# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

Filed: March 25, 2026

| | | |
|---|---|---|
| GUSTAVO VEGA, | * | |
| Petitioner, | * | No. 21-1794V |
| v. | * | Special Master Young |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |

*Laura Levenberg*, Muller Brazil, Dresher, PA, for Petitioner.
*Debra A. Filteau Begley*, United States Department of Justice, Washington, DC, for Respondent.

## DECISION ON ENTITLEMENT[1]

On September 2, 2021, Gustavo Vega ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program").[2] Pet., ECF No. 1. Petitioner alleged that he suffered from "Guillain-Barré Syndrome ("GBS") an off-table cause-in-fact injury, resulting from adverse effects of the human papillomavirus ("HPV") vaccination" he received on January 14, 2020. *Id.*

A careful analysis and weighing of all the evidence and testimony presented in this case in accordance with the applicable legal standards[3] reveals that Petitioner has failed to provide

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

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

preponderant evidence that the HPV vaccine he received on January 14, 2020, caused him to develop GBS. Accordingly, Petitioner is not entitled to an award of compensation.

## I. Procedural History

Petitioner filed his petition, a declaration, and medical records on September 2, 2021. Pet.; Pet'r's Exs. 1–7, ECF No. 1. Petitioner filed additional medical records on October 15, 2021, and June 16, 2022. Pet'r's Ex. 9, ECF No. 13; Pet'r's Exs. 10–11, ECF No. 20. On January 9, 2023, Respondent filed his Rule 4(c) report arguing against compensation. ECF No. 24.

On April 3, 2023, Petitioner filed an expert report from Joseph Jeret, M.D. Pet'r's Ex. 12, ECF No. 27. Petitioner filed additional medical records on April 18, 2023, and May 16, 2023. Pet'r's Ex. 27, ECF No. 34; Pet'r's Ex. 68, ECF No. 36. On April 20, 2023, Petitioner filed an expert report from Omid Akbari, Ph.D. Pet'r's Ex. 28, ECF No. 35. Respondent filed an expert report from Andrew MacGinnitie, M.D., Ph.D., on August 28, 2023, and an expert report from Matthew Brier, M.D., Ph.D., on October 6, 2023. Resp't's Ex. A, ECF No. 39; Resp't's Ex. C, ECF No. 41. On December 15, 2023, Petitioner filed supplemental reports from Dr. Jeret and Dr. Akbari. Pet'r's Ex. 69–70, ECF No. 42. On March 26, 2024, Respondent filed supplemental reports from Dr. Brier and Dr. MacGinnitie. Resp't's Exs. E–F, ECF No. 46. Petitioner filed a second supplemental report from Dr. Jeret on April 9, 2024. Pet'r's Ex. 86, ECF No. 48. That same day, Petitioner filed a status report indicating he did not intend to file additional expert reports. ECF No. 49.

The parties agreed to resolve entitlement with a ruling on the record. Informal Comm., docketed Apr. 25, 2024. On June 24, 2024, Petitioner filed a motion for a ruling on the record. Pet'r's Mot., ECF No. 51. Respondent filed a response on September 19, 2024, and Petitioner filed a reply on October 21, 2024. Resp't's Response, ECF No. 54; Pet'r's Reply, ECF No. 55.

This matter is now ripe for consideration.

## II. Factual Background

### A. Medical History

Petitioner's pre-vaccination medical history included two prior craniotomies for excision of an acoustic neuroma that left him with mild left-sided facial paralysis and left-sided hearing loss and tinnitus. *See* Pet'r's Ex. 3 at 49. On December 11, 2019, Petitioner saw his primary care provider ("PCP") where he received his first HPV vaccine but declined a flu vaccine. Pet'r's Ex. 2 at 42, 44.

On January 14, 2020, Petitioner returned to his PCP for possible side-effects of flushing and back pain since starting a new medication.[4] Pet'r's Ex. 2 at 10–11. The medication was

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

[4] The medication is not relevant to the issue here.

discontinued. *Id.* At this visit, Petitioner received his second HPV vaccine, at issue here, and he again declined a flu vaccine. *Id.* at 11.

Eleven days later, on January 25, 2020, Petitioner presented to the emergency department ("ED") for the inability to bear weight on both his legs and "trouble gripping with both hands" since 7:00am the day before. Pet'r's Ex. 3 at 58. He reported that he first noticed his symptoms when attempting to squeeze a tube of toothpaste; he also had difficulty typing on a keyboard. *Id.* at 59. That morning, he woke up with his legs feeling "heavy" and weak. *Id.* The problem was described as "gradually worsening." *Id.* Petitioner further reported a recent upper respiratory tract infection. *Id.* at 62. Neurological examination was positive for weakness. *Id.* at 60. He was "[u]nable to hold the 'okay' sign" and "unable to rapidly tap fingers." *Id.* at 61. He also had decreased grip strength and ankle flexion.[5] *Id.* at 61–62. GBS was suspected, and Petitioner was admitted for further evaluation and treatment. *Id.* at 51, 62.

Following his January 26, 2020 admission, Petitioner was evaluated by neurologists Kristie Bauman and Valeriya Levitan. Pet'r's Ex. 3 at 77. His documented history included a recent HPV vaccination and a cold in December, although a separate note from Dr. Levitan stated that Petitioner had "no preceding illness." *Id.* at 77–78. On examination, Petitioner demonstrated weakness in his upper and lower extremities, absent reflexes at his biceps and triceps, and reduced reflexes in his legs. *Id.* at 79. His sensation was largely intact except for a small area of numbness on his left foot, and "mildly decreased" vibration sense in his bilateral toes. *Id.* His gait was abnormal. *Id.* Drs. Bauman and Levitan concluded that Petitioner's symptoms were most consistent with GBS, although his "gait instability [was] out of proportion to his weakness and reported sensory loss." *Id.* at 80. Ganglioside antibody and other testing was ordered, and a five-day course of intravenous immunoglobulin ("IVIG") was started. *Id.* at 81.

A lumbar puncture on January 29, 2020, revealed elevated protein of 49 mg/dl (normal 15-40 mf/dL), and ganglioside antibody testing revealed elevated GM1 and GD1a. Pet'r's Ex. 3 at 153, 155, 172. These results confirmed a diagnosis of GBS. *Id.* On January 30, 2020, following five IVIG treatments, Petitioner was discharged to inpatient rehabilitation. *Id.* at 51–57, 110. Petitioner's weakness plateaued while hospitalized but remained severe. *Id.* at 53. He struggled to walk and complained of pain in his legs and lower back. *Id.*

Petitioner remained at the rehabilitation facility for 48 days, until March 20, 2020. Pet'r's Ex. 11 at 7–15. At discharge, he could walk with a rolling walker and while wearing bilateral ankle-foot orthoses ("AFOs"), but he still required assistance with stairs and many activities of daily living. *Id.* at 10. Outpatient physical therapy ("PT") and occupational therapy ("OT") were recommended. *Id.* at 10, 297. Outpatient psychiatric therapy for significant anxiety and depression was also recommended. *Id.* at 323–34.

After discharge from the inpatient rehab facility on April 17, 2020, Petitioner presented to neurologist Dr. Elina Zakin. Pet'r's Ex. 4 at 1. His history of present illness stated Petitioner had a "recent admission for acute onset weakness of distal > proximal LE > UE in Jan[uary] 2020, diagnosed with [acute inflammatory demyelinating polyneuropathy ("AIDP")] (in the setting of

[5] Petitioner also exhibited a left-sided facial droop, which was his "baseline" caused by prior removal of an acoustic neuroma. Pet'r's Ex. 3 at 58, 61.

recent HPV vaccination) who presents to establish care for AIDP." *Id.* He reported that prior to vaccination, he was healthy and running. *Id.* at 2. Two days after the hepatitis and HPV vaccinations, he "started to experience sudden onset weakness of finger flexors." *Id.* At this visit Petitioner still had complaints of weakness and noted increased drooling with the onset of GBS symptoms. *Id.* at 2–3. Dr. Zakin explained that Petitioner likely had the acute motor axonal neuropathy ("AMAN") variant of GBS, given his predominant motor weakness in his distal muscles with no sensory abnormalities. *Id.* at 4. Dr. Zakin recommended an electromyogram/nerve conduction study ("EMG/NCS"), and told Petitioner to continue his home exercise program. *Id.*

On May 7, 2020, Petitioner presented to NYU Medical Center for an initial outpatient PT evaluation. Pet'r's Ex. 4 at 29. Petitioner subsequently completed 50 physical therapy sessions through April 1, 2021. *See id.* at 29–116; Pet. Ex. 3 at 389–736.

