# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

**No. 20-884V**

Filed: November 19, 2025

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ERIC SIMMONS,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

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*Christina Ciampolillo*, Conway, Homer, P.C., Boston, MA, for Petitioner.
*Emilie Williams*, U.S. Department of Justice, Washington, DC, for Respondent.

## RULING ON ENTITLEMENT[1]

**Shah**, Special Master:

On July 21, 2020, Eric Simmons ("Petitioner") 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 human papillomavirus ("HPV") and meningococcal vaccines he received on July 31, 2017. *Id*. at 1, 17.

[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. Hereinafter, for ease of citation, all "§" references to the Vaccine Act 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 demonstrated he is entitled to compensation. Briefly, Petitioner preponderantly showed that the HPV vaccine can cause GBS with epidemiologic evidence and a reliable molecular mimicry theory. He further demonstrated, through treating physician statements, expert opinions, and other evidence, a logical sequence of cause and effect between the vaccine and his condition, and that his GBS symptoms began within a reasonable timeframe after vaccination.

## I. PROCEDURAL HISTORY

After filing the petition, Petitioner filed affidavits and medical records. Exs. 1-11. On January 4, 2021, Respondent filed a Rule 4(c) Report ("Report"), recommending that entitlement be denied. Report at 2, 9 (ECF No. 23).

On January 7, 2021, former Special Master Katherine E. Oler held a status conference to inform the parties that, in her view, it appeared that "Petitioner suffered from [GBS] after his HPV and meningococcal vaccinations, and . . . onset of GBS appears to have been anywhere between 30 to 42 days after vaccination." ECF No. 24 at 1. Special Master Oler also noted that there was litigative risk on both sides and that, although non-binding, Respondent had settled similar cases in the past. *Id*.

On February 8, 2021, Respondent stated he intended to continue defending this case. ECF No. 25. Petitioner filed an expert report from Norman Latov, M.D., Ph.D., along with medical literature and Dr. Latov's curriculum vitae on April 9, 2021. Exs. 12-36. On October 25, 2021, Respondent filed an expert report by Andrew MacGinnitie, M.D., Ph.D., along with medical literature and Dr. MacGinnitie's curriculum vitae. Exs. A & Tabs 1-30, B. Respondent filed an expert report by Michael Kruer, M.D., and his curriculum vitae, on March 10, 2022. Exs. C, D.

On March 28, 2022, Special Master Oler held a second status conference. ECF No. 42. She again noted that onset appeared to have occurred between 30 to 42 days after vaccination and that there "does not appear to be any antecedent infection, and Respondent's expert does not raise that possibility." *Id*. at 1. She pointed out that there was litigative risk on both sides and that it would be beneficial for the parties to engage in settlement negotiations, noting that, although not binding, Respondent has settled similar cases in the past. *Id*.

Respondent declined to entertain settlement and requested that the Court schedule further proceedings. ECF No. 43. Petitioner filed a supplemental expert report from Dr. Latov and additional medical literature on August 19, 2022. Exs. 37-52. Respondent filed a supplemental expert report from Dr. MacGinnitie on October 21, 2022. Ex. E.

Special Master Oler held a third status conference on October 25, 2022, to inform the parties that a ruling on the record would be appropriate for this case and that, "without rendering a tentative conclusion under Rule 5," her view was that there was "no obvious impediment to Petitioner's claim." ECF No. 50 at 1. She noted that there was "no evidence of prior viral infection that could have caused Petitioner's [GBS]" and that onset for Petitioner's condition arose in within

the appropriate timeframe. *Id*. In her view, this case turned on whether she credited Petitioner's theory of causation under *Althen* prong one. *Id.*

Petitioner filed additional medical literature on December 28, 2022. Ex. 41. On January 10, 2023, Respondent confirmed that the record was complete for briefing. ECF No. 54. On February 10, 2023, Petitioner filed a second supplement expert report from Dr. Latov, along with additional medical literature, and stated the record was complete for briefing. Exs. 53-55; ECF No. 57.

Petitioner filed a motion for a ruling on the record ("Motion") on June 28, 2023. ECF No. 61. Respondent filed a response ("Response") on September 25, 2023, and Petitioner filed a reply brief ("Reply") on November 16, 2023. ECF Nos. 64, 66. The parties agreed that the record was complete on November 20, 2023. ECF No. 67. This case was reassigned to me on August 14, 2024. ECF No. 68. This matter is now ripe for adjudication.

## II. FACT EVIDENCE

In 2017, Mr. Simmons was a high school student entering his senior year. Ex. 10 ("Pet. Affidavit") at 1. Prior to the subjection vaccination, his medical history was significant for obesity, prehypertension, mild intermittent asthma without complications, peanut and tree nut allergies, and eczema. Ex. 2 at 107, 111, 136-38, 170; Ex. 6 at 2.

On July 31, 2017, Petitioner received the second dose of the HPV vaccination series, as well as a meningococcal vaccination, during a routine physical with David Rice, M.D., his primary care provider ("PCP"). Ex. 1 at 2; Ex. 2 at 131-32, 134, 138. During this visit, Dr. Rice counseled Petitioner on nutrition and physical activity and created an obesity management plan for him. Ex. 2 at 138. Petitioner's exam was normal. *Id.* He was told to follow up in two to three months to recheck his weight and blood pressure. *Id*. at 138-39.

On August 17, 2017, Petitioner presented as a new patient to allergist Joel Hartman, M.D., for an evaluation of environmental and food allergies. Ex. 5 at 2. Petitioner reported an allergic reaction to peanuts that occurred when he was four years old, as well as rhinitis, an asthma diagnosis at age two, and that his family owned two dogs and four cats. *Id*. He had no other complaints. *Id.* A physical exam was normal. *Id.* at 4. A skin test revealed significant reactivity to tree, grass, and weed pollen, along with dust mites, cockroaches, dogs, cats, molds, peanuts, and tree nuts. *Id*. at 4-5. Given his "multiple positive reactions and the clinically significant symptoms," Dr. Hartman believed Petitioner would be a good candidate for allergen immunotherapy. *Id*. at 5. Dr. Hartman advised him to avoid peanuts and tree nuts, prescribed Patanase and epinephrine, and instructed him to follow up in three to four months or sooner if needed. *Id*. at 5-6. Petitioner subsequently received allergy injections for grass, trees, dogs, dust mites, and weeds on August 30, September 7, and September 13, 2017. *Id*. at 9-10.

According to his affidavit, executed on August 23, 2020, Petitioner started his senior year of high school on August 28, 2017. Pet. Affidavit at 1. "In early September 2017, around the Labor Day Holiday," he noticed that his "legs were feeling sore and they hurt." *Id*. He was "unable to walk normally" and noticed that the bottom of his feet "were starting to feel numb." *Id*. Initially,

he thought that his legs were sore from a new exercise routine that he had started; however, it soon became too painful for him to walk or stand for any length of time. *Id*. at 1-2. He fell twice while he was at school and eventually had trouble standing and walking on his own; he "needed to hold onto family members for help." *Id*. at 2.

Petitioner's mother, Mrs. Jill Simmons, submitted an affidavit executed September 1, 2020. Ex. 11 ("Mrs. Simmons Affidavit"). "In September 2017, not long after school started," she noticed that Petitioner was "dragging his feet when he walked." *Id*. at 1. She said that when she picked up her son from school, he "always stood under a specific tree" where she met him; however, "[o]ne day" she noticed that he was sitting rather than standing. *Id*. This continued for several days. *Id.* Petitioner explained that his feet and legs hurt, and it was too painful to stand. *Id*. He had recently started climbing stairs for exercise, and they both thought the exercise might be the cause of his leg pain. *Id*. Mrs. Simmons, however, soon noticed that her son was "walking differently"; he "was kicking his legs out when he stepped forward and stomping his feet down on the ground." *Id*.

Petitioner experienced a fall at school on September 12, 2017. Mrs. Simmons Affidavit at 2. He was only able to walk holding onto people on either side of him; "[h]is gait was heavy and clumsy, and he tripped over his feet occasionally." *Id*. at 3. Thereafter, when he walked through the house, he would hold onto the walls or furniture for assistance. *Id*. Mrs. Simmons reported that her son was unable to return to school on his own because he needed assistance to walk, and the district would not allow her to assist him at school. *Id*. He decided to continue his senior year of high school at home. *Id*.

On September 13, 2017, 44 days after the subject vaccinations, Petitioner saw Dr. Rice. Ex. 2 at 173-89. His chief complaint was pain in both calves and numbness. *Id.* at 176. Dr. Rice noted "[o]nset following a first time and intense bout of stair climbing" involving walking up and down a flight of stairs about six times. *Id*. After that session, Petitioner's legs "felt like jelly." *Id.* After starting his exercise regimen, Petitioner reported that he developed pain in both calf muscles, numbness in his legs, his feet were "tingly," his muscles felt weak, he had "some mild low back pain," and he had "nearly fallen a couple of times." *Id*. He stopped exercising after his symptoms occurred; however, his symptoms did not improve, and he felt that the weakness and numbness had increased. *Id*. He denied having any illnesses over the past month. *Id.*

On exam, Dr. Rice observed that Petitioner's deep tendon reflexes ("DTRs") were "reduced to trace" and that he reported "decreased sharp sensation of all areas of legs bilaterally up to lower thighs." Ex. 2 at 177. He had "moderate foot pronation," walked somewhat stiffly, and had "difficulty standing on toes for more than just a few seconds." *Id*. Dr. Rice's assessment was bilateral leg weakness, and he urgently referred Petitioner to a neurologist for leg weakness, altered sensation, and decreased DTRs. *Id*.

