# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

**No. 20-1490V**

Filed: November 7, 2025

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PEGGY MCCARTER,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

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*Nancy R. Meyers, Esq.*, Turning Point Litigation, Greensboro, N.C., for petitioner.
*Sarah C. Duncan, Esq*., U.S. Department of Justice, Washington, D.C., for respondent.

## DECISION[1]

**Roth**, Special Master:

On October 28, 2020, Peggy McCarter ("petitioner") filed a timely petition pursuant to the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10 *et seq*.[2] ("Vaccine Act" or "the Program"). Petitioner initially alleged that she suffered from Guillain-Barré Syndrome ("GBS") as the result of an influenza ("flu") vaccine she received on October 24, 2018. Petition, ECF No. 1. She later filed an amended petition, alleging the flu vaccination caused her to develop polyneuropathy. Amended Petition, ECF No. 26.

Upon careful evaluation of all the evidence submitted, I find that petitioner has not provided preponderant evidence that the flu vaccine she received caused and/or contributed to the injury alleged.

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

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

## I. Procedural History

The petition was filed on October 28, 2020 and was assigned to the Special Processing Unit ("SPU") after petitioner filed her medical records. ECF Nos. 1, 9. Respondent filed his Rule 4(c) Report on April 15, 2021 recommending against compensation. ECF No. 14.

The case was reassigned to the undersigned on September 10, 2021. ECF No. 22. Petitioner filed her amended petition on April 20, 2022. ECF No. 26. She filed an expert report and medical literature on May 9, 2022. Petitioner's Exhibit ("Pet. Ex.") 10-26, ECF No. 29.

Respondent filed responsive expert reports on September 8, 2022, along with supportive medical literature. Respondent's Exhibit ("Resp. Ex.") A-D, ECF Nos. 31-37. Petitioner filed her supplemental expert report on October 24, 2022. Pet. Ex. 27, ECF No. 38. She filed additional medical literature on November 3, 2022. Pet. Ex. 28-38, ECF No. 39. Respondent filed his responsive expert reports on January 30, 2023. Resp. Ex. E-F, ECF No. 41.

A Rule 5 Conference was held on May 16, 2023. The parties were encouraged to discuss a reasonable resolution or advise whether they preferred a hearing or a Ruling on the Record. ECF No. 45.

On June 27, 2023, the parties filed a joint status report, advising they would like to proceed with a Ruling on the Record and requested a briefing schedule. ECF No. 46.

Petitioner filed her Motion for Ruling on the Record on October 10, 2023. Motion, ECF No. 48. Respondent filed his Response on November 16, 2023. Response, ECF No. 50. Petitioner filed a Reply on December 11, 2023. Reply, ECF No. 52.

The matter is now ripe for ruling.

## II. Factual Background

### A. Medical History Prior to the Flu Vaccine

Petitioner's medical history was significant for anxiety, hypertension, asthma, diverticulitis, vertigo, seasonal allergies, GERD, insomnia, cardiac pacemaker placement, venous insufficiency, sick sinus syndrome, obstructive sleep apnea, and depression. Pet. Ex. 2 at 2-28, 291, 311, 399, 429. In January of 2018, petitioner reported neck pain with some early left cervical radicular symptoms developing over the past week. Her primary care physician ("PCP") noted her history of lumbosacral back pain with radiculopathy.[3] *Id*. at 552. She was diagnosed with subacute maxillary sinusitis, left otalgia, and left cervical radiculopathy.[4] *Id.* at 552-53.

[3] Radiculopathy refers to disease of the nerve roots, such as from inflammation or impingement by a tumor or a bony spur. Radiculopathy, DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1547 (33rd ed. 2020) [hereinafter *Dorland's*].

[4] Cervical radiculopathy refers to radiculopathy of cervical nerve roots, often with neck or shoulder pain. Cervical radiculopathy, *Dorland's* at 1547.

She received the subject flu vaccine on October 24, 2018.[5] Pet. Ex. 2 at 2.

**B. Medical History Following the Flu Vaccine**

On October 26, 2018, petitioner contacted EMS with complaints of a two-day history of nausea, weakness, diarrhea, and fatigue. Pet. Ex. 3 at 14-15. EMS documented a slightly elevated temperature and lower extremity weakness. *Id.* She stated she received a flu shot on Wednesday and her symptoms began that evening. She was transported by EMS to Piedmont Medical Center for admission and was given Tylenol enroute. *Id.* at 15, 287. In the emergency room, she complained of bilateral leg aching and weakness when she tried to walk. She also reported a cough and nasal drainage. *Id*. at 287. The admission note documented a flu vaccine on Wednesday then feeling "generally ill" with body aches and fatigue by Thursday. *Id.* at 213, 287-91. On examination, she was alert, had normal speech and motor with no neurological deficits. She could not ambulate but could stand at the side of the bed. Lab work showed elevated white blood cell ("WBC") count. *Id*. at 289.

On October 27, 2018, petitioner was seen via telehealth by neurologist Dr. Renga. Pet. Ex. 3 at 218-19. Petitioner reported that she received flu and pneumonia vaccines on October 24, 2018 and began developing leg weakness and a burning sensation the following evening. She had a sore throat, vomiting, and a low-grade fever. She was unable to stand. The exam notes included "[s]ymmetric sensations" with sensation intact bilaterally. The impression was non-specific weakness. Her recent flu vaccination was noted, but GBS was a "low possibility." Dr. Renga recommended physical therapy ("PT") and diagnostic testing to rule out GBS. *Id.* at 219.

On that same day, the attending physician noted continued complaints of lower extremity weakness and pain with some paresthesia. Pet. Ex. 3 at 283. On examination, she had minimally decreased strength and deep tendon reflexes of both lower extremities but was unable to move her legs extensively; however, she was noted to be moving her legs under the blankets while conversing. *Id*. at 284. Her complaint of weakness was noted to be symmetric. *Id*. at 285. The impression was generalized weakness of unclear etiology. GBS was unlikely "given presentation . . . and lack of progression." *Id*.

Petitioner was evaluated by the PT that day for weakness and leg pain. Pet. Ex. 3 at 362. She had 3/5 bilateral lower extremity strength. *Id*. at 364. The PT wrote that she "[a]ppeared inconsistent/self-limiting at times" but was agreeable to PT. *Id*. at 363. The PT notified the RN that a psychiatric evaluation may be needed if the inconsistent and self-limiting behaviors continued. *Id*. at 363, 366.

Two days later, on October 28, 2018, petitioner had significantly improved without any treatment and was able to walk. Etiology remained unclear. Pet. Ex. 3 at 280-82.

On October 29, 2018, she complained of shortness of breath, severe lower extremity weakness, dizziness, lightheadedness, and "reaction to her influenza vaccine on her left arm with pain". WBC count had normalized, and neurological exam was normal. Pet. Ex. 3 at 277-79. Spinal CT scans showed degenerative changes in the lumbar spine. *Id*. at 227, 392-95. The physician

[5] She also received a pneumonia vaccine, which is not the subject of this claim.

noted the PT's suspicion for self-limiting behavior because of the inconsistencies in strength deficits. *Id*. at 279. GBS was noted as "highly unlikely" because she did not have progressive, symmetrical weakness. She refused a lumbar puncture and could not undergo spinal MRI because she had a pacemaker. Her symptoms were thought to be secondary to a viral illness, given her vomiting and abdominal pain; functional symptomology could not be ruled out. *Id*.

On October 30, 2018, petitioner transitioned to Encompass for short-term rehabilitation for neurologic conditions and "[d]isuse" myopathy.[6] Pet. Ex. 4 at 37; Pet. Ex. 3 at 227. She reported that her weakness and fatigue began the evening of vaccination and progressed to pain and numbness by Friday when she presented to the hospital. *Id*. On examination, she had difficulty standing without assistance, her left patellar and Achilles reflexes were +2, and she had trace right patellar and Achilles reflexes. Pet. Ex. 4 at 204. She reported that her upper extremity weakness had resolved, but she still had residual lower extremity weakness worse on the right. *Id*. at 195. The impression included "weakness possibly secondary to a viral prodrome". *Id*. at 207. She reported improvement by November 5, 2018, was walking with a rolling walker, and had modified independence for transfers and ambulation. She showed 4+/5 strength in her right leg and 5-/5 in her left leg. *Id.* at 161. Petitioner was discharged home on November 6, 2018, with a diagnosis of acute myopathy. *Id.* at 64.

Petitioner began outpatient PT on November 12, 2018. Pet. Ex. 5 at 2. At her initial session, she reported that she woke up on October 26 and was unable to feel or move her legs. She thought she had regressed since her hospital discharge with difficulty standing from a chair, climbing stairs, walking, dressing herself, and being easily fatigued. She reported 6/10 pain in her right hip, thigh, and lower leg. *Id.* She had 22 PT sessions through February of 2019. *Id.* at 12-77.

Petitioner presented to her PCP, Dr. Tuttle, on November 19, 2018. She reported receipt of a flu shot on October 24 and "within 2 days developed pain and weakness of her lower extremities", was hospitalized for GBS, with a telehealth neurology visit confirming the diagnosis. She declined a lumbar puncture. At the time of the visit, she was using a walker but was mostly wheelchair-bound. Pet. Ex. 2 at 751-52.

On November 28, 2018, petitioner was evaluated as a new patient by neurologist Dr. Reeves. Pet. Ex. 2 at 771. She reported receipt of a flu shot on a Wednesday, extreme fatigue by Thursday, and an inability to move from the neck down on Friday morning. She reported being unable to walk when she was discharged from the hospital. *Id*. She had diffuse weakness in the proximal and distal upper extremities and in the lower extremities on examination. *Id.* at 772. Light touch sensation was intact. Pinprick sensation was decreased below the knee on the right leg, in the left foot, and in the fingertips of the right hand. *Id*. The impression was "acute onset of weakness", improving. *Id.* at 773. It was noted that petitioner's condition was "not the slowly ascending weakness typically seen with [GBS]" but GBS was not ruled out given her history. An EMG study was recommended. Dr. Reeves also noted that if petitioner "does have evidence of GBS, likely proceed with IVIG to help with her recovery." *Id.*

Petitioner returned to Dr. Reeves for EMG testing on December 26, 2018. Pet. Ex. 6 at 6. She was slowly improving, was able to walk unassisted and no longer had weakness, but she had

[6] Myopathy is any disease of a muscle. Myopathy, *Dorland's* at 1206.

decreased balance. *Id*. Dr. Reeves interpreted the EMG as consistent with "generalized demyelinating sensory peripheral neuropathy."[7] *Id.* at 7. Dr. Reeves noted the symptoms may be residual from GBS, although there was no obvious motor involvement on EMG, and her symptoms had nearly completely resolved. She was to continue with PT and vitamin B-12. Dr. Reeves' plan did not include a recommendation for IVIG. *Id*.