On June 26, 2020, Petitioner presented to Dr. Zakin for a follow-up visit. Petitioner complained of continued numbness and tingling in his hands and feet. Pet'r's Ex. 4 at 117. Petitioner underwent an EMG, which showed "changes consistent with an acquired motor axonopathy or neuronopathy predominantly involving the lower extremities, with sparing of sensory nerve involvement." *Id.* at 121. The EMG also showed evidence of ongoing severe axonal denervation. *Id.*

Petitioner returned to Dr. Zakin on September 2, 2020. Pet'r's Ex. 4 at 189. He was still taking Gabapentin for his pain. *Id.* He had undergone an EMG of his face in August 2020, and it revealed a chronic partial and inactive left facial neuropathy. *Id.* at 193. Dr. Zakin further explained that Petitioner had "residual left facial palsy" from his prior brain surgeries. *Id.* Dr. Zakin noted Petitioner was improving, more stable, and his gait normal, although he continued to rely on a walker. *Id.* Continued PT was recommended. *Id.*

During a follow-up virtual visit with Dr. Zakin on October 15, 2020, Dr. Zakin discussed with Petitioner his "very slow course of improvement," which was "expected in motor variants of GBS." Pet'r's Ex. 4 at 249. Dr. Zakin ordered monthly IVIG treatments for three months. *Id.* Petitioner underwent IVIG infusions in December 2020, January 2021, and February 2021. *Id.* at 361, 398, 429. On February 22, 2021, Petitioner reported a slight improvement in his balance when he spoke with Dr. Zakin. *Id.* at 429. Dr. Zakin recommended additional IVIG over the next four months and continued adherence to home exercises. *Id.* at 434, 459.

On July 12, 2021, Petitioner had a televisit with Dr. Zakin. Pet'r's Ex. 8 at 509. He reported continued improvement and that he kept up with PT during the Spring of 2021. *Id.* He still used AFOs and a rolling walker, and he reported severe foot pain after walking for more than one hour. *Id.* Dr. Zakin noted improved balance and recommended continued aggressive performance of his home exercises. *Id.* at 512–13.

Petitioner saw Dr. Zakin on January 27, 2022, and he asked a number of questions about his prognosis. Pet'r's Ex. 68 at 12. He felt his strength was stable, that his ankle and foot strength had improved, but that he sometimes dropped objects unexpectedly. *Id.* He also reported episodic blurred vision, intermittent palpitations and shortness of breath, insomnia, right knee pain, and he wanted to discuss whether he should return to his prior "high stress" job. *Id.* Petitioner expressed

concern over getting the COVID vaccine for "heightened auto-immunity." *Id.* He also stated that he was still taking gabapentin at night for neuropathic pain. *Id.* Examination documented mildly reduced strength in his bilateral lower extremities and a tremor in his upper extremities. *Id.* at 15. Petitioner still demonstrated left-sided facial weakness. *Id.* His reflexes were normal throughout, and sensory testing was normal, except for a "small patch of numbness to all modalities at medial ankle on the left." *Id.* He could rise spontaneously from a chair, and while his gait was improved, it was wide-based and he held onto a walker. *Id.* at 16. Dr. Zakin explained that many of his questions concerned symptoms that were not neurologic, and she referred him for an eye examination, recommended he discuss anxiety with his counselor, and ordered a brace for Petitioner's knee. *Id.* at 17–18. She stated that Petitioner was improving, and ordered additional PT. *Id.*

## III. Experts

### A. Expert Qualifications

#### 1. Petitioner's Expert, Dr. Omid Akbari, Ph.D.

Dr. Akbari is an immunologist. Pet'r's Ex. 28 at 1. He received his Ph.D. in cellular and molecular immunology from the National Institute of Medical Research in London, United Kingdom, and completed a postdoctoral fellowship at Stanford University. Pet'r's Ex. 29 at 1. He is currently a Professor of Immunology at Keck School of Medicine, University of Southern California. Pet'r's Ex. 28 at 1. His research is "focused on the role of immune tolerance and how immune cells induce autoimmune and allergic diseases." *Id.* In his laboratory "there are multiple research studies which are relevant to the issue of understanding the medical theories involved in regard to how a vaccination can result in an appropriate or dysregulated immune responses causing inflammation." *Id.* "The known mechanisms of pathogenesis involved in these injuries involve the same mechanisms and cellular pathways that are investigated in [his] laboratory." *Id.* Dr. Akbari serves as an editor and reviewer for several journals and has numerous publications. *Id.*; Pet'r's Ex. 29 at 5, 9–16.

#### 2. Petitioner's Expert, Dr. Joseph Jeret, M.D.

Dr. Jeret is a board-certified neurologist. Pet'r's Ex. 12 at 1. He received his M.D. from SUNY Downstate Medical Center where he subsequently completed a neurology residency and a fellowship in clinical neurophysiology. *Id.*; Pet'r's Ex. 13 at 1. He is employed by Optumas as a physician in the neurology department and on staff at South Nassau Community Hospital and Mercy Medical Center. Pet'r's Ex. 12 at 1. He also maintains a neurology practice. *Id.* He "routinely care[s] for patients with varied neurologic illnesses, including spine disease and nerve disease. It is not uncommon for [his] patients to have complex clinical presentations and multiple diagnoses. Specifically, [he has] diagnosed patients with GBS, CIDP, and other peripheral nerve disorders." *Id.* Dr. Jeret has extensive publications in neurology. Pet'r's Ex. 13 at 2–7.

### 3. Respondent's Expert, Andrew MacGinnitie, M.D., Ph.D.

Dr. MacGinnitie is board certified in allergy/immunology and pediatrics. Resp't's Ex. A at 2. He received his Ph.D. in pathology and his M.D. from the University of Chicago Pritzker School of Medicine. *Id.* at 1. Thereafter, he completed a pediatrics residency and an allergy/immunology fellowship at Boston Children's Hospital. *Id.* at 1–2. He is currently an Attending Physician as well as the Clinical Chief for the Division of Immunology at Boston Children's Hospital. *Id.* at 1. He does "not routinely care for patients with GBS except when they have concurrent allergic and/or immunologic issues." *Id.* at 2. Dr. MacGinnitie has published numerous articles. Resp't's Ex. B at 12–18.

### 4. Respondent's Expert, Matthew Brier, M.D., Ph.D.

Dr. Brier is a board-certified neurologist. Resp't's Ex. D at 3. He received his Ph.D. in neuroscience and his M.D. from Washington University in St. Louis. *Id.* at 1; Resp't's Ex. C at 1. Thereafter, he completed a fellowship in multiple sclerosis and neuroimmunology as well as a residency at Washington University. Resp't's Ex. C at 1. He is currently an Assistant Professor of Neurology and Radiology at Washington University and an Attending Neurologist at Barnes Jewish Hospital. *Id.* He cares for patients with immunologic and autoimmune disorders of the nervous system as well as patients with general neurology conditions, including GBS "and its mimics." *Id.* He has published numerous articles. Resp't's Ex. D at 6–12.

## B. Expert Reports

### 1. Dr. Akbari's First Report[6]

Dr. Akbari spent much of his first report overviewing the immune system. Pet'r's Ex. 28 at 7–12. He summarized:

> [T]he main functions of the immune system are to recognize and subsequently eliminate foreign antigens, to induce immunologic memory, and to develop tolerance to self-antigens. Effective immunologic homeostasis relies on a continual balance among several factors, including T helper cell activation and suppression by regulatory T cells. Innate like lymphocytes are fast acting immune cells that may react to foreign stimuli such as vaccines. When homeostasis is disrupted and the

[6] Dr. Akbari spent much of his first report discussing vaccines not relevant to this petition. *See, e.g.*, Ex. 28 at 5 ("My research into your particular question concluded that the theory of molecular mimicry supports that Tdap vaccination can plausibly cause an adverse immune cross reaction and result in the onset of GBS."), 7 ("Tdap . . . causes a local inflammatory reaction."), 16 ("A second criterion for implicating molecular mimicry. . . is to demonstrate the plausibility of cross-reactivity by autoreactive T-cells or autoantibodies with a microbial antigen, or in this case to a component of the Tdap vaccine."), 18 ("[T]he role of Treg/Teff after immunization with flu and also the role of Tregs in controlling unwanted immune response are well established."), 20 (discussing B and T cell responses to tetanus toxoid), 20-21 (referring to "the potential for crossreactivity from tetanus toxoid to self-peptides in some people"). Because Dr. Akbari did not compare the Tdap and flu vaccines to the HPV vaccine, I will not elaborate on these opinions further.