The following day, September 14, 2017, Petitioner presented to neurologist Paul Burke, M.D., for "acute onset of bilateral leg weakness and numbness." Ex. 2 at 192. Petitioner reported that about two weeks earlier, he suddenly noticed bilateral numbness in his calves, which ascended his legs and was followed by weakness. *Id*. He also had dyspnea on exertion, which was new. *Id*. On exam, Petitioner had a wide-based gait and mute plantar responses; "2+ bilateral lower leg

edema"; "1+ [b]iceps, areflexic triceps, patellar and Achilles reflexes"; decreased strength in his lower extremities and temperature sensation loss; and sinus tachycardia[3] with a blood pressure of 150/71. *Id*. at 194. Dr. Burke performed an EMG/NCS, which was abnormal and showed "electrophysiological evidence of a length dependent motor and sensory axonal neuropathy or polyradiculoneuropathy." *Id*. at 194, 197.

Dr. Burke's assessment was acute motor and sensory axonal neuropathy, tachycardia, dyspnea on exertion, and edema of both legs. Ex. 2 at 194. He believed Petitioner had "acute inflammatory polyradiculoneuropathy such as an axonal variant of [GBS] or [acute motor sensory axonal neuropathy ("AMSAN")]," and he commented that "[p]ossibly the HPV vaccine 6 weeks ago was a trigger." *Id*. Other possible etiologies included West Nile virus, Zika virus, HIV seroconversion, Lyme disease, toxin exposures, or polyradiculopathy due to infiltrating processes such as lymphoma, sarcoid disease, or neoplastic disease. *Id*. Petitioner also had significant tachycardia, which Dr. Burke noted could be a sign of "instability which would be consistent with an inflammatory neuropathy." *Id*. at 194-95. After consulting with other pediatric neurologists, Dr. Burke sent Petitioner to Brenner's Pediatric Emergency Department ("ED"). *Id*. at 195.

That day, Petitioner presented to the ED for bilateral leg numbness, weakness, and swelling since his HPV vaccination.[4] Ex. 2 at 208. Petitioner reported that numbness began in his left calf and spread to his foot and knee and his lower right leg. *Id.* at 209. He had tingling and intermittent calf pain. *Id.* He had collapsed the day before due to sudden leg weakness. *Id*. He reported receiving the HPV vaccine on July 31 and an allergy shot one week prior to the onset of his symptoms. *Id*. He had no recent travel, tick exposure, or sick contacts. *Id.* On exam, he had decreased sensation to both legs and an impaired gait. *Id.* at 211. He was referred for a lumbar puncture and admitted. *Id.* at 212.

Later that day, Petitioner was seen by hospitalist Elizabeth Halvorson, M.D. Ex. 2 at 218. He explained that his leg numbness had begun two to three weeks earlier. *Id.* "It began in the left calf, then extended both distally to the foot and proximally to the knee." *Id.* It then spread to the right calf. *Id*. He also reported tingling in both feet. *Id.*

On exam, Petitioner had "bilateral [lower extremity] mild weakness, patellar reflex intact, difficult to elicit [A]chilles reflex, [lower extremity] sensation decreased, impaired gait, [and] bilateral pitting edema." Ex. 2 at 220. Dr. Halvorson noted that the two leading differential diagnoses for Petitioner were GBS and acute disseminated encephalomyelitis, which she

[3] Sinus tachycardia: tachycardia originating in the sinus node; it is normal during exercise or anxiety and occurs abnormally associated with shock, hypotension, hypoxia, congestive heart failure, fever, and various high output states. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=112009&searchterm=sinus+tachycardia (last visited July 15, 2025); Tachycardia: excessive rapidity in the action of the heart; the term is usually applied to a heart rate above 100 beats per minute in an adult and is often qualified by the locus of origin as well as by whether it is paroxysmal or nonparoxysmal. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=48715 (last visited July 15, 2025).

[4] The ED arrival account incorrectly stated the vaccination was given two weeks earlier; it was actually given 45 days earlier. Ex. 1 at 2; Ex. 2 at 208.

abbreviated as "ADM." *Id*. She commented that most GBS patients will report a preceding bout of respiratory or gastrointestinal symptoms, but Petitioner did not. *Id.* She further noted that immunizations can trigger GBS, and Petitioner had just received his "first" HPV vaccination and recent allergy shots. *Id.* She stated: "ADM is also a demyelinating disease that can occur both post-infectious and post-vaccination," but again, Petitioner did not experience "any prodromal symptoms prior to weakness." *Id*. at 221. ADM is most commonly associated with the measles, mumps, and rubella vaccine, which Petitioner did not receive. *Id*.

While hospitalized, Petitioner underwent an EMG and an MRI of the spine. Ex. 2 at 214. The MRI showed "showed moderate canal stenosis at L1-L2, advanced canal stenosis at L2-L3, and moderate to advanced stenosis of L4-L5"; these findings were "thought to be chronic in nature" and not related to Petitioner's symptoms. *Id*. at 214-15; 295. The EMG "showed de-axonal pattern consistent with axonal variant of [GBS]." *Id*. at 214-15, 433-37.

Petitioner's symptoms improved after five days of IVIG treatment and inpatient physical therapy ("PT"). Ex. 2 at 214-15. He was discharged on September 19, 2017, with a diagnosis of AMSAN. *Id.* at 214. At the time of discharge, he was ambulating well, experiencing fatigue only with extended effort. *Id.*

On September 20, 2017, Petitioner reported for outpatient PT. Ex. 2 at 462-73. He attended weekly PT for the next 12 weeks. *Id.* at 513-31, 546-52, 579-88, 636-50, 670-76, 716-22, 729-43, 762-71.

On September 29, 2017, Petitioner followed up with Dr. Rice. Ex. 2 at 502. Dr. Rice noted that Petitioner was recently diagnosed with GBS and underwent five days of IVIG treatment. *Id*. He was still "very limited with his mobility" and had difficulty going to school because of the distance between classes. *Id.* He had stayed home at the end of the week due to leg pain and weakness, and he fell once at home while walking on uneven terrain. *Id*. at 502-03. Dr. Rice recommended that Petitioner continue to be homeschooled because he did not have "the physical strength and coordination required to walk long distances, carry a book bag, and negotiate steps." *Id*. at 504. He also recommended against Petitioner taking that year's influenza ("flu") vaccine. *Id.*

On October 12, 2017, Petitioner followed up with Dr. Burke. Ex. 2 at 535. Petitioner reported that he had less numbness in his left foot than at his initial visit; however, his leg strength was unchanged, and he felt that he had developed a "new numbness in his hands." *Id*. at 537. On exam, his muscle bulk and tone were normal, and he had 4/5 strength in his right hip flexors, knee extensors, knee flexors, and bilaterally in his ankles. *Id*. at 538. He had vibratory and temperature sensation loss in his legs, his reflexes were areflexic, and his plantar responses were mute. *Id*. at 539. Dr. Burke recommended that Petitioner see the neurosurgical department to address his lumbar stenosis and discussed GBS and the recovery process. *Id.*

On October 23, 2017, Petitioner saw neurosurgeon Jonathan Lee Wilson, M.D., "for evaluation of likely incidentally documented L2 compression fracture with associated spinal stenosis." Ex. 2 at 557. Dr. Wilson noted that Petitioner "underwent this imaging during a workup for ongoing diagnosis of [GBS] presumed secondary to HPV vaccine" and had also undergone "an

EMG/PNCV and lumbar puncture with findings consistent with [GBS]." *Id*. Dr. Wilson reviewed the September 15, 2017 lumbar spine MRI and noted that it showed "an old compression deformity at L2 with about 50% anterior vertebral body height loss," as well as "mild to moderate central canal stenosis at the L 1-2 and L 2-3 levels." *Id*. at 560. The assessment was lumbar stenosis without neurogenic claudication and a compression fracture in the L2 lumbar vertebra. *Id*. at 561. Dr. Wilson opined that Petitioner's "symptoms fit the most with [GBS] versus resolving cauda equina syndrome." *Id*.

Petitioner followed up with Dr. Burke on October 26, 2017, for repeat NCV and EMG studies. Ex. 2 at 600. Dr. Burke noted that since his last visit, Petitioner had developed "increased weakness of ankle dorsiflexors and grip strength," as well as "more numbness of his hands and of his abdomen." *Id*. The exam was notable for "0/5 bilateral ankle dorsiflexion and 4/5 finger extensors, finger spreaders and finger flexors." *Id*. Compared to prior studies, Dr. Burke's impression was that Petitioner had worsening sensory and motor responses; however, there was some evidence of reinnervation occurring on the EMG, indicating improvement. *Id*. Dr. Burke opined that Petitioner's numbness and weakness was due to AMSAN, not lumbar polyradiculopathy. *Id*.

Petitioner saw Dr. Burke again on November 13, 2017. Ex. 2 at 654-57. On exam, he had decreased bilateral ankle dorsiflexion with bilateral foot drop, and he remained areflexic. *Id*. at 657. Dr. Burke continued to assess acute inflammatory polyradiculoneuropathy with axonal features, lumbar degenerative disc disease, and stenosis. *Id*. He recommended a second course of IVIG, which Petitioner completed on an outpatient basis from November 20-21, 2017. *Id*. at 687-88, 703-04.