On February 22, 2019, petitioner presented to Dr. Tuttle. Pet. Ex. 2 at 840-41. Dr. Tuttle noted a mild case of GBS after receiving a flu vaccination and that she was doing well. She was walking unassisted. She had some paresthesia and decreased sensation in her right hand and arm and paresthesia and tenderness in her right leg. *Id*.

On March 26, 2019, petitioner followed up with Dr. Reeves, who noted "generalized demyelinating sensory peripheral neuropathy with no evidence of motor involvement on EMG but with some weakness." On examination, she had continued, mild right-side weakness "with some inconsistency" not present on the left. Pet. Ex. 6 at 4-5.

Petitioner returned to PT on June 12, 2019, and reported improvement in energy and endurance following medication and pacemaker adjustment. She also reported some new pain on her right shoulder blade. Pet. Ex. 5 at 96.

On June 18, 2019, petitioner followed up with Dr. Reeves. Pet. Ex. 6 at 2. The visit notes included that an EMG in December of 2018 showed demyelinating sensory neuropathy but no motor abnormalities. It was also noted that the EMG was performed a few months after her initial presentation. She had some residual weakness but only on the right side. On examination, her motor strength revealed weakness of 4+/5 on the right with "some inconsistency". *Id.* The impression included "evidence of sensory nerve damage on EMG." IVIG was not ordered. *Id*. at 3.

Roughly seven months later, on January 20, 2020, petitioner returned to the neurology clinic and was seen by Dr. Mandell for "possible post-vaccine neuropathy." Pet. Ex. 7 at 2. She provided a history of flu vaccine then developing numbness and weakness in her arms and legs three days later. She also reported some residual numbness in her hands, a tendency to drop things, and right leg weakness. *Id.* On examination, she had decreased sensation to pinprick in the toes of her right foot and her vibratory sense was reduced in the right foot compared to the left. *Id*. at 3. Her reflexes were 3 in the right arm, 2+ in the left arm, and 2+ bilaterally at the knees and ankles. The impression included weakness and numbness three days post-vaccination. *Id*. Dr. Mandell noted that the "temporal sequence would seem to implicate the vaccine", but a three-day onset is very quick where "normally it would take days to weeks." He added that petitioner's reflexes were well-preserved and "brisk", but he was not sure what her reflexes were when she presented in October of 2018. *Id*. He wrote that "post vaccine [GBS]/demyelinating neuropathies are extremely rare but do occur." *Id*. at 4.

Petitioner underwent repeat EMG on February 21, 2020, which showed mild sensory deficits. Dr. Mandell included petitioner's history of bilateral carpal tunnel. He opined that

[7] Sensory peripheral neuropathy is a functional disturbance or pathologic change of sensory nerves in the peripheral nervous system. Neuropathy, *Dorland's* at 1250; Sensory neuropathy, *Dorland's* at 1252.

petitioner most likely had axonal polyneuropathy that "she seemed to get acutely a few days after [the] vaccine", but the "[c]ause and effect is unclear." Pet. Ex. 7 at 5.

### C. Petitioner's Affidavit

Petitioner affirmed she was very active prior to receiving the subject vaccine, walking three miles daily, swimming, and skiing. Pet. Ex. 9 at 2. She received prior flu vaccines without event. *Id*.

She received the subject flu vaccine on the morning of October 24, 2018, as well as a pneumonia vaccine. She did not have any side effects that day. Pet. Ex. 9 at 2. The next day, she recalled being a "little more fatigued than normal" but still felt fine. The following day, though, she woke up with no feeling in her legs. Her husband called EMS. She denied any fever, nausea, or diarrhea. *Id*.

When she arrived at the hospital, she was unable to stand because she could not feel her legs. She was admitted and "attended to during [her] stay by a PA" and only "spoke with a neurologist [] via the TV." Pet. Ex. 9 at 2.

## III. Expert Opinions

### A. Petitioner's Expert, Dr. Steinman[8]

Dr. Steinman opined that the components of the 2018-2019 influenza vaccine initiated an immune response that cross-reacted via molecular mimicry to antigens known to be targeted in polyneuropathy. He further opined that a two-day onset of symptoms is medically reasonable. Pet. Ex. 10 at 1, 6-7.

Dr. Steinman summarized petitioner's medical history beginning with her presentation to the hospital on October 26, 2018 and discharge to inpatient rehabilitation on October 30, 2018 with a diagnosis of generalized weakness. Pet. Ex. 10 at 4-5. Dr. Steinman included petitioner's visit to Dr. Reeves on November 28, 2018, at which Dr. Reeves noted that petitioner's symptoms were not typical of GBS but "it is possible since there was no active viral infection". *Id*. at 5-6; Pet. Ex. 6 at 8-10. An EMG performed on December 26, 2018 showed findings consistent with generalized sensory polyneuropathy. Pet. Ex. 10 at 6; Pet. Ex. 6 at 15-22. At a visit with Dr. Mandell in January of 2020, Dr. Mandell discussed whether petitioner's symptoms were "vaccine related or not", with the temporal sequence seemingly implicating the vaccine, although the onset was quite fast. Pet. Ex. 10 at 6; Pet. Ex. 7 at 2-4. A repeat EMG on February 21, 2020 showed axonal polyneuropathy. Pet. Ex. 10 at 6; Pet. Ex. 7 at 5.

Dr. Steinman opined that petitioner's diagnosis was polyneuropathy, consistent with her treating providers. Pet. Ex. 10 at 6. He added that the Brighton criteria used for diagnosing GBS has limitations, as discussed in *Sejvar*. Pet. Ex. 27 at 1-2; Resp. Ex. A Tab 1.[9] While useful as a

[8] Dr. Steinman received his M.D. from Harvard University in 1973. Pet. Ex. 14. He is a board-certified neurologist and is a Professor of Neurology and Pediatrics at Stanford University. Pet. Ex. 10. He is well known to the Court.
[9] James J. Sejvar et al., *Guillain–Barré syndrome and Fisher syndrome: Case definitions and guidelines*

guide, the Brighton criteria do "not fit the reality of the treating physicians who must weigh all the data in their medical practice in evaluation of this actual case." *Id*. He also addressed petitioner's EMG/NCS findings submitting that the electromyographer found the findings consistent with generalized sensory polyneuropathy. Pet. Ex. 10 at 24; Pet. Ex. 27 at 2.

Therefore, Dr. Steinman argued that petitioner's case "most closely resembles" acute sensory demyelinating polyneuropathy, a variant of GBS. He relied on *Uncini & Yuki* to explain that over the last decade some patients have presented with normal reflexes and normal NCS studies but were still thought to "have a possible GBS variant." Pet. Ex. 27 at 2-5; Pet. Ex. 28.[10] *Uncini & Yuki* observed 22 patients with acute onset paresthesias, only two of whom did not have absent or diminished tendon reflexes, and only 5 who responded to IVIG, steroids, or plasma exchange. Pet. Ex. 28. Thus, the preservation of reflexes and the fact that the treating physicians did not attempt immunotherapy were not "reason[s] to exclude this diagnosis". Pet. Ex. 27 at 4.

Dr. Steinman disagreed that petitioner more likely had conversion disorder. Pet. Ex. 27 at 5. He expressed his hesitancy with a diagnosis of conversion disorder because its history "is fraught with implications about 'hysteria.'" Plus, he would not overrule a diagnosis made by treating providers. *Id*.

For *Althen* prong one, Dr. Steinman submitted that the flu vaccine can trigger GBS via molecular mimicry between the vaccine and myelin basic protein and/or contactin-1. He explained that molecular mimicry is a cross-reactive response, wherein T cells that routinely respond to viruses begin to attack a corresponding self-component. Pet. Ex. 10 at 7; Pet. Ex. 19.[11] It is one of the main causal mechanisms proposed in the pathogenesis of GBS. Pet. Ex. 27 at 4-5; Pet. Ex. 29.[12] This response can occur even in the absence of exact homology. Pet. Ex. 10 at 10; Pet. Ex. 19. In fact, the researchers in *Gautam III*[13] were able to induce paralysis in animal models where only 5 out of 10 amino acids were identical. Pet. Ex. 24.[14] *Fujinami & Oldstone* induced experimental encephalomyelitis ("EAE") in animals where the hepatitis B virus polymerase shared only 6 amino acids with myelin basic protein. Pet. Ex. 39.[15] Dr. Steinman has been involved in studies where they were able to trigger EAE with only 4 of 11 (*Gautam II*) and 5 of 11 (*Gautam I*) amino acids being identical. Pet. Ex. 10 at 10-12; Pet. Ex. 22;[16] Pet. Ex. 23.[17] Finally, he

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*for collection, analysis, and presentation of immunization safety data*, 29 Vaccine 599 (2011), filed as "Resp. Ex. A Tab 1".

[10] Antonino Uncini, MD & Nobuhiro Yuki, MD, PhD, *Sensory Guillain-Barre syndrome and related disorders: an attempt at systemization*, 45 Muscle & Nerve 464 (2012), filed as "Pet. Ex. 28".

[11] Lawrence Steinman, *Autoimmune Disease*, 269 Sci. Am. 106 (1993), filed as "Pet. Ex. 19".

[12] Nobuhiro Yuki, *Guillain-Barre syndrome and anti-ganglioside antibodies: a clinician-scientist's journey*, 88 Proc. of Japan Acad., Series B 299 (2012), filed as "Pet. Ex. 29".

[13] Petitioner filed three articles with the lead author listed as Anand Gautam. For ease of reference herein, Exhibit 22 is referred to as *Gautam I*; Exhibit 23 is referred to as *Gautam II*; and Exhibit 24 is referred to as *Gautam III*.

[14] Anand M. Gautam et al., *A polyalanine peptide with only five native myelin basic protein residues induces autoimmune encephalomyelitis*, 176 J. Experimental Med. 605 (1992), filed as "Pet. Ex. 24".

[15] Robert S. Fujinami & Michael B. A. Oldstone, *Amino acid homology between the encephalitogenic site of myelin basic protein and virus: mechanism for autoimmunity*, 230 Sci. 1043 (1985), filed as "Pet. Ex. 39".

[16] Anand M. Gautam et al., *A viral peptide with limited homology to a self-peptide can induce clinical signs of experimental autoimmune encephalomyelitis*, 161 J. Immunology 60 (1998), filed as "Pet. Ex. 22".