> immune system responds in favor of activation, as it would when you stimulate it with a vaccine, the host becomes susceptible to autoimmunity. In GBS, research indicates that this balance is being disrupted leaving the host susceptible to an adverse autoimmune reaction upon stimulation of the immune system from infection or vaccination, mediated by activation of inflammasome.

*Id.* at 10.

Dr. Akbari proposed the theory of molecular mimicry to explain how the HPV vaccine can cause GBS. Pet'r's Ex. 28 at 5, 12–15. He explained molecular mimicry is "the mechanism by which an immune stimulated response by infection or other method, i.e. vaccination, can trigger cross-reactive antibodies or T cells that cause the symptoms of autoimmune disease." *Id.* at 6.

"One important criterion that can implicate molecular mimicry as a causal mechanism is the identification of T cells or antibodies in patients with the disease." Pet'r's Ex. 28 at 15. "Current research indicates that autoimmunity in GBS is most likely mediated by antibodies directed against myelin antigens, along with autoreactive T cells and macrophages that invade the myelin sheath, axonal membranes, and/or the nodes of Ranvier." *Id.* According to Dr. Akbari, GBS patients "have increased levels of pathogenic autoreactive T cells and antibodies present in the peripheral blood and cerebral spinal fluid [("CSF")]." *Id.* "Antibodies against gangliosides have been found in the serum of GBS patients during the acute phase of the disease." *Id.* (citing Pet'r's Ex. 54;[7] Pet'r's Ex. 55).[8] "Anti-glycan antibodies directed against gangliosides are considered a major immune effector which can induce damage to intact nerve fibers and are considered to be a major contributor in the pathogenesis of GBS." *Id.* (citing Pet'r's Ex. 54).

Dr. Akbari also raised regulatory T-cell dysfunction, inflammasome activation secondary to vaccine adjuvants, and a combination of these with molecular mimicry as mechanisms of causation. *See generally* Pet'r's Ex. 28. He opined that "[r]egulatory T cells play an important role in induction of GBS after vaccination." *Id.* at 17. "HPV vaccine is shown to stimulate the [regulatory T cells] and [effector T cells] in normal individuals after immunization. However, in some individuals, after vaccination, pathogenic [effector T cells] cells are able to differentiate further, exert robust effector function and cause severe injury such as GBS." *Id.* at 18. Dr. Akbari also argued that adjuvants, including aluminum, "facilitate the induction of immune responses mainly by triggering pathways such as inflammasome." *Id.* at 9.

Specific to the HPV vaccine causing GBS, Dr. Akbari cited several articles to show an association. Pet'r's Ex. 28 at 19–20. Miranda et al.[9] was a cohort study designed to evaluate whether HPV vaccination could induce or trigger autoimmune diseases. Pet'r's Ex. 63 at 1. Over two million girls aged 13–16 years were included in the study and followed over four years using French nationwide databases. *Id.* Among the 2,252,716 girls, 37% received an HPV vaccine and

---

[7] Gang Zhang et al., *Erythropoietin Enhances Nerve Repair in Anti-Ganglioside Antibody-Mediated Models of Immune Neuropathy*, 6 PLOS ONE e27067 (2011).
[8] Hugh J. Willison & Nobuhiro Yuki, *Peripheral Neuropathies and Anti-Glycolipid Antibodies*, 125 BRAIN 2591 (2002).
[9] 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).

4,096 autoimmune diseases occurred during a mean follow-up time of 33 months. *Id.* Forty-three of the autoimmune diseases identified were GBS. *Id.* at 4. The authors concluded that there was not an increased risk of autoimmune disease after HPV vaccination except for GBS. *Id.* at 1, 4, 6. The association between HPV vaccination and GBS was "particularly marked in the first [two] months following vaccination." *Id.* at 4. "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."[10] *Id.*

Souayah et al.[11] used data from the Vaccine Adverse Event Reporting System ("VAERS") to identify 69 cases of GBS within six weeks of HPV vaccination. Pet'r's Ex. 64 at 1. The authors did not draw a conclusion as to causation and suggested further surveillance is warranted. *Id.* at 1, 4. Boender et al.[12] reviewed 25 studies and found that "[i]n 22 studies, no increased risk of GBS was observed, while in three studies a "signal of increased risk of GBS after HPV vaccination was identified." Pet'r's Ex. 16 at 1. The authors concluded: "The absolute and relative risk of GBS after HPV vaccination is very low and lacks statistical significance." *Id.* Dr. Akbari then cited a Centers for Disease Control and Prevention ("CDC") report[13] that found between December 2014 and December 2017, "when approximately 28 million doses of Gardasil had been given out in the United States, there were [four] confirmed reports of GBS." Pet'r's Ex. 28 at 19.

Dr. Akbari further opined that host susceptibility "is a significant factor the development of autoimmune disease, including neuropathy such as GBS, independent of the initiating pathologic cause." Pet'r's Ex. 28 at 20. "[T]he decreased number of T regulatory cells most likely is a main factor resulting in a person's predisposition to develop autoimmune diseases after immunization." *Id.*

In the conclusion section of his first report, Dr. Akbari raised the mechanism of prime and recall response to help demonstrate a logical sequence of cause and effect. Pet'r's Ex. 28 at 22. He wrote that "if the same stimuli [that are produced in the first dose a vaccine] such as the second dose of vaccine are given a few weeks to a few months after the initial dose, the memory cells begin to replicate and produce cytokines in a significantly short period of time." *Id.* Dr. Akbari explained that as a result of this "secondary exposure to a given antigen triggers an immune response that is more vigorous and robust often resulting in the onset of demyelinating disease and GBS." *Id.* Thus, Dr. Akbari concluded that, "the timing between receipt of the second vaccine [the one subject to this petition] and development of demyelinating disease is approximately 11 days, which is an appropriate timeframe for the immune-mediated mechanism of causing neuropathy." *Id.*

---

[10] Over half of the HPV vaccine GBS group also had an infection (a gastrointestinal or respiratory illness) that preceded that onset of GBS, and over half of those cases occurred more than six months after vaccination. Pet'r's Ex. 63 at 5 tbls. 4–5.

[11] Nizar Souayah et al., *Guillain-Barré Syndrome After Gardasil Vaccination: Data from Vaccine Adverse Event Reporting System 2006-2009*, 29 VACCINE 886 (2011).

[12] T. Sonia Boender et al., *Risk of Guillain-Barré Syndrome After Vaccination Against Human Papillomavirus: A Systemic Review and Meta-Analysis, 1 January 2000 to 4 April 2020*, 27 EURO SURVEILLANCE (2022).

[13] This CDC report was not filed as an exhibit.

**2. Dr. Jeret's First Report**

Dr. Jeret opined Petitioner's GBS developed 10 days after his second HPV vaccination. Pet'r's Ex. 12 at 7. He explained that vaccines "stimulate the body's immune system to create antibodies," but on rare occasions, it "'gets the wrong message' and will attack an unintended target by creating an autoimmune response against peripheral nerve myelin." *Id.* at 8. Dr. Jeret explained this is called molecular mimicry and "is the basis for GBS after flu and other vaccines." *Id.* at 8, 10. "The same molecular mimicry is the basis for GBS after HPV vaccine. Vaccines like Gardasil may be more likely to trigger GBS because of the high antigenicity of the vaccine's recombinant proteins and other components of the vaccine." *Id.* at 8.