On December 27, 2017, Petitioner followed up with Dr. Burke, noting that he had experienced improvement of strength in all four extremities since his second round of IVIG. Ex. 2 at 748. Dr. Burke recommended that Petitioner continue with PT. *Id*. at 750.

Petitioner finished outpatient PT on December 29, 2017. Ex. 2 at 766. He had made improvements in ankle strength and ambulation. *Id*.

On March 6, 2018, Petitioner saw Dr. Burke, who noted no "new issues." Ex. 2 at 797. The exam remained the same, and Dr. Burke recommended that Petitioner continue home PT. *Id*. at 799.

Petitioner followed up with Dr. Rice on December 17, 2018. Ex. 2 at 849-55. He had "regained 'most' of his strength," but he still had some areas of numbness in his calves. *Id*. at 850. He was able to walk without limp or limitation and could walk up steps without difficulty. *Id*. Dr. Rice noted that Petitioner developed GBS "about 7 weeks after HPV and MCV4." *Id*. at 854. Although Petitioner's exam was normal, Dr. Rice was concerned about his inactivity and social isolation. *Id*. at 853-54. He encouraged regular exercise and involvement in activities where Petitioner could engage with others. *Id*. at 854.

On May 30, July 10, and October 23, 2019, Petitioner saw Kimberly B. Wright, P.A., in the Novant Health Family Medicine clinic, for depression. Ex. 4 at 9-10, 37-38, 55-56. His

diagnosis was mild depression, and he was prescribed bupropion, which worked well. *Id*. at 11, 55.

On February 5, 2020, Petitioner saw P.A. Wright for an annual exam. Ex. 4 at 97-108. His exam was normal, with no complaints of numbness or weakness. *Id*.

## III. EXPERT EVIDENCE

### A. Norman Latov, M.D., Ph.D.: First Expert Report

Dr. Latov submitted three reports in this case. Ex. 12 ("First Latov Rep."); Ex. 37 ("Second Latov Rep."); Ex 53 ("Third Latov Rep.").

Dr. Latov earned his M.D. and Ph.D. from University of Pennsylvania School of Medicine in 1975 and is board certified in psychiatry and neurology. Ex. 13 ("Latov CV") at 1. He is currently a Professor in the Department of Neurology and Neuroscience at the Weill Medical College of Cornell University and an attending neurologist at New York Presbyterian Hospital. *Id.* at 2. He is a member of the American Neurological Association and American Academy of Neurology. *Id.* He serves as an editorial board member for *BMC Neurology*. *Id.* at 3. He has more than 200 publications related to autoimmune neuropathies in peer-reviewed journals. First Latov Rep. at 1. Dr. Latov's laboratory is "credited with the discovery of anti-MAG and anti-GM1 ganglioside antibodies, and the development of assays that are currently used for testing patients with suspected autoimmune neuropathies." *Id.*

Dr. Latov concluded that Petitioner suffered from GBS caused by the meningococcal and HPV vaccines he received on July 31, 2017. First Latov Rep. at 3. He opined that Petitioner's "clinical presentation, course, tests, and response to treatment were most consistent with the diagnosis of the axonal form of [GBS]." *Id*. He noted that Petitioner was diagnosed with GBS by his treating physicians. *Id*.

GBS is "an autoimmune disease that targets the peripheral nerves." First Latov Rep. at 3 (citing Mazen M. Dimachkie & Richard J. Barohn, *Guillain-Barré Syndrome and Variants*, 31 NEUROL CLIN. 491 (2013) (Ex. 16) ("Dimachkie & Barohn"). Following the 1976-76 swine flu vaccination program, vaccination became recognized as a trigger of GBS. *Id.* (citing Lawrence B. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, U.S., 1976-1977*, 110 AMERICAN J. EPIDEMIOLOGY 105 (1979) (Ex. 29) ("Schonberger"). The risk of developing GBS was found to be significantly elevated for ten weeks following vaccination. *Id.* Further, GBS has been reported "following other vaccinations that stimulate or modulate the immune system to cause autoimmune disease." *Id.* at 3-4 (citing Eitan Israeli et al., *Guillain-Barré Syndrome— A Classical Autoimmune Disease Triggered by Infection or Vaccination*, 42 CLINICAL REV. ALLERGY & IMMUNOLOGY 121 (2012) (Ex. 20) ("Israeli")).

Dr. Latov cited a 2006 paper published in the Centers for Disease Control's ("CDC's") Morbidity and Mortality Weekly Report ("MMWR"), which found a small increased risk for GBS after meningococcal vaccination. First Latov Rep. at 4 (citing *Update: Guillain-Barré Syndrome Among Recipients of Menactra Meningococcal Conjugate Vaccine – United States, June 2006-*

*September 2006*, 55 MORBIDITY AND MORTALITY WKLY. REP. 1120 (2006) (Ex. 34) ("MMWR 2006")). Regarding the HPV vaccine, he cited a large cohort study in France that "revealed an increased risk detected following vaccination." *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. 25) ("Miranda")). He also pointed out that the 1976 swine influenza ("flu") vaccine, seasonal flu vaccine, and tetanus vaccine had been associated with GBS in the literature. *Id.* at 3-4.

Dr. Latov explained that "[v]accines can induce autoimmune disease" via molecular mimicry, bystander activation, or a combination of both mechanisms. First Latov Rep. at 4 (citing COMMITTEE TO REVIEW ADVERSE EFFECTS OF VACCINES, ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al., eds., 2012) (Ex. 19) ("IOM 2012"). The Acute Motor Axonal Neuropathy ("AMAN") variant of GBS is an example of GBS caused by molecular mimicry, which has been reported to follow infection with *Campylobacter jejuni* ("*C. jejuni*"). *Id*. (citing Maojun Zhang et al., *Association Study Between an Outbreak of GBS in Jilin, China, and Preceding Cj Infection*, 7-8 FOODBORNE PATHOGENS & DISEASE 913 (2012) (Ex. 36) ("Zhang")). *C. jejuni* has lipopolysaccharides ("LPS") that are "similar in structure to gangliosides in the peripheral nerve, so that immune reactivity to the [*C. jejuni*] LPS during infection can cross react with gangliosides in peripheral nerve to cause an inflammatory neuropathy, as has been demonstrated in experimental animal studies." *Id.* (citing Caporale et al., *Experimental axonopathy induced by immunization with Campylobacter jejuni lipopolysaccharide from a patient with Guillain-Barré syndrome*, 174 J. OF NEUROIMMUNOLOGY (2006) (Ex. 15) ("Caporale")). Also, experimental allergic neuritis ("EAN") is an example of GBS caused by molecular mimicry following vaccination in experimental animals. *Id*. In humans, vaccination with the original Semple anti-rabies vaccine was found to induce GBS through molecular mimicry. *Id.* (citing Emanuel Appelbaum, Morris Greenberg & Jack Nelson, *Neurological Complications Following Antirabies Vaccination*, 151 J. AM MED. ASS'N 181 (1953) (Ex. 14) ("Appelbaum"); Thiravat Hemachudha et al., *Immunologic Studies of Rabies Vaccination-Induced Guillain Barré Syndrome*, 38 NEUROLOGY 375 (1988) (Ex. 18) ("Hemachudha"); Ramon S. Javier et al., *Semple Rabies Vaccine: Presence of Myelin Basic Protein and Proteolipid Protein and its Activity in Experimental Allergic Encephalomyelitis*, 93 J. NEUROLOGICAL SCI. 221 (1989) (Ex. 21) ("Javier")). Finally, molecular mimicry "has been shown to be responsible for narcolepsy following the pandemic 2009 H1N[1] influenza infection or vaccination with the Pandemrix vaccine, by cross reactivity with orexin (hypocretin) in neuronal cells in genetically susceptible individuals." *Id.* (citing Birgitte R. Kornum, Juliette Faraco & Emmanuel Mignot, *Narcolepsy with hypocretin/orexin deficiency, infections and autoimmunity of the brain*, 21 NEUROBIOLOGY 897 (2011) (Ex. 23) ("Kornum"); Guo Luo et al., *Autoimmunity to hypocretin and molecular mimicry to flu in type 1 narcolepsy*, 115 PNAS E12323 (2018) (Ex. 24) ("Luo")).

Dr. Latov further opined that bystander activation may occur under circumstances by which "infection or immunization can stimulate the immune system to overcome immune tolerance, allowing activation of the autoreactive cells, resulting in autoimmune disease." First Latov. Rep. at 5. He referenced several animal studies as examples of bystander activation causing EAE. *Id*. (citing Axel Nogai et al., *Lipopolysaccharide Injection Induces Relapses of Experimental Autoimmune Encephalomyelitis in Nontransgenic Mice via Bystander Activation of Autoreactive CD4+ Cells*, 175 J. OF IMMUNOLOGY 959 (2005) (Ex. 27) ("Nogai"); Pauline Soulas et al.,

*Autoantigen, innate immunity, and T cells cooperate to break B cell tolerance during bacterial infection*, 115 J. CLIN. INVEST. 2257 (2005) (Ex. 32) ("Soulas"); Joan Goverman et al., *Transgenic Mice that Express a Myelin Basic Protein-Specific T Cell Receptor Develop Spontaneous Autoimmunity*; 72 CELL 551 (1993) (Ex. 17) ("Goverman")). He pointed out that the meningococcal vaccine is "composed of oligosaccharides that are conjugated to diphtheria toxoid carrier protein, which is also an immune stimulant," and the HPV vaccine "uses amorphous aluminum hydroxyphosphate sulfate as adjuvant." First Latov Rep. at 5 (citing Markus Knuf *et al.*, *Comparative Effects of Carrier Proteins on Vaccine-Induced Immune Response*, 29 VACCINE 4881 (2011) (Ex. 22) ("Knuf")).

