on May 19, 2023, at which Drs. Simpson, Tompkins, and Donofrio testified. Minute Entry dated 5/19/2025; Tr. at 3. The parties filed post-hearing briefs. ECF Nos. 70, 72, 75.

The parties agreed that the record was complete on January 9, 2024. ECF No. 76. This case was reassigned to me on August 13, 2024. ECF No. 77. This matter is now ripe for adjudication.

II. FACT EVIDENCE

Petitioner was born May 20, 1999. Ex. 2 at 26. He had a pre-vaccination history of asthma. *Id.* at 24. He received his first meningococcal vaccination on September 6, 2011. Ex. 1 at 1.

According to his affidavit, executed May 16, 2019, Petitioner graduated from high school in the spring of 2017. Ex. 4 ("Affidavit") at 1. He planned to attend the University of Texas beginning in August 2017. *Id.*

Petitioner received his second meningococcal vaccine and his first HPV vaccine on July 3, 2017. Ex. 2 at 12; Ex. 1 at 1. On August 4, 2017, he received his second HPV vaccine at an appointment for a tuberculosis test. Ex. 2 at 28; Ex. 1 at 1.

According to his affidavit, Petitioner "did not notice anything unusual" after receiving the meningococcal and HPV vaccinations on July 3, 2017, and he continued playing sports. Affidavit at 1. However, the day after he received his second HPV vaccine on August 4, 2017, he "felt fatigued" and his "calf muscles were sore and cramping." *Id.* at 2. Though he initially assumed his symptoms were "due to playing soccer earlier that week," the cramping continued and he "noticed it was difficult to climb stairs." *Id.*

On August 7, 2017, Petitioner saw his primary care provider ("PCP"), Kaneez R. Khan, M.D., at Parkland Hospital, for muscle cramps in his right lower leg and extremity weakness. Ex. 2 at 42, 45. He reported the myalgias had been present for three days. *Id.* at 45. He also reported having a decreased range of motion and difficulty walking. *Id.* He had not taken any pain medications. *Id.* He denied having any injury but explained he had walked long distances over the weekend during a college tour. *Id.* He denied lower back pain, fatigue, weakness, weight loss or gain, fevers, or night sweats. *Id.* He reported having received the second dose of the HPV vaccine the same day as his tuberculosis test. *Id.* Dr. Khan noted that Petitioner was "attributing [his symptoms] to the HPV vaccination." *Id.*

On exam, Petitioner had normal range of motion in his spine, shoulders, elbows, wrists, fingers, hips, knees, and ankles, with no active swelling, tenderness, or synovitis in his joints and no soft tissue nodules. Ex. 2 at 46. He complained of pain in his calf and lower thigh. *Id.* Dr. Khan assessed "[m]uscle spasms of both lower extremities," and he recommended Petitioner take 400 mg of Ibuprofen every 8 hours for three days and then as needed. *Id.* at 46-47. Dr. Khan also noted that the HPV vaccine likely did not cause Petitioner's myalgias and that "[e]xtensive walking vs other etiology for myalgia/myositis [was] possible." *Id.* at 47.

On August 16, 2017, Petitioner presented to Khalida Yasmin, M.D., for muscle weakness of the extremities following his HPV vaccination. Ex. 2 at 120. He reported that the day after the August 4, 2017 HPV vaccination, he developed pain in his lower extremities, was unable to walk, needed assistance to be helped up, and had since had difficulty walking and had fallen several

times at home. *Id.* at 123. He was also feeling weakness in his right hand, was unable to squeeze a lemon, and tended to drop things. *Id*. On exam, Petitioner had an abnormal, "wobbly" gait and poor balance. *Id.* at 126. Dr. Yasmin's assessment was lower extremity and right arm weakness. *Id*. Petitioner was referred to the Parkland Hospital Emergency Department ("ED"). *Id*. at 127.

Later that day, Petitioner was seen by Katherine Gaston, M.D., at the ED. Ex. 2 at 173. Petitioner reported that his symptoms began the day after receiving his HPV vaccine on August 4, 2017, and since then he was unable to "squeeze well with his hands or flex his toes." *Id*. He also reported that he had "fallen down the stairs because his legs gave out beneath him." *Id*. On exam, he had decreased grip strength and decreased strength in both forearms, ankles, and toes. *Id.* at 175. He could not stand on his toes or squat. *Id.* Dr. Gaston's differential diagnosis included GBS. *Id.* She recommended a lumbar puncture, lab tests, and a neurology consultation. *Id.*

On August 17, 2017, Petitioner was seen as an inpatient by neurologist Michelle Devine, M.D. Ex. 2 at 181. Dr. Devine noted that Petitioner had received the second dose of the HPV vaccine on August 4. *Id.* He reported that the next day, he developed bilateral calf cramping, and by August 8, he began having difficulty walking. *Id.* One to two days later, he noticed hand weakness. *Id.* He reported no sick contacts or symptoms of infection in the last three months. *Id.* On exam, he had normal reflexes but demonstrated decreased foot clearance bilaterally while walking. *Id.*at 185. Dr. Devine suspected GBS or a myopathy. *Id.*

Also on August 17, Petitioner was examined by neurologist Hsueh-Sheng Chiang, M.D., Ph.D., who documented absent upper extremity reflexes but intact reflexes in the lower extremities. Ex. 2 at 197. Later that same day, attending neurologist Darin T. Okuda, M.D., examined Petitioner and found he had a "reduction in his overall strength" in all extremities but intact ankle and patellar reflexes. *Id.* at 195. Those findings, along with Petitioner's cerebrospinal fluid ("CSF") results, led Dr. Okuda to diagnose "GBS post immunization against HPV." *Id*. The plan was to begin plasmapheresis ("PLEX") therapy.[3] *Id.*

Petitioner saw the transfusion medicine attending physician, James D. Burner, M.D. Ex. 2 at 186. Dr. Burner noted that Petitioner received the HPV vaccine on August 4, 2017, his symptoms began "within a day," and he was "admitted with suspected GBS." *Id*. Petitioner completed his fifth course of PLEX therapy on August 25, 2017. *Id*. at 884. At that point, he was able to walk but remained weak. Affidavit at 3. His strength gradually improved with physical and occupational therapy, his gait improved from wobbly and wide-based to normal and requiring no assistance, and he was able to briefly stand on his toes. Ex. 2 at 884. He was discharged from the hospital on August 25, 2017. *Id.* at 883.

On September 27, 2017, Petitioner saw neurologist Khalil Salim Husari, M.D., for a follow-up. Ex. 2 at 946. Dr. Husari noted that Petitioner was "about 85% back to his baseline" but reported some difficulty with running, playing soccer, and writing. *Id*. The HPV vaccine was listed under Petitioner's allergies. *Id.* An exam revealed bilaterally reduced reflexes in the biceps,

[3] Dr. Chiang similarly noted that Petitioner's "[s]ymptoms arose after his second shot of HPV vaccine." Ex. 2 at 197.

triceps, brachioradialis, and ankles. *Id.* at 948. Dr. Husari's impression was that Petitioner had made a "good recovery so far" and was going to continue with physical therapy and add occupational therapy. *Id*. Dr. Husari commented:

> He will continue to be off any immunotherapy as we think that it was an isolated event. Regarding the question of Flu vaccination, data are not clear and there is no clear consensus. But we would avoid Flu vaccination this season at least and possibly [a] couple of years.

Id. He did not make any specific recommendation about any other future vaccinations.[4]

Petitioner followed up with Dr. Husari again on February 21, 2018. Ex. 2 at 998. Dr. Husari noted that Petitioner "continues to do well, denies any new neurological symptoms, [] continues to make slow progress," and was "back in college now." *Id*. Although he had slightly reduced strength in his fingers bilaterally, he otherwise had normal strength on examination. *Id*. at 999. He also had a normal-based gait, stride, stance, and arm swing and was able to walk on his heels, toes, and tandem. *Id*. at 999-1000. He still had bilaterally reduced reflexes in the biceps, triceps, brachioradialis, and ankles. *Id.* at 999. Dr. Husari's assessment was that Petitioner as "doing well" and he wished him "good luck with college" and told him to "avoid vaccination for the next few years." *Id*. at 1000-01.

There are no relevant medical records after February 2018.

Petitioner began classes at his local community college in the fall of 2018, a year later than he planned. Affidavit at 3. At the time his affidavit was executed in 2019, he had "mostly recovered" from GBS, though he fatigued "much more quickly" than he did before his injury. *Id*.

III. EXPERT EVIDENCE

A. Expert Reports

1. David M. Simpson, M.D.: First Expert Report

Dr. Simpson submitted two reports in this case. Ex. 6 ("First Simpson Rep."); Ex. 19 ("Second Simpson Rep.").

Dr. Simpson earned his M.D. from the State University of New York at Buffalo in 1979. Ex. 7 ("Simpson CV") at 1. He is licensed in New York and certified by the National Board of Medical Examiners, the American Board of Psychiatry and Neurology, with subspecialties in Clinical Neurophysiology and Neuromuscular Medicine, and the American Board of Electrodiagnostic Medicine. *Id*. Currently, Dr. Simpson is a Professor of Neurology and the

[4] Dr. Husari's notes from the September 27, 2017 visit were reviewed and affirmed by the attending neurologist, Lauren Phillips, M.D. Ex. 2 at 949.

Director of the Neuromuscular Division and Clinical Neurophysiology Laboratories at the Icahn School of Medicine at Mount Sinai, where he has worked since 1984. *Id*. at 2. He also serves on the editorial board for *AIDS Patient Care* and *Current HIV/AIDS Reports* and as an ad-hoc reviewer for more than 50 journals. *Id*. at 14-15. In a given month, Dr. Simpson sees "two to three patients" diagnosed with GBS. Tr. at 10.

Dr. Simpson opined that it is more likely than not that the HPV and meningococcal vaccinations caused Petitioner to develop GBS. First Simpson Rep. at 5. He cited four possible biological mechanisms by which a vaccine could lead to neurological illness, either by autoimmunity or direct neurotoxic effects. *Id.* at 3 (citing Thomas J. Safranek et al., *Reassessment of the Association between [GBS] and Receipt of Swine Influenza Vaccine in 1976-1977: Results of a Two-State Study*, 133 AM. J. OF EPIDEMIOLOGY 940 (1991) (Ex. 8) ("Safranek")). The first proposed mechanism, molecular mimicry, rests on the supposition that the epitopes of the vaccine can result "in the development of immune antibodies and/or T cells that could cross-react with epitopes on myelin or axonal glycoproteins" and lead to neuronal damage. *Id*. (citing Michael C. Levin et al., *Neuronal Molecular Mimicry in Immune-Mediated Neurologic Disease*, 44 ANN. NEUROL. 87 (1998) (Ex. 9) ("Levin")). Molecular mimicry is "widely accepted" as a mechanism for causing the development of autoimmunity generally and GBS specifically. *Id*. at 4. This mechanism has been proven to lead to the development of GBS, most notably in the setting of *Campylobacter jejuni* ("*C. jejuni*") infection. *Id*. (citing Levin). Furthermore, it is "generally accepted" that vaccination "can serve as the causal antecedent" for GBS and chronic inflammatory demyelinating polyneuropathy ("CIDP"). *Id*. at 4-5.

Second, Dr. Simpson explained that a vaccine could cause injury by directly damaging the myelin membranes or axons. First Simpson Rep. at 3. Third, immune complex formation caused by vaccination could cause injury by producing a vasculitis type syndrome. *Id*. (citing Maria Antonia Pou et al., *Development of Autoimmune Diseases After Vaccination*, 14 J. OF CLINICAL RHEUMATOLOGY 243 (2008) (Ex. 10) ("Pou")). Fourth, a vaccine could promote the loss of self-tolerance, resulting in the alteration of immunoregulatory mechanisms. *Id*. at 4.