Dr. Jeret relied on the World Health Organization ("WHO") causality assessment criteria in Collet et al.[14] to conclude that it is "very likely/certain that [Petitioner's] HPV vaccine caused his GBS" because the "clinical event occurred with a plausible relationship subsequent to vaccine administration" and there was "no concomitant disease or drug that [could] explain his development of GBS." Pet'r's Ex. 12 at 10 (citing Pet'r's Ex. 17 at 3 tbl. 2). The criteria for causation used in Collet et al. consists of (1) biological plausibility, (2) "time elapsed between the vaccine administration and the onset of the adverse events," and (3) "whether other factors (drugs, chemicals[,] or underlying disease) could account for the adverse symptoms."[15] Pet'r's Ex. 17 at 1. In Petitioner's case, Dr. Jeret believed there "was no preceding viral or bacterial illness that were recognized triggers." Pet'r's Ex. 12 at 10. While he opined that *C. jejuni* is "probably the most clearly-defined illness that precipitates GBS," trivial upper respiratory infections ("URIs") are not listed as triggers for GBS. *Id.* at 9 (citing Pet'r's Ex. 18).[16] "Thus, [Petitioner's] preceding URI—whose timing is not specified, whose associated symptoms are not specified, and which is never diagnosed as influenza or considered by any of the practitioners to have been significant—did not play a role in his subsequent development of GBS." *Id.* Like Dr. Akbari, Dr. Jeret cited Miranda et al. and Souayah et al. for identifying cases of GBS after HPV vaccination. Pet'r's Ex. 12 at 8. Dr. Jeret also commented that he did not believe that Petitioner's medications played any role in the development of his GBS. *Id.* at 7.

Dr. Jeret opined that "GBS beginning 3–42 days after the vaccine would be consistent with a causal relationship based on molecular mimicry," and reiterated Petitioner's GBS began 10 days after vaccination. Pet'r's Ex. 12 at 11.

[14] J.P. Collet et al., *Monitoring Signals for Vaccine Safety: The Assessment of Individual Adverse Event Reports by an Expert Advisory Committee*, 78 BULLETIN OF THE WORLD HEALTH ORGANIZATION 178 (2000).

[15] This is not the standard used in the Vaccine Program.

[16] Marinos C. Dalakas, *Guillain-Barré Syndrome: The First Documented COVID-19-Triggered Autoimmune Neurologic Diseases*, 7 NEUROLOGY: NEUROIMMUNOLOGY & NEUROINFLAMMATION 781 (2020). Dr. Jeret also cited a GBS fact sheet from Yale Medicine online; however, this was not filed as an exhibit.

### 3. Dr. MacGinnitie's First Report

Dr. MacGinnitie agreed that GBS is the proper diagnosis, but he disagreed that the HPV vaccine was the cause. *See generally* Resp't's Ex. A. He also agreed that molecular mimicry can be a trigger for autoimmune disease and cited Peterson & Fujinami[17] for criteria. *Id.* (citing Resp't's Ex. A, Tab 1).

**TABLE 3.2 Criteria for Establishment of a Cause-and-effect Relationship Between Molecular Mimicry and Autoimmune Disease**

1. Similarity between a host epitope and an epitope of a microorganism or environmental agent.
2. Detection of antibodies or T cells cross-reactive with both epitopes in patients with the autoimmune disease.
3. Epidemiological link between exposure to the environmental agent or microbe and development of autoimmune disease.
4. Reproducibility of autoimmunity in an animal model following sensitization with the epitopes, infection with the microbe or exposure to the environmental agent.

Resp't's Ex. A, Tab 1 at 6 tbl. 3.2. While Dr. MacGinnitie opined these criteria do not need to be satisfied in every case, he noted Petitioner, through Dr. Jeret and Dr. Akbari, did not provide "evidence that fulfills any of these criteria." Resp't's Ex. A at 8.

In addressing the first criterion, Dr. MacGinnitie opined that GBS "can be caused by the generation of antibodies against carbohydrate antigens expressed by the bacteria *C. jejuni*, which also recognize similar molecules called gangliosides on human nerve axons." Resp't's Ex. A at 8. "These antibodies then mediate damage to the nerve cells resulting in GBS symptoms." *Id.* He noted that Petitioner tested positive for two gangliosides, GM1 and GD1a, and that the latter is associated with GBS. *Id.* (citing Pet'r's Ex. 3 at 155–56). However, he opined that Gardasil does not contain any carbohydrates that could be cross-reactive with human gangliosides. *Id.* at 9. He explained that Gardasil contains the L1 proteins of different HPV serotypes produced in yeast. *Id.* (citing Resp't's Ex. A, Tab 5 (Gardasil package insert); Resp't's Ex. A, Tab 6)).[18] "These L1 proteins spontaneously self-assemble into multimers, termed virus-like particles (VLPs) which are subsequently purified, removing any contaminating yeast material. Thus, the resulting vaccine should not contain any components that would cross react with gangliosides." *Id.* "Without any vaccine component cross-reactive with human gangliosides, there is no evidence that molecular mimicry between vaccine components and gangliosides could have resulted in [Petitioner's] GBS nor his positive testing for anti-ganglioside antibodies." *Id.*

Next, Dr. MacGinnitie opined there is no evidence of cross-reactive antibodies or T-cells in this case. Resp't's Ex. A at 9. Similarly, he opined there is no animal model linking the HPV vaccine to GBS. *Id.* Lastly, he found no large scale, population-based studies supporting HPV vaccination as a trigger for GBS. *Id.* Dr. MacGinnitie concluded that "[i]n the absence of even a single criteria being fulfilled, any immune stimulus (infections, exposure to allergen, vaccination)

[17] Lisa K. Peterson & Robert S. Fujinami, *Molecular Mimicry in* AUTOANTIBODIES 13 (2nd ed., Elsevier 2007).
[18] Rashi Yadav et al., *Virus-Like Particle-Based L2 Vaccines Against HPVs: Where Are We Today?*, 12 VIRUSES 18 (2020).

could be hypothesized to trigger any autoimmune disease." *Id.* at 11. Dr. MacGinnitie further criticized the literature cited by Dr. Akbari and Dr. Jeret. *Id*. at 9–11. He described reliance on VAERS as "problematic" because it is based on active reporting with no controls or auditing process to verify diagnosis. *Id.* at 10. Next, Dr. MacGinnitie argued that the Boender et al. and Gee et al.[19] articles found "no conclusion can be drawn about an increased or decreased risk of GBS post-vaccination," and that there is "no evidence of an increased risk of GBS following 4vHPV," respectively. *Id.* (first citing Pet'r's Ex. 16, then quoting Pet'r's Ex. 20 at 1) Dr. MacGinnitie acknowledged that the Miranda et al. study "did find an increased rate of GBS in the [two] months after vaccination, but [he noted that] this was a relatively small study." *Id.* Dr. MacGinnitie further noted that this finding could not be replicated in larger, epidemiologic studies, and it "was included in the meta-analysis by Boender et al. along with the negative studies and no statistically increased overall risk was found." *Id.* These studies, according to Dr. MacGinnitie, provide the best evidence against causation. *Id.* However, given the rare occurrence of GBS, and the limits of epidemiology detecting incidence rates in such small populations, Dr. MacGinnitie added that "it is impossible to completely rule out a very small increased (or decreased) risk." *Id.*

Dr. MacGinnitie found no basis for Dr. Akbari's theories based on regulatory T-cell dysfunction and inflammasome activation by aluminum adjuvants. Resp't's Ex. A at 12–14. Indeed, he opined the aluminum adjuvant used in the Gardasil vaccine "is safe and actually decreases systemic inflammation after vaccination." *Id*. at 14 (citing Resp't's Ex. F, Tab 9).[20] Moreover, Dr. MacGinnitie pointed out that Petitioner did not exhibit symptoms such as fever or malaise in the days after vaccination that would be indicative of substantial system inflammation. *Id.* at 13.