Addressing timing, Dr. Latov cited the Schonberger study, which found a significantly elevated risk of GBS in the flu-vaccinated population. First Latov Rep. at 5. The increased risk was greatest within the five-week period after vaccination but was present for approximately nine to ten weeks. *Id.* Petitioner's symptoms began approximately four weeks after vaccination, which was within the time frame of elevated risk. *Id.* Dr. Latov found no other potential causes that might have induced Petitioner's GBS. *Id.*

Dr. Latov concluded that Petitioner's GBS was caused by the subject HPV and meningococcal vaccines through either molecular mimicry, bystander activation, or a combination of both mechanisms. First Latov Rep. at 6.

**B. Andrew MacGinnitie, M.D., Ph.D.: First Expert Report**

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

Dr. MacGinnitie earned both his Ph.D. and M.D. from the Pritzker School of Medicine at the University of Chicago in 1996 and 1998, respectively. Ex. B ("MacGinnitie CV") at 1. He served as an Associate Professor at Harvard Medical School and as an attending physician at the Children's Hospital of Boston, specializing in Pediatrics Allergy/Immunology.[5] *Id.* at 2. He was also a consultant at DBV Technologies and Clinical Chief in the Division of Immunology at Boston's Children Hospital. *Id.* He was the Co-Director of Boston Children's Hospital Asthma Executive Committee; the Chairman of Boston Children's Hospital Allergy/Immunology Fellowship, Clinical Competency Committee; and a member of Boston Children's Hospital Clinical Operations Leadership Group, Clinical Outcomes Committee, Health Quality and Outcomes Forum, Physicians Organization Congress, Joint Satellite Oversight Committee, and Medical Peer Review Committee. *Id.* at 3. He is a member of the Clinical Immunology Society and Fellow at the American Academy of Allergy, Asthma, and Immunology. *Id.* at 4.

[5] Beginning in January 2024, after the record in this case was completed, Dr. MacGinnitie began a new position as a Pediatric Allergy Immunologist Specialist and Professor of Pediatrics at the Medical College of Wisconsin. *See* CHILDREN'S WISCONSIN, https://childrenswi.org/medical-professionals/latest-news/andrew-macginnitie-md (last visited Nov. 7, 2025).

Dr. MacGinnitie did not dispute the GBS diagnosis but concluded that Petitioner's GBS was unrelated to the HPV and meningococcal vaccines he received on July 31, 2017. First MacGinnitie Rep. at 5, 10. He disagreed that the mechanistic theories proposed by Dr. Latov reliably explained how the subject vaccines could have caused Petitioner's GBS. *Id.* at 5.

First, Dr. MacGinnitie opined that there is "no evidence of sequence or conformational homology between components of the HPV and meningococcal vaccines and proteins or other molecules expressed on human nerve tissues." First MacGinnitie Rep. at 5. Although he agreed that there is some evidence for molecular mimicry in human autoimmune disease, in those cases, the "pathogens [all] have carbohydrate sequences (antigens) in common with peripheral nerve tissue." *Id.* at 6 (quoting Robert K. Yu et al., *Ganglioside Molecular Mimicry and Its Pathological Roles in Guillain-Barré Syndrome and Related Diseases*, 74 INFECTION AND IMMUNITY 6517 (2006) (Ex. A, Tab 3) ("Yu")). Dr. Latov did not provide any evidence that there was "similar cross-reactivity between any component" of either vaccine received by Petitioner and human peripheral nerves. *Id.* at 6.

In any event, mere sequence similarity is not sufficient to show molecular mimicry as a trigger of autoimmune disease, because "there is substantial overlap between human and microbial proteins (and presumably carbohydrate and lipid antigens)." First MacGinnitie Rep. at 6 (citing Darja Kanduc et al., *Massive peptide sharing between viral and human proteomes*, 29 PEPTIDES 1755 (2008) (Ex. A, Tab 4) ("Kanduc") (finding that "each of 30 viral genomes studied had multiple instances of 5, 6 and even 7 amino acids peptides identical to human peptides."); Brett Trost et al., *Bacterial peptides are intensively present throughout the human proteome*, SELF/NONSELF 71 (2010) (Ex. A, Tab 5) ("Trost") (concluding that "all bacterial proteomes . . . share hundreds of nonamer sequences with the human proteasome.")). This point was also made by the IOM report on the adverse effects of vaccines. *Id.* (citing COMMITTEE TO REVIEW ADVERSE EFFECTS OF VACCINES, ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al., eds., 2012) (Ex. A, Tab 6) ("IOM 2011")).

Second, "Dr. Latov's arguments for influenza and rabies vaccine as triggers of GBS by analogy are flawed." First MacGinnitie Rep. at 7. The flu infection is itself a trigger of GBS, "making the idea that vaccination would also be a trigger much more plausible." *Id.* "This is not the case for HPV or meningitis[,] neither of which have been linked to development of GBS after wild-type infection[.]" *Id.* The Semple rabies vaccine included rabies-infected nerve tissue from other mammals. *Id.* Because there is significant homology between nerve proteins in humans and other mammals, "it is not surprising that this vaccine . . . could trigger autoimmunity against neural tissue." *Id.* "Neither HPV [nor] meningococcal vaccines contain tissue from other mammals." *Id.*

Third, the studies showing an increased risk of narcolepsy following H1N1 flu vaccination are of dubious relevance here. First MacGinnitie Rep. at 7. The H1N1 vaccine formulation associated with narcolepsy contained a powerful adjuvant, and the studies involving that formulation of the vaccine did not consistently show an increased risk of narcolepsy. *Id.* at 7-8. (citing Daniel Weibel et al., *Narcolepsy and Adjuvanted Pandemic Influenza A (H1N1) 2009 Vaccines – Multi-Country Assessment*, 36 VACCINES 6202 (2018) (Ex. A, Tab 11) ("Weibel")). Studies involving the non-adjuvanted formulation of the vaccine showed no association with

narcolepsy. *Id.* at 7. (citing Duffy et al., *Narcolepsy and influenza A(H1N1) pandemic 2009 vaccination in the United States*, 83 NEUROLOGY 1823 (2014) (Ex. A, Tab 9) ("Duffy"); Young J. Choe, Geun-Ryang Bae & Duk-hyoung Lee, *No association between influenza A(H1N1) pdm09 vaccination and narcolepsy in South Korea: An ecological study*, 30 VACCINE 7439 (2012) (Ex. A, Tab 10) ("Choe")). Also, "wild-type H1N1 infection has been shown to trigger narcolepsy, adding biologic plausibility to influenza vaccine as a trigger, similar to its association with GBS." *Id.* at 8 (citing Weibel; Fang Han et al., *Narcolepsy Onset is Seasonal and Increased Following the 2009 H1H1 Pandemic in China*, 70 AM. NEUROLOGICAL ASS'N 410 (2011) (Ex. A, Tab 16) ("Han")). Dr. MacGinnitie did not believe that a modest increase of incidence of narcolepsy following a specific influenza vaccine was "supportive of vaccination as a trigger GBS in this case."[6] *Id.*

Fourth, Dr. MacGinnitie opined that there is no evidence of bystander activation driving GBS in those receiving the subject vaccines. First MacGinnitie Rep. at 8. The studies referenced by Dr. Latov involved mice "genetically manipulated so that all the animal's T-cells express[ed] a specific T-cell receptor. Various immune stimuli, none similar to the vaccines [Petitioner] received, were able to activate these T-cells, thereby breaking tolerance." *Id.* (citing Goverman; Nogai; Soulas). Further, Petitioner did not report any symptoms consistent with inflammation after vaccination. *Id.* Therefore, "the vaccinations [Petitioner] received are not a notable inflammatory stimulus and there is no evidence that they would be sufficient to trigger 'bystander activation.'" *Id.* at 9. Also, Dr. Latov did not provide evidence for his assertion that diphtheria toxoid or alum are notably powerful immune stimulants; neither are known to be linked to autoimmune disease. *Id.*

Fifth, Dr. MacGinnitie argued that the "[e]pidemiology does not show an association" between the subject vaccinations and GBS. First MacGinnitie Rep. at 9. Dr. Latov omitted language from the 2006 article linking Menactra vaccination and GBS that acknowledged a larger study would be necessary to provide a more conclusive finding. *Id.* (citing MMWR 2006). Two larger studies have failed to provide evidence of an increased risk of GBS associated with the meningococcal vaccine. *Id.* (citing Priscilla Velentgas et al., *Risk of Guillain-Barré Syndrome After Meningococcal Conjugate* Vaccination, 21 PHARMACOEPIDEMIOLOGY AND DRUG SAFETY 1350 (2012) (Ex. A, Tab 23) ("Velentgas"); Weiling K. Yih et al., *No Risk of Guillain-Barré Syndrome Found After Meningococcal Conjugate Vaccination in Two Large Cohort* Studies, 21 PHARMACOEPIDEMIOLOGY AND DRUG SAFETY 1359 (2012) (Ex. A, Tab 24) ("Yih")).