[17] Anand M. Gautam et al., *Minimum structural requirements for peptide presentation by major histocompatibility complex class II molecules: Implications in induction of autoimmunity*, 91 Proc. of Nat'l Acad. of Sci. USA 767

referenced *Lanz* which demonstrated molecular mimicry between EBNA-1 and a protein called GlialCAM as a trigger for multiple sclerosis ("MS"). Pet. Ex. 10 at 12-15; Pet. Ex. 11;[18] Pet. Ex. 27 at 12. Further, these studies showed that the amino acids do not need to be consecutive to induce neuroinflammation. Pet. Ex. 10 at 11.

Through his searches, Dr. Steinman was able to find homologies between the flu vaccine and myelin basic protein ("MBP") and contactin-1 associated with autoimmune responses in GBS and inflammatory neuropathy. Pet. Ex. 10 at 15-21; Pet. Ex. 20;[19] Pet. Ex. 21;[20] Pet. Ex. 27 at 11-12. He first identified the components of the 2018-2019 flu vaccine, then used BLAST followed by an Immune Epitope Database ("IEDB") search to identify the following homologies with MBP: GYAADLKS (5 of 8 identical amino acids) and GTCYPGDFIDY (5 of 11 identical amino acids). He used the same process to identify the following homologies with contactin-1, "known to be targeted in GBS": RVRDQRGNVLL (6 of 11 identical amino acids) and LIIGKEDRRYGP (5 of 12 identical amino acids). Pet. Ex. 10 at 7, 15-21; Pet. Ex. 18;[21] Pet. Ex. 21; Pet. Ex. 27 at 17-19.

He conceded that sequence homology alone is insufficient to trigger molecular mimicry. Pet. Ex. 27 at 13. However, he was involved in a clinical trial where "we tested injection of molecular mimics into humans, and it caused widespread unwanted immune reactions, including worsening of disease", forcing them to stop the clinical trial. *Id*. at 14-15; Pet. Ex. 36.[22] Without doing research on actual human specimens, Dr. Steinman explained that the results he provided are the best available evidence to show how a potential mimic in the vaccine could cause the disease in question. Pet. Ex. 27 at 14.

He defended the literature he cited in support of his theory. Pet. Ex. 27 at 15; Pet. Ex. 11;[23] Pet. Ex. 22;[24] Pet. Ex. 23;[25] Pet. Ex. 24.[26] Specifically, he explained that *Lanz* did not have to rely on BLAST searches because they "actually had human specimens . . . to work with." His approach in this case "is applied to analysis of molecular mimics, when we are unable to get actual specimens." Pet. Ex. 27 at 16; Pet. Ex. 11.

Based on the literature he provided, the homology he found between the subject vaccine and MBP and contactin-1 is sufficient to "provide a meaningful clinical neuroinflammatory response". Pet. Ex. 10 at 16. Additionally, the mimics were identified on the IEDB, a resource

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(1994), filed as "Pet. Ex. 23".

[18] Tobias V. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis Bind EBV EBNAI and GlialCAM*, Nature doi:10.1038/s41586-022-04432-7 (2022), filed as "Pet. Ex. 11".

[19] R.A.C. Hughes et al., *Immune responses to myelin antigens in Guillain-Barré syndrome*, 6 J. Neuroimmunology 303 (1984), filed as "Pet. Ex. 20".

[20] Yumako Miura et al., *Contactin 1 IgG4 associates to chronic inflammatory demyelinating polyneuropathy with sensory ataxia*, 138 Brain 1484 (2015), filed as "Pet. Ex. 21".

[21] Centers for Disease Control and Prevention, Frequently Asked Flu Questions 2018-2019 Influenza Season, filed as "Pet. Ex. 18".

[22] Ludwig Kappos et al., *Induction of a non-encephalitogenic type 2 T helper-cell autoimmune response in multiple sclerosis after administration of an altered peptide ligand in a placebo-controlled, randomized phase II trial*, 6 Nature Med. 1176 (2000), filed as "Pet. Ex. 36".

[23] Lanz et al., *supra* note 18.

[24] Gautam et al., *supra* note 16.

[25] Gautam et al., *supra* note 17.

[26] Gautam et al., *supra* note 14.

provided by the National Institute of Health, as key targets of the immune response to the flu vaccine. *Id*. at 17-21. Taken together, they support that flu vaccine could trigger polyneuropathy. *Id*. at 24-25; Pet. Ex. 27 at 20.

For *Althen* prongs two and three, Dr. Steinman argued that a two-day onset was medically reasonable and is characteristic of a recall response to petitioner's prior flu vaccinations. Pet. Ex. 10 at 24. Relying on *Schonberger* which studied the 1976 swine flu vaccination campaign, he submitted that a recall response can produce neurologic symptoms between 2-4 days. *Id*.; Pet. Ex. 26.[27]

Dr. Steinman also discussed *Langmuir*, noting that *Langmuir* included data in weekly intervals, while *Schonberger* used 2-day intervals. Pet. Ex. 27 at 6-8. He submitted that petitioner's rapid onset of symptoms was medically reasonable due to a recall response, having received several prior flu vaccines. *Id*. at 8; Pet. Ex. 30.[28] *Tan* "show[ed] that influenza vaccination induces the recall of memory B cells that express antibodies that previously underwent affinity maturation against prior years' seasonal influenza". Pet. Ex. 27 at 10; Pet. Ex. 32.[29] Further, *Kardjito & Grange* studied skin test reactivity to tuberculin with the "most marked" reactions occurring at 24 hours. Pet. Ex. 27 at 9-10; Pet. Ex. 31.[30] Similarly, *Lai* showed that recall responses can occur as early as 6 hours after an antigenic challenge. Pet. Ex. 27 at 10; Pet. Ex. 33.[31]

In sum, Dr. Steinman believed "[t]he timing is correct" and onset within "approximately two days" post-vaccination is reasonable given that she had at least five prior flu vaccines, setting her up for a recall response. Pet. Ex. 27 at 20-21.

### B. Respondent's Expert, Dr. Callaghan[32]

Dr. Callaghan opined that petitioner did not have GBS or axonal neuropathy but likely conversion disorder. Resp. Ex. A at 5.

Dr. Callaghan detailed petitioner's medical history following the vaccine. He noted that throughout petitioner's hospitalization in October of 2018, providers documented GBS as a "low possibility" and that "functional symptomatology" could not be excluded. The records also included her ability to move her legs while conversing but an inability to do so when examined. Further, the PT records include some self-limiting behaviors. Her symptoms improved without

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[27] Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. Epidemiology 105 (1979), filed as "Pet. Ex. 26".

[28] S. Fazekas de St. Groth, M.D. & R.G. Webster, Ph.D., *Disquisitions on Original Antigenic Sin*, 124 J. Experimental Med. 331 (1966), filed as "Pet. Ex. 30".

[29] Yann-Chong Tan et al., *Sequencing Antibody Repertoires Provides Evidence for Original Antigenic Sin Shaping the Antibody Response to Influenza Vaccination*, 151 Clinical Immunology 55 (2014), filed as "Pet. Ex. 32".

[30] T. Kardjito & J.M. Grange, *Immunological and Clinical Features of Smear-Positive Pulmonary Tuberculosis in East Java*, 61 Tubercle 231 (1980), filed as "Pet. Ex. 31".

[31] Wendy Lai et al., *Transcriptional Control of Rapid Recall by Memory CD4 T Cells*, 187 J. Immunology 133 (2011), filed as "Pet. Ex. 33".

[32] Dr. Callaghan received his M.D. from the University of Pennsylvania in 2004 and his M.S. in Clinical Research Design and Statistical Analysis from the University of Michigan in 2011. He is board-certified in neurology and in electrodiagnostic medicine. He is an Early Career Professor and Associate Professor in Neurology at the University of Michigan Medical School and the Health System. Resp. Ex. B.

intervention, she was able to ambulate, was discharged to inpatient rehab, where she had an uneventful stay and met all rehabilitation goals. An EMG in December of 2018 showed generalized sensory neuropathy with no motor involvement and a normal needle exam. She continued to report weakness on only the right side. A repeat EMG in February of 2020 showed normal motor responses, normal F responses, bilateral carpal tunnel, and right peroneal neuropathy at the knee; the needle exam showed chronic neurogenic changes in a proximal to distal gradient. Resp. Ex. A at 1-5.

Dr. Callaghan referenced the Brighton diagnostic criteria for GBS, which requires bilateral and flaccid weakness of limbs, reduced or absent deep tendon reflexes in weak limbs, monophasic illness pattern, progressive worsening of symptoms over 12 hours to 4 weeks, plateauing of symptoms after reaching maximum severity, and the absence of another condition that accounts for symptoms of weakness. Motor nerve conduction studies are used to diagnose GBS. Resp. Ex. A at 5; Resp. Ex. A Tab 1;[33] Resp. Ex. A Tab 3;[34] Resp. Ex. E at 1. He conceded that the "Brighton criteria are not perfect" but maintained that they are the best and most often used criteria to diagnose GBS. Resp. Ex. E at 1.

Because petitioner failed to meet several of the Brighton criteria, Dr. Callaghan argued that she did not have GBS. Specifically, petitioner did not have reduced or absent deep tendon reflexes in weak limbs. Rather, her neurologist documented normal reflexes over several visits and even described her reflexes as well-preserved and "brisk". Resp. Ex. A at 5. He noted that Dr. Steinman agreed that reduced reflexes were never documented. Resp. Ex. E at 2. She received no treatment such as IVIG for GBS. She had two EMG/NCS studies, both of which showed "completely normal motor nerve conduction studies and needle electromyography, which is completely inconsistent with the persistent weakness she described". Resp. Ex. A at 5; Resp. Ex. E at 1. He submitted that the slowing of distal latencies in petitioner's sensory nerves without changes in amplitudes seen on EMG was likely because she was not properly "warmed" beforehand. Resp. Ex. A at 5. Dr. Callaghan specifically disputed that petitioner had sensory GBS, which according to *Uncini & Yuki* relied on by petitioner requires "exclusive sensory symptoms". Resp. Ex. E at 2; Pet. Ex. 28.[35] The medical records show that petitioner's complaints were not exclusively sensory, but rather that she complained of weakness. Resp. Ex. E at 2.