As to the URI, Dr. MacGinnitie believed that "[g]iven timing of [Petitioner's] GBS with onset in January, when influenza is widespread in the community, it is certainly possible that the URI [Petitioner] reported to [the ED physician] was influenza and this was the trigger of his GBS, given that influenza, unlike HPV, is a known trigger of GBS." Resp't's Ex. A at 14. However, he pointed out that many cases of GBS do not have an identified trigger. *Id.*

#### 4. Dr. Brier's First Report

Dr. Brier analyzed the citations cited by Petitioner's experts and concluded they only show that there exists "cases of GBS that may be temporally preceded by HPV vaccine." Resp't's Ex. C at 8. He referred to the WHO 2019 manual for "Causal Assessment of an Adverse Event Following Immunization."[21] *Id.* at 9. According to Dr. Brier, the manual defines adverse events following immunization as having two levels of scientific basis: population and individual. *Id.* He highlighted the necessary criteria they use and their applicability to this case:

[19] Julianne Gee et al., *Risk of Guillain-Barré Syndrome Following Quadrivalent Human Papillomavirus Vaccine in the Vaccine Saftey Datalink,* 35 VACCINE 5756 (2017).
[20] Harm HogenEsch, *Mechanism of Immunopotentiation and Safety of Aluminum Adjuvants*, 3 FRONTIERS IMMUNOLOGY 406 (2013).
[21] This was not filed as an exhibit.

- Temporal relationship: The present data do show that some cases of GBS may follow HPV vaccination, however the present data do not show that this temporal association is beyond what would be observed by chance.
- Strength of Association: The existing data show, at best, a weak association which falls well below the standard for statistical significance.
- Dose response relationship: There is no demonstration of a dose response.
- Consistency of evidence: Consistency, to the extent it exists, demonstrates no association between HPV vaccination and GBS.
- Specificity: The studies cited are population based and do not control for confounding causes. Moreover, the studies which show an association generally do not conform to established diagnostic criteria.
- Biological plausibility: I defer this to the immunology expert but would note that no compelling mechanism known to exist in humans in response to HPV vaccine has been clearly demonstrated.

*Id.* According to Dr. Brier, this precludes Petitioner's GBS being caused by the HPV vaccine for failure to meet these criteria. *Id.* Dr. Brier acknowledged that this is not the standard Petitioner needs to meet here. *Id.* at 10. However, even without using these criteria, Dr. Brier "did not see any reliable evidence in this case establishing, to a more likely than not standard, that the HPV vaccine can cause GBS, or did cause [Petitioner's] GBS." *Id.* Dr. Brier believed Petitioner's GBS is idiopathic. *Id.*

### 5. Dr. Akbari's Supplemental Report

In response to the critiques from Respondent's experts, Dr. Akbari detailed more of his molecular mimicry theory. *See generally* Pet'r's Ex. 70. He "conducted an extensive analysis of the sequences present in the Gardasil [v]accine" to "shed light on the specific molecular components that might trigger molecular mimicry and, subsequently, the development of autoimmune reactions." *Id.* at 2. He conducted a BLAST search to "investigate potential molecular mimics between the vaccine component and the [m]yelin basic protein [("MBP")]." *Id.* Dr. Akbari cited Cornblath et al.[22] for his opinion that MBP "plays a pivotal role as the main target in immune attacks leading to GBS."[23] *Id.* (citing Pet'r's Ex. 74). Cornblath et al. noted several studies had been done measuring immunoreactive MBP ("iMBP") in the CSF of patients with central nervous system ("CNS") disease and sought to determine iMBP levels in patients with peripheral neuropathies, including GBS. Pet'r's Ex. 74 at 1. The authors found that "MBP-like material is present in the CSF of most patients with longstanding demyelinating polyradiculoneuropathies." *Id.* The question remained, what is the source of this material. *Id.* They hypothesized that "the most likely source is P1 protein from [peripheral nervous system] myelin breakdown in the spinal roots." *Id.* Cornblath et al. compared their findings to studies of patients with CNS disease and found that "[i]n patients with peripheral neuropathy, confusing findings may result." *Id.* at 2. They concluded that "there is no method of determining the origin of iMBP in CSF." *Id.*

[22] David R. Cornblath et al., *Immunoreactive Myelin Basic Protein in Cerebrospinal Fluid of Patients with Peripheral Neuropathies*, 20 ANNALS NEUROLOGY 370 (1986).

[23] Dr. Akbari wrote: "Please note that while GBS is indeed a complex disease with various antigens involved, it's worth highlighting that approximately 50 percent of GBS patients exhibit the presence of antibodies against [MBP]." Pet'r's Ex. 70 at 4 (citing Pet'r's Ex. 74).

Dr. Akbari cited the Gardasil vaccine package insert to show the ingredients include purified virus-like particles ("VLPs") of the major capsid (L1) protein of HPV. Pet'r's Ex. 70 at 2 (citing Pet'r's Ex. 18). He explained that these "VLPs are purified, and an aluminum-containing adjuvant is subsequently introduced to enhance the immune response." *Id.* at 3. Dr. Akbari then opined that HPV shares sequences that are "identical or highly similar with myelin proteins." *Id.* Dr. Akbari did not include his search parameters in his report but provided the following results:

> The Human Myelin Basic Protein is: E N P V VHFF KN I VT P RT P
>
> L1 HPV11: Q M F A RHFF NR AGT VGE
>
> When we ask the blast software to align and notify any homology, the following result is coming up:
>
> E N P V VHFF KN I VT P RT P  Identical regions highlighted in yellow
>
> Q M F A RHFF NR AGT VGE P  Tolerated regions highlighted in green

*Id.* Dr. Akbari opined "there are five amino acids featuring three consecutive matches at critical binding points, a phenomenon supported by studies indicating that such homology is adequate to trigger molecular mimicry." *Id.* He also found there are three amino acids whose similarity is "tolerated." *Id.* He relied on Hausman et al.[24] to state "the replacement of asparagine (N) with arginine (R) at position 94 is tolerated; in other words, these substitutions are recognized by a T cell clone responsive to myelin basic protein, albeit with reduced potency." *Id.* (citing Pet'r's Ex. 73). "Furthermore, the isoleucine (I) to alanine (A) substitution at position 95 is well-tolerated, providing us with a total of seven out of twelve consecutive amino acids featuring exact matches or permissible substitutions. Remarkably, these substitutions still facilitate the stimulation of a human T cell clone reactive to gangliosides." *Id.* He stated the same is true for HPV type 18 L1, with four amino acid identities to myelin basic protein. *Id.* at 3–4. He cited Gautam et al.[25] to explain the significance of seven out of 12 amino acid identities as well as the adequacy of five out of 12 amino acid identities "to provoke an immune response directed at myelin basic protein" and "to induce clinical demyelination." *Id.* at 4 (citing Pet'r's Ex. 76).

Dr. Akbari said the goal of this analysis was to "shed light on the scientific underpinnings of how the HPV vaccine might influence the breakdown of tolerance to gangliosides and potentially act as a trigger to induce GBS." Pet'r's Ex. 70 at 3. He concluded:

> Based on an overwhelming preponderance of evidence, it is unequivocal that the Gardasil vaccine was the predominant instigator of GBS in [Petitioner]. This determination is unequivocally rooted in a scrupulously [sic] analysis of the vaccine's constituents and their compelling potential to incite grievous pathological immune responses through the unequivocal mechanism of molecular mimicry.

[24] Stefan Hausman et al., *Structural Features of Autoreactive TCR That Determine the Degree of Degeneracy in Peptide Recognition*, 162 J. IMMUNOLOGY 338 (1999).

[25] Anand M. Gautam et al., *A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis*, 161 J. IMMUNOLOGY 60 (1998).

*Id.* at 6.