[6] Dr. MacGinnitie also noted that further studies on the potential cross-reactivity between flu hemagglutinin protein and hypocretin, the protein suspected to be targeted in narcolepsy, have produced inconsistent results. First MacGinnitie Rep. at 8 (citing De la Herran-Arita et al., Retraction of the research article: *CD4+ T Cell Autoimmunity to Hypocretin/Orexin and Cross-Reactivity to a 2009 H1N1 Influenza A Epitope in Narcolepsy*, 6 SCI. TRANSLATIONAL MED. 247 (2014) (Ex. A, Tab 13) ("De la Herran-Arita"); Daniela Latorre et al., *T cells in patients with narcolepsy target self-antigens of hypocretin neurons*, 562 NATURE 63 (2018) (Ex. A, Tab 14) ("Latorre"); Syed S. Ahmed et al., *Antibodies to influenza nucleoprotein cross-react with human hypocretin receptor 2*, 7 SCI. TRANSLATIONAL MED 294 (2015) (Ex. A, Tab 15) ("Ahmed"); Fang Han et al., *Narcolepsy Onset Is Seasonal and Increased following the 2009 H1N1 Pandemic in China*, 70 ANN. NEUROL. 410 (Ex. A, Tab 16) ("Han")).

Similarly, although the Miranda study showed an increased risk of GBS after HPV vaccination in France, six other large studies "have shown no increased risk of GBS across a variety of countries indicating a true relationship is unlikely." *Id*.; *see* Nick Andrews et al., *No increased risk of Guillain-Barré syndrome after human papilloma virus vaccine: A self-controlled case-series study in England*, 35 VACCINE 1729 (2017) (Ex. A, Tab 25) ("Andrews"); Lisen Arnheim-Dahlstrom et al., *Autoimmune, neurological, and venous thromboembolic adverse events after immunisation of adolescent girls with quadrivalent human papillomavirus vaccine in Denmark and Sweden: cohort study*, BMJ 1 (2013) (Ex. A, Tab 26) ("Arnheim-Dahlstrom"); Julianne Gee et al., *Risk of Guillain-Barré Syndrome following quadrivalent human papillomavirus vaccine in the Vaccine Safety Datalink*, 35 VACCINE 5756 (2017) (Ex. A, Tab 27) ("Gee"); L. Grimaldi-Bensouda et al., *Autoimmune disorders and quadrivalent human papillomavirus vaccination in young female subjects*, 275 J. OF INTERNAL MED. (2014) (Ex. A, Tab 28) ("Grimaldi-Bensouda"); C. Chao et al., *Surveillance of autoimmune conditions following routine use of quadrivalent human papillomavirus vaccine*, 271 J. OF INTERNAL MED. (2011) (Ex. A, Tab 29) ("Chao"); Jozica Skufca et al., *The association of adverse events with bivalent human papilloma virus vaccination: A nationwide register-based cohort study in Finland*, VACCINE (2018) (Ex. A, Tab 30) ("Skufca").

Lastly, Dr. MacGinnitie noted that Petitioner did not have an adverse reaction to previous doses of the HPV or meningococcal vaccines. First MacGinnitie Rep. at 9-10. There is no evidence provided by Dr. Latov that explains why Petitioner received these vaccinations previously and did not have an adverse reaction. *Id.*

**C. Michael Kruer, M.D.: Expert Report**

Dr. Kruer submitted one report in this case. Ex. C ("Kruer Rep.").

Dr. Kruer earned his M.D. from the University of Arizona College of Medicine in 2004. Ex. D ("Kruer CV"). He is a board-certified pediatric neurologist and fellowship-trained clinical neuroimmunologist. Kruer Rep. at 1; Kruer CV at 1. Currently, Dr. Kruer is the Director of Cerebral Palsy and Pediatric Movement Disorders Program and the attending neuroimmunologist at the Barrow Neurological Institute at Phoenix Children's Hospital. Kruer CV at 1. His research includes the role of autoimmunity in neurologic disease, with a focus on autoantibodies directed against brain proteins that may lead to disease. Kruer Rep. at 1. He also serves on the editorial boards for the *Journal of Child Neurology* and *Pediatric Neurology* and is a reviewer for several journals. Kruer CV at 2. Clinically, Dr. Kruer has treated adults and pediatric patients with central nervous system and peripheral nervous system autoimmunity, including GBS. Kruer Rep. at 1.

Dr. Kruer noted that although Petitioner's symptoms were ultimately attributed to GBS, his course was "somewhat unusual" in that he "showed only equivocally elevated protein in his [cerebrospinal fluid ("CSF")], and his spinal cord MRI did not show nerve root enhancement as typical for GBS." Kruer Rep. at 2. Petitioner's physicians considered polyradiculopathy as an alternative diagnosis to GBS because of Petitioner's disappointing initial response to IVIG treatment. *Id*.

In response to Dr. Latov's citation of data connecting the 1976-77 swine flu and rabies vaccines to GBS, Dr. Kruer noted that "all vaccines are not created equal." Kruer Rep. at 2. GBS is not included on the Vaccine Injury Table for the HPV and meningococcal vaccines, and the MMWR article Dr. Latov cited "[did] not support a connection between the meningococcal vaccine and GBS." *Id*. at 2-3. Furthermore, studies done after the Miranda study in France did not show a significant association between GBS and HPV vaccination. *Id*. at 3.

Dr. Kruer questioned the relevance of the molecular mimicry theory in this case, "given that neither the meningococcal [n]or HPV vaccines have been clearly linked to GBS." Kruer Rep. at 3. Similarly, bystander activation was not a reliable causation theory "because bystander activation is typically considered insufficient to trigger autoimmune disease by itself."[7] *Id.*

Regarding the temporal relationship between the administration of the vaccines and GBS, Dr. Kruer opined that the four-week interval between vaccination and Petitioner's symptoms is "of uncertain significance" because a connection between the meningococcal and/or HPV vaccines and GBS "has not been clearly established." Kruer Rep. at 4. And the lack of evidence of alternative causation is not compelling because "only about two-thirds of GBS cases have a clear antecedent." *Id*.

**D. Dr. Latov: Supplemental Expert Report**

1. Response to Dr. Kruer's Expert Report

First, responding to Dr. Kruer's characterization of Petitioner's GBS diagnosis, Dr. Latov noted that all of Petitioner's physicians diagnosed him with GBS, and "[n]o other condition would have explained his illness." Second Latov Rep. at 1. Petitioner's CSF protein was elevated to a range consistent with GBS patients, his MRI was consistent with a diagnosis of axonal GBS, and he made significant improvement following his GBS diagnosis. *Id*. Petitioner's neurosurgeon and neurologist "agreed that polyradiculopathy was not the cause of his symptoms" and that the "electrodiagnostic testing was consistent with AMSAN." *Id*.

Second, although the Vaccine Injury Table does not include GBS following meningococcal or HPV vaccinations as a covered injury, "this does not prove that there is no association." Second Latov Rep. at 1. In general, the "available studies do not take into account the under reporting of adverse effects when data is voluntary, reliant on self-reporting, or retrospective, and there is no correction factor for under reporting." *Id*. (citing Anjana E. Sharma et al., *What Safety Events Are Reported For Ambulatory Care? Analysis of Incident Reports from a Patient Safety Organization*, JT COMM J QUAL PATIENT SAF., Author Manuscript (2020) (Ex. 49) ("Sharma"); David J. Cullen et al., *The Incident Reporting System Does Not Detect Adverse Drug Effects: A Problem for Quality Improvement*, 21 J. OF QUALITY IMPROVEMENT 541 (1995) (Ex. 39) ("Cullen"); Jesse A. Berlin et al., *Adverse Event Detection in Drug Development: Recommendations and Obligations Beyond Phase*, 98 AM. J. OF PUBLIC HEALTH 1366 (2008) (Ex. 38) ("Berlin"); *Kelly Estrada-*

[7] Dr. Kruer cited Yovana Pacheco et al., *Bystander activation and autoimmunity*, 103 J. AUTOIMMUNITY 102301 (2019), but Respondent did not file this article.

*Orozco, Hospital Adverse Event Reporting Systems: A Systemic Scoping Review of Qualitative and Quantitative Evidence*, 17 J. PATIENT SAF. 1866 (2021) (Ex. 41) ("Estrada-Orozco"). Furthermore, the fact that GBS is not included on the Table for the subject vaccines likely creates a bias against reporting, because the events would not be understood by doctors or patients to be causally linked. *Id*. In a related example, in California, reporting of adverse events to VAERS significantly increased after non-medical immunization exemptions were eliminated, "as there was then a reason for reporting." *Id*. (citing Anne M. Hause et al., *Association between Vaccine Exemption Policy Change in California and Adverse Event Reporting*, PEDIATR INFECT DIS J., Author Manuscript (2021) (Ex. 44)).

Third, in response to reports that patients given the meningococcal vaccine do not show a "higher incidence of GBS than the background rate in the normal population," Dr. Latov reiterated that current studies are "likely to underestimate" adverse incidents due to the under-reporting biases discussed above. Second Latov Rep. at 2.

Fourth, on the potential applicability of molecular mimicry, Dr. Latov explained:

> The Menac[t]ra vaccine is composed of bacterial oligosaccharides that are cross reactive with human glycolipids. In particular, the paragloboside and Nacetyllactosamine sequences, are present in both meningococcus oligosaccharides and peripheral nerve tissue, with paragloboside having been identified as an antigen for autoantibodies in a patient with [GBS], with the antibody titers falling following the acute illness.