Dr. Callaghan further argued that there was no evidence to support axonal neuropathy. The first EMG showed a normal sural sensory amplitude and the second showed a low-normal amplitude. "If the petitioner had an axonal neuropathy, these studies would be expected to be abnormal". Resp. Ex. A at 5. Additionally, slowing of distal latencies in the sensory nerves would not be seen in axonal neuropathy. *Id*. Likewise, the evidence did not support small fiber neuropathy GBS, which would present with neuropathic pain and no weakness. Resp. Ex. E at 2. Petitioner consistently complained of leg weakness which is inconsistent with both sensory GBS and small fiber neuropathy GBS. Finally, leg weakness in conjunction with normal EMG needle findings is inconsistent with a peripheral nerve cause. *Id*.

[33] Sejvar et al., *supra* note 9.
[34] Arthur K. Asbury, MD & David R. Cornblath, MD, *Assessment of Current Diagnostic Criteria for Guillain-Barre Syndrome*, 27 Annals of Neurology S21 (1990), filed as "Resp. Ex. A Tab 3".
[35] Uncini & Yuki, *supra* note 10.

Dr. Callaghan argued that the most likely diagnosis was conversion disorder. He provided the Diagnostic and Statistical Manual of Mental Disorders ("DSM-5") criteria for diagnosing conversion disorder: "(1) ≥ 1 symptom of altered voluntary motor or sensory function, (2) clinical evidence of discordance between symptom(s) and recognized neurological or medical conditions, (3) results in clinically significant distress or impaired social, occupational, or other functioning, or warrants medical evaluation, (4) symptom(s) not better explained by other medical or mental disorder." Resp. Ex. A at 5; Resp. Ex. A Tab 2.[36]

Dr. Callaghan argued that there was a "clear discordance between [petitioner's] symptoms and medical condition given severe weakness with normal motor nerve conduction and needle [EMG] testing." Resp. Ex. A at 5. Further, petitioner clearly had distress, prompting frequent medical evaluations in the months following her hospitalization. Nevertheless, she had three neurological examinations that were inconsistent with the symptoms she described. In the first, the neurologist noted that she moved her legs while conversing but was unable to move them when examined. The second noted intermittent, severe weakness of the lower extremities on examination, alternating with a normal neurological exam. The third noted that for dorsiflexion, she could move her foot, but she could not move it back as expected based on gravity when let go. Finally, during her hospitalization, her physicians consistently noted self-limiting behavior and that her subjective complaints did not align with objective findings. *Id.*; Resp. Ex. E at 1. Finally, her EMG/NCS findings were "very supportive of a conversion disorder" because "any cause of peripheral nerve weakness would reveal abnormalities on the needle EMG", but both of petitioner's were normal. Resp. Ex. E at 2.

Dr. Callaghan disagreed with Dr. Steinman's opinion that petitioner did not have conversion disorder because her treating providers did not explicitly diagnose the condition, noting that he provided no other support for that opinion. Resp. Ex. E at 2. He also argued that Dr. Steinman ignored the several notations throughout the medical records where providers noted inconsistencies between petitioner's complaints and their findings. *Id*.

Dr. Callaghan briefly addressed Dr. Steinman's proposed causation theory and opinion on onset. Resp. Ex. A at 5-6. He argued that onset within two days would be "too fast" for the development of GBS after vaccination. *Id*. at 6. Additionally, petitioner reported onset as one day after the vaccine. *Id*. There was no support provided that showed flu vaccine triggers a recall response that then causes GBS within one day post-vaccination. Resp. Ex. E at 2. He disagreed with Dr. Steinman's use of *Schonberger*, arguing that the mere demonstration that GBS cases occurred within one day of flu vaccination "does not mean that this is higher than the background rate seen in those without vaccination." *Id.*; Pet. Ex. 26.[37] Additionally, *Langmuir* found that the risk of GBS is near zero until 3.5 days post-vaccination and concluded that the risk interval is between 3.5 days and 42 days after vaccination. *Id.*; Resp. Ex. A Tab 4.[38]

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[36] Anthony Feinstein, MD, PhD, *Conversion Disorder*, 24 Behav. Neurology & Psychiatry 861 (2018), filed as "Resp. Ex. A Tab 2".

[37] Schonberger et al., *supra* note 27.

[38] Alexander D. Langmuir et al., *An Epidemiologic and Clinical Evaluation of Guillain-Barre Syndrome Reported in Association with the Administration of Swine Influenza Vaccines*, 119 J. Epidemiology 841 (1984), filed as "Resp. Ex. A Tab 4".

Further, Dr. Callaghan submitted that MBP and contactin-1 are involved in MS and CIDP, respectively, but it is unclear how either applies to GBS or to this case. Resp. Ex. A at 6. Dr. Steinman provided two articles that discussed GBS patients with antibodies to MBP and contactin-1, but neither article supports these proteins playing any role in the pathogenesis of GBS. Resp. Ex. E at 2; Pet. Ex. 35;[39] Pet. Ex. 37.[40]

### C. Respondent's Expert, Dr. Matloubian[41]

Dr. Matloubian summarized petitioner's medical history, emphasizing petitioner's complaints of weakness, nausea, fatigue, and diarrhea two days post-vaccination and that her symptoms began the night she received the vaccine. Resp. Ex. C at 2. He also noted that during her initial hospitalization, she was noted to be moving her legs under the blanket while conversing but was unable to move them extensively on examination. *Id*. at 3. She demonstrated self-limiting behavior, had intact sensation, and had inconsistent strength deficits. *Id*. at 3, 5. GBS was noted to be "highly unlikely", given the lack of symmetrical progressive weakness and rapid symptom improvement. *Id*. at 3-4.

Because he is not a neurologist, Dr. Matloubian deferred to Dr. Callaghan on the exact diagnosis but agreed that petitioner did not have GBS. Resp. Ex. C at 5; Resp. Ex. F at 1. He described GBS as "a monophasic disease, meaning that it reaches nadir at some point followed by slow recovery." Resp. Ex. C at 6. Generally, GBS symptoms progress over two weeks and reach nadir around four weeks after onset. The medical records here show less than 18 hours between onset to nadir of symptoms, making a diagnosis of GBS highly unlikely. *Id*.; Resp. Ex. C Tab 1;[42] Resp. Ex. F at 2; Pet. Ex. 28.[43] He argued that the timing of onset and the rapid resolution without any medical intervention were inconsistent with GBS. Additionally, *Chandrashekhar & Dimachkie* note that asymmetric weakness and fever at the time of onset make a diagnosis of GBS "doubtful." Both were consistent with petitioner's clinical symptoms. *Id*. Dr. Matloubian noted that several treating providers documented inconsistencies in strength deficits and in her symptomology. Resp. Ex. C at 6.

Addressing Dr. Steinman's causation theory, Dr. Matloubian argued that neither MBP nor contactin-1 have been identified as targets of autoimmunity in GBS. Resp. Ex. C at 7; Resp. Ex. F at 2. MBP is associated with MS, a demyelinating disease of the central nervous system, while GBS is a disease of the peripheral nervous system. Dr. Matloubian explained that *Hughes*, relied upon by Dr. Steinman, was published in 1984 and found a few patients with GBS who had

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[39] Kai W. Wucherpfennig et al., *Recognition of the Immunodominant Myelin Basic Protein Peptide by Autoantibodies and HLA-DR2-restricted T Cell Clones from Multiple Sclerosis Patients*, 100 J. Clinical Investigation 1114 (1997), filed as "Pet. Ex. 35".

[40] Bibiana Bielekova et al., *Encephalitogenic potential of the myelin basic protein peptide (amino acids 83-99) in multiple sclerosis: Results of a phase II clinical trial with an altered peptide ligand*, 6 Nature Med. 1167 (2000), filed as "Pet. Ex. 37".

[41] Dr. Matloubian received both his M.D. and Ph.D. in Virology from the University of California, Los Angeles in 1996. He is board-certified in internal medicine and rheumatology. He is a Clinical Professor in Medicine at the University of California, San Francisco. Resp. Ex. D.

[42] Swathy Chandrashekhar, MD & Mazen M Dimachkie, MD, UpToDate: Guillain-Barré syndrome in adults: Pathogenesis, clinical features, and diagnosis, Up To Date (2022), filed as "Resp. Ex. C Tab 1".

[43] Uncini & Yuki, *supra* note 10.

autoantibodies to MBP. *Id*.; Pet. Ex. 20.[44] However, more recent literature makes no mention of MBP as a self-antigen involved in GBS or similar polyneuropathies, suggesting that the observation in *Hughes* did not withstand the test of time and further scientific rigor. Resp. Ex. C at 7; Resp. Ex. C Tab 1;[45] Resp. Ex. C Tab 2;[46] Resp. Ex. C Tab 3;[47] Resp. Ex. F at 2. Likewise, IgG4 antibodies to contactin-1 are associated with CIDP, a disease distinct from GBS. Resp. Ex. C at 7; Resp. Ex. F at 2; Pet. Ex. 21.[48] Rather, scientists, including Dr. Steinman, have identified autoantibodies against gangliosides, which are "molecules consisting of sugars attached to lipid (fat) molecules", as being associated with development of GBS. *Id*.; Resp. Ex. F at 2, 3; Pet. Ex. 29.[49]

Even if he accepted MBP or contactin-1 as relevant to the development of GBS, Dr. Matloubian took issue with Dr. Steinman's opinion that there is molecular mimicry between MBP/contactin-1 and flu antigens. Resp. Ex. C at 8. Dr. Steinman's sequence homology searches did not provide any evidence that petitioner had T cells that can recognize MBP or contactin-1. Further, sequence homology alone is not sufficient to trigger molecular mimicry, and the IOM specifically notes that "[m]any such homologies exist, and the vast majority of these are not associated with biologically relevant autoimmune phenomena or actual human disease". *Id*.; Resp. Ex. C Tab 4.[50]

Dr. Steinman argued that the 5 of 8 identical amino acids are "above the threshold needed" for neuroinflammation. Pet. Ex. 10 at 16. Dr. Matloubian argued that this threshold is one that Dr. Steinman "patched together" and is not accepted by the scientific community at large. Resp. Ex. C at 10. He argued that Dr. Steinman's theory in this case "is neither scientifically correct, nor generalizable, nor a true representation of the conclusions of the authors of the referenced studies." Other studies have shown that altering a single amino acid of 8 dramatically reduces the ability of the peptide to activate T cells. *Id*.; Resp. Ex. C Tab 9.[51] Dr. Matloubian also questioned the search parameters of Dr. Steinman's IEDB searches because the searches he conducted of the identified sequences without any constraints did not produce any "relevant hits" to suggest that it was an immune target on influenza hemagglutinin. Resp. Ex. C at 11; Resp. Ex. F at 3.