Next, Dr. Akbari addressed "the inquiries posed by Dr. MacGinnitie concerning the roles of cytokines and the mechanisms through which molecular mimicry and the inflammasome contribute to the process of demyelination." Pet'r's Ex. 70 at 8. He explained cytokines are "signaling molecules within the immune system [that] play a pivotal role in orchestrating immunes response" and "central players" for demyelination. *Id.* He said cytokines "can act as mediators in the cascade of events that lead to the destruction of myelin," which is characteristic of GBS. *Id.* "For example, pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β) can contribute to the activation of immune cells and the subsequent attack on myelin." *Id.* "Moreover, the inflammasome, a complex of proteins responsible for initiating an inflammatory response, can be closely linked to demyelination. It serves as a platform for the activation of pro-inflammatory cytokines like IL-1β and IL-18, which further exacerbate the immune response and can lead to tissue damage, including demyelination." *Id.*

Dr. Akbari proposed that the "HPV vaccination is capable of inducing a robust inflammasome activation, and the consequences include the production of active caspase-1, converting inactive precursor cytokines of the IL-1 family (including IL-1a and IL-1b), to their active forms." Pet'r's Ex. 70 at 9 (citing Pet'r's Ex. 77).[26] Dr. Akbari added that "[b]esides IL-1 and inflammasome, another vaccine-induced pro-inflammatory cytokine is IL-6. It is important to also mention that Tregs are able to suppress inflammasome activation and actively regulate proinflammatory cytokines, suggesting that impaired Tregs can also contribute to the induction of demyelinating diseases." *Id.* In support of this assertion, Dr. Akbari cited the Haas et al.[27] article, which took revelations discovered in prior studies regarding Treg dysfunction in multiple sclerosis patients and sought to determine the cause for new treatments for that condition. Pet'r's Ex. 81. The authors found that "diminished suppressive Treg potencies in [multiple sclerosis] are related to disequilibrium in the homeostatic composition of circulating Treg and most likely result from an altered thymic release of newly formed T cells into the periphery." *Id.* at 7. They were hopeful "that pharmacologic modulation of the thymus and the reconstruction of peripheral Treg homeostasis" could be a "promising treatment strategy in [multiple sclerosis] and other autoimmune disorders." *Id.* at 8. As to the applicability to GBS, Dr. Akbari suggested "that the prevalence of newly generated regulatory T cells (Treg) is critical for Treg suppressive function and determines Treg dysfunction in demyelinating disease." Pet'r's Ex. 70 at 9.

Specifically relevant to the HPV vaccine causing GBS, Dr. Akbari stated:

> All we are asserting is that the HPV vaccine has the capability to cause GBS. As demonstrated earlier, the combination of segments of the HPV virus and alum adjuvant potentiates the effects of antigens, sometimes more than 100 times, and in certain individuals, this combination can result in the development of GBS.

---

[26] Virginie Petrilli et al., *The Inflammasome: A Danger Sensing Complex Triggering Innate Immunity*, 19 CURRENT OP. IMMUNOLOGY 615 (2007).

[27] Jurgen Haas et al., *Prevalence of Newly Generated Naïve Regulatory T Cells (Treg) is Critical for Treg Suppressive Function and Determines Tred Dysfunction in Multiple Sclerosis*, 179 J. IMMUNOLOGY 1322 (2007).

Pet'r's Ex. 70 at 10. He again referenced Miranda et al., Souayah et al., and Boender et al. *Id.* at 11. Dr. Akbari argued that "[o]verall, these studies collectively suggest that HPV can potentially lead to GBS." Pet'r's Ex. 70 at 11.

Lastly, Dr. Akbari again commented on his opinion that Petitioner developed GBS after his second dose of the HPV vaccine. Pet'r's Ex. 70 at 11–12, 16. He explained:

> In [i]mmunology the requirement of a second (or possibly third or multiple) signal to induce pathogenic disease is often essential. These requirements also explain how molecular mimicry can facilitate both immune tolerance and immune autoreactivity. Scientific theories are neither absolutely false nor absolutely true. They are always somewhere in between. Some theories are better, more credible, and more accepted than others, and that is why we always state "more likely than not" or based on "preponderance of evidence."

*Id.* at 16. He added that when a "subsequent stimulus, such as the second dose of a vaccine, is administered a few weeks to a few months after the initial dose, these memory cells are awakened. They begin to replicate and produce cytokines in significantly higher quantities." *Id.* at 12. According to Dr. Akbari, this "booster effect triggers a more vigorous and robust immune response, which, in some cases, can lead to adverse effects like GBS." *Id.* Dr. Akbari found this important since Petitioner "did not experience any adverse effects after his first dose but developed GBS 11 days after the second dose. This temporal relationship suggests that the HPV vaccination could indeed be a contributing factor to the development of GBS in his case." *Id.*

**6. Dr. Jeret's Supplemental Reports**

Dr. Jeret filed two supplemental reports disagreeing with Dr. Brier. Pet'r's Exs. 69, 86. He focused on statistical issues with medical literature, particularly how GBS is rare and some cases may go unreported. Pet'r's Ex. 69 at 1–2. His opinion remained the same as his original report. *Id.* at 2–3; Pet'r's Ex. 86 at 2.

**7. Dr. MacGinnitie's Supplemental Report**

Dr. MacGinnitie criticized Dr. Akbari's BLAST search and opined the "degree of sequence similarity between HPV L1 proteins and [MBP] [] identified by Dr. Akbari is not notable." Resp't's Ex. F at 2. Dr. MacGinnitie pointed out that Dr. Akbari did not provide the details of the searches he conducted or the official results. Without that information, he undertook a BLAST search himself using HPV11 and HPV18 L1 proteins versus human MBP. *Id.* "Initially, [he] got no matches, but when [he] loosened the stringency of the search (using the Expect Value discussed below) it returned a single match for each search with the one for HPV 11 shown here." *Id.*

sp|P02686|MBP_HUMAN Myelin basic protein OS=Homo sapiens OX=9606 GN=MBP PE=1 SV=3
Sequence ID: Query_7139155 Length: 304 Number of Matches: 1
Range 1: 260 to 266

| Score | Expect | Method | Identities | Positives | Gaps | Frame |
|---|---|---|---|---|---|---|
| 13.9 bits(24) | 9.6() | Compositional matrix adjust. | 5/7(71%) | 5/7(71%) | 0/7(0%) | |

```
Query  469  GYRGRTS  475
            GY GR S
Sbjct  260  GYGGRAS  266
```

*Id.* Dr. MacGinnitie explained the Expect Value, "which evaluates how unlikely a particular match is – in other words, how probable or improbable it is that such a match could have occurred by chance." *Id.* at 3. "The built in 'cutoff' is an expect value less than or equal to 0.05 and using this cutoff [Dr. MacGinnitie] got zero matches." *Id.* "Only when [he] increased the cutoff to 10 (meaning 10 such matches between [two] sequences would be expected by chance) did [he] obtain a match." *Id.* Despite Dr. MacGinnitie finding this homology, he opined "[s]equence similarity alone is not enough to demonstrate molecular mimicry as a trigger of disease." *Id.* at 4. The Institute of Medicine ("IOM")[28] discussed this and wrote that "[m]any such homologies exist, and the vast majority of these are not associated with biologically relevant autoimmune phenomenon or actual human disease." *Id.* (quoting Resp't's Ex. F, Tab 6 at 10). Kanduc et al.[29] stated that if sequence homology was enough to induce molecular mimicry, then "viral infections should be a practically infinite source or autoimmunity diseases since this study demonstrates that viral 5-mer matches are disseminated throughout practically all the human proteome and each viral match is repeated almost more than 10 times." Resp't's Ex. F, Tab 3 at 11. Dr. MacGinnitie summarized, "Dr. Akbari has not identified significant homology between the HPV L1 protein in Gardasil and human MBP nor is a simple sequence homology enough to trigger autoimmunity." Resp't's Ex. F at 6.

Dr. MacGinnitie added that "gangliosides and proteins are distinct classes of molecules so there is no reason to think that T-cells that recognize MBP would also recognize gangliosides." Resp't's Ex. F at 6 (citing Resp't's Ex. F, Tab 14).[30] Dr. MacGinnitie then cited Ziganshin et al.[31] for the proposition that autoantibodies against MBP are not present in patients with GBS and criticized Dr. Akbari's interpretation of Cornblath et al. for saying otherwise. *Id.* at 7 (citing Resp't's Ex. F, Tab 17; Pet'r's Ex. 74). "Thus, even if Dr. Akbari were correct that HPV vaccination could trigger production of an autoimmune response to MBP, and [Dr. MacGinnitite] do[es] not agree with this assertion, it would not explain development of GBS." *Id.* Dr. MacGinnitie noted "that antibodies against MBP are typically associated with multiple sclerosis, a demyelinating disorder of the central, not peripheral nervous system." *Id.* Dr. MacGinnitie reiterated that Petitioner "had positive testing for [two] gangliosides, GM1 and GD1a with the

[28] Inst. of Med., *Adverse Effects of Vaccines: Evidence Causality* (Kathleen Stratton et al. eds., 2012).
[29] Darja Kanduc et al., *Massive Peptide Sharing Between Viral and Human Proteomes*, 29 PEPTIDES 1755 (2008).
[30] Jon D. Laman et al., *Guillain-Barré Syndrome: Expanding the Concept of Molecular Mimicry*, 43 TRENDS IMMUNOLOGY 296 (2022).
[31] Rustam H. Ziganshin et al., *The Pathogenesis of the Demyelinating Form of Guillain-Barre Syndrome (GBS): Proteo-Peptidomic and Immunological Profiling of Physiological Fluids*, 15 MOLECULAR & CELLULAR PROTEOMICS 2366 (2016).

latter being associated with GBS." *Id.* at 8 (citing Pet'r's Ex. 3 at 155–56). "Thus, development of these anti-ganglioside antibodies provides a clear immunologic explanation for development of diseases, with no need to invoke autoimmunity against proteins such as MBP." *Id.*

#### 8. Dr. Brier's Supplemental Report

Dr. Brier submitted a supplemental report criticizing Dr. Jeret's opinions and addressing "how one weighs evidence in the setting of imperfect data." Resp't's Ex. E at 1. He compared different types of literature filed including large epidemiologic studies and case reports. *Id.* at 1–4. He also further discussed diagnostic criteria, although he agreed GBS is the correct diagnosis. *Id.* at 4–6. His opinion remained that "there is insufficient evidence to infer that HPV vaccination causes GBS." *Id.* at 6.