Dr. Simpson opined that there is "ample support in the medical literature" for a connection between various vaccinations and demyelinating neuropathies. First Simpson Rep. at 4 (citing J. Pritchard et al., *Risk of relapse of [GBS] or chronic inflammatory demyelinating polyradiculoneuropathy following immunisation*, 73 J. NEUROL. NEUROSURG. PSYCHIATRY 348 (2002) (Ex. 11) ("Pritchard"); Lawrence B. Schonberger, *[GBS] Following Vaccination in the National Immunization Program, United States, 1976-1977*, 110 AM. J. OF EPIDEMIOLOGY 105 (1979) (Ex. 12) ("Schonberger"); INSTITUTE OF MEDICINE (U.S.) VACCINE SAFETY COMMITTEE, ADVERSE EVENTS ASSOCIATE WITH CHILDHOOD VACCINES: EVIDENCE BEARING ON CAUSALITY, 46-47 (Kathleen R. Stratton et al. eds., 1994) (Ex. 13) ("Stratton")). The "outbreak" of cases of GBS during the swine flu vaccination program in 1976 was the most notable example of a connection between GBS and vaccination. *Id*. (citing Safranek). Moreover, "[n]umerous case reports have documented the development of GBS and CIDP following flu vaccination." *Id*. (citing Schonberger; Stratton). Additionally, in a survey conducted in the United Kingdom, 11 of 311 patients with GBS who received new immunizations developed recurrence of symptoms like weakness or fatigue. *Id.* (citing Pritchard at 348).

Additionally, "[m]edical literature supports the association of HPV vaccine and neurological adverse events." First Simpson Rep. at 4. For example, a 2015 report described 53 patients who suffered numerous neurological symptoms following HPV vaccine, "including 57% with limb weakness and 66% with neuropathic pain." *Id.* (citing Louise Brinth et al., *Suspected side effects to the quadrivalent human papilloma vaccine*, 62 DANISH MED. J. 1 (2015) (Ex. 14) ("Brinth"). The Miranda study from France showed a significantly increased risk of GBS in the HPV-vaccinated group. *Id.* (citing Sara Miranda et al., *Human papillomavirus vaccination and risk of autoimmune diseases: A large cohort study of over 2 million young girls in France*, 35 VACCINE 4761 (2017) (Ex. 15) ("Miranda")).

Similarly, the meningococcal vaccine has been associated with neurological adverse events. First Simpson Rep. at 4. In October 2005, the Centers for Disease Control and Prevention ("CDC") stated five GBS cases following this vaccination had been reported to the Vaccine Adverse Event Reporting System ("VAERS"). *Id.* (citing *Guillain-Barré Syndrome Among Recipients of Menactra Meningococcal Conjugate Vaccine--- United States, June--July 2005*, 54 MORBIDITY AND MORTALITY WKLY. REP. 1023 (2005) (Ex. 16) ("MMWR 2005"). In 2006, the CDC reported "9 more such cases, 8 of which met the surveillance definition of GBS[.]" *Id.* (citing CDC, *Guillain-Barré Syndrome Among Recipients of Menactra Meningococcal Conjugate Vaccine ---United States, June 2005--September 2006*, 55 MORBIDITY AND MORTALITY WKLY. REP. 1120 (2006) (Ex. 17) ("MMWR 2006")). The relative risk of developing GBS after the meningococcal vaccine was 1.78, a significant increase. *Id.*

Furthermore, according to a report from the National Vaccine Information Center, there is evidence that co-administration of the HPV and Menactra vaccines increases the risk of GBS. First Simpson Rep. at 4 (citing *Analysis Shows Greater Risk of GBS Reports When HPV Vaccine Is Given with Meningococcal and Other Vaccines*, NATIONAL VACCINE INFORMATION CENTER, (August 15, 2007), https://www.nvic.org/news-media-reports/press-releases/analysis-shows-greater-risk-of-gbs-reports (Ex. 18) ("NVIC")). The report described a 1130% increased risk of GBS when the HPV was co-administered with the meningococcal vaccine, as compared to HPV vaccination alone. *Id.* These findings were statistically significant. *Id.*

Dr. Simpson agreed with the GBS diagnosis, based on Petitioner's clinical presentation, lab tests, and response to PLEX treatment. First Simpson Rep. at 5. He pointed out that Petitioner was "neurologically asymptomatic prior to his receipt of the HPV and Menactra vaccines" and developed neurological symptoms one day after receiving the second HPV vaccination. *Id.* He concluded this timing was medically appropriate. *Id.* The accepted timing of an immune-mediated response following vaccination is up to 42 days after vaccination. *Id.* (citing Stratton). Moreover, the Schonberger study showed "an increased incidence of GBS up to ten weeks after vaccination." *Id.* (citing Schonberger).

Finally, Dr. Simpson noted that Petitioner's "treating physicians repeatedly documented a causal association" between the subject vaccinations and the onset of GBS. *Id.* He opined that the second dose of the HPV vaccine might have "provided a 'booster' effect, which may have increased the probability of neurological symptoms due to an autoimmune response." *Id.* There were no alternative explanations for Petitioner's GBS. *Id.*

2. Peter D. Donofrio, M.D.: First Expert Report

Dr. Donofrio submitted two reports in this case. Ex. A ("First Donofrio Rep."); Ex. C ("Second Donofrio Rep.").

Dr. Donofrio earned his M.D. from The Ohio State University School of Medicine in 1975. Ex. A, Tab 1 ("Donofrio CV") at 1. He is licensed in Ohio, Michigan, North Carolina, and Tennessee and board certified in Internal Medicine, Psychiatry and Neurology, Electrodiagnostic Medicine, and Neuromuscular Medicine. *Id*. at 2. Currently, Dr. Donofrio is retired; however, before his retirement he was the Vice-Chair of Clinical Affairs for the Department of Neurology, Director of the Neuromuscular Division, and a Professor of Neurology at Vanderbilt University School of Medicine. *Id*.; Tr. at 130.

Dr. Donofrio has published on GBS, CIDP, and other neuropathies, and he served on the Medical Advisory Board for the GBS/CIDP International Foundation. Donofrio CV at 8, 13-31; First Donofrio Rep. at 1. While he was practicing medicine, he saw between 300-500 patients per year, with roughly two patients per month being admitted for GBS. Tr. at 131. He has treated patients who presented with GBS after vaccinations. *Id*. at 132.

Dr. Donofrio took issue with the diagnosis of GBS/acute inflammatory demyelinating polyneuropathy ("AIDP") in Petitioner. First Donofrio Rep. at 7. He explained:

> [GBS] is a monophasic illness that commonly follows within 7-10 days after a gastrointestinal or respiratory illness. The neurological examination requirements to fulfill the diagnosis include a progressive motor weakness of more than one limb and areflexia (loss of tendon jerks). Universal areflexia is the rule, though distal areflexia with definite hyporeflexia of the biceps and knee jerks will suffice if other features are consistent.

Id. at 5 (citing Arthur K. Asbury & David R. Cornblath, *Assessment of Current Diagnostic Criteria for [GBS]*, 27 ANN. NEUROL. S21 (1990) (Ex. A, Tab 2) (Ex. 20) ("Asbury & Cornblath"); James J. Sejvar, *[GBS] and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data*, 29 VACCINE 599 (2011) (Ex. A, Tab 3) ("Sejvar"); Hugh J. Willison et al., *Guillain-Barré syndrome*, 388 LANCET 717 (2016) (Ex. A, Tab 4) ("Willison")). In Dr. Donofrio's view, Petitioner did not meet the GBS criteria because he did not have areflexia at any time. *Id*. Most of his treating examiners found that his reflexes were normal, and some even found enhanced reflexes in his legs. *Id.*

Dr. Donofrio further pointed out that most patients with GBS undergo EMG/NCS testing that shows evidence of "nerve demyelination in the form of conduction velocity slowing, prolonged distal latencies, conduction block and temporal dispersion, and prolong F-wave latencies." First Donofrio Rep. at 5. But these studies were not performed on Petitioner, so no such finding was made in his case. *Id.* Additionally, patients presumed to have GBS commonly

undergo imaging studies of the spinal cord to rule out myelopathy,[5] which could cause weakness and increased reflexes in the arms and legs. *Id*. Petitioner did not have such imaging. *Id*. at 6. Thus, it is possible his symptoms were caused by a transient myelopathy of the cervical spinal cord, rather than GBS. *Id*. at 5-6.

Dr. Donofrio also commented that Petitioner did not report sensory symptoms such as numbness, tingling, or paresthesias of the arms and legs. First Donofrio Rep. at 6. This is rare in GBS. *Id*. However, he did have elevated serum CPK[6] and serum aldolase, which could "suggest a muscle disorder or myopathy."[7] *Id*.

Dr. Donofrio generally noted that many illnesses can mimic GBS, including an acute or subacute myelopathy. First Donofrio Rep. at 6 (citing Kerry H. Levin, *Variants and Mimics of [GBS]*, 10 THE NEUROLOGIST 61 (2004) (Ex. A, Tab 5) ("Levin")). Although Petitioner's history eliminated most alternatives, Dr. Donofrio opined that "the differential diagnosis of GBS is broad and should be considered in someone whose presentation did not meet criteria for GBS." *Id*. In his view, "an acute or subacute myelopathy would be a leading consideration" for Petitioner's diagnosis. *Id*.

Next, Dr. Donofrio opined that the timing of the onset of Petitioner's symptoms "casts doubt on the relationship to the vaccination." First Donofrio Rep. at 6. The Vaccine Injury Table ("Table") recognizes GBS following influenza ("flu") vaccination only where the onset of GBS occurs "not less than 3 days and not more than 42 days" after vaccination. *Id*.; 42 C.F.R. § 100.3(c)(15). Petitioner's GBS symptoms began only 24 hours after receiving the second dose of the HPV vaccine, which is outside the Table timeframe. *Id*.

Dr. Donofrio also critiqued the clinical data supplied by Dr. Simpson. He noted that VAERS reports of GBS following vaccination are "weak" data, because the reporting process is passive, and the information is difficult to verify. First Donofrio Rep. at 6 (citing Penina Haber & Frank DeStefano, Letter to the Editor, 109 CLINICAL IMMUNOLOGY 359 (Dec. 2003) (Ex. A, Tab

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[5] Myelopathy: 1. any of various functional disturbances or pathologic changes in the spinal cord, often referring to nonspecific lesions in contrast to the inflammatory lesions of myelitis. 2. a pathological condition of the bone marrow. DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=32732&searchterm=myelopathy (last visited Jan. 15, 2026) ("DORLAND'S"). Dr. Donofrio defined "myelopathy" as "a generic term for spinal cord disorders." First Donofrio Rep. at 5.

[6] The experts seemingly used creatine kinase ("CK") and creatine phosphokinase ("CPK") interchangeably throughout their reports and testimony. *See e.g.* Tr. at 20 (Dr. Simpson discussing CPK); Second Simpson Rep. at 5 (Dr. Simpson addressing Petitioner's "elevated serum CK"); First Donofrio Rep. at 3 (noting Petitioner's CK values during the "Review of medical records) and 6 (noting that Petitioner had "elevated serum CPK). Dorland's Medical Dictionary Online uses these terms synonymously. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=11584 (last visited Jan. 15, 2026). For consistency, I will refer to "CK" in this opinion.

[7] Myopathy: any disease of a muscle. DORLAND'S, https://www.dorlandsonline.com/dorland/definition ?id=32891&searchterm=myopathy (last visited Jan. 15, 2026).