Dr. Matloubian explained that MHC/HLA molecules are different from person to person and determine what peptides are presented to the body's T cells. Resp. Ex. C at 9. For example, his own immune system will "see" different peptides from a flu vaccine than what petitioner's immune system would see. "[W]ithout knowing what MHC/HLA molecules petitioner had, there is no way for [Dr. Steinman] or anyone else to predict which peptides" petitioner's T cells would see. Therefore, "there is no basis for a theory that molecular mimicry occurs at the T cell level"

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[44] Hughes et al., *supra* note 19.
[45] Chandrashekhar & Dimachkie, *supra* note 42.
[46] Nortina Shahrizaila et al., *Guillain-Barré syndrome*, 397 Lancet 1214 (2021), filed as "Resp. Ex. C Tab 2".
[47] Nobuhiro Yuki, M.D., Ph.D. & Hans-Peter Hartung, M.D., *Guillain–Barré Syndrome*, 366 N. Engl. J. Med. 2294 (2012), filed as "Resp. Ex. C Tab 3".
[48] Miura et al., *supra* note 20.
[49] Yuki, *supra* note 12.
[50] K. Stratton et al., *IOM Committee to Review Adverse Effects of Vaccines: Evidence and Causality*, Nat'l Acad. Sci. (2011), filed as "Resp. Ex. C Tab 4".
[51] Mark A. Daniels et al., *Thymic selection threshold defined by compartmentalization of Ras/MAPK signalling*, 444 Nature 724 (2006), filed as "Resp. Ex. C Tab 9".

here. *Id*. at 9, 11-12. Further, without actual experimentation, there is no way to know what peptides are antigenic in petitioner. *Id*. at 9; Resp. Ex. C Tab 7.[52] Dr. Matloubian later clarified that he was not asking petitioner or her expert to perform experiments to prove molecular mimicry; but noted that Dr. Steinman's theory had significant limitations that render it more likely than not that the sequences identified do not represent true T cell epitopes. Resp. Ex. F at 2.

Finally, Dr. Matloubian argued that Dr. Steinman's theory applies only to T cells because it is extrapolated from studies on T cells and cannot be extrapolated further to apply to B cells as well. Resp. Ex. C at 12.

Regarding timing, Dr. Steinman opined that onset within 6 hours of vaccination was medically reasonable. Resp. Ex. F at 3. He relied on *Kardjito & Grange*, which studied tuberculin skin tests in patients with active tuberculosis ("TB"), to support his opinion of a rapid onset of memory B and T cells within just hours. However, Dr. Matloubian argued that the rapid skin responses noted in this study were not due to activation of memory B or T cells. *Id*.; Pet. Ex. 31.[53] The patients studied had active TB with an active T and B cell response; in contrast, a memory or recall response occurs after an infection has been cleared. Resp. Ex. F at 3-4. The authors also noted their belief that the observed rapid skin reactions were due to the formation of immune complexes between TB antigens from the skin test and the preexisting antibodies the patients developed in response to the active TB infection. *Id*. at 4. Relying on *Abbas* and *Lee*, Dr. Matloubian submitted that antibody secreting cells typically appear in circulation around day 4 after vaccination and peak between days 5 and 8 with secondary exposure; it would take several additional days for cross-reactive antibodies to bind to gangliosides on peripheral nerves and cause damage, resulting in symptomatic GBS. Thus, the rapid onset in this case was not medically reasonable to implicate the vaccine as a cause. *Id*. at 4-5; Resp. Ex. C Tab 5;[54] Resp. Ex. F Tab 4.[55]

Dr. Matloubian cited to petitioner's medical records to show that she reported onset of weakness less than 48 hours after vaccination. Resp. Ex. C at 12. He argued this is "highly incompatible with B and T responses" even if it were part of a recall response. *Id*.; Resp. Ex. F at 5. He further argued that petitioner's treaters did not think the onset was consistent with vaccine-induced GBS. Resp. Ex. C at 12; Pet. Ex. 7 at 3.

## IV. Parties' Arguments

### A. Petitioner's Motion for Ruling on the Record

[52] Scheherazade Sadegh-Nasseri, *A step-by-step overview of the dynamic process of epitope selection by major histocompatibility complex class II for presentation to helper T cells [version 1; referees: 4 approved]*, 5 F1000 Rsch. (2016), filed as "Resp. Ex. C Tab 7".
[53] Kardjito & Grange, *supra* note 30.
[54] A. K. Abbas et al., Antigen Presentation to T Lymphocytes and the Functions of Major Histocompatibility Complex Molecules, in *Cellular and Molecular Immunology*, Elsevier 117, filed as "Resp. Ex. C Tab 5".
[55] F. Eun-Hyung Lee et al., *Circulating Human Antibody-Secreting Cells during Vaccinations and Respiratory Viral Infections Are Characterized by High Specificity and Lack of Bystander Effect*, 186 J. Immunology 5514 (2011), filed as "Resp. Ex. F Tab 4".

Petitioner summarized her medical history and all three experts' opinions. *See generally* Motion. She asserted that her diagnosis is polyneuropathy. *Id*. at 5-9, 23-25.

Petitioner argued that she has identified a vaccine-related injury as required by the Act and that respondent misconstrued the law regarding diagnosis in his causation analysis. Motion at 9-10. Petitioner acknowledged that it is sometimes necessary for a special master to first determine which injury is best supported by the evidence prior to conducting an *Althen* analysis. For example, in *Broekelschen v. Sec'y of Health & Human Servs*., the petitioner had symptoms of two "completely different conditions". The Federal Circuit held that it was appropriate for the special master to first determine which diagnosis was best supported by the evidence prior to analyzing causation because "the question of causation turn[ed] on which injury [the petitioner] suffered." 618 F.3d 1339, 1346 (Fed. Cir. 2010). Similarly, in *Lombardi v. Sec'y of Health & Human Servs.*, the petitioner alleged three dissimilar disorders, respondent proposed an additional five disorders that could explain the symptoms, and none of the treating providers agreed on the diagnosis. 656 F.3d 1343, 1352-53 (Fed. Cir. 2011). However, determining the appropriate diagnosis is a "narrow prerequisite" that does not apply in this case. Motion at 10.

Here, petitioner's treating physicians all agreed that she suffers from polyneuropathy. Motion at 11. Petitioner's expert also agreed with that diagnosis. Further, none of her treaters mentioned the possibility of conversion disorder as a viable diagnosis. *Id*. Thus, in the absence of the "unique features in *Broekelschen* and *Lombardi* that prompted the Federal Circuit to create a limited prerequisite to the *Althen* test", there is no need to determine petitioner's diagnosis as a threshold matter prior to analyzing causation. *Id*. Rather, petitioner argued that the undersigned should determine causation under *Althen* and then, after petitioner has proven a prima facie case, determine whether respondent has met his burden in proving alternate cause. *Id*.

Petitioner concluded that she has satisfied her burden under *Althen* and respondent has failed to prove an alternative cause was the sole substantial factor in bringing about the injury. Motion at 15-23. Thus, she is entitled to compensation. *Id*. at 25.

**B. Respondent's Response**

Respondent summarized petitioner's medical history and the experts' opinions. *See* Response at 1-16. He disputed that petitioner ever had GBS, citing to clinical findings like preserved reflexes, improvement without IVIG treatment, a normal EMG, and onset of symptoms within two days. *Id*. at 14-15. Respondent's expert Dr. Callaghan explained that conversion disorder would better explain petitioner's symptoms. *Id*. at 15.

Respondent argued that petitioner has not preponderantly established her injury, which "is a prerequisite to any causation analysis." Response at 19-20; *Broekelschen*, 618 F.3d at 1346; *Lombardi*, 656 F.3d at 1352. He disagreed with petitioner's characterization of her treaters' opinions and with her interpretation of vaccine caselaw. Response at 20. The Federal Circuit in *Lasnetski v. Sec'y of Health & Human Servs*. affirmed that it is petitioner's burden to first prove her injury. 696 Fed. App'x 497, 504 (Fed. Cir. 2017). Likewise, in *Simanski v. Sec'y of Health & Human Servs*., compensation was denied where GBS and CIDP were the diagnoses alleged. The minor child was initially treated for possible GBS, but later records showed that a genetic disease

was the diagnosis. 601 Fed. App'x 982-85 (Fed. Cir. 2015). The petitioners did not allege an alternative claim for the genetic disease, but respondent offered evidence to support the genetic disease as the diagnosis. *Id*.

Here, respondent argued there is a "fundamental disagreement between the parties as to what injury or condition petitioner even suffers. Consequently, this Court does need to first determine whether there is preponderant support for the injury petitioner alleges." Response at 22. Further, petitioner's treaters do not agree on her injury with several specifically noting that her symptoms were inconsistent with typical GBS. *Id*. at 22-23. Petitioner has therefore confused respondent's argument that there is a different diagnosis for petitioner's condition as an argument for alternative cause. *Id*. at 25.

Finally, respondent argued that even if the Court were to determine petitioner proved by preponderant evidence that she had the injury alleged, she cannot satisfy her burden under *Althen*. Respondent argued through his experts that myelin basic protein and contactin-1 are not relevant autoantigens in GBS generally or in acute sensory demyelinating polyneuropathy specifically. Response at 37-38. Further, petitioner's expert failed to demonstrate molecular mimicry between the flu vaccine and self-structures. *Id*. at 39-40. The contemporaneous medical records show petitioner's onset of symptoms as the evening she received the subject vaccination, which is far too quick to implicate the vaccine as the cause. *Id*. at 27-28, 33, 41. Additionally, there is evidence that petitioner was suffering from a viral prodrome at the time her symptoms began. *Id*. at 34.

In conclusion, respondent argued that petitioner failed to meet her burden in proving causation and compensation should be denied. Response at 42.

### C. Petitioner's Reply

In her Reply, petitioner distinguished the two cases respondent cited to support his argument that the validity of the injury must first be analyzed prior to causation. She argued that *Lasnetski* was dissimilar to the instant case because the petitioner in *Lasnetski* failed to identify any injury or specific diagnosis and alleged only symptoms. The Federal Circuit held that merely listing symptoms was insufficient to satisfy the Act's requirement of an identifiable injury. 696 Fed. App'x at 501-04; Reply at 2. The petition in *Simanski*, on the other hand, was dismissed because the petitioner in that case alleged GBS as the injury despite the prevailing diagnosis of a genetic disorder from the treating physicians. Because the petitioner's medical records contradicted the claimed injury, the Federal Circuit held that the special master need not reach the question of causation. 601 Fed. App'x at 985-88; Reply at 3.