## IV. Applicable Legal Standards

### A. Burden of Proof

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" *Rooks v. Sec'y of Health & Hum. Servs.*, 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, *reprinted in* 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991).

### B. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); *Capizzano*, 440 F.3d at 1319–20. Here, Petitioner does not assert a Table claim; therefore, Petitioner must prove a vaccine he received caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

A petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific

explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. A petitioner is not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano,* 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1325. However, Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). Furthermore, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. *See Waterman v. Sec'y of Health & Hum. Servs.*, 123 Fed. Cl. 564, 573–74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. *Moberly*, 592 F.3d at 1322; *see also de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1351 (Fed. Cir. 2008). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322.

Next, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *see also Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. *Shyface*, 165 F.3d at 1351. The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.,* 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted).

Lastly, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. For example, if a petitioner's theory involves a process that takes several days to develop after vaccination, an injury that occurred within a day of vaccination would not be temporally consistent with that theory. Conversely, if the theory is one that anticipates a rapid development of a reaction post-vaccination, the development of the alleged injury weeks or months

post-vaccination would not be consistent with that theory. *See de Bazan*, 539 F.3d at 1352. Causation-in-fact cannot be inferred from temporal proximity alone. *See Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992); *Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983) ("[w]ithout more, [a] proximate temporal relationship will not support a finding of causation").

In determining whether Petitioner is entitled to compensation, the special master shall consider all materials in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. *See Moberly*, 592 F.3d at 1325–26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); *Althen*, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor). A petitioner who satisfies her burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

## IV. Discussion

### A. *Althen* Prong One

Dr. Akbari opined that "molecular mimicry and induction of inflammasome are credible medical theor[ies] causally linking the HPV vaccination to injuries that [Petitioner] experienced." Pet'r's Ex. 70 at 16. Dr. Jeret opined "molecular mimicry is the basis for GBS after HPV vaccine." Pet'r's Ex. 12 at 8. I find Petitioner failed to provide preponderant evidence of a sound and reliable medical theory explaining how the HPV vaccine can cause GBS.

I note at the outset that Dr. Akbari referred to the Tdap vaccine throughout his first report. *See, e.g.*, Pet'r's Ex. 28 at 5 ("My research into your particular question concluded that the theory of molecular mimicry supports that Tdap vaccination can plausibly cause an adverse immune cross reaction and result in the onset of GBS."), 16 ("A second criterion for implicating molecular mimicry. . . is to demonstrate the plausibility of cross-reactivity by autoreactive T-cells or autoantibodies with a microbial antigen, or in this case to a component of the Tdap vaccine."). Dr. Akbari never compared the Tdap vaccine to the HPV vaccine or explained how evidence relevant to one is applicable to the other. It appears these references may have been copied from another expert report in a different case. *See* also Pet'r's Ex. 70 at 13 (referencing conditions (pericarditis and rheumatoid arthritis) and doctors (Dr. LaRue and Dr. Bates) not involved in this case). I will not address his opinions on other vaccines or other conditions to the extent he did not compare them to the vaccine and injury at issue in this case. An expert may "extrapolate from existing data" where the reasons for extrapolation are transparent and persuasive. *K.O. v. Sec'y of Health & Hum. Servs.*, No. 13-472V, 2016 WL 7634491 (Fed. Cl. July 7, 2016) (quoting *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009)).

Overall, I find Dr. Akbari's opinions as to *Althen* prong one are underdeveloped and conclusory in nature. When evaluating whether petitioners have carried their burden of proof, special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), *mot. for rev. denied, decision aff'd*, 141 Fed. Cl. 138, *aff'd*, 945 F.3d 1362 (Fed. Cir. 2020). I will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." *Prokopeas v. Sec'y of Health & Hum. Servs.*, No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting *Moberly*, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. *See id.*

Dr. Akbari's proposed theory was confusing as he focused on molecular mimicry but inexplicably injected inflammasome and T-cell dysfunction into his arguments. He did not provide needed context for a relationship between either of these concepts and his main theory; nor did he propose alternative theories (with evidentiary support) that center on either. *See e.g.*, Pet'r's Ex. 70 at 9 (Dr. Akbari writing that "inflammasome and IL-1 are capable of suppressing the Treg function tremendously," but that "[i]f you add IL-1 to Tregs, the cells are not able to exert their suppressive effect," yet "the induction of inflammasome by HPV vaccine is capable of suppressing Tregs and increas[ing] the chance of induction of demyelinating disease"). These vague, conclusory, and contradictory statements muddle any persuasive argument for a biological mechanism. Moreover, while Respondent discussed Dr. Akbari's other potential theories in his responsive brief, Petitioner chose not to rebut any of those criticisms in his reply brief and maintained his focus on the molecular mimicry theory. I find any other alternative theories raised by Dr. Akbari lack the detail sufficient to determine reliability or reasonableness based on a preponderant standard. Accordingly, I too will focus my analysis on molecular mimicry.

Dr. Akbari conducted a BLAST search to attempt to show homology between components of the HPV vaccine and MBP as the human antibody target for GBS. Dr. Akbari asserted he found five amino acids featuring three consecutive matches at critical binding points and opined that such homology is adequate to trigger molecular mimicry. Additionally, he opined there were three amino acids whose similarity is "tolerated." Pet'r's Ex. 70 at 2. Dr. MacGinnitie disputed the relevancy of any association between MBP and GBS and opined that gangliosides, which are associated with GBS, should have been applied instead.[32] Nonetheless, even if MBP was relevant to GBS, Dr. MacGinnitie opined the HPV vaccine would not likely induce GBS. First, Dr. Akbari did not file his search parameters, and Dr. MacGinnitie was unable to replicate Dr. Akbari's alleged homology results. Second, even if he could, Dr. MacGinnitie believed the linear peptide homologies of four (HPV 11) and five (HPV 18) were insignificant.

Kanduc et al. and the IOM state that homology alone is not enough to induce molecular mimicry and subsequently, autoimmune diseases. However, we do not reach the issue of homology here as Petitioner applied his molecular mimicry theory to MBP, and Petitioner has not proved by preponderant evidence that MBP is the target antigen in GBS.

[32] The significance of gangliosides in this case is discussed more below in *Althen* Prong two.