*Id*. (citing Chao-Ming Tsai, *Molecular Mimicry of Host Structures by Lipooligosaccharides of Neisseria Meningitidis: Characterization of Sialylated and Nonsialylated Lacto-N-Neotetraose Structures in Lipooligosaccharides Using Monoclonal Antibodies and Specific Lectins*, 491 ADV. EXP. MED. BIOLOGY 525 (2001) (Ex. 51) ("Tsai"); Jane Dodd & Thomas M. Jessell, *Cell Surface Glycoconjugates and Carbohydrate-binding Proteins: Possible Recognition Signals in Sensory Neurone Development*, 124 J. EXP. BIOLOGY 225 (1986) (Ex. 40) ("Dodd & Jessell"); A.A. Ilyas et al., *Serum Antibodies to Gangliosides in Guillain-Barré Syndrome*, 23 ANN. NEUROL. 440 (1988) (Ex. 45) ("Ilyas")). Similarly, the HPV vaccine is a "recombinant vaccine, corresponding to the purified virus like particles of the major capsid L1 protein of HPV." *Id*. Dr. Latov noted that although cross reactivity with nerve tissue has not been investigated, "HPV has been reported to share peptide sequences with human proteins." *Id*. (citing Kanduc).

Lastly, Dr. Latov disagreed with Dr. Kruer's proposition that "bystander activation is typically considered insufficient to trigger autoimmune disease by itself." Second Latov Rep. at 2. In several animal studies, bystander activation was shown to trigger autoimmune disease. *Id*. Two human studies also showed autoimmunity triggered by this mechanism: (1) GBS and other autoimmune diseases were triggered by "check point inhibitors that boost immunity to cancer cells by interfering with signals that prevent autoimmunity"; and (2) autoimmune meningitis and encephalitis were triggered by "therapeutic OKT3 antibodies that stimulate autoreactive T-cells." *Id*. (citing Erum Khan *et al.*, *CNS and PNS manifestation in immune checkpoint inhibitors: a systemic review*, 432 J. OF NEUROLOGICAL SCIS. 1 (2022) (Ex. 46) ("Khan"); C. Sgro, *Side-effects*

*of a monoclonal antibody, muromonab CD3/orthoclone OKT3: bibliographic review*, 105 TOXICOLOGY 23 (1995) (Ex. 50) ("Srgo")).

2. Response to Dr. MacGinnitie's First Expert Report

In response to the comment that he failed to explain "why vaccination would have activated cells triggering peripheral nervous tissues and thereby triggered GBS as opposed to some other autoimmune disease," Dr. Latov noted that the "occurrence of any particular immune disease" is complex and depends on a variety of factors, including host susceptibility, genetic predisposition, immunological memory, antigen presenting molecules, T and B cell receptor repertoire, and environmental and triggering factors. Second Latov Rep. at 2-3. Also, "many patients do not present with multiple contemporaneous autoimmune diseases." *Id*. at 3. For example, checkpoint inhibitors can induce GBS or myasthenia gravis; however, they do not often occur in the same patient. *Id*.

Discussing the criticism that the studies relating to bystander activation do not concern vaccines, Dr. Latov noted that vaccination "provides an immune stimulus that can activate pre-existing autoreactive cells." Second Latov Rep. at 3. In the Goverman study, EAE was induced in mice that were injected with pertussis, an immune adjuvant that has a similar mechanism as the diphtheria toxoid carrier protein used in the Menactra vaccine. *Id.* (citing Goverman; S. Pecetta et al., *Carrier priming effect of CRM is related to an enhanced B and T cell activation in meningococcal serogroup A conjugate vaccination. Immunological comparisons between CRM and diphtheria toxoid*, 34 VACCINE 2334 (2016) (Ex. 47) ("Pecetta")).

In response to the remark that "all bacterial proteosome share hundreds of nonamer sequences with the human proteasome," Dr. Latov noted that "[t]he fact that autoimmunity does not always occur does not mean that it never occurs." Second Latov Rep. at 3. He explained that introducing a "cross reactive antigen" does not trigger autoimmunity on its own; autoimmunity requires a trigger such as active infection, immunization, or disruption of suppressor functions. *Id*. Moreover, the development of autoimmunity depends on additional individual factors such as major histocompatibility complex ("MHC") type, innate immunity, immunologic memory, existing T and B-cell receptor repertoire, and others. *Id*. For example, only a small percentage of people that received the Semple rabies vaccine that contains nerve tissue developed encephalomyelitis. *Id*.

On the topic of the Semple rabies vaccine, which contained animal nerve tissue, Dr. Latov stated that molecular mimicry occurs when there is a structural homology "between an exogenous agent, such as a vaccine or infection, and a self or auto-antigen that is subsequently targeted." Second Latov Rep. at 3. "Induction of immune reactivity against the foreign agent results in cross reactivity with the self-antigen, with subsequent tissue damage and autoimmune disease." *Id*. The Semple rabies vaccine had proteins that are "foreign," rather than "self," because it was grown in either "sheep or goat brain that has proteins that are similar, but not identical to human tissue." *Id*. This proved the concept of molecular mimicry. *Id*.

With respect to Dr. MacGinnitie's comment that the "relationship between narcolepsy and influenza vaccination is complex," Dr. Latov elaborated that the risk of narcolepsy "increased 5 to

14 fold in children and adolescents and 2 to 7 fold in adults" following the Pandemrix vaccine, noting that the patients did not have an H1N1 infection. Second Latov Rep. at 3. Although there is a debate regarding the specific antigen(s) that were targeted, the Pandemrix cases were all positive for "HLA class II," which is most consistent with an autoimmune etiology. *Id*. (citing Tomi Sarkanen et al., *Narcolepsy Associated with Pandemrix Vaccine*, 18 CURRENT NEUROLOGY & NEUROSCIENCE REPS. 43 (2018) (Ex. 48) ("Sarkanen")).

Dr. Latov reiterated that bystander activation is "a generally accepted scientifically plausible mechanism that would explain the development of [an] autoimmune disease following vaccination." Second Latov Rep. at 4. However, costly and impractical laboratory experiments would be required to determine whether bystander activation was responsible for Petitioner's GBS. *Id*.

In response to the comment that "vaccination is a minor stimulus," Dr. Latov explained that a sufficiently stimulatory vaccination that can induce an immune response against a foreign antigen can also induce an immune response against a self-antigen. Second Latov Rep. at 4. For example, the antigen in the swine flu or Semple rabies vaccines was sufficient to induce GBS in susceptible individuals. *Id*.

Lastly, in response to Dr. MacGinnitie's observation that Petitioner did not have an adverse reaction to previous dose of the same vaccines, Dr. Latov noted that "[i]mmunization can prime the immune system to a subsequent challenge with the same antigen." Second Latov Rep. at 4. He explained that immunological memory is the immune system's ability to "recognize an antigen that the body has previously encountered and initiate a corresponding immune response." *Id*. For example, keyhole limpet hemocyanin ("KLH") and *C. jejuni* lipopolysaccharide ("Cj-LPS"), both of which have the oligosaccharide GalGalNAc, do not induce anti-GalGalNAc antibodies by themselves; however, immunization with "KLH and later with Cj-LPS, results in generation of antibodies to GalGalNAc." *Id*. citing (Itzhak Wirguin *et al.*, *Induction of anti-GM1 ganglioside antibodies by Campylobacter jejuni lipopolysaccharides*, 78 J. OF NEUROIMMUNOLOGY 138 (1997) (Ex. 52) ("Wirguin")).

**E. Dr. MacGinnitie: Supplemental Expert Report**

Dr. MacGinnitie reiterated that there is "no evidence of molecular mimicry as a cause of disease in the case." Second MacGinnitie Rep. at 1. Although he concurred with Dr. Latov that molecular mimicry can trigger autoimmune disease in humans, Dr. MacGinnitie opined that there is no evidence of molecular mimicry in this case, and the evidence cited by Dr. Latov is not relevant here. *Id*. For example, the Semple rabies vaccine's ability to cause central nervous system ("CNS") disease is not relevant because of the "vast differences between injecting CNS homogenates from other species in the rabies vaccines" compared to "the highly purified proteins and carbohydrates in the" HPV and meningococcal vaccines. *Id*. Similarly, the Tsai article Dr. Latov cited focused on meningococcal lipopolysaccharide ("LPS"), which is not contained in Menactra and is therefore not relevant to this case. *Id*. at 2; *see* Tsai. The Dodd & Jessell article was about the expression of molecules on nerve tissue and made no mention of meningococcus or vaccines. *Id*.; *see* Dodd & Jessell. The Kanduc article demonstrated "massive overlap between

all viruses and human proteins and [did] not provide any support for molecular mimicry as a trigger of illness in this case." *Id*. at 3; *see* Kanduc.

As for Dr. Latov's bystander activation theory, Dr. MacGinnitie noted that the animal studies cited by Dr. Latov are not relevant here because the studies involved genetically manipulated mice "with T-cells expressing T-cell receptors known to target specific proteins." Second MacGinnitie Rep. at 3. Dr. MacGinnitie also reiterated that there is no evidence of significant immune activation in Petitioner after vaccination. *Id*.

Dr. MacGinnitie disagreed that Goverman, in which EAE was triggered in mice by injecting pertussis, was applicable here. Second MacGinnitie Rep. at 3. He disputed the contention that pertussis "has a similar mechanism of action as the diphtheria toxoid carrier protein used in the Menactra meningococcus vaccine." *Id.* He explained:

> First, while both act via a process called ADP-ribosylation, pertussis (PT) toxin acts by altering G-proteins which are keys in intracellular signaling, while diphtheria toxins (DT) blocks transcription of RNA into protein by binding to transcription factors. More importantly, Menactra contains diphtheria toxoid not DT. The toxoid has is denatured by treatment with formaldehyde which renders it biochemically inactive.