Petitioner maintained that she provided a sound and reliable theory of causation and that her symptoms began "within two days" of the subject vaccine. Reply at 6-9. She submitted that respondent has "made no effort to show an alternative cause" and in fact confirmed that his "discussion of conversion disorder was offered . . . to avoid an *Althen* analysis altogether". *Id*. at 9-10.

## V. Standards for Adjudication

**A. Causation**

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii); *see also Wright v. Sec'y of Health & Human Servs*., 22 F.4th 999, 1006 (Fed. Cir. 2022) (defining the term "residual effects" in the Act, as "detrimental conditions within the patient, such as lingering or recurring signs and symptoms" of the alleged vaccine injury, which are compensable). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352 (Fed. Cir. 1999). Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, a petitioner must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs*., 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that a petitioner show by preponderant evidence that a vaccination they received caused their 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. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each *Althen* prong requires a different showing. Under the first prong, 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 (citation omitted). To satisfy this prong, a

petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id*. at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, a petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs*., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *Id*. at 961 (citation omitted), and "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," *Paluck v. Sec'y of Health & Human Servs*., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375).

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

**B. Diagnosis**

As a threshold matter, petitioner must establish that she actually suffered the injury alleged in the petition. *See Broekelschen*, 618 F.3d at 1346. As the Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of the petitioner's injury before engaging in the *Althen* analysis. *Id*. Thus, where "the existence and nature of the injury itself is in dispute, it is the special master's duty to *first determine* which injury is best supported" by the evidence. *Lombardi*, 656 F.3d at 1352 (affirming a special master's decision to dismiss a petition when the petitioner could not establish that she had any of the three diagnoses alleged) (citing *Broekelschen*, 618 F.3d at 1345) (emphasis added).

**C. Fact Finding**

The process for making determinations in Vaccine Program cases regarding factual issues begins with analyzing the medical records, which are required to be filed with the petition. § 11(c)(2). Medical records created contemporaneously with the events they describe are generally

considered to be more trustworthy. *Cucuras v. Sec'y of Health & Human Servs*., 993 F.2d 1525, 1528 (Fed. Cir. 1993); *but see Kirby v. Sec'y of Health & Human Servs*., 993 F.3d 1378, 1382-83 (Fed. Cir. 2021) (clarifying that *Cucuras* does not stand for proposition that medical records are presumptively accurate and complete). While not presumed to be complete and accurate, medical records made while seeking treatment are generally afforded more weight than statements made by petitioners after-the-fact. *See Gerami v. Sec'y of Health & Human Servs*., No. 12-442V, 2013 WL 5998109, at *4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013) (finding that contemporaneously documented medical evidence was more persuasive than the letter prepared for litigation purposes), *mot. for rev. denied*, 127 Fed. Cl. 299 (2014). Indeed, "where later testimony conflicts with earlier contemporaneous documents, courts generally give the contemporaneous documentation more weight." *Campbell ex rel. Campbell v. Sec'y of Health & Human Servs*., 69 Fed. Cl. 775, 779 (2006); *see United States v. U.S. Gypsum Co*., 333 U.S. 364, 396 (1948).

In short, "the record as a whole" must be considered. § 13(a).

**D. 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 & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. "In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Id*. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly ex rel. Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010). The *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely 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"). 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*, 618 F.3d at 1347 (citing *Lampe*, 219 F.3d at 1362). And 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 ex rel. Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

**E. Medical Literature**

Finally, although this decision discusses some but not all the literature in detail, the undersigned reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human 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 & Human 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).

## VI. Discussion

### A. *Althen* Prongs and Diagnosis

The parties disagreed on both diagnosis and the legal standard to be applied. Petitioner argued that she has satisfied the Act's requirement by providing an identifiable injury, supported by the opinions of her medical expert and treating physicians. *See generally* Motion; Reply. Petitioner further argued that a determination of diagnosis is only required in limited, unique circumstances, where a petitioner alleges only symptoms—rather than an injury—or where treaters have not agreed on a diagnosis. Neither is the case here. Motion at 9-11; Reply at 2-3. Therefore, petitioner argued that it is unnecessary to determine diagnosis in this case. Motion at 11. Rather, the court must first determine causation then address respondent's argument of conversion disorder as an alternative cause. *Id*.

Respondent argued that there is a "fundamental disagreement between the parties as to what injury or condition petitioner even suffers. Consequently, this Court does need to first determine whether there is preponderant support for the injury petitioner alleges." Response at 22. Respondent further argued that petitioner has confused respondent's suggestion of a different diagnosis with the assertion of an alternate cause of petitioner's injury. *Id*. at 25. Petitioner argued in her Reply that respondent offered conversion disorder in order "to avoid an *Althen* analysis altogether". Reply at 9-10.

Because a finding on diagnosis is not the only dispositive issue in this case, I will address the parties' arguments on diagnosis within the prong two analysis.

#### 1. Petitioner Has Provided a Sound and Reliable Medical Theory.

The first *Althen* prong requires petitioner to provide a "reputable medical theory" demonstrating that the vaccine received *can* cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id.* at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

Petitioner submitted that flu vaccination can cause GBS or similar variants like polyneuropathy. Pet. Ex. 10 at 1, 6-7; Pet. Ex. 27 at 2-5. Dr. Steinman submitted that the mechanism by which this can occur is molecular mimicry between the components of the flu vaccine and MBP and/or contactin-1. Pet. Ex. 10 at 7; Pet. Ex. 27 at 4-5. In support of his theory, he identified homologies between the flu vaccine and both MBP and contactin-1 using BLAST searches and further filtration using IEDB. Pet. Ex. 10 at 7, 15-21; Pet. Ex. 27 at 17-19.

Respondent argued that petitioner did not provide sufficient evidence to show that a flu vaccine could trigger molecular mimicry and ultimately cause GBS, particularly within only two days. Resp. Ex. A at 5-6; Resp. Ex. C at 7; Resp. Ex. E at 2; Resp. Ex. F at 2. Drs. Callaghan and Matloubian argued that the current literature does not support MBP and contactin-1 playing a role in the pathogenesis of GBS. *Id*. Further, Dr. Matloubian submitted that Dr. Steinman's threshold of 5 of 8 identical amino acids is arbitrary and not accepted by the scientific community. Resp. Ex. C at 10. Without knowing the MHC/HLA molecules an individual has, there is no way to know what peptides their T cells would recognize. *Id*. at 9, 11-12. Respondent also submitted that even a recall response that triggers molecular mimicry and results in symptomatic GBS could not occur within only two days. Resp. Ex. F at 3-5.

The association between flu vaccine and GBS is well-established in the Vaccine Program. *See, e.g., Chinea v. Sec'y of Health & Human Servs*., 144 Fed. Cl. 378, 383 (2019) (citations omitted); *Strong v. Sec'y of Health & Human Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018); *Stitt v. Sec'y of Health & Human Servs.,* No. 09-653V, 2013 WL 3356791 (Fed. Cl. Spec. Mstr. May 31, 2013); *Stewart v. Sec'y of Health & Human Servs.*, No. 06-777V, 2011 WL 3241585, at *16 (Fed. Cl. Spec. Mstr. July 8, 2011); *see also Barone v. Sec'y of Health & Human Servs.,* No. 11-707V, 2014 WL 6834557 (Fed. Cl. Spec. Mstr. Nov. 12, 2014). In fact, flu / GBS is an on-Table claim where symptoms manifest within a specified timeframe.[56] 42 C.F.R. § 100.3(a). Accordingly, for purpose of analysis, I find that there is preponderant evidence to support prong one.

**2. Petitioner Failed to Demonstrate a Logical Sequence of Cause and Effect.**

To satisfy the second prong of *Althen*, a petitioner must establish 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 need only be "logical and legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 548-49; *accord Capizzano*, 440 F.3d at 1326. In other words, even if the vaccination can cause the injury alleged, petitioner must show "that it did so in [this] particular case." *Hodges*, 9 F.3d at 962 n.4 (citation omitted).

Generally, "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". *Paluck*, 786 F.3d at 1385 (internal citations omitted). However, special masters are directed to consider the evidence as a whole and are not bound by the notes of treating physicians within the medical record. *See Snyder*, 88 Fed. Cl. at 746 n.67 ("there is nothing ... that mandates that the

[56] The Qualifications and Aids to Interpretation recognize four subtypes of GBS, which include acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor and sensory neuropathy, and Fisher Syndrome. The first three subtypes mirror the Brighton criteria in defining an on-Table GBS. 42 C.F.R. §100.3(c)(15). The presumption of causation afforded by the Table only applies where the vaccine corresponds with the injury alleged. If the injury alleged is different from what is listed on the Table, a petitioner must prove causation-in-fact. *Grant v. Sec'y of Health & Human Servs*., 956 F.2d 1144, 1147-48 (Fed. Cir. 1992) ("Simple similarity to conditions or time periods listed in the Table is not sufficient evidence of causation" in an off-Table claim).

testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted").

In addressing prong two, both parties largely focused on diagnosis. Petitioner alleged that she suffers from acute sensory demyelinating polyneuropathy, an atypical form of GBS, which explains why her symptoms did not clearly align with the Brighton criteria. Motion at 5-9, 23-25; Pet. Ex. 27 at 2-5. She relied on her medical records, the progression of her symptoms, and Dr. Steinman's opinions. She argued that Dr. Steinman opined that GBS is the correct diagnosis and consistent with the opinions of petitioner's treating providers, despite failing to satisfy the Brighton criteria. Pet. Ex. 10 at 6; Pet. Ex. 27 at 1-2. According to Dr. Steinman, petitioner's condition "most closely resembles" acute sensory demyelinating polyneuropathy. Based on *Uncini & Yuki*, some patients are thought to "have a possible GBS variant" despite having normal reflexes and normal NCS studies. Pet. Ex. 27 at 2-5; Pet. Ex. 28.[57] Dr. Steinman referenced a November 28, 2018 record in which Dr. Reeves wrote that petitioner's symptoms were not typical of GBS, but GBS "is possible since there was no active viral infection". Pet. Ex. 10 at 5-6; Pet. Ex. 6 at 8-10. Dr. Steinman also opined that petitioner's December 26, 2018 EMG was consistent with generalized sensory polyneuropathy which is "sensory in nature, so it is no wonder that [petitioner] exhibited somewhat normal motor function". Motion at 24; Pet. Ex. 27 at 2-5; Pet. Ex. 6 at 7, 15-22; Pet. Ex. 10 at 6. Dr. Steinman opined that a repeat EMG on February 21, 2020 showed axonal polyneuropathy. Pet. Ex. 10 at 6; Pet. Ex. 7 at 5. He added that while the Brighton criteria are useful, it has limitations when applied to actual patients in clinical practice. Pet. Ex. 27 at 1-2; Resp. Ex. A Tab 1.[58] Finally, Dr. Steinman argued that petitioner's quick onset was due to a recall response, having received prior seasonal flu vaccines, and does not negate a GBS diagnosis. Motion at 24; Pet. Ex. 27 at 5-11. Summarily, Dr. Steinman opined that "[t]he timing is correct; and, the flu vaccine was noted in the medical records." Pet. Ex. 10 at 26.