6) ("Haber & DeStefano")). Also, the Miranda study investigators used claim and billing codes to identify "GBS" patients and did not verify the diagnoses through a medical record review. *Id.* at 6-7. "This analysis would not be able to determine the accuracy of any diagnosis." *Id.* at 7.

3. S. Mark Tompkins, Ph.D.: Expert Report

Dr. Tompkins submitted one report in this case. Ex. B ("Tompkins Rep.").

Dr. Tompkins earned his Ph.D. in immunology from Emory University in 1997. Ex. B ("Tompkins CV") at 1. He is a Professor in the University of Georgia's Center for Vaccine and Immunology and a Professor/Assistant Department Head in the Department of Infectious Diseases. Tompkins CV at 1-2.

Dr. Tompkins's research focuses on host-pathogen interactions, and he has co-authored more than 90 peer-reviewed papers. Tr. at 85; Tompkins Rep. at 1. He received the Charles C. Shepard Laboratory Science award for a publication he co-authored in the *Journal of Virology*, entitled "Engineering Enhanced Vaccine Cell Lines to Eradicate Vaccine-Preventable Disease: the Polio End Game." Tompkins CV at 3. He serves on the editorial board of the *Journal of Virology* and is an ad hoc reviewer for several journals, including the *Journal of Immunology* and the *Proceedings of the National Academy of Science* ("PNAS"). *Id*. at 5; Tr. at 89.

Dr. Tompkins opined that Dr. Simpson provided no sound mechanism for how the HPV and Menactra vaccines could have caused Petitioner's GBS. Tompkins Rep. at 4. Dr. Tompkins responded to Dr. Simpon's report by answering four questions:

1) What is the etiology of GBS?
2) Is there evidence for molecular mimicry between HPV or Menactra vaccines and immune targets of GBS?
3) Are the HPV or Menactra vaccines associated with GBS?
4) Does the timing of HPV and Menactra vaccines support a temporal association with onset of symptoms?

Id.

First, Dr. Tompkins commented that "despite decades of research, the precise causes of GBS are still debated." Tompkins Rep. at 4 (citing Bianca van den Berg et al., *[GBS]: pathogenesis, diagnosis, treatment and prognosis*, 10 NEUROLOGY 469 (2014) (Ex. B, Tab 2) ("van den Berg"); NAT'L INSTS. OF HEALTH, *[GBS] Fact Sheet* (2018) (Ex. B, Tab 3) ("NIH"); Hong-Liang Zhang, *Th1/Th2/Th17/Treg cytokines in [GBS] and experimental autoimmune neuritis*, 24 CYTOKINE & GROWTH FACTORS REV. 443 (2013) (Ex. B, Tab 5) ("Zhang")). Some investigators believe it is antibody-mediated, while others contend it can be mediated by T cells and cytokines. *Id.* (citing Zhang).

Dr. Tompkins generally agreed that there are several "hypothesized mechanisms for triggering autoimmune disease," including the four mechanisms that Dr. Simpson discussed: molecular mimicry, neurotoxic effects, immune complex formation, and loss of self-tolerance.

Tompkins Rep. at 4. Molecular mimicry is a "hypothesis for a mechanism of triggering autoimmune disease" where an infectious agent expresses an epitope that resembles a host epitope or a molecular mimic. *Id.*

> During the immune response to the infection, the host immune system primes T cell and/or B cell responses to the infectious epitope resembling the self-epitope. If the immune response is sufficiently robust, it will 'break tolerance,' meaning T cells or B cells that were previously not able to respond to the self-epitope would be expanded by the infection and licensed to respond.

Id. This, in turn, could lead to tissue damage and autoimmunity. *Id.* at 4-5. However, "there is still a paucity of data proving the phenomena." *Id.* at 5.

Dr. Tompkins pointed out several problems with the molecular mimicry hypothesis: (1) because infections also produce self-antigens and inflammation, it is "difficult to separate the antigen that is priming the autoimmune response;" (2) "there is growing recognition that immune cell activation is regulated by multiple mechanisms;" and (3) although the "homology between linear peptides in humans and pathogenic organisms is common," it is not indicative of the potential to prime autoimmune disease. Tompkins Rep. at 5. Also, Dr. Simpson failed to explain how the HPV or meningococcal vaccines express homologous epitopes that "activate self-reactive B or T cells to break tolerance and elicit autoimmune disease." *Id.*

Dr. Tompkins noted that Dr. Simpson's molecular mimicry theory of GBS rests on the 1976 flu vaccine and *C. jejuni*. Tompkins Rep. at 5. However, though the 1976 swine flu vaccine was associated with GBS, "there is no association with the current influenza vaccines" and there is "not a demonstrated mechanism." *Id.* (citing Helmar C. Lehmann, *[GBS] after exposure to influenza virus*, 10 LANCET INFECT. DIS. 643 (2010) (Ex B, Tab 10) ("Lehmann")). Moreover, a "systematic review" of factors associated with the development of GBS did not find HPV or *N. meningitidis* were associated with GBS. *Id.* at 6.[8] Also, *C. jejuni* infection is associated with the acute motor axonal neuropathy ("AMAN") form of GBS, and this relationship can be replicated in animal models. *Id.* (citing van den Berg, Lehmann, and Nobuhiro Yuki et al., *Carbohydrate mimicry between human ganglioside GM1 and* Campylobacter jejuni *lipooligosaccharide causes [GBS]*, 101 PNAS 11404 (2004) (Ex. B, Tab 4) ("Yuki 2004")). This has not been demonstrated for the HPV or meningococcal vaccines. *Id.*

With respect to other proposed mechanisms, Dr. Tompkins noted that "mercury and other toxins can damage neuronal tissues and have been associated with neurological disorders and autoimmune disease." Tompkins Rep. at 5 (citing William Crow et al., *Mercury as an environmental stimulus in the development of autoimmunity – A systemic review*, 16 AUTOIMMUNITY REVS. 72 (2017) (Ex. B, Tab 7)). However, there is no evidence that vaccines

[8] Dr. Tompkins cited Virginia K. Wachira, Henry M. Peixoto & Maria R.F. de Oliveira, *Systematic review of factors associated with the development of Guillain-Barré syndrome 2007-2017: what has changed?*, 24 TROP. MED. INT. HEALTH. 132 (2019), but Respondent did not file this article.

containing Thimerosal are associated with neurological or autoimmune diseases. *Id*. Moreover, according to the package inserts, the HPV and meningococcal vaccines do not contain Thimerosal. *Id*. (citing Menactra, Meningococcal (Groups A, C, Y, and W-135) Polysaccharide Diphtheria Toxoid Conjugate Vaccine (Package Insert) (Revised 2018) (Ex. B, Tab 8); Gardasil, Human Papillomavirus 9-valent Vaccine, Recombinant (Package Interest) (Revised 2020) (Ex. B, Tab 9)). Similarly, although immune complexes are "implicated in several autoimmune disease," there is "no evidence that autoantibodies are forming immune complexes to trigger inflammation in GBS." *Id*.

Turning to the clinical data, Dr. Tompkins critiqued the Brinth paper supplied by Dr. Simpson, noting that it was a retrospective analysis of patients with symptoms that occurred within a month of the first or second dose of the HPV vaccine and "provide[d] no evidence of causality or even statistical comparison with other groups." Tompkins Rep. at 6. He pointed out that Miranda was the only study to identify a statistically significant increased risk of GBS, and that study triggered "several large safety studies including millions of doses of [the] HPV vaccine, which in turn "provide[d] compelling evidence that the HPV vaccine is safe and there is no epidemiological association with HPV vaccination and GBS." *Id*. at 6-7 (citing WORLD HEALTH ORGANIZATION, *Weekly epidemiological record*, No. 28, 393-404 (July 14, 2017) (Ex. B, Tab 12) ("WHO"); Julianne Gee et al., *Risk of [GBS] following quadrivalent human papillomavirus vaccine in the Vaccine Safety Datalink*, 35 VACCINE 5756 (2017) (Ex. B, Tab 13) ("Gee")).

As for the meningococcal vaccine, Dr. Tompkins noted that the MMWR reports from 2005 and 2006 are case reports and "have no data to support causality, only a temporal association between vaccination and onset of symptoms." Tompkins Rep. at 7. These reports present data from VAERS, which has known weaknesses and cannot be used to determine vaccine causation. *Id*. (citing Michael M. McNeil et al., *The Vaccine Safety Datalink: success and challenges monitoring vaccine safety*, 32 VACCINE 5390 (2014) (Ex. B, Tab 14) ("McNeil"); Tom T. Shimabukuro et al., *Safety monitoring in the [VAERS]*, 33 VACCINE 4395 (2015) (Ex. B, Tab 15) ("Shimabukuro")). Thus, the MMWR reports "do not provide substantial support for an association" between the meningococcal vaccine and GBS. *Id*. Also, later studies did not report an association between the meningococcal vaccine and GBS. *Id*. (citing Nicola Principi & Susanna Esposito, *Do Vaccines Have a Role as a Cause of Autoimmune Neurological Syndrome?*, 8 FRONTIERS IN PUB. HEALTH 1 (2020) (Ex. B Tab 16) ("Principi & Esposito")).[9]

In response to Dr. Simpson's reference to the NVIC report, Dr. Tompkins opined that there were "several considerable weaknesses" with the report, including that there were no named authors, it was not peer-reviewed, it cited only two references, and it did not provide "an accurate representation of the information" that it referenced. Tompkins Rep. at 7. In Dr. Tompkins's opinion, the NVIC report was "an unreliable source of information." *Id*. at 7-8 (referencing Lenny Grant et al., *Vaccination Persuasion Online: A Qualitative Study of Two Provaccine and Two*

[9] Dr. Tompkins also cited Priscilla Velentgas et al., *Risk of Guillain-Barré syndrome after meningococcal conjugate vaccination*, 21 PHARMACOEPIDEMIOL. DRUG SAF. 1350 (2012) and Weiling K. Yih, Eric Weinraub & Martin Kulldorff, *No risk of Guillain-Barré syndrome found after meningococcal conjugate vaccination in two large cohort studies*, 21 PHARMACOEPIDEMIOL. DRUG SAF. 1359 (2012), but Respondent did not file these articles.

Vaccine-Skeptical Websites, 17 J. OF MED. INTERNET RSCH. e133 (2015) (Ex. B, Tab 19) ("Grant")).