Cornblath et al. did not support the finding that MBP plays a pivotal role in the development of GBS; in fact, it did not study the presence of MBP antibodies in GBS. Rather, it studied the presence of MBP itself (the protein, not the antibodies) in the CSF of patients with GBS and concluded that "MBP-like material is present in the CSF of most patients with longstanding demyelinating polyradiculoneuropathies and probably reflects detection of peripheral nervous system P1 protein." Pet'r's Ex. 74 at 1. Like here, Cornblath et al. has been used to support this assertion previously in the Program and was likewise found to be inapplicable to vaccine-caused GBS. *See Blackburn v. Sec'y of Health & Hum. Servs.*, 2015 WL 425935 (Fed. Cl. Spec. Mstr. 2015) (Dr. Steinman implicated an immune attack on MBP to show how the HPV vaccine caused GBS/CIDP. Dr. Steinman conceded that Cornblath et al. did not support the proposition that MBP is the target antigen in GBS and the Court ultimately rejected Dr. Steinman's theory, dismissing the case.). Ziganshin et al. tested serum from GBS patients and controls for MBP antibodies and found no "statistically significant difference." Resp't's Ex. F, Tab 17 at 7. They did however find that gangliosides GM1 and GD1a were targets in the AMAN variant of GBS. Petitioner did not respond to Dr. MacGinnitie's opposition to Dr. Akbari's theory on this point or apply his theory to these gangliosides. Thus, the target antigen of GBS is not established here and Dr. Akbari's conflicting opinions, on this point and others, are not as persuasive without further explanation or supporting authority.[33]

Dr. Jeret also proposed molecular mimicry but only discussed it generally and did not provide context for its application to GBS caused by the HPV vaccine. He asserted that the mechanism that leads to GBS following the flu vaccine can be applied to HPV vaccination without further explanation. To the extent that molecular mimicry is offered as a theory, it must be supported by a sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548. There must also be some degree of selectivity. *See W.C. v. Sec'y of Health & Hum. Servs.,* 704 F.3d 1352, 1360 (2013) (finding that a petitioner cannot prevail by simply invoking a biological term, or by showing that the mechanism is a valid theory to explain how *other* triggers may have induced *other* diseases and determining that a petitioner must produce additional evidence that the mechanism can cause that vaccine to cause a specific disease); *Caves v. Sec'y of Health & Hum. Servs.,* 100 Fed. Cl. 119, 135 (2011), *aff'd*, 463 F. App'x. 932 (2012); *McKown v. Sec'y of Health & Hum. Servs.,* No. 15-1451, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019). Petitioner does not have to provide a specific mechanism, but it must be detailed enough to apply to the administered vaccine and alleged injury in this case. Otherwise, any vaccination, by nature of its purpose to illicit an immune response, could be asserted as the cause of any autoimmune disease that later developed in an individual, and *Althen* prong one "would be rendered meaningless." *See Caves,* 100 Fed. Cl. at 135; *see also McKown,* 2019 WL 4072113, at *50 ("[M]erely chanting the words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or the vaccine in question.").

Several studies were cited regarding an association, or lack thereof, between the HPV vaccine and GBS. For example, Souayah et al. used data from VAERS to conclude there was an increased risk of GBS after vaccination when they identified 69 cases of GBS within six weeks of HPV vaccination. Miranda et al. found an increased risk of GBS after HPV vaccination. And while

[33] Because Petitioner has not provided preponderant evidence of the target antigen here, we do not reach the issue of sequence homology and whether it is sufficient.

Boender et al. (cited by both parties) found "a signal of increased risk of GBS after HPV vaccination," they concluded that the "absolute and relative risk of GBS after HPV vaccination is very low and lacks statistical significance." Pet'r's Ex. 84 at 1. While epidemiologic studies are not required to be successful, when the petitioner files epidemiologic studies himself concluding there is no association between the subject vaccine and injury, that evidence is evaluated along with all other evidence that is submitted in favor of or against Petitioner's arguments.

After consideration of the evidence, I find that Petitioner has not presented preponderant evidence of a sound and reliable medical theory to explain how the HPV vaccine can cause GBS. Therefore, Petitioner does not meet his burden pursuant to *Althen* prong one.

**B. *Althen* Prong Two**

Both parties agreed the diagnosis is GBS. But a successful argument pursuant to *Althen* prong two is heavily dependent on a reliable causation theory. Petitioner's inability to meet his burden demonstrating how the HPV vaccine can cause GBS effectively precludes him from being able to show that his symptoms were actually caused by the vaccine according to said theory. Moreover, neither Dr. Akbari nor Dr. Jeret analyzed Petitioner's clinical presentation in the context of their theory. This alone hinders any meaningful discussion on prong two.

Petitioner tested positive for the GM1 and GD1a ganglioside antibodies when he was diagnosed with GBS. Petitioner's treating physicians explained these were associated with his type of GBS, the AMAN variant. *See, e.g.*, Pet'r's Ex. 3 at 155 (test results), 51 (Dr. Talmasov noting an association between GM1/GD1a and GBS); Pet'r's Ex. 4 at 4 (Dr. Zakin noting Petitioner's elevated GM1 and GD1A in association with his GBS), 189, 245, 305, 349, 386 (Dr. Zakin consistently stating Petitioner had a motor variant of GBS associated with a positive GM1 antibody). Dr. MacGinnitie also opined that studies show that GM1 and GD1a antibodies are associated with AMAN; "[t]hus, development of these anti-ganglioside antibodies provides a clear immunologic explanation for development of diseases, with no need to invoke autoimmunity against proteins such as MBP." Resp't's Ex. F at 8. Neither Dr. Akbari nor Dr. Jeret addressed this.

While Petitioner's neurologist Dr. Zakin noted Petitioner's GBS was "in the setting of recent HPV vaccination," she did not opine as to how the two were causally connected, if at all. Pet'r's Ex. 4 at 1.

In considering the reliability of a petitioner's evidence of a prima facie case, the special master may consider alternative causes for a petitioner's condition that are reasonably raised in the record, even if the respondent does not pursue a formal alternative cause argument. *Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349, 1358 (Fed. Cir. 2010). Thus, in weighing a petitioner's case-in-chief, a special master may consider evidence that the petitioner's alleged injury could have been caused by alternative causes. *Id.*

During Petitioner's hospitalization, it was noted that Petitioner had an HPV vaccine and a cold[34] prior to the onset of symptoms. Dr. Jeret opined that Petitioner's "preceding URI—whose

[34] Although one of the doctors noted there was no preceding illness. Pet'r's Ex. 3 at 77–78.

timing is not specified, whose associated symptoms are not specified, and which is never diagnosed as influenza or considered by any of the practitioners to have been significant—did not play a role in his subsequent development of GBS." Pet'r's Ex. 12 at 9. Dr. MacGinnitie opined it was "certainly possible" that Petitioner's URI was influenza and "was the trigger of his GBS, given that influenza, unlike HPV is a known trigger of GBS." Resp't's Ex. A at 14. This is not preponderant evidence of actual causation for either the alleged cold or the HPV vaccine in Petitioner's case.

As indicated above, Petitioner did not apply molecular mimicry, or any theory, in the context of gangliosides, particularly those involved in GBS. For these reasons, I find Petitioner has failed to provide preponderant evidence of a logical sequence of cause and effect between his HPV vaccination and his GBS.

### C. *Althen* Prong Three

Petitioner asserted that the onset of his GBS was 10–11 days after vaccination. Dr. Akbari proposed a mechanism of prime and recall response to explain how 10 days is an appropriate temporal association, between Petitioner's HPV vaccination and the onset of his symptoms, consistent with causation. *See* Pet'r's Ex. 28 at 22; Pet'r's Ex. 70 at 11–12, 16. He wrote that "if the same stimuli [that are produced in the first dose a vaccine] such as the second dose of vaccine are given a few weeks to a few months after the initial dose, the memory cells begin to replicate and produce cytokines in a significantly short period of time." Pet'r's Ex. 28 at 22. Dr. Akbari pointed out the significance of this since Petitioner "did not experience any adverse effects after his first dose but developed GBS 11 days after the second dose." Pet'r's Ex. 70 at 12. Thus, according to Dr. Akbari, "the timing between receipt of the second vaccine and development of demyelinating disease . . . is an appropriate timeframe for the immune-mediated mechanism of causing neuropathy." Pet'r's Ex. 28 at 22. Dr. Jeret opined "GBS beginning 3–42 days after the vaccine would be consistent with a causal relationship based on molecular mimicry" and that Petitioner's GBS began 10 days after vaccination. Pet'r's Ex. 12 at 11.

Respondent "does not contest that [P]etitioner's GBS began within a timeframe that is reasonable to infer causation." Resp't's Response at 29. Based on the medical literature filed, this would be a medically acceptable timeframe. However, and as discussed above, since Petitioner has not provided preponderant evidence that the HPV vaccine can cause GBS, Petitioner ultimately fails to show preponderant evidence that his vaccine caused his condition.

## V. Conclusion

After a careful review of the record, Petitioner has failed to provide preponderant evidence that his January 14, 2020 HPV vaccine caused his GBS. Accordingly, Petitioner's claim is **DENIED**. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a). [35]

[35] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.

**IT IS SO ORDERED.**

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