Respondent disagreed that GBS, or any variant thereof, was the correct diagnosis and suggested instead that petitioner suffered from conversion disorder. Response at 14-15; *see generally* Resp. Ex. A; Resp. Ex. C; Resp. Ex. E; Resp. Ex. F. He argued that petitioner's reflexes were well preserved and "brisk" rather than reduced or absent as seen in GBS. Her condition improved without treatment. Her NCS study was normal. The time between onset of symptoms and nadir was less than 24 hours rather than the 4-6 weeks as seen in sensory GBS variants. Response at 14-16. Throughout the course of her treatment, she was referred to as having "generalized weakness" with GBS consistently considered a low possibility. The etiology of her symptoms was documented as unclear or possibly secondary to a viral illness. *Id*. at 22-23.

Respondent relied on Dr. Callaghan's opinions to dispute GBS as the correct diagnosis. Dr. Callaghan noted that throughout petitioner's October 2018 hospitalization, her treating physicians documented GBS as a "low possibility". Resp. Ex. A at 1-5. The records show well preserved and "brisk" reflexes, that her condition rapidly improved without any treatment or intervention, and normal NCS studies are "completely inconsistent with the persistent weakness she described". *Id*. at 5; Resp. Ex. E at 1. Dr. Callaghan maintained that the Brighton criteria are the best and most often used criteria to diagnose GBS. Thus, it is significant that petitioner failed to meet several of the criteria. *Id*. He also disputed Dr. Steinman's suggestion that petitioner had

[57] Uncini & Yuki, *supra* note 10.
[58] Sejvar et al., *supra* note 9.

sensory GBS because sensory GBS requires "exclusive sensory symptoms" and petitioner complained of weakness. Resp. Ex. E at 2; Pet. Ex. 28.[59] Dr. Callaghan concluded that complaints of leg weakness in conjunction with normal EMG/NCS findings were inconsistent with any peripheral nerve cause. Resp. Ex. E at 2.

Dr. Callaghan opined that petitioner more likely suffered from conversion disorder, given the inconsistencies in her subjective complaints and the objective findings. Resp. Ex. A at 1-5; Resp. Ex. E at 1-2. He cited several notations throughout the medical records where providers documented inconsistent strength deficits and self-limiting behavior in support of this opinion. Resp. Ex. E at 2.

Similarly, Dr. Matloubian questioned GBS as the correct diagnosis. He noted that petitioner's treaters documented GBS as "highly unlikely" given the lack of symmetrical progressive weakness, rapid symptom improvement, and timing of onset after vaccination. Resp. Ex. C at 3-4, 5, 12; Resp. Ex. F at 1. He also noted that the time between onset and nadir was less than 18 hours, when it is usually four weeks for GBS. Resp. Ex. C at 6; Resp. Ex. C Tab 1;[60] Resp. Ex. F at 2; Pet. Ex. 28.[61] Petitioner's rapid improvement of symptoms without treatment was also inconsistent with GBS. Additionally, she had asymmetric weakness and a fever at the time of onset, both of which were inconsistent with GBS. Resp. Ex. C at 6. R

In summary, respondent argued that conversion disorder was a more likely diagnosis. But, to the extent that petitioner had weakness, that weakness was not caused by GBS, sensory or otherwise, or any other peripheral nerve cause. Although, respondent's two arguments against GBS as the correct diagnosis were somewhat incongruous,[62] his arguments effectively cut against GBS as the diagnosis.

The Federal Circuit has recognized that the opinions of treating providers are generally owed a level of deference. *See, e.g.*, *Capizzano*, 440 F.3d at 1326. While this recognition is most often invoked in vaccine cases within a prong two analysis of causation, it has also been considered "especially apt" in determining diagnosis, since "no person is better qualified to opine on [a petitioner's] condition . . . than the physicians that treated [them] at that time." *Smith v. Sec'y of Health & Human Servs*., No. 15-1194V, 2018 WL 6242453, at *1, *5 (Fed. Cl. Spec. Mstr. Oct. 31, 2018). I find the opinions of petitioner's treating providers quite persuasive on the issues of diagnosis and prong two more broadly in this case.

Furthermore, it is widely accepted that contemporaneous medical records constitute trustworthy (though not infallible) evidence. *Cucuras*, 993 F.2d at 1528; *Kirby*, 993 F.3d at 1382-83. Consistent with the caselaw, I find the contemporaneous medical records from the time petitioner received the subject flu vaccine through her discharge from the hospital to be more

[59] Uncini & Yuki, *supra* note 10.
[60] Chandrashekhar & Dimachkie, *supra* note 42.
[61] Uncini & Yuki, *supra* note 10.
[62] Respondent's arguments were internally inconsistent in that he disputed the existence of petitioner's symptoms in offering conversion disorder as a more likely explanation for her symptoms while also maintaining that weakness as a symptom rules out sensory GBS, which requires exclusively sensory symptoms. However, these arguments could reasonably be viewed as arguments made in the alternative, which is how I interpreted them herein.

reliable than the medical records following her discharge when she presented reporting—inaccurately—that she had been diagnosed with GBS.

Petitioner's medical records support the following: two days after the subject vaccination, petitioner contacted EMS and reported a two-day history of nausea, weakness, diarrhea, and fatigue. Pet. Ex. 3 at 14-15. EMS records include a slightly elevated temperature and lower extremity weakness. *Id.* Upon presentation to the hospital, petitioner was alert, with normal speech, sensory, and motor function, and no neurological deficits. *Id*. at 289. The examination from a neurological consultation included "[s]ensation intact bilaterally" with the impression being non-specific weakness and with GBS a "low possibility." *Id.* at 219. A physician's progress note included continued complaints of lower extremity weakness and difficulty ambulating. *Id*. at 283. Petitioner reported vomiting witnessed by nurses but denied fever, although she reported that she felt hot and had some chills. She complained of bilateral lower extremity pain with occasional paresthesia. WBC levels were elevated. *Id*. On physical examination, she had minimally decreased strength and reflexes and was unable to move on examination, but she was noted to be moving her extremities under the blanket while conversing. *Id*. at 284. The impression was generalized weakness with no clear etiology. She had a fever and minimal leukocytosis. *Id*. at 285. A physician's note included that petitioner underwent physical and occupational therapy evaluation that morning and was noted to have some self-limiting behavior with inconsistent strength deficiencies. Additional testing was required. *Id*. at 285, 366. GBS was unlikely "given presentation . . . and lack of progression." *Id*. at 285. An RN was notified for a possible psychiatric evaluation. *Id*. at 366.

The hospitalist's progress note for October 28, 2018, documented petitioner's report that she felt "much better today," was able ambulate, had no complaint of any myalgias or paresthesias, and her weakness had significantly improved. Pet. Ex. 3 at 280. The etiology remained unclear. *Id*. at 282. The record included that "GBS seems highly unlikely at this point given symptomatology. Perhaps patient had viral syndrome. She did have vomiting and some abdominal pain. cantNOT (sic) rule out functional symptomatology." *Id*.

On October 29, 2018, petitioner reported various complaints including shortness of breath, leg weakness, reaction to flu vaccine on her arm with pain, dizziness, and lightheadedness. However, physical examination was normal with significantly improved strength in all extremities. Pet. Ex. 3 at 277-79. The assessment included "no clear etiology at this time", likely some limiting behavior with inconsistent strength deficiencies per PT, intermittent weakness of the lower extremities, unable to do MRI due to pacemaker even if devices are pacemaker-compatible, and noted to not have progressive, symmetrical weakness so GBS is unlikely. She refused lumbar puncture. *Id*. at 279. Viral syndrome due to vomiting and abdominal pain was suspected, and functional symptomatology could not be ruled out. *Id*.

As evidenced by the contemporaneous medical records, the treaters who observed petitioner's symptoms did not diagnosis her with GBS or a variant thereof. Rather, they specifically noted that GBS was "highly unlikely" based on physical findings and objective testing. They suspected a viral syndrome based on presentation. Pet. Ex. 3 at 219, 279. She did not satisfy the six Brighton criteria.[63] Petitioner did not have symmetrical, progressive weakness of the limbs;

[63] *Supra* note 56.

did not have reduced or absent deep tendon reflexes; did not have a monophasic illness; did not have progressively worsening symptoms over 12 hours to 4 weeks; did not have a plateau of symptoms after reaching maximum severity; and there was evidence of other conditions that could explain her symptoms of weakness. Her reported asymmetrical weakness was inconsistent in that she could not move her legs on examination but she was noted to be moving her legs while conversing; she had near normal reflexes; she reported improvement then "relapse" the following day; the time between onset and nadir was less than 24 hours without medical intervention; and several providers raised the possibility of self-limiting behavior, inconsistencies between what was reported and what was found on examination, and attributed her condition to possible viral infection due to elevated WBC, fever, and abdominal pain. Pet. Ex. 3 at 14-15, 213, 280-82, 284-85, 277-79, 280-82, 284, 287-89; Pet. Ex. 4 at 161, 195, 204; Pet. Ex. 5 at 2; Pet. Ex. 6 at 2, 4-5; Resp. Ex. A Tab 1.[64] Additionally, even the motor nerve conduction studies, which are used to diagnose GBS, performed later were normal. Resp. Ex. A Tab 1; Pet. Ex. 6 at 15; Pet. Ex. 7 at 6. Thus, petitioner's symptoms were inconsistent with those associated with GBS and failed to meet the Brighton criteria.

Dr. Steinman postured that as a clinician, the Brighton criteria are somewhat limited when applied to real patients. Pet. Ex. 27 at 1-2; Resp. Ex. A Tab 1.[65] Further, her symptoms would not fit perfectly within the Brighton criteria because she had a rare variant of GBS. Motion at 24; Pet. Ex. 27 at 2-5. These arguments would have been more persuasive if petitioner had met at least one or more of the Brighton criteria or had some objective finding consistent with GBS, but she did not. Where a patient fails to satisfy diagnostic criteria or meet any of the criteria of the condition alleged, the logical conclusion is that the patient does not have that condition.