Lastly, Dr. Tompkins addressed whether the timing of the subject vaccinations supports a temporal association with the onset of Petitioner's symptoms. Tompkins Rep. at 8. Petitioner received his first dose of the HPV vaccine and the meningococcal vaccine on July 3, 2017, and the second dose of the HPV vaccine on August 4, 2017. *Id.*; Ex. 1 at 1. He reported that his symptoms began one day after receiving his second HPV dose. Tompkins Rep. at 8; *see* Ex. 2 at 173. Dr. Tompkins noted that the interval between vaccination and the increased risk of GBS is six weeks. Tompkins Rep. at 8 (citation not filed). He opined that "the timing of the primary vaccinations and onset of symptoms falls within an accepted window of increased risk." *Id.* He further opined that although the timing of Petitioner's injury falls within the "feasible window for onset of symptoms," the immunological data on responses to HPV vaccination does "not support a causal effect at specific times post-vaccination." *Id.*

4. Dr. Simpson: Second Expert Report

First, Dr. Simpson addressed Dr. Donofrio's contention that the preservation of deep tendon reflexes ("DTRs") in Petitioner excluded GBS/AIDP as a diagnosis. Second Simpson Rep. at 3. He opined that although "it is true that depressed or absent DTRs are a cardinal feature of classical GBS," there are reports of atypical forms of GBS with preserved DTRs. *Id.* (citing Asbury & Cornblath). For example, the Unver paper reported three GBS cases where DTRs "were consistently preserved in serial neurological exams." *Id.* (citing O. Unver et al., *Atypical [GBS] with preserved deep tendon reflexes: report of three cases*, 19S EUR. J. OF PAEDIATRIC NEUROLOGY S61 (2015) (Ex. 21) ("Unver")). Similarly, Papathanasiou & Markakis reported an atypical presentation of GBS where one patient had normal reflexes and two patients had increased DTRs. *Id.* (citing Athanasios Papathanasiou & Ioannis Markakis, *Clinical Heterogeneity of [GBS] in the Emergency Department: Impact on Clinical Outcome*, 2016 CASE REPORTS IN EMERGENCY MEDICINE 1 (2016) (Ex. 22) ("Papathanasiou & Markakis")). Another study by Yuki showed "continuity between GBS patients with hypoexcitable or absent DTRs ('DTR-reduced patients') and those with normal or exaggerated DTRs ('DTR-preserved patients'), and demonstrated the clinical, serological, and electrophysiological features in the latter." *Id.* (citing Nobuhiro Yuki et al., *[GBS] associated with normal or exaggerated tendon reflexes*, 259 J. Neurol. 1181, 1182 (2010) (Ex. 24) ("Yuki 2010")).

Dr. Simpson noted that although electrodiagnostic studies "provide helpful data" for diagnosing GBS, nerve conduction studies are "supportive" and not a "mandatory criteria." Second Simpson Rep. at 4 (citing Asbury & Cornblath). The Table says that electrophysiologic findings of GBS are "supportive, but not required." *Id.* (citing 42 C.F.R. § 100.3(c)(15)(iv)). Thus, the "lack of testing . . . does not invalidate" Petitioner's GBS diagnosis. *Id.* Similarly, nerve biopsy is "only rarely performed in patients with GBS" and is not a classic method to diagnose GBS. *Id.* (citing 42 C.F.R. § 100.3(c)(15)(iv); Asbury & Cornblath).

Next, in response to Dr. Donofrio's opinion that it is possible that Petitioner had myelopathy, which could not be ruled out because spinal cord imaging studies were not performed, Dr. Simpson opined that the "variably preserved reflexes" that Petitioner exhibited are a reported

phenomenon in GBS. Second Simpson Rep. at 4. Petitioner "had no other features consistent with myelopathy, such as sphincter involvement, spasticity, or a segmental sensory level," and therefore spinal cord imaging was "not a critical diagnostic test" for Petitioner. *Id*. Moreover, Petitioner's treating neurologists did not advance myelopathy in their differential diagnosis. *Id.*

Concerning the fact that Petitioner did not have sensory symptoms or signs, Dr. Simpson again noted that these are not required to diagnose GBS. Second Simpson Rep. at 4 (citing Asbury & Cornblath). Rather, mild sensory symptoms or signs are supportive of classical GBS. *Id.* A "paucity of sensory findings" is particularly characteristic of the AMAN variant of GBS. *Id*.

Addressing the fact that Petitioner had "modest" elevated serum CK levels during his hospitalization, Dr. Simpson noted that such elevations are "nonspecific and may be seen in a range of neuromuscular disorders affecting muscle motor neuron, peripheral nerve, and muscle." Second Simpson Rep. at 5 (citing Nizar Chahin & Eric J. Sorenson, *Serum Creatine Kinase Levels in Spinobulbar Muscular Atrophy and Amyotrophic Lateral Sclerosis*, 40 MUSCLE & NERVE 126 (2009) (Ex. 26) ("Chahin & Sorenson")). Furthermore, elevated serum CK levels are "not uncommon in patients with GBS." *Id*. (citing M. Nagappa et al., *[GBS] in the elderly: Experience from a tertiary-care hospital in India*, 46 J. OF CLINICAL NEUROSCIENCE 45 (2017) (Ex. 27) ("Nagappa"); Seok-Jin Choi et al., *HyperCKemia in [GBS]*, 83 EUR. NEUROL. 451 (2020) (Ex. 28) ("Choi")). Also, Petitioner's CK levels dropped to 231 Units/L on August 23, 2017; this was also consistent with GBS. *Id*.; Ex. 2 at 298.

As for the "[m]any illnesses that can mimic GBS" that Dr. Donofrio highlighted, Dr. Simpson agreed with Dr. Donofrio that "most of these disorders have been eliminated" by Petitioner's medical history. Second Simpson Rep. at 5. Notably, Petitioner's "consensus diagnosis was GBS" among his treating physicians, including his treating neurologists. *Id*.

Regarding onset, Dr. Simpson opined that July 3, 2017 -- the day Petitioner received the first HPV vaccine and the meningococcal vaccine -- should be the date from which onset is measured. Second Simpson Rep. at 5. He noted that the one-month onset of Petitioner's symptoms was within the appropriate time frame for Table GBS related to the flu vaccine. *Id*.

In response to Dr. Tompkins's comment that Petitioner's primary immune response to the initial vaccinations on July 3, 2017, likely had diminished by the time his GBS symptoms began, Dr. Simpson pointed out that "extrapolations from theoretical and unproven associations between the trajectory of cytokine levels and the accepted interval risk between vaccinations and GBS are inappropriate." Second Simpson Rep. at 5.

Finally, with respect to the epidemiologic and other clinical data, Dr. Simpson noted that he provided "several studies supporting an association between both [the] HPV and meningococcal vaccines and GBS." Second Simpson Rep. at 6. Notably, the rare effects of vaccines are often not detected in initial trials. *Id*. at 5 (citing Stratton (noting that even very large epidemiological studies may not detect or rule out rare events)). As the Brinth paper noted, "[p]ost-licensure monitoring may be superior to pre-licensure reviews in detecting rare adverse events." Brinth at 1.

5. Dr. Donofrio: Supplemental Expert Report

First, Dr. Donofrio noted that Dr. Simpson "acknowledged that depressed or absent deep tendon reflexes ("DTRs") are a cardinal feature of classic GBS." Second Donofrio Rep. at 1. Dr. Simpson, however, also cited numerous reports of atypical GBS with preserved DTRs. *Id*. Although Drs. Donofrio and Simpson agreed that most alternative differential diagnoses that were eliminated by Petitioner's medial history, Dr. Donofrio opined that "the differential should still be considered in the setting of normal to enhanced DTRs." *Id*.

Dr. Donofrio also agreed with Dr. Simpson that nerve conduction studies are helpful but not needed for a GBS diagnosis, and the lack of a nerve conduction study does not invalidate a GBS diagnosis. Second Donofrio Rep. at 1. He explained that absent a nerve conduction study, all other aspects of Petitioner's presentation would need to be consistent with GBS; including the presence of areflexia. *Id*. Additionally, nerve conduction studies are "necessary to differentiate the different types of GBS such as AIDP, AMAN, and AMSAN." *Id*.

Disagreeing with Dr. Simpson, Dr. Donofrio opined that in "the setting of normal and increased DTRs," spinal cord imaging "should be done." Second Donofrio Rep. at 1.

Dr. Donofrio reiterated that Petitioner did not exhibit areflexia; therefore, his "diagnosis [was] not confirmed by examinations throughout his illness." Second Donofrio Rep. at 3. Furthermore, the diagnosis of AIDP documented in Petitioner's records could not be confirmed, as there was no electrodiagnostic testing. *Id.*

B. Expert Testimony

1. Dr. Simpson's Testimony

Dr. Simpson was qualified as an expert in neurology, neuromuscular medicine, electrodiagnostic medicine, and clinical neurophysiology. Tr. at 15. He concluded that Petitioner was correctly diagnosed with GBS, and his condition was "causally related" to the HPV and meningococcal vaccinations he received on July 3, 2017. *Id*. at 22.

Dr. Simpson described GBS as an autoimmune disorder that presents with acute or sub-acute progressive muscle weakness and, on occasion, sensory symptoms and signs. Tr. at 16. The clinical findings for GBS include (1) objective muscle weakness in the limbs; (2) on occasion, bulbar musculature weakness, meaning weakness with chewing, swallowing, and, in "very severe cases," breathing; and (3) abnormal and reduced muscle and tendon reflexes. *Id*. Testing for GBS can include EMG and nerve conduction studies and CSF analysis, which often reveals "elevated spinal fluid protein with absent or a low number of white blood cells," termed albuminocytologic dissociation. *Id*. at 17.

Dr. Simpson opined that before the subject vaccinations, Petitioner was "in excellent health" and had no "recorded neurologic symptoms or signs." Tr. at 17. The onset of Petitioner's symptoms began approximately 31 days after his initial vaccinations on July 3, 2017. *Id.* at 17-18. At that time, he had muscle aches and pains and general fatigue. *Id*. at 18. As Petitioner's

symptoms progressed, he developed muscle weakness, which included "difficulty raising his arms, gripping objects, and walking." *Id*.

On August 16, 2017, Petitioner presented to the ED, where it was documented that he "had objective significant muscle weakness in the arms and the legs." Tr. at 18. Dr. Simpson noted that during Petitioner's early hospitalization, his examiners "repeatedly documented" muscular weakness in his arms and legs, which "evolved over the first several days." *Id.* Petitioner was "very quickly diagnosed with [GBS]" and began five treatments of PLEX for GBS. *Id*. at 18-19. He "respond[ed] well to the treatment with improvement in his neurological function." *Id*. at 19. Additionally, CSF analysis revealed that Petitioner had "markedly elevated protein" and a "very low white blood cell count." *Id*. There was a "virtual consensus very soon" among the treating physicians that the proper diagnosis was GBS, and myopathy was "quickly disregarded" as a diagnosis. *Id*. at 19-20.

Dr. Simpson opined that Petitioner's GBS was "causally related" to the HPV and meningococcal vaccines he received on July 3, 2017, with an approximate onset of neurological symptoms four weeks later. Tr. at 22. GBS is an autoimmune disorder involving the "body's immune system self-reacting against its own organ systems and its own tissues." *Id.* at 23. Although it is not always known what triggers autoimmunity, it is "well represented" in the medical literature that there are numerous factors that "could contribute as triggers to autoimmunity," including hereditary or genetic predispositions, infections, or vaccinations. *Id*. The "two best examples of triggers" for GBS are (1) *C. jejuni*, a gastrointestinal illness, and (2) the 1976 swine flu vaccine; molecular mimicry is the mechanism that triggers autoimmunity in both cases. *Id*. 24-26. Molecular mimicry is a theory that "there are antigens that are present on an exogenous agent that cross-react with innate human antigens on tissue." *Id*. at 26.

With respect to homology between components of the HPV vaccine and host tissue, Dr. Simpson cited the Kanduc and Phelan studies, which identified approximately 82 cross-reactive epitopes on the viral protein. Tr. at 27; *see* Darja Kanduc, *Quantifying the possible cross-reactivity risk of an HPV16 vaccine*, 8 J. OF EXPERIMENTAL THERAPEUTICS & ONCOLOGY 65 (2009) (Ex. 32) ("Kanduc"); Jody Phelan et al., *A potential antigenic mimicry between viral and human proteins linking Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) with autoimmunity: The case of HPV immunization*, 19 AUTOIMMUNITY REVIEWS 102487 (2020) (Ex. 33) ("Phelan"). One of the identified epitopes was found to cause decreased nerve conduction in animals. *Id.* at 27-28.