*Id*.[8]

Additionally, though Dr. Latov opined that GBS and other autoimmune diseases can be triggered by checkpoint inhibitors, Petitioner "was not treated with a checkpoint inhibitor or OKT3." Second MacGinnitie Rep. at 3. Moreover, checkpoint inhibitors are "signals across the entire immune system, and it is therefore not surprising that they could trigger autoimmunity."[9] *Id*. Further, the antibodies induced by OKT3 can cause a cytokine release syndrome ("CRS"). *Id.* By contrast, the vaccinations that Petitioner received "only contain specific proteins and carbohydrates designed to elicit a specific immune response against those components." *Id*. The adjuvant alum in Gardasil acts locally to increase the strength of the immune response, not to

[8] Dr. MacGinnitie cited Seema Mattoo & James D. Cherry, *Molecular pathogenesis, epidemiology, and clinical manifestations of respiratory infections due to Bordetella pertussis and other Bordetella subspecies*, 18 CLIN. MICROBIOL. REV. 326 (2005); R.K. Holmes, *Biology and molecular epidemiology of diphtheria toxin and the tox gene*, 181 J. INFECT. DIS. S156 (2000); and "Menactra Prescribing Information, 2018." Respondent did not file these materials.

[9] Dr. MacGinnitie cited Jennifer B. Jacob, Mark K. Jacob & Prahlad Parajuli, *Review of immune checkpoint inhibitors in immuno-oncology*, 91 ADV. PHARMACOL. 111 (2021), but Respondent did not file the article.

activate systemic immunity.[10] *Id*. Similarly, Petitioner did not receive OKT3 and "there is no evidence . . . that vaccination with Gardasil or Menactra can lead to CRS." *Id*. at 4.

Lastly, Dr. MacGinnitie opined that even if it is accepted that the influenza vaccine can trigger narcolepsy, it is "minimally informative" in this case because Petitioner received different vaccines and developed a different disease. Second MacGinnitie Rep. at 4. Further, the Sarkanen study that Dr. Latov cited in support of his interpretation was published "without consideration of the SOMNIA study, the most detailed study of any possible relationship" between the influenza vaccine and narcolepsy. *Id*. (referencing Sarkanen).

**F. Dr. Latov: Second Supplemental Expert Report**

Regarding molecular mimicry, Dr. Latov explained:

> The Menactra vaccine contains groups A, C, Y and W Meningococcal capsular polysaccharides. Serogroups W and V contain alternating sequence of D-galactose or D-glucose and sialic acid, and serogroup C contains sialic acid homopolymers. The D-galactose and sialic acid sequence is also present in several peripheral nerve gangliosides including GM1, GM3, GM2, GD1a, and GT1a, and sialic acid homopolymers are present in disialosyl gangliosides including GD3, GD2, GD1b, GT1b, Gb1b and GT1a, with gangliosides having been shown to be antigenic targets in GBS following C. jejuni infection.

Third Latov Rep. at 1 (citing Odile B. Harrison et al., *Description and Nomenclature of Neisseria meningitidis Capsule Locus*, 19 EMERGING INFECTIONS DISEASES 566 (2013) (Ex. 55); John A. Goodfellow & Hugh J. Willison, *Gangliosides and Autoimmune Peripheral Nerve Diseases*, 156 PROG. MOL. BIOL. TRANSL. SCI. 355 (2018) (Ex. 54)).[11] Although the question of whether the oligosaccharides in the Menactra vaccine or the homologous sequence in the HPV vaccine can induce autoimmune disease has not been thoroughly investigated or scientifically proven, he provided a "plausible" and "scientifically sound theory" that explains Petitioner's development of GBS following vaccination. *Id*.

Next, Dr. Latov noted that checkpoint inhibitor and OKT3 are examples of bystander activation causing diseases in humans and that he provided them as a response to the argument that "such mechanisms do not exist." Third Latov Rep. at 1. Additionally, he explained that cytokine release by itself "does not fully explain the clinical syndrome that followed treatment with OKT3." *Id*.

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[10] Dr. MacGinnitie cited, but Respondent did not file, Arnaud M. Didierlaurent et al., *AS04, an aluminum salt- and TLR4 agonist-based adjuvant system, induces a transient localized innate immune response leading to enhanced adaptive immunity*, 183 J. IMMUNOL. 6186 (2009).

[11] Dr. Latov also cited the Menactra Package Insert, but Petitioner did not file it.

Lastly, the case of Pandemrix influenza vaccine causing narcolepsy was cited as an example of molecular mimicry causing disease. Third Latov Rep. at 1. Notably, Dr. MacGinnitie recognized that molecular mimicry has been established as a mechanism for causing GBS following *C. jejuni* infection. *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.2 (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) ("It 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 do not dispute that Mr. Simmons developed GBS, and that diagnosis is supported by the medical records. The parties dispute whether the HPV and meningococcal vaccines he received on July 31, 2017, caused his GBS.

### A. *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 theories

Dr. Latov proffered two mechanistic theories: molecular mimicry and/or bystander activation. First Latov Rep. at 4.

*a. Molecular mimicry*

Dr. Latov explained that molecular mimicry involves a structural homology and cross-reaction between an exogenous agent and self. First Latov Rep. at 4. He cited the well-known theory that molecular mimicry between gangliosides and *C. jejuni* bacteria causes the AMAN subtype of GBS. *Id.*; *see* IOM 2012 at 71-72 ("One example of molecular mimicry as the likely mechanism causing clinical autoimmune disease is found in the subtype of [GBS] characterized by acute motor axonal neuropathy (AMAN). Approximately one fourth of patients with GBS have had [*C. jejuni*] infection in the preceding few weeks, compared to only 1-2 percent of controls."); Zhang at 918 (using serology to link *C. jejuni* infection to cases of GBS in China, and noting the cross-reactivity between several strains of *C. jejuni* and gangliosides involved in GBS). He also noted that EAN in animal models can be induced by immunization with peripheral nerve tissues. First Latov Rep. at 4; *see* Caporale at 17 (reporting that rabbits immunized with LPS containing GM1 and GD1a epitopes developed a strong anti-LPS and antiganglioside response and GBS-like symptoms); Soliven at 283-84 (discussing animal models of AMAN and related diseases induced by antibodies against glycolipids); Willison at 546 (discussing EAN animal model).

With respect to vaccine-induced neurological injury, Dr. Latov cited the Semple rabies vaccine as one that could induce GBS and encephalomyelitis via molecular mimicry. First Latov Rep. at 4. That cross-reaction was believed to be due to the presence of animal neural protein in the vaccine. *See* Javier at 228; Hemachudha at 376; Appelbaum at 188. Additionally, the 2009 H1N1 flu vaccine was associated with narcolepsy based on a purported cross-reaction with orexin in neuronal cells. First Latov Rep. at 4; Kornum at 900 (observing that molecular mimicry was one mechanism thought to cause observed cases of narcolepsy following H1N1 vaccination); Luo at E12323 (study concluding molecular mimicry between flu antigens and neuronal tissue was a likely cause of narcolepsy). Lastly, Dr. Latov generally asserted that the HPV vaccine contains purified virus particles, and the HPV virus has been reported to share peptide sequences with human proteins. Second Latov Rep. at 2 (citing Kanduc).

Dr. MacGinnitie did not dispute the notion that GBS and other human diseases can be caused via molecular mimicry, nor did he challenge that *C. jejuni* infection can cause GBS in this way. *See* Second MacGinnitie Rep. at 1 ("I concur with Dr. Latov that molecular mimicry can trigger human autoimmune disease, with GBS triggered by *C. jejuni* infection as one example for which there is substantial evidence."). He argued, however, that Dr. Latov's Semple rabies vaccine example was inapposite because the vaccine contained actual neuronal tissue from animals, making a cross-reaction with human tissue unsurprising. First MacGinnitie Rep. at 7. He further commented that the H1N1 vaccine/narcolepsy connection was unclear, because the vaccine was given during a pandemic and might have been administered to H1N1-infected individuals, confounding the analysis, and because a publication reporting a purported cross-reactive mimic (authored by the corresponding author in Luo) was retracted, and other studies investigating this hypothesis did not lead to a consensus. *Id.* at 8.

Dr. MacGinnitie also opined that, even if sequence similarity between the subject vaccines and host tissues were shown, that would not be sufficient to show cross-reactivity capable of causing autoimmunity, as "there is substantial overlap between human and microbial proteins (and presumably carbohydrate and lipid antigens)." First MacGinnitie Rep. at 6; *see also* Second

MacGinnitie Rep. at 3 (although Kanduc found homologies between HPV and human proteins, that study "demonstrate[d] massive overlap between all viruses and human proteins and [did] not provide any support for molecular mimicry as a trigger of illness in this case.").

Dr. MacGinnitie has offered reasonable critiques of Dr. Latov's position. Most notably, sequence homologies among human, viral, and bacterial proteins have been shown to be ubiquitous in nature, diminishing the weight of homology alone as proof of molecular mimicry-driven disease. *See* Kanduc at 1755 ("Importantly, the massive viral to human peptide overlapping calls into question the possibility of a direct causal association between virus-host sharing of amino acid sequences and incitement to autoimmune reactions through molecular recognition of common motifs."); Trost at 71 ("All bacterial proteomes, independent of their pathogenicity, share hundreds of nonamer sequences with the human proteome. This overlap is very widespread, with one third of human proteins sharing at least one nonapeptide with one of these bacteria."). I nonetheless conclude, however, that the molecular mimicry theory proffered by Petitioner is reliable for purposes of establishing *Althen* prong one in this case. The experts agreed that molecular mimicry is a recognized mechanism for GBS, including in the well-established example of *C. jejuni* infection. The evidence also demonstrated that molecular mimicry could explain vaccine-induced GBS, as in the case of the Semple rabies vaccine. As Dr. Latov persuasively explained, that vaccine's ability to induce neurological injury supported the molecular mimicry theory, because the immune system reacted to a foreign, but similar, antigen. Second Latov Rep. at 3.