Petitioner's clinical presentation was not consistent with acute sensory demyelinating polyneuropathy either. Dr. Steinman referred to petitioner's December 26, 2018 EMG as showing generalized sensory polyneuropathy, which is "sensory in nature"; this study was noted to not show any motor involvement. Pet. Ex. 27 at 2-5; Pet. Ex. 6 at 6-7. Dr. Steinman relied on *Uncini & Yuki* to support his opinion that inflammatory demyelinating neuropathy can indeed be purely sensory. Pet. Ex. 27 at 2-3. The authors state the "[t]he possibility that some patients diagnosed with an acute sensory neuropathy could actually have [GBS] has been repeatedly advanced in the literature". Pet. Ex. 28 at 1.[66] Acute sensory demyelinating polyneuropathy is one of the three types of sensory GBS. *Id*. The authors defined sensory GBS as "an acute, monophasic, widespread neuropathy characterized clinically by exclusive sensory symptoms and signs that reach their nadir in 6 weeks". *Id*. Based on this study, Dr. Steinman argued that normal motor function would not exclude a sensory type of GBS. Pet. Ex. 27 at 2-5. However, here, petitioner's complaints were not of exclusively sensory symptoms, and, in fact, her examinations showed sensory to be intact. Pet. Ex. 3 at 219, 289. Additionally, petitioner's symptoms reached nadir within 24 hours. *Id*. at 14-15, 218-19, 287. Dr. Steinman also failed to explain how the December 2018 EMG showed normal motor function when petitioner's primary subjective complaint throughout her hospitalization and later treatment was leg weakness. *Id*. at 287, 289, 291, 293, 298, 366, 468. Thus, even using the definition of sensory GBS as set forth in the literature Dr. Steinman relied on, petitioner's clinical picture was inconsistent with that diagnosis.

[64] Sejvar et al., *supra* note 9.
[65] *Id*.
[66] Uncini & Yuki, *supra* note 10.

It is noteworthy that Drs. Callaghan and Matloubian focused on the provider opinions and medical records from the time EMS was called through petitioner's hospitalization. Dr. Steinman, however, credited the physician's records and opinions following petitioner's discharge from the hospital when she presented reporting a history of hospitalization for GBS. *See* Pet. Ex. 10 at 5-6. Though Dr. Steinman is correct that petitioner's EMGs were interpreted as consistent with sensory neuropathy and later axonal polyneuropathy, these EMGs were performed approximately two months and 1.5 years, respectively, after onset and at a time when her symptoms were largely resolved. Pet. Ex. 6 at 6-7; Pet. Ex. 7 at 5. Also notable are the visit notes that accompany each EMG. In her first appointment with Dr. Reeves on November 28, 2018, Dr. Reeves' plan was that if petitioner "does have evidence of GBS, likely proceed with IVIG to help with her recovery." Pet. Ex. 2 at 773. Despite follow up appointments with Dr. Reeves[67] after the December 2018 EMG, he did not recommend IVIG, suggesting that he did not believe there was evidence of GBS that IVIG would help to alleviate. *See generally* Pet. Ex. 6. Additionally, Dr. Mandell specifically evaluated petitioner in 2020 to assess whether her symptoms were "vaccine related or not" and cautioned that, though possible, the onset after vaccination was quick, she had brisk reflexes, and "[c]ause and effect [between her symptoms and the vaccine] is unclear." Pet. Ex. 7 at 2-5. Thus, petitioner's argument is undercut by her own providers' medical records.

Because respondent's experts' opinions were more aligned with the providers who had the benefit of observing, examining, and treating petitioner at the time she experienced symptoms, and because the medical records clearly indicate those treaters did not believe petitioner had GBS, I find that petitioner has not proven by preponderant evidence that she suffered from GBS, acute sensory demyelinating polyneuropathy, or other similar condition(s).[68] Accordingly, the evidence submitted to support the injury alleged as well as prong two of *Althen* is insufficient to satisfy petitioner's burden.

**3. Petitioner Failed to Show an Appropriate Temporal Relationship.**

The third *Althen* prong requiring an appropriate temporal relationship contains two parts. First, a petitioner must establish the "timeframe for which it is medically acceptable to infer causation" and second, she must demonstrate that the onset of the disease occurred in this period. *Shapiro v. Sec'y of Health & Human Servs.,* 101 Fed. Cl. 532, 542-43 (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). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury utilized to satisfy the first prong. *Id.* at 542; *Koehn v. Sec'y of Health & Human Servs*., No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Petitioner argued that onset of GBS within two days of flu vaccination is medically reasonable in the context of a recall response. Pet. Ex. 10 at 24. Dr. Steinman offered *Tan* to show that the flu vaccine can induce a recall response in an individual who previously received flu

[67] Dr. Reeves is the physician who interpreted the December 2018 EMG/NCS. See Pet. Ex. 6 at 6-7.

[68] Because petitioner failed to provide preponderant evidence of the alleged diagnosis, it is unnecessary to analyze respondent's proposed alternative diagnosis of conversion disorder.

vaccines. Pet. Ex. 27 at 10; Pet. Ex. 32.[69] Additionally, *Schonberger* showed that a recall response can produce neurologic symptoms between 2-4 days. Pet. Ex. 10 at 24; Pet. Ex. 26;[70] Pet. Ex. 27 at 6-8. Dr. Steinman argued that *Schonberger* was more instructive on timing than the *Langmuir* study because *Schonberger* presented the data in two-day intervals, while *Langmuir* used seven-day intervals. Pet. Ex. 27 at 7; Pet. Ex. 26; Resp. Ex. A Tab 4.[71] Dr. Steinman offered *Kardjito & Grange*, which studied responses to tuberculin skin-tests and showed the "most marked" skin reactivity was at 24 hours post-test. Pet. Ex. 27 at 9-10; Pet. Ex. 31.[72] Finally, *Lai* showed recall responses as early as 6 hours after an antigenic challenge. Pet. Ex. 27 at 10; Pet. Ex. 33.[73] Dr. Steinman pointed to the medical records as showing that petitioner received annual flu vaccinations for several years prior to receiving the subject flu vaccine. As such, she had a recall response to the subject flu vaccine, so manifestation of symptoms within only two days was medically reasonable. Motion at 24; Pet. Ex. 27 at 20-21.

Respondent argued that the onset of symptoms less than two days after vaccination is not medically appropriate to implicate the vaccine as a cause. Resp. Ex. A at 6; Resp. Ex. F at 4-5. Dr. Matloubian addressed the literature relied on by Dr. Steinman, concluding that the articles did not support his opinion on timing. He pointed out that *Kardjito & Grange* studied tuberculin skin tests in patients with active TB; thus, the rapid skin responses were not because of a recall response but rather due to an active T and B cell response. Resp. Ex. F at 3-4; Pet. Ex. 31.[74] He also discussed *Lai* as unsupportive of Dr. Steinman's opinion because it involved experiments in a test tube and with a high concentration of antigen-specific T cells rapidly exposed to a high concentration of the specific antigen. That scenario is not comparable to the immune response to a vaccine. Resp. Ex. F at 4-5; Pet. Ex. 33.[75] Dr. Matloubian provided *Abbas* and *Lee*, which showed antibodies appear in circulation 4 days post-vaccination and peak between days 5 and 8 with secondary exposure. Additional time is necessary for the cross-reactive antibodies to bind to gangliosides on the peripheral nerves and even more time is required to cause damage that results in clinical symptoms. Resp. Ex. F at 4-5; Resp. Ex. C Tab 5;[76] Resp. Ex. F Tab 4.[77] Finally, *Langmuir* found that the risk of GBS is near zero until 3.5 days post-vaccination, so onset before that is not medically appropriate. Resp. Ex. E at 2; Resp. Ex. A Tab 4.[78] Dr. Callaghan noted that *Langmuir* specifically addressed the appropriate time interval for GBS after flu vaccine, while *Schonberger* did not. *Id*.

While it is undisputed that the flu vaccine can induce a recall response, the evidence presented in this case does not show it can do so the same day as reported by petitioner to her providers at various times. Dr. Steinman provided some support for his opinion on the rapid onset seen here, but it was effectively rebutted by respondent's experts. *Kardjito & Grange* was not informative on the issue of recall response because the patients studied had active infections. Pet.

[69] Tan et al., *supra* note 29.
[70] Schonberger et al., *supra* note 27.
[71] Langmuir et al., *supra* note 38.
[72] Kardjito & Grange, *supra* note 30.
[73] Lai et al., *supra* note 31.
[74] Kardjito & Grange, *supra* note 30.
[75] Lai et al., *supra* note 31.
[76] Abbas et al., *supra* note 54.
[77] Lee et al., *supra* note 55.
[78] Langmuir et al., *supra* note 38.

Ex. 31.[79] *Lai* was unhelpful because antigen-specific activation was required for rapid recall. Pet. Ex. 33.[80] The articles petitioner relied on to support the rapid onset of symptoms were too attenuated to the facts herein to be persuasive. Dr. Steinman's point about the presentation of data in *Schonberger* versus *Langmuir* is well-taken. Pet. Ex. 27 at 7; Pet. Ex. 26;[81] Resp. Ex. A Tab 4.[82] Nevertheless, *Langmuir* reanalyzed the data used in *Schonberger* and still found the risk interval of GBS after flu vaccine to start at 3.5 days.

Additionally, Dr. Steinman's opinion that petitioner's symptoms began two days after vaccination was inconsistent with her own statements to providers in the days following her flu vaccine. She initially reported to EMS that her symptoms began the evening of vaccination. Pet. Ex. 3 at 14-15, 287. When discharged to short-term rehabilitation, she again reported that her weakness and fatigue began the evening of vaccination and progressed to pain and numbness by Friday when she presented to the hospital. Pet. Ex. 4 at 37; Pet. Ex. 3 at 227. Dr. Steinman failed to address the medical records that were inconsistent with his opinion on a two-day onset, ultimately rendering his opinion less persuasive.

Based on the evidence as a whole, petitioner failed to provide preponderant evidence to support prong three.

## VII. Conclusion

Upon careful evaluation of all the evidence submitted in this matter, I find that petitioner has failed to show she is entitled to compensation under the Vaccine Act. **The Clerk shall enter judgment consistent with this Decision.**[83]

**IT IS SO ORDERED**.

**s/ Mindy Michaels Roth**
Mindy Michaels Roth
Special Master

[79] Kardjito & Grange, *supra* note 30.
[80] Lai et al., *supra* note 31.
[81] Schonberger et al., *supra* note 27.
[82] Langmuir et al., *supra* note 38.
[83] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party filing a notice renouncing the right to seek review.