Regarding the clinical evidence of a possible causal relationship between the HPV vaccine and GBS, Dr. Simpson offered two studies: Brinth and Miranda. Tr. at 28-29. In Brinth, the authors found an association between the HPV vaccine and "a wide range of complications, including neurologic complications," where "a fair percentage" of the cases reported muscle weakness as a primary symptom. *Id*. at 28. Dr. Simpson opined that the Brinth study provided "some basis for the potential neurologic complications relating to HPV." *Id*. at 28-29. In Miranda, a French study, the authors compared the risk of GBS in the HPV-vaccinated adolescent population with an unvaccinated adolescent population; "they found statistical evidence of an increased risk of [GBS] relating to HPV [vaccination]." *Id*. at 29.

With respect to the meningococcal vaccine, Dr. Simpson noted that case reports "at least raised the concern" of a causal connection between the vaccine and GBS. Tr. at 29. The CDC, in a pair of reports from 2005 and 2006, reported 17 cases of GBS in patients "within a short time frame" after receiving the meningococcal vaccine and concluded that "there was an excess risk of [GBS] related to Menactra [meningococcal] vaccination." *Id*. at 29-30.

Addressing the appropriate timing for the onset of GBS following vaccination, Dr. Simpson noted that "different analyses report different conclusions." Tr. at 30. For example, the Schonberger article discussed a 10-week risk period following flu vaccination, the IOM recognized a six-week risk period, and the Vaccine Table set forth an onset period of 3-42 days after flu vaccination. *Id*. at 31. Dr. Simpson opined that Petitioner's onset of GBS was "approximately 31 to 32 days" after his vaccinations and "well within" the Table's risk period. *Id*.

Dr. Simpson testified that there "were no alternative causes" for Petitioner's GBS. Tr. at 32. He considered "any antecedent events that may act as triggers" for GBS. *Id*. Infection and vaccination are the "two most important" possible triggers for GBS, but Petitioner did not have an underlying infection that might have increased his risk of GBS. *Id*. at 32-33. Instead, "the only antecedent trigger that was present in [Petitioner's] case was the vaccination series one month earlier." *Id*. at 33. Additionally, Petitioner's treating physicians were "very confident" in their GBS diagnosis because they initiated PLEX treatment "very quickly," on the first day of admission and before diagnostic test results were available. *Id*. at 33-34.

On cross, Dr. Simpson opined that he did not believe MRI imaging or electrodiagnostic studies were essential to diagnose GBS in Petitioner. Tr. at 41-42. Furthermore, increased or normal reflexes would not rule out GBS on a differential diagnosis. *Id*. at 46-47. The fact that Petitioner recovered quickly did not suggest he merely experienced a placebo effect to PLEX therapy and did not have GBS. *Id*. at 57-58. Overall, although there is no "universally accepted set of criteria" for GBS, Dr. Simpson incorporated the medical literature and his own clinical experience to "establish a degree of confidence" that Petitioner had GBS. *Id*. at 65-67.

On redirect, Dr. Simpson was shown and read into the record several excerpts from the medical literature regarding the criteria for diagnosing GBS. Tr. at 69-75.[10] He explained that Petitioner's "clinical presentation with this rapid onset of evolving weakness . . . would make

[10] Dr. Simpson quoted Willison et al., which noted that "[GBS] is a remarkably clinically diverse disorder and includes several clinically distinctive variants, forms, frustes, and atypical cases." Tr. at 70 (quoting Willison at 722). Also, "[m]ost patients have or develop[] reduced tendon reflexes in the affected limbs. Reflexes can initially be normal especially in pure motor and axonal forms of the disorder or in a few cases, even be hyper-reflexic." *Id*. at 71 (quoting Willison et al. at 721). Unver et al. noted that "[a]lthough hypo- or areflexia is necessary for clinical diagnosis of GBS, preserved deep tendon reflexes do not exclude the diagnosis of [GBS]. The electrophysiological studies play a very important role in differentiating it from other causes." *Id*. at 71-72 (quoting Unver et al. at S62). Papathanasiou & Markakis similarly reported that "[c]urrent data suggest that 5% of patients with AIDP and 20% of patients with AMAN have preserved or exaggerated DTRs." *Id*. at 72-73 (quoting Papathanasiou & Markakis at 2). And Yuki et al. stated: "The presence of hyperreflexia in an appropriate clinical and electrophysiological context should not induce delay in treatment." *Id*. at 75 (quoting Yuki 2010 at 1189).

myopathy extremely unlikely" as a diagnosis. *Id*. at 77. He noted that Petitioner's clinical presentation and "consensus diagnosis from all his treating physicians was [GBS] with no alternatives plausible." *Id*. at 32.

2. Dr. Tompkins's Testimony

Dr. Tompkins was qualified as an expert in immunology and molecular pathogenesis. Tr. at 91. He first explained the difference between the 1976 swine flu vaccine and the current flu vaccine. *Id*. at 91-94. Current vaccines no longer use the whole virus, as was previously done; rather, they use "split" virus, resulting in much a purer product. *Id*. at 93.

As for the components of the HPV and meningococcal vaccines, Dr. Tompkins explained that the HPV vaccine is a recombinant vaccine that is made from the cloned "L" protein of human papillomavirus and expressed in yeast. Tr. at 94. The highly purified viral particles are then adsorbed to alum adjuvant and mixed to "however many valencies that they have." *Id*. Although it is "not truly synthetic," the process is "very controlled." *Id.* The meningococcal vaccine is a polysaccharide vaccine made by purifying the outside of the bacterium and attaching to the diphtheria toxoid. *Id*. The result is "a good immune response to the meningococcal outer layer." *Id*. at 95. The meningococcal vaccine does not include an adjuvant. *Id.*

Dr. Tompkins explained that the molecular mimicry theory hypothesizes that something from outside the cell, such as a pathogen, has enough identity with "self" that if an immune response is elicited from the pathogen, "there's a potential for a cross-reactive immune response where T-cells or antibodies to that pathogen might now recognize and damage self because it thinks it's not self." Tr. at 95-96. The theory does not suppose "simply a linear stretch of amino acids. It is identity that an antibody or a T-cell would recognize from a pathogen." *Id*. at 96. Dr. Tompkins described proving molecular mimicry as "a multistep process" in which there would need to be "that same identity" from the pathogen inside the self, and the autoimmune response would need to be detected. *Id*. at 96-97.

Regarding Dr. Simpson's specific molecular mimicry theory, Dr. Tompkins noted that the HPV and meningococcal vaccines are different from the *C. jejuni* infection and 1976 swine flu vaccine examples cited by Dr. Simpson. Tr. at 99. Dr. Simpson did not provide "any description of identity for HPV or the MCD-4 vaccine with a self-antigen or a specific antigen that might be associated with autoimmune disease." *Id*. By contrast, for the 1976 swine flu vaccine and GBS, there are data around potential targets as well as antibodies that can elicit autoimmune disease. *Id*. This is also the case for *C. jejuni* infection. *Id.* Furthermore, infection and vaccination are "too different" to reliably compare. *Id*. at 99-100. Dr. Tompkins opined that the criteria for establishing molecular mimicry between the subject vaccines and GBS were not satisfied. *Id*. at 101.

Next, Dr. Tompkins addressed Dr. Simpson's second theory of causation: "neurotoxic effect." Tr. at 101-02. Dr. Tompkins explained that such an effect is generally described as "some sort of contaminant;" i.e., "something that is damaging the cell because of what it is." *Id*. at 102. Moreover, a neurotoxic effect does not involve an immune response, other than the inflammation that is secondary to the toxicity. *Id*. He opined that there is no indication that the subject vaccines are neurotoxic. *Id*.

Dr. Tompkins then addressed Dr. Simpson's "immune complex formation" theory of causation. Tr. at 103. He explained that immune complex formation involves antibodies circulating throughout the host that can penetrate tissues. *Id*. An antibody will form an "immune complex" if it binds to a multi-complex protein, virus, or bacterium. *Id*. Immune complexes can do "a variety of things," including trigger inflammation, accumulate and deposit at different sites within the body, and cause kidney issues. *Id*. Dr. Tompkins opined that there is "no evidence that immune complexes are involved in GBS" or that molecular mimicry causes these complex formations. *Id*. at 104.

Dr. Tompkins then addressed Dr. Simpson's last theory of causation, loss of self-tolerance. Tr. at 104. He testified that "the whole goal of the immune system is to differentiate self from nonself, and if it sees nonself, to respond." *Id*. Within the immune system there are "potentially autoreactive" B and T cells that circulate but "never do anything because we have lots of checks, which we call tolerance." *Id*. at 108. The immune system's tolerance can break down with infections and cause the autoreactive cells to "start damaging the self." *Id*. At the most basic level, loss of self-tolerance occurs when "a very high level of inflammation" causes such a breakdown. *Id*. Dr. Tompkins explained that loss of self-tolerance does not "actually have anything to do with molecular mimicry." *Id*. He opined that the steps that he outlined for loss of self-tolerance were not met in this case. *Id*. at 109.

Dr. Tompkins opined that there is no association between the HPV vaccine and GBS. Tr. at 109. He referenced the June 7-8, 2017 meeting of the WHO's Global Advisory Committee on Vaccine Safety ("GACVS"). *Id*.; *see* WHO. In response to the findings of the French Miranda study, the GACVS reviewed other studies that found no association between HPV vaccine and GBS. Tr. at 113. The GACVS stated: "Both the UK and US studies concluded, based on their respective data, that a risk of [less than one] case of GBS per million doses of vaccine could now be excluded." WHO at 399.

Regarding the appropriate timing of GBS following vaccination, Dr. Tompkins testified that he would consider the antibody response to the vaccine, as well as T cell responses, responses in the lymph nodes, and cytokine production. Tr. at 114-16. He opined that the timing of GBS onset was not shown to be appropriate in this case. *Id*. at 116-17.

Next, Dr. Tompkins further discussed "sequences" as related to the molecular mimicry theory. Tr. at 117. He explained that sequences can "be an epitope," which is "something that is recognized by the immune system . . . something that a T-cell would recognize," for example, a peptide, which is a string of amino acids. *Id*. The process itself, however, is "complex" and can be highly variable. *Id*. at 117-18. He opined that having a sequence of five, eight, or 20 amino acids that are identical between a myelin protein and the "L-1 of the [HPV] does not in any way suggest that the peptide from the [HPV] is going to activate a cell in a host" and cause a reaction. *Id*. at 118. Additionally, there are "post-translational modifications" that can change the specificity in a way that cannot necessarily be predicted. *Id*. at 118-19. He explained that "the fact that you can identify a reactive epitope that matches HPV and matches any protein in a human is not at all surprising." *Id*. at 119. Also, "having a string of five amino acids . . . is not necessarily indicative"

of cross-reactivity. *Id*. at 120. It is unsurprising that the Kanduc paper identified roughly 80 epitopes that were found in the human proteome. *Id*. at 121.

On cross, Dr. Tompkins agreed that homologous sequences are "fairly common" in nature and frequently cross-react. Tr. at 124. He agreed that genetic and host factors are important in the development of human disease, and that while the vaccines recommended by the CDC are generally safe, in "rare circumstances vaccination could cause autoimmune disease," including adverse neurological events. *Id.* at 124-25. He explained that to conclude at an "experimental level" that there is a causal relationship between a vaccine and an autoimmune condition, he would need to identify "T-cells or antibodies from an individual with the disease that are reactive to self-identity . . . and are associated with that disease." *Id*. at 125-27. At the population level, epidemiological studies that show an association would "connect[] the final piece to really show that causality." *Id*. at 127. Lastly, he clarified that the appropriate window of onset of GBS following vaccination would be from two to 42 days. *Id.* at 127-18.

3. Dr. Donofrio's Testimony

Dr. Donofrio was qualified as an expert in neurology. Tr. at 138. He opined that there was no relationship between the subject vaccinations and Petitioner's injury. *Id*. at 139.