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.").

*b. Bystander activation*

According to Dr. Latov, bystander activation may occur under circumstances by which "infection or immunization can stimulate the immune system to overcome immune tolerance, allowing activation of the autoreactive cells, resulting in autoimmune disease." First Latov Rep. at 5. He opined that the diphtheria toxoid conjugated to meningococcal vaccine, and/or the aluminum adjuvant in the HPV vaccine, could induce GBS in this way. *Id.* He referenced several animal studies as examples of bystander activation causing EAE, as well as cases of GBS in humans taking checkpoint inhibitors for cancer or using OKT3 antibodies to prevent allograft rejection. In Khan, for instance, GBS was reported as a common adverse event following use of checkpoint inhibitors. Khan at 6. In Sgro, meningitis and encephalitis were reported as side-effects of OKT3 treatment. Srgo at 26.

As Dr. MacGinnitie persuasively countered, however, there are meaningful distinctions between the mechanisms of these treatments and vaccines. Second MacGinnitie Rep. at 3-4. Checkpoint inhibitors are designed to block inhibitory signals across the entire immune system, which does not occur with vaccination. *Id.* at 3. Similarly, OKT3 antibodies are associated with rapid increases in inflammatory cytokines and can cause a cytokine release syndrome ("CRS"), leading to neurological complications; there is no evidence that the subject vaccines can cause CRS. *Id.* at 3-4; Srgo at 25 (discussing the clinical consequences of CRS). Overall, Dr. Latov's invocation of the bystander activation theory was less persuasive than his molecular mimicry theory.

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, *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 is appropriate to assess in a Vaccine Program case.

The parties produced epidemiologic data on the potential causal association between HPV vaccine and GBS. Dr. Latov cited 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.*

Dr. MacGinnitie noted that the Miranda study has been criticized because it relied on administrative data rather than patient medical records. First MacGinnitie Rep. at 9 (citing Gee). As noted, the Miranda authors acknowledged this limitation and reported that they used "broader ICD-codes including non-autoimmune disorders" to capture cases that might not have been properly coded. Miranda at 4767. They commented that this method might have resulted in an underestimation of the hazard ratios. *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 of the data 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. Neither Dr. MacGinnitie nor Dr. Kruer argued the study's methodology was invalid or that its findings should be discarded.

Respondent's experts offered several other studies as a counterweight to Miranda. The Andrews study retrospectively assessed the risk of GBS in 12-18-year-old girls in England. Andrews at 1729. The investigators collected data from the National Health Service, measuring the incidence of GBS within three months of HPV vaccination as compared to outside that time window. *Id.* at 1730. Approximately 10.4 million doses of HPV vaccine were given to the study population. *Id.* at 1731. The authors reported no significant association between HPV vaccination and the development of GBS in the relevant period. *Id.* In Arnheim-Dahlstrom, the authors investigated the association of a range of adverse events with HPV vaccination. Arnheim-Dahlstrom at 1. Like Miranda, the study was prospective, following more than 200,000 HPV-vaccinated girls for a 180-day period. *Id.* Unlike Miranda, though, this study did not specifically evaluate the potential association between GBS and HPV vaccination. Instead, it more broadly evaluated cases of "paralysis" and "neurological events" and found no significant association with HPV vaccination during the relevant period. *Id.*

In Gee, the authors 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.* Grimaldi-Bensouda was a case-control study that measured the risk of autoimmune diseases in HPV-vaccinated patients. Grimaldi-Bensouda at 398. The study recorded no GBS cases in those patients. *Id.* at 404 (Table 2). In Chao, about 189,000 HPV-vaccinated women were followed for 180 days to assess the risk of autoimmune conditions. Chao at 193. The study found no signal of potential harm for any autoimmune disease, including GBS. *See id.* at 197 (Table 1). Finally, in Skufca, the investigators performed a case-control study evaluating a number of adverse outcomes following HPV vaccination in Finnish girls. Skufca at 2. The vaccine was not statistically significantly associated with a higher risk of GBS, though a non-significant increased risk was found. *Id.* at 6 (Fig. 3).

These studies do provide 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 Tdap vaccination caused the petitioner to develop neuromyelitis optica ("NMO"). *Farrell v. Sec'y of Health & Hum. Servs.*, No. 19-301V, 2025 WL 2409187, *1 (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)).

This reasoning is persuasive. 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.

**B. *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).

In this case, the medical records revealed that Petitioner began experiencing symptoms near the beginning of September 2017, or about one month after his vaccinations. Ex. 2 at 192. There is no indication that he experienced any illness in the period before his symptoms developed, nor was there any evidence of any other cause of his GBS. *See id.* (denying to the neurologist any recent colds, flu, tick bites, sick contacts, or recent travel); *id.* at 176 (denying to his pediatrician any illnesses over the past month). The treating physicians focused on the HPV vaccine as a possible causal factor. Petitioner's neurologist, Dr. Burke, remarked: "Possibly the HPV vaccine 6 weeks ago was a trigger" of his GBS. *Id.* at 194, 197. Hospitalist Dr. Halvorson also noted that the "[o]nly recent change[] is that he had his first HPV shot 7/31." *Id.* at 218. She commented that immunizations are possible triggers of GBS and that Petitioner "did just receive his first HPV vaccine (7/31) and recent allergy shots." *Id.* at 220. PCP Dr. Rice recommended against Petitioner taking the flu vaccine that year. *Id.* at 504.[12]

[12] During Petitioner's emergency department visit on September 14, 2017, Elizabeth Mitchell Caudill, M.D., noted "HPV vaccine on 7/31, allergy shot one week prior to symptoms." Ex. 2 at 209. That same

Respondent argues that the clinical course fails to substantiate the mechanistic theories advanced by Petitioner for causation. Response at 15. He specifically notes that Petitioner did not complain of any symptoms after his vaccination indicating an unusual inflammatory response was occurring, calling into question the applicability of the bystander activation theory here. *Id.*; *see also* First MacGinnitie Rep. at 8. Respondent's experts did not, however, suggest that these facts show the molecular mimicry theory, which I have credited here, is inapplicable.[13]

Given the clinical course, treating physician comments, and absence of any alternative cause, I conclude that Petitioner has preponderantly proven *Althen* prong two.

### C. *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.

When he first saw Dr. Burke for his GBS symptoms on September 14, 2017, Petitioner reported experiencing symptoms about two weeks earlier. Ex. 2 at 192. The same day, Petitioner reported to hospitalist Dr. Halvorson that he began experiencing symptoms about two to three weeks before. *Id.* at 218. Petitioner averred in his affidavit that his symptoms began in early September. Pet. Affidavit at 1. These reports place the onset of symptoms from about 24 to 34 days after the subject vaccinations. Consistent with that, Dr. MacGinnitie stated that "it seems clear to me that [Petitioner] developed an axonal variant of GBS with onset roughly 2 weeks prior to his visit to his PCP, Dr. Rice on September 13, 2017 and one month after receipt of HPV and meningococcal vaccines." First MacGinnitie Rep. at 5.

Dr. Latov contended that the Schonberger study supported the onset of GBS for up to nine to ten weeks after flu vaccination, with the highest risk at the five-week mark. First Latov Rep. at 5. He opined that Petitioner's GBS onset fell within the appropriate timeframe after the subject vaccinations. *Id.* Neither Dr. Kruer nor Dr. MacGinnitie directly disputed this.[14] Instead, Dr.

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day, Benjamin Neil Lisle, M.D., noted: "[Petitioner] denies any recent infections, sick contacts, or surgeries, he does report having received the HPV vaccination 6w ago." *Id*. at 224. On October 12, 2017, Dr. Burke again noted that Petitioner "received the HPV vaccine on July 31st, 2017. *Id*. at 535. Petitioner's patient history from his October 23, 2017 visit with Jonathan Lee Wilson, M.D., noted that "[Petitioner] had the vaccine on 7/31/2017 and developed symptoms in his distal lower extremities including sensory/motor in early September." *Id*. at 557.

[13] Dr. MacGinnitie also noted that Petitioner had received prior doses of both of the subject vaccines and did not have any adverse reaction. First MacGinnitie Rep. at 9-10. Dr. Latov persuasively countered, however, that prior immunizations can sensitize the immune system and trigger a response to a future dose of the same antigen. Second Latov Rep. at 4 (citing Wirguin). This argument was not rebutted.

[14] For GBS following seasonal flu vaccination, the Vaccine Injury Table recognizes "3-42 days (not less than 3 days and not more than 42 days)" as an acceptable timeframe for onset. 42 C.F.R. § 100.3.

Kruer stated that the onset of Petitioner's symptoms was of "uncertain significance" because the causal connection between GBS and the subject vaccines had not been "clearly established." Kruer Rep. at 4. As noted, though, a Vaccine Program petitioner does not have the burden of showing a "clearly established" causal link between vaccine and injury. Thus, Dr. Kruer's comment is an unpersuasive rebuttal to Dr. Latov's opinion. As such, I conclude that 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