Dr. Donofrio testified that Petitioner's medical history did not "necessarily" support GBS as a diagnosis, because his case did not meet the criteria for GBS set forth in Asbury & Cornblath. Tr. at 139-40. The most "critical" features required to diagnose GBS include "progressive motor weakness of more than one limb" and areflexia. *Id*. at 141. The weakness can range from minimal impairment to total paralysis in all four extremities; most patients experience weakness in all four extremities. *Id*. Also, universal areflexia is the rule, "though distal areflexia with definite hyperreflexia in the biceps, in the arms, and the knees will suffice if other features are consistent." *Id*. Another important feature in GBS is progression of weakness that "is usually symmetrical, and then mild sensory symptoms or signs." *Id*. at 141-42. Petitioner "did not have any sensory symptoms or signs" or cranial nerve involvement. *Id*. at 142.

Dr. Donofrio noted that Petitioner had weakness in the upper and lower extremities, but normal reflexes and even hyperreflexia in the lower extremities. Tr. at 143. He was "very much bothered" by the reflex findings; such symptoms would cause him to "wonder if the patient had a spinal cord process in the neck, in the cervical spine." *Id*.

Dr. Donofrio opined that, though an MRI would not be required to diagnose GBS "if there are strong features of the illness and laboratory testing that would support the diagnosis without an MRI," a nerve conduction study "would be critical in a patient with presumed [GBS] if we were not doing imaging studies[.]" Tr. at 142. Because Petitioner did not undergo electrodiagnostic testing, the diagnosis of AIDP, which requires particular electrodiagnostic findings, should not have been assigned. *Id.* at 145-46. Also, it was surprising that Petitioner improved after just one

day of PLEX therapy, leading Dr. Donofrio to further question the GBS diagnosis.[11] *Id.* at 149-50.

Regarding causation, Dr. Donofrio testified that the evidence did not substantiate the molecular mimicry theory advanced by Dr. Simpson or the general contention that the subject vaccines could cause GBS. Tr. at 155.

On cross, Dr. Donofrio agreed that most alternative diagnoses for Petitioner could be eliminated by considering his presentation. Tr. at 158-59. Petitioner did not have a sensory level, bowel or bladder dysfunction, or spasticity. *Id.* at 159. Furthermore, despite his improvement after PLEX, at the time of discharge on August 24, 2017, Petitioner still had "noticeable deficits in strength." *Id*. at 161. And one month later, on September 27, 2017, Petitioner still had diminished reflexes in the upper extremities but not his knees, along with mild weakness in the wrist and fingers but normal strength elsewhere. *Id*.; *see* Ex. 2 at 948-49.

Dr. Donofrio further agreed that an August 16, 2017 exam found that Petitioner had "lost reflexes" in the right triceps and brachial radialis and had hyperreflexia at the knee and ankle. Tr. at 162-63. Some "atypical" GBS patients have preserved reflexes, as discussed in several articles. *Id*. at 163 (referencing Unver et al.; Papathanasiou & Markakis; Yuki 2010).

Dr. Donofrio testified that he would accept a causal association between a vaccination and GBS "if there was strong epidemiological evidence" and appropriate timing of onset. Tr. at 163-64. He agreed that "rare events are not easily detected by epidemiologic evidence or studies." *Id*. at 165. The molecular mimicry theory was "biologically plausible." *Id.* at 166. The onset of Petitioner's symptoms about 31-33 days after vaccination was within the 42-day range identified in the literature. *Id.* at 167.

During examination by Special Master Oler, Dr. Donofrio explained that diagnosing myelopathy "depends on the localization." Tr. at 168.

> In the cervical spine, it would be weakness in four limbs. There may be spasticity after a while. It's often not there in the first few days, hyperreflexia, lower extremities, and usually the upper extremities. Bilateral Babinski responses when you scrape the bottom of the foot, and that would be the clinical diagnosis. And then the -- you know, the standard of care now would be an MRI scan looking for pathology in the neck.

Id. at 168-69. Petitioner did not have spasticity, but it may have been too early for it to develop, and not all patients develop spasticity. *Id*. at 169. Petitioner also did not have bilateral Babinski

[11] In response to questioning from Special Master Oler, Dr. Donofrio explained the difference between PLEX and IVIG treatments for GBS. Tr. at 169-70. Both treatments are "the same in terms of efficacy," but IVIG is "the standard of care" because it is easy to administer intravenously, whereas PLEX requires sophisticated equipment that many smaller hospitals do not have. *Id*. at 170.

responses. *Id*. Nonetheless, Dr. Donofrio was "as comfortable with the diagnosis [of myelopathy] as . . . with the other conditions in the differential diagnosis." *Id*. Overall, without a nerve conduction study showing either "an axon loss or demyelinating neuropathy," he could not diagnose GBS. *Id*.

IV. APPLICABLE LAW

A. Petitioner's Burden in Vaccine Program Cases

Under the Vaccine Act, a petitioner may prevail in one of two ways. First, he may show that he suffered a Table injury within the time provided in the Table. § 11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed in the Table, he may demonstrate that he suffered an "off-Table" injury that was caused-in-fact by his vaccination. § 11(c)(1)(C)(ii).

For both Table and non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. § 13(a)(1). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1322 n.24 (Fed. Cir. 2010); *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). The petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or the opinion of a competent physician. § 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health and Human Services*. 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires a petitioner to establish by preponderant evidence that the vaccination caused his injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278.

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

certain." *Id.* at 548-49; *Bunting v. Sec'y of Health & Hum. Servs.,* 931 F.2d 867, 873 (Fed. Cir. 1991).

A petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Despite their expertise, special masters are not empowered by statute to conclusively resolve what are complex scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. However, this does not negate or reduce a petitioner's ultimate burden to establish his entitlement to compensation by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs*., 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 (stating that "medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, the existence of medical records and/or statements of treating physician views does not require the special master to adopt their conclusions *per se*. § 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct–that it must be accepted in its entirety and cannot be rebutted."). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs*., 100 Fed. Cl. 742, 749 (2011) (it was not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Hum. Servs*., No. 06-522V 2011 WL 1935813, *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically acceptable temporal relationship." *Id.* Thus, a petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also be consistent with the theory for how the relevant vaccine can cause the alleged injury (*Althen* prong one's requirement). *Id.* at 1352;

Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff'd without op.,* 503 F. App'x 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs*., No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Analysis of Fact Evidence

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

Medical records created contemporaneously with the events they describe are generally trustworthy, because they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions," where "accuracy has an extra premium." *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1382 (Fed. Cir. 2021) (quoting *Cucuras*, 993 F.2d at 1528). Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *See generally Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475 at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony, especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, there are situations in which compelling oral testimony could be more persuasive than written medical 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*, 2005 WL 6117475, at *19 ("Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent.") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, the special master should assess each witness's credibility when determining the weight their

testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In deciding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony, a rational analysis must be explicated. *Burns*, 3 F.3d at 417.

C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* 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 play a slightly different role in Vaccine Program cases than they do in other federal judicial proceedings. Those factors are employed by judges to exclude evidence that is unreliable and potentially confusing to a jury. In Vaccine Program cases, these factors are generally used to assess the reliability and weight of scientific evidence. *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 persuasiveness of expert testimony already admitted[.]"). The flexible use of the *Daubert* factors to evaluate persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 743.

Respondent frequently offers one or more experts of his own to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting

Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997)). 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. Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis special masters must employ in Vaccine Program cases. *Id.* at 1325-26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[T]his court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. Consideration of Medical Literature

Finally, although this decision discusses some but not all the medical literature in detail, I have reviewed and considered all the medical records and literature submitted in this matter. *See Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Hum. Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'") (citation omitted), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

V. ANALYSIS

In this case, the parties dispute whether Mr. Rodriguez developed GBS, as well as whether the HPV and meningococcal vaccines he received on July 3 and/or August 4, 2017, caused his purported GBS. As discussed below, I conclude Petitioner has preponderantly proven both the GBS diagnosis and HPV vaccine causation.

A. Diagnosis

As a threshold matter, a petitioner must establish that he suffered the injury for which he seeks compensation. *Broekelschen*, 618 F.3d at 1346. "The function of a special master is not to 'diagnose' vaccine-related injuries, but instead to determine 'based on the record as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [petitioner]'s injury.'" *Andreu*, 569 F.3d at 1382 (quoting *Knudsen*, 35 F.3d at 549). "Although the Vaccine Act does not require absolute precision, it does require the petitioner to establish an injury – the Act specifically creates a claim for compensation for 'vaccine-related injury or death.'" *Stillwell v. Sec'y of Health & Hum. Servs*., 118 Fed. Cl. 47, 56 (2014) (quoting 42.U.S.C. § 300aa-11(c)). Accordingly, the Federal Circuit has concluded that it is "appropriate for the special master to first determine what injury, if any, [is] supported by the evidence presented in the record" before applying a causation analysis pursuant to *Althen*. *Lombardi v. Sec'y of Health & Hum. Servs*., 656 F.3d 1343, 1351-53 (Fed. Cir. 2011).

The medical records indicate that prior to the subject vaccinations, Petitioner had no contributory medical history. He received the HPV and meningococcal vaccines on July 3, 2017.

Ex. 2 at 12; Ex. 1 at 1. Thirty-two days later, on August 4, 2017, he received a second dose of HPV vaccine. Ex. 2 at 28; Ex. 1 at 1.

On or about August 4 or 5, 2017, Petitioner developed cramping in his calf muscles and fatigue. *Compare* Ex. 2 at 45 (reporting to his PCP on August 7, 2017, that he had been experiencing myalgias in his legs for three days – since August 4); *with* Affidavit at 2 (Petitioner averring that he developed cramping in his calves one day after the August 4 vaccination). By August 7, 2017, he reported extremity weakness, resulting in difficulty walking. Ex. 2 at 45. At that time, he was assessed with muscle spasms. *Id.* at 47.

Nine days later, on August 16, 2017, Petitioner reported having difficulty walking, needing assistance to stand up, and having fallen several times. Ex. 2 at 123. He also reported right hand weakness. *Id.* He exhibited a wobbly gait and poor balance on exam. *Id.* at 126. When he was seen by neurologist Dr. Devine on August 17, he exhibited normal reflexes in the biceps, triceps, brachioradialis, and ankles, along with hyperreflexia in the patellae, but he had abnormalities in walking. *Id.* at 185, 195. On examination by neurologist Dr. Chiang later that day, Petitioner had absent reflexes in the upper extremities, but normal reflexes in the lower extremities. *Id.* at 197. That same day, attending neurologist Dr. Okuda confirmed Petitioner had a "reduction in his overall strength" in all his extremities but that his ankle and patellar reflexes were preserved. *Id.* at 195. Petitioner's CSF results showed albuminocytologic dissociation, consistent with GBS. *Id.* Dr. Okuda diagnosed "GBS post immunization against HPV." *Id.*

Petitioner's GBS diagnosis was confirmed by other treating physicians, including several neurologists. *See* Ex. 2 at 186-87 (infusion specialist Dr. Burner noting the diagnosis of "suspected GBS" and ordering PLEX therapy); *id.* at 197 (neurologist Dr. Chiang recording an impression of GBS that arose after the second dose of HPV vaccine); *id.* at 946 (neurologist Dr. Husari noting a history of GBS and observing that Petitioner had returned to 85% of baseline by September 27, 2017). None of Petitioner's treating physicians diagnosed or strongly suspected another condition. He improved in response to PLEX therapy, a common GBS treatment. *Id.* at 884. At his follow-up with Dr. Husari in late September 2017, Petitioner continued to exhibit reduced reflexes in several locations and had an impaired ability to squat. *Id.* at 948. He also had reduced reflexes and impaired squatting at his February 2018 follow-up with Dr. Husari. *Id.* at 999-1000.

Despite the clear consensus among Petitioner's treating providers, as well as Dr. Simpson, Dr. Donofrio opined that he could not conclude Petitioner had GBS. First Donofrio Rep. at 7; Second Donofrio Rep. at 1; Tr. at 139-40. Dr. Donofrio was particularly troubled by the fact that Petitioner had exams showing normal or overactive reflexes instead of absent or decreased reflexes. Tr. at 143. Also, Petitioner did not have MRI imaging or electrodiagnostic testing, which could have ruled out other conditions like myelopathy. *Id.* at 142. Without electrodiagnostic testing, the assignation of the specific GBS subtype of "AIDP" as a diagnosis would technically be inaccurate. *Id.* at 145-46. Petitioner also lacked sensory symptoms or signs, did not have cranial nerve involvement, and recovered more quickly with PLEX therapy than one would expect in a GBS patient. *Id.* at 142, 150.

Importantly, Dr. Donofrio did not affirmatively rule out GBS in Petitioner; instead, he suspected Petitioner had myelopathy instead of GBS based on his clinical presentation. Tr. at 158-59. He agreed that most other alternative diagnoses could be ruled out. *Id.*

According to the Asbury & Cornblath paper submitted by both parties' experts, universal areflexia is the "rule" in classic GBS, though in some cases distal areflexia with hyporeflexia of the biceps and knee jerks "will suffice if other features are consistent." Asbury & Cornblath at S21. The other "required" feature of classic GBS is progressive motor weakness in more than one limb. *Id.* Other features are characterized as "strongly supportive" of a GBS diagnosis. *Id.* The supportive clinical symptoms are a nadir of weakness within four weeks, symmetry of symptoms, mild sensory symptoms or signs, cranial nerve involvement, recovery beginning two to four weeks after nadir, autonomic dysfunction, and absence of fever at the onset of symptoms. *Id.* at S21-S22. The "strongly" supportive CSF features are elevated CSF protein and counts of 10 or fewer mononuclear leukocytes per cubic millimeter. *Id.* at S22. Finally, about 80% of patients will have abnormal electrodiagnostic testing. *Id.*

Dr. Donofrio did not dispute that Petitioner developed weakness in more than one limb, but he observed that Petitioner had normal or hyperactive reflexes on some exams, particularly in his lower extremities. Tr. at 143. Dr. Simpson agreed this was unusual. *Id.* at 46. However, as noted, Petitioner did at times exhibit absent or decreased reflexes. Ex. 2 at 197. He continued to have reduced/absent reflexes in some anatomical locations about six months after he was discharged. *Id.* at 999. Furthermore, Dr. Simpson cited several articles describing cases of atypical GBS involving normal and/or hyperactive reflexes. *See* Unver at S61-S62 (reporting three cases of GBS involving preserved reflexes); Papathanasiou & Markakis at 1 (reporting three cases of GBS, two of which involved hyperreflexia and one of which involved preserved reflexes); Yuki 2010 at 1181 (study comparing GBS patients with preserved and/or hyperactive reflexes to patients with absent/reduced reflexes).

Regarding the other clinical supporting symptoms/signs, Petitioner did not have sensory symptoms or signs or cranial nerve involvement, but he did have a nadir of weakness within four weeks of onset, followed by recovery over the next month. *See* Ex. 2 at 883-84, 948-49. He did not have a fever at onset. *Id.* at 125. Significantly, his CSF results were consistent with GBS, as the treating physicians and Dr. Simpson concluded. *Id.* at 197; Tr. at 16.

As for Petitioner's elevated serum CK levels, Dr. Donofrio opined that "their elevation suggest[s] a muscle disorder or myopathy." First Donofrio Rep. at 6. However, Dr. Simpson testified that although elevated serum CK level are "potentially consistent with a muscle disease like myopathy . . . it was very quickly determined that" the CK levels were incidental, the treating physicians ruled out myopathy, and Petitioner was "treated for the consensus diagnosis [of GBS]." Tr. at 20.

It is also true that Petitioner did not have electrodiagnostic testing or MRI imaging to confirm his diagnosis. But none of the treating physicians doubted the diagnosis based on the lack of such testing. The Asbury & Cornblath criteria do not require such testing to confirm a GBS diagnosis.

As to Petitioner's recovery after initiating PLEX treatment, although he did experience some degree of improvement relatively quickly, he had not recovered completely even one month later. *See* Ex. 2 at 884, 948. Dr. Simpson was persuasive in arguing the overall clinical course was consistent with GBS.

Dr. Donofrio opined that Petitioner more likely had a myelopathy than GBS. However, Dr. Simpson pointed out that Petitioner did not have several features consistent with myelopathy, such as Babinski responses, sphincter involvement, spasticity, or a segmented sensory level. Second Simpson Rep. at 3; Tr. at 168-69. Thus, I am not persuaded Petitioner's case was more consistent with myelopathy than with GBS, particularly given the clear agreement among his treating neurologists and the medical literature filed regarding GBS presentation.

I therefore conclude that Petitioner has preponderantly established that he suffered from the injury alleged, GBS.

B. *Althen* Prong One

In the context of the Program, "to establish causation, the standard of proof is preponderance of evidence, not scientific certainty." *Langland v. Sec'y of Health & Hum. Serv.*, 109 Fed. Cl. 421, 441 (Fed. Cir. 2013). Petitioner's burden under *Althen*'s first prong is to provide a medical theory causally connecting the vaccination and the injury. *Id.* This theory, again, must be sound and reliable. *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019).

As addressed below, I conclude that Petitioner has provided a reliable medical theory for how the HPV vaccine can cause GBS. Given that conclusion, I do not need to resolve whether Petitioner has also satisfied *Althen* prong one as to the meningococcal vaccine.

1. Causation Theory

Dr. Simpson discussed several mechanistic theories for how the subject vaccines could cause GBS, but his testimony focused on the molecular mimicry theory of causation. *See* Tr. at 171. He commented that molecular mimicry is widely accepted as a mechanism for causing autoimmune disease, including GBS. First Simpson Rep. at 3. For example, he claimed molecular mimicry has been shown to cause GBS following *C. jejuni* infection, as well as after the 1976 swine flu vaccine. *Id.*; Tr. at 24-26. Specific to this case, he cited two studies finding numerous epitopes on the HPV viral protein, one of which was found to decrease nerve conduction in animals. Tr. at 27-28; *see* Kanduc, Phelan.

Dr. Tompkins did not dispute that molecular mimicry is the prevailing theory for GBS caused by *C. jejuni*, but he argued there is no demonstrated mechanism, including molecular mimicry, for GBS caused by the swine flu vaccine. Tompkins Rep. at 3-6. Moreover, the mimicry leading to *C. jejuni*-caused GBS has been demonstrated experimentally in animals, which is not the case with either of the subject vaccines; further, infection and vaccination are "too different" immunologically to reliably compare. *Id.* at 4; Tr. at 99-100. Also, Dr. Simpson failed to identify

any specific homology between either of the subject vaccines and host tissues involved in GBS. Tr. at 99.

I conclude that Petitioner has proffered a reliable causation theory for purposes of establishing *Althen* prong one in this case. The experts agreed that molecular mimicry is a recognized mechanism for the pathogenesis of GBS, including in the well-established example of *C. jejuni* infection. Furthermore, as discussed below, this record contains reliable epidemiologic data supporting a potential association between the HPV vaccine and GBS, lending credence to the molecular mimicry theory as applied here. *See Harris v. Sec'y of Health & Hum. Servs.*, No. 18-944V, 2023 WL 2583393, at *22 (Fed. Cl. Feb. 21, 2023) ("Because petitioners in this program are allowed to prove their cases circumstantially, and because experts in this program are permitted to engage in at least some extrapolation, the fact that GBS is well accepted as an autoimmune condition with a wide variety of suspected antigenic triggers, inclusive of antigens from both infection and vaccination, provides meaningful evidence supporting petitioner's burden of proof with respect to *Althen* prong one. That is, even before addressing any vaccine-specific evidence, this general understanding of GBS pathophysiology constitutes a reasonably strong starting premise for a claim that vaccines beyond the flu vaccine can be implicated as triggers of GBS.").

2. Epidemiologic Data

Although a petitioner does not need epidemiologic data to prevail on *Althen* prong one, such data can be considered and given weight by the special master. In *Tullio*, Special Master Moran explained at length why it is appropriate to give such data consideration in Vaccine Program cases. *Tullio v. Sec'y of Health & Hum. Servs.*, No. 15-51V, 2019 WL 7580149, at *6 (Fed. Cl. Spec. Mstr. Dec. 19, 2019). First, the Federal Circuit "has endorsed special masters weighing epidemiological studies that investigated whether a vaccination is associated with an increased incidence or worsening of a disease." *Id.* (discussing *W.C.*, 704 F.3d at 1361). Second, other legal authorities have considered such data in assessing related causation questions. *Id.* at *7. Third, scientists rely on epidemiologic data to make determinations about causation. *Id.* at *7-8. I agree that epidemiologic data can be important circumstantial evidence of a causal exposure-injury association and are appropriate to assess in a Vaccine Program case.

a. Miranda

Dr. Simpson submitted the Miranda study, which investigated a potential association between HPV vaccination and certain autoimmune diseases, including GBS, in a population of about 2.2 million girls between the ages of 13 and 16. Miranda at 4761. Using data from French national databases, the investigators prospectively followed girls vaccinated with HPV until either December 31, 2013, the date of one of 14 events of "interest" (including GBS), change in health insurance, or death, whichever occurred first. *Id.* The study found a "strong and robust association . . . between HPV vaccination and GBS, which was particularly marked in the first months following vaccination." Miranda at 4766. For example, in the first two months after HPV

vaccination, the adjusted hazard ratio for development of GBS was 5.35, a statistically significant finding. *Id.* at 4765 (Table 4). The authors explained:

> [T]he association between HPV vaccination and GBS was particularly marked in the first 2 months following vaccination and then tended to decrease for longer exposure windows, reaching non-significancy beyond 12 months after vaccination. Consistent results were obtained with an alternative approach using SCCS [self-control case series] method . . . This association did not differ with the type of HPV vaccine or whether or not GBS was preceded by a recent history of gastrointestinal or respiratory tract infection, and remained consistent when the analysis censored observations at the first of any other vaccination during the follow-up . . . Further adjustment for seasonality and calendar year yielded similar results: aHR of 3.94 [95% CI: 1.82–8.56] and 4.05 [95% CI: 1.86–8.80], respectively.
>
> Assuming a causal relationship, and based on our estimated aHR of 3.96, 15 of the 19 exposed cases of GBS in our study would be attributable to HPV vaccination, thus leading to an estimated attributable number of cases of 1.8 per 100,000 girls vaccinated (95% CI [1.1–2.0]).

Id. at 4764. The authors further commented that the significant association between HPV vaccination and GBS

> remained very robust across several sensitivity analyses and alternative SCCS design, notably when adjusting for calendar year or seasonality, or when considering the history of recent gastrointestinal or respiratory infections, which censoring when other vaccines occurred, or when limiting analysis to a period prior pandemics vaccination. This suggests that our results are unlikely to be explained by classical confounders in GBS, such as 2009-2010 A(H1N1) influenza virus or vaccination, seasonal variations and/or previous respiratory tract infections.

Id. at 4766.

The authors acknowledged that their study had limitations. Miranda at 4767. They were unable to validate GBS cases through medical record review, because the information they reviewed was anonymized. *Id.* Also, they limited their analysis to cases severe enough to warrant significant medical intervention like hospitalization, which could have led to an underestimation of the true GBS risk. *Id.* They noted that HPV-vaccinated girls had higher overall use of healthcare than unvaccinated girls, which could have artificially elevated the reported risk. *Id.* They reported attempting to adjust for these and other factors in their analysis. *Id.* They concluded: "An

increased risk of GBS after HPV vaccination is possible, but further studies are warranted to confirm this finding." *Id.*

Overall, I find Miranda to be persuasive evidence of a causal association between HPV vaccination and GBS. The study was large, controlled, and prospective. The investigators ran several sensitivity analyses and applied other study designs to confirm their finding of an HPV vaccine/GBS association. They attempted to adjust for the acknowledged limitations with the study, some of which they surmised could have led to an underestimation of the risk. While Dr. Donofrio criticized the study's use of billing codes instead of medical records to identify GBS cases, neither Dr. Donofrio nor Dr. Tompkins argued the study was invalid or that its findings should be discarded. *See* First Donofrio Rep. at 6-7.

b. Brinth

Brinth described 53 Danish patients who were referred to the Frederiksberg Hospital Syncope Unit and reported side effects after HPV vaccination. Brinth at 1. All the patients were under evaluation for autonomic nervous system dysfunction believed to be associated with the HPV vaccine. *Id.* The side effects described in the two months following vaccination included 35 reports of neuropathic pain; 30 reports of limb weakness; and 35 reports of motor weakness. *Id.* at 3. The authors characterized these symptoms as likely related to postural orthostatic tachycardia syndrome ("POTS"), not GBS. *Id.* None of the patients were diagnosed with GBS. Thus, the relevance of Brinth to this case is marginal.

c. NVIC

In his reports, Dr. Simpson cited the NVIC report, which compared VAERS reports following HPV vaccination to those following co-administration of HPV and meningococcal vaccines. NVIC at 1. The authors reported a statistically significant increased risk of reported adverse events, including a 1130% increased risk of reported GBS, following co-administration of HPV and meningococcal vaccines compared to vaccination with HPV alone. *Id.* The report also described a case of GBS following HPV, meningococcal, and varicella vaccination in an 18-year-old girl. *Id.* at 2. Because this analysis did not appear to be peer-reviewed and relied on VAERS reports, which are voluntarily provided and often unverifiable, it carries limited weight.

d. WHO

Dr. Tompkins pointed out that Miranda was the only study to find a statistically significant increased risk of GBS following HPV vaccination and prompted "several large safety studies including millions of doses of [the] HPV vaccine," which in turn "provide[d] compelling evidence that the HPV vaccine is safe and there is no epidemiological association with HPV vaccination and GBS." Tompkins Rep. at 6-7. He cited a 2017 WHO "safety update of HPV vaccines," which reported that studies done in response to Miranda's findings had found no association between HPV vaccination and GBS. WHO at 398-99.

e. Gee

Dr. Tompkins also cited Gee, which prospectively studied a population of about 2 million male and female HPV-vaccinated patients over the course of 10 years, using data from the Vaccine Safety Datalink ("VSD") in the U.S. Gee at 5757. The authors found no statistically significant association between HPV vaccination and onset of GBS within the 42 days thereafter. *Id.*

f. Conclusion

The studies referenced by Dr. Tompkins provide some counterevidence suggesting the lack of a causal association between HPV vaccination and GBS. But they do not supersede Miranda or render it unreliable evidence. Again, in the Vaccine Program, the petitioner need not produce *any* epidemiological data to meet *Althen* prong one, much less prove that the *weight* of epidemiology favors their causal claim.

In the recent case of *Farrell*, Special Master Moran considered a claim that a tetanus-diphtheria-acellular pertussis ("Tdap") vaccination caused neuromyelitis optica ("NMO"). *Farrell v. Sec'y of Health & Hum. Servs.*, No. 19-301V, 2025 WL 2409187, at *1 (Fed. Cl. Spec. Mstr. July 29, 2025). In addressing *Althen* prong one, the special master concluded the petitioner met his burden "largely due to a supporting epidemiologic study." *Id.* at *4. The study investigated the risk of a relapse of NMO within 30, 60, and 90 days of a vaccination. *Id.* It found a statistically significant increased risk of relapse in these timeframes in patients who were not on preventative immunotherapy. *Id.* Additionally, five patients in the study had new onset of NMO, including two who received either Tdap or tetanus-diphtheria ("Td") vaccines. *Id.* Special Master Moran stated: "The presence of an epidemiologic study finding that vaccinations are associated with an increased incidence of a disease is strong evidence favoring an award of compensation." *Id.* at *6 (citing *In re Swine Flu Immunization Prods. Liab. Litig.*, 508 F. Supp. 897, 907 (D. Colo. 1981) ("Where, as here, the exact organic cause of a disease cannot be scientifically isolated, epidemiologic data becomes highly persuasive"), *aff'd sub nom. Lima v. United States*, 708 F.2d 502 (10th Cir. 1983); *In re Agent Orange Prod. Liab. Litig.*, 611 F. Supp. 1223, 1239 (E.D.N.Y. 1985) (stating that in mass tort cases, "epidemiologic studies on causation assume a role of critical importance"), *aff'd sub nom. In re Agent Orange Prod. Liab. Litig.*, MDL No. 381, 818 F.2d 187 (2d Cir. 1987)).

Although Special Master Moran's decision is not binding, it is instructive here. The record before me includes epidemiological data supporting a potential causal association between the HPV vaccine and GBS, along with reliable evidence of the molecular mimicry theory of GBS causation. I conclude that this is adequate to meet *Althen* prong one here.

C. *Althen* Prong Two

Althen prong two requires proof of "a logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278. The sequence of cause and effect must be "'logical' and legally probable, not medically or scientifically certain." *Andreu*, 569 F.3d at 1380 (quoting *Knudsen*, 35 F.3d at 548-49). A petitioner is not required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition,

or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1325. Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second *Althen* prong. *Id.* Further, special masters are expected to consider the views of treating doctors. *Id.* at 1326. Such views are often persuasive because the doctors have direct experience with the patient whom they are diagnosing -- but they are not necessarily dispositive of the causation question. *See McCulloch v. Sec'y of Health & Hum. Servs.*, No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015).

There is no evidence suggesting Petitioner's pre-vaccination medical history is relevant. Petitioner received his first dose of the HPV vaccine, along with a second dose of the meningococcal vaccine, on July 3, 2017. Ex. 2 at 12; Ex. 1 at 1. On August 4, 2017, he received his second HPV dose. Ex. at 28; Ex. 1 at 1. On August 7, 2017, he reported three days of muscle cramps in his right lower leg, along with extremity weakness. Ex. 2 at 42, 45. He denied any injury or recent fevers. *Id.* at 45. At the time of his initial presentation, his PCP did not suspect his HPV vaccination was responsible for his symptoms. *Id*.

When Petitioner was first seen by neurologist Dr. Devine on August 17, 2017, he reported that his calf cramps began August 5, the day after his second HPV vaccine dose. Ex. 2 at 181. He reported no sick contacts or symptoms of infections in the previous three months. *Id.* Later the same day, when Dr. Okuda, the attending neurologist, examined Petitioner and reviewed his CSF findings, he assessed "GBS post immunization against HPV." *Id.* at 195. The infusion specialist, Dr. Burner, also noted that Petitioner had received the HPV vaccine on August 4, 2017, with symptoms beginning "within a day" after the vaccination. *Id.* at 186. None of the physicians identified any potential cause of Petitioner's GBS aside from the HPV vaccine. Neurologist Dr. Husari, who followed Petitioner after his discharge from the hospital, advised him to "avoid vaccination in the next few years." *Id.* at 1000-01.

Respondent argues that *Althen* prong two is not satisfied because Petitioner's claim is "based entirely on a '*post hoc ergo propter hoc*' line of reasoning;" that is, that Petitioner's symptoms started sometime after the subject vaccinations and were therefore caused by the vaccinations. ECF No. 72 ("Resp.'s Post-Hearing Brief") at 17 (emphasis in original). He maintains that, although the contemporaneous medical records referenced the subject vaccines, they were "based upon the history provided by [P]etitioner and his timing of events. As such, the treating physician statements were limited by the information that was provided to them by [P]etitioner himself, and a treating physician's opinion on vaccine causation is only as strong as its underlying basis." *Id.* at 16-17 (citing *Moberly*, 592 F.3d at 1323; *Perreira v. HHS*, 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994)). Notably, Respondent does not dispute the accuracy of what Petitioner reported to the treating physicians.

Based on this record, I conclude Petitioner has preponderantly satisfied *Althen* prong two. Although Petitioner's treating neurologists did not explicitly attribute his GBS to the HPV vaccine, Dr. Okuda characterized his condition as "post immunization." Other treating physicians consistently referenced the fact that Petitioner's symptoms followed his second HPV vaccination. There is no evidence in the record disputing Petitioner's contemporaneous reports concerning his vaccinations and the subsequent onset of his symptoms; thus, Respondent's suggestion that the

treating physicians based their views on incorrect information does not have merit. There was no evidence suggesting any other cause of Petitioner's GBS.

D. *Althen* Prong Three

Althen prong three contains two parts. First, a petitioner must establish the "timeframe for which it is medically acceptable to infer causation," and second, he must demonstrate that the onset of the disease occurred in this period. *Shapiro,* 101 Fed. Cl. at 542-43.

Petitioner received HPV and meningococcal vaccinations on July 3, 2017, and he received a second dose of HPV on August 4, 2017. Ex. 2 at 12, 28; Ex. 1 at 1. He reported developing muscle cramping on August 4 or 5, 2017. *Compare* Ex. 2 at 45 (reporting to his PCP on August 7, 2017, that he had been experiencing myalgias in his legs for three days – since August 4); *with* Affidavit at 2 (Petitioner averring that he developed cramping in his calves one day after the August 4 vaccination). Thus, Petitioner developed an initial symptom indicative of GBS about 32-33 days after the first dose of HPV, and either the day of or one day after the second dose.

Dr. Donofrio contended that the onset of Petitioner's GBS was inappropriate because it occurred only one day after his *second* HPV vaccination, outside the 3-42-day timeframe prescribed by the Vaccine Injury Table for GBS following flu vaccination. First Donofrio Rep. at 6. But when asked during cross examination if he agreed "that the onset of Petitioner's symptoms approximately 31 to 33 days following vaccination . . . is an appropriate time frame," he testified that "[i]t is within the 42-day range." Tr. at 167. Respondent's immunologist, Dr. Tompkins, also admitted that "the timing of the *primary* vaccinations and onset of symptoms [fell] within an accepted window of increased risk." Tompkins Rep. at 8 (emphasis added). Dr. Simpson likewise measured onset from the time of the first HPV vaccination and concluded that the onset of symptoms about one month later was appropriate. Tr. at 31.

I agree that onset in this case should be measured from the date of the first HPV dose (July 3, 2017), as any immune response triggered by the HPV vaccine likely started at the time Petitioner first received it. The experts ultimately appeared to agree that the onset of Petitioner's symptoms fell within a medically acceptable timeframe after that dose. I therefore conclude Petitioner has preponderantly proven *Althen* prong three.

VI. CONCLUSION

Upon careful evaluation of all the evidence submitted in this matter, including the medical records, the testimony, the experts' opinions, and the medical literature, I conclude that Petitioner has shown by preponderant evidence that he is entitled to compensation under the Vaccine Act. **His petition is therefore GRANTED.** An order regarding damages will issue shortly.

IT IS SO ORDERED.

s/ Jennifer A. Shah
Jennifer A. Shah
Special Master