In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: December 12, 2025

REBECCA RAY,	*	PUBLISHED
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Petitioner,	*	No. 20-321V
	*	
v.	*	Special Master Nora Beth Dorsey
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Guillain-Barre Syndrome ("GBS"); Chronic Inflammatory Demyelinating Neuropathy ("CIDP").
	*	
Respondent.	*	
	*	

David John Carney, Green & Schafle, LLC, Philadelphia, PA, for Petitioner.
Ryan D. Pyles, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On March 23, 2020, Rebecca Ray ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018)[2] alleging that the influenza ("flu") vaccination she received on October 9, 2018, was the cause-in-fact of her Guillain-Barre Syndrome ("GBS"). Petition at Preamble (ECF No. 1). Petitioner filed an amended petition on October 16, 2020, wherein she alleged a Table Injury for GBS and in the alternative, a cause-in-fact claim for GBS. Amended

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

("Am.") Petition at Preamble (ECF No. 17). Respondent argued against compensation and recommended that the case be dismissed. Respondent's Report ("Resp. Rept.") at 1, 6 (ECF No. 21).

Subsequently, the Chief Special Master dismissed Petitioner's Table Claim for GBS for "failure to establish onset within the 3-42 day time period following the administration of vaccination." Findings of Fact and Conclusions of Law Dismissing Table Case ("Findings of Fact") dated Jan. 13, 2021, at 2 (ECF No. 22). In dismissing the Table claim, the Chief Special Master found "Petitioner's GBS occurred on December 22, 2018 (over 70 days—or ten weeks—after her flu vaccination." Id. at 6. The Chief Special Master warned "Petitioner [was] unlikely to establish causation in this case even for a non-Table claim, since a timeframe of ten weeks has never been deemed medically acceptable for vaccine-caused GBS." Id. Petitioner was encouraged to "strongly consider seeking dismissal of what remain[ed] of her case." Id.

Thereafter, following briefing on an off-Table claim and an amended petition alleging a causation-in-fact claim of chronic inflammatory demyelinating polyneuropathy ("CIDP") following the October 9, 2018 flu vaccination, this case was reassigned to the undersigned for adjudication of Petitioner's non-Table claim of CIDP following the flu vaccine. Order dated Apr. 13, 2021 (ECF No. 28);[3] Second Am. Petition at Preamble (ECF No. 31). Petitioner was then allowed ample time to obtain expert reports and medical literature to address the issue of a ten-week onset.

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[4] the undersigned finds that Petitioner has failed to provide preponderant evidence that her flu vaccine caused her GBS/CIDP based on the fact that onset occurred more than 10 weeks (74 days) after vaccination, an onset period too long to establish a medically acceptable temporal association. Thus, Petitioner has not satisfied her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

[3] The Chief Special Master again emphasized "a timeframe of ten weeks has *never been deemed medically acceptable* for vaccine caused GBS" and limited Petitioner to a non-Table case of CIDP. Order dated Apr. 13, 2021.

[4] While the undersigned has reviewed all the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); Simanski v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'"), aff'd, 601 F. App'x 982 (Fed. Cir. 2015); Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

I. ISSUES TO BE DECIDED

Diagnosis is at issue; Petitioner asserts Petitioner's condition is GBS/CIDP, whereas Respondent contends Petitioner's diagnosis is CIDP alone and not GBS. Joint Submission, filed Feb. 11, 2025, at 4 (ECF No. 106).

Regarding onset, the parties acknowledge the Chief Special Master ruled that onset of Petitioner's symptoms began December 22, 2018, more than ten weeks post-vaccination. Joint Submission at 4 (citing Findings of Fact at 5). However, Petitioner maintains that her symptoms "began prior to December 22, 2018." Id. "However, even if the Court maintains that the onset of symptoms began on December 22, 2018, [P]etitioner maintains that she has established causation in fact under Althen." Id. Respondent disagrees with Petitioner and asserts "that the Court's factual finding [as to onset] is correct." Id.

Respondent also questions the date of vaccination, noting "there is ambiguity" regarding the date (whether October 8 or 9, 2018). Joint Submission at 5. The parties agree, however, that the "difference in dates" related to vaccine administration does not "materially affect[] the parties' respective positions or application of the evidence to the facts of this case." Id.

The parties agree that Petitioner's prior medical history is not contributory to her claim here, and thus, she asserts no claim for significant aggravation. Joint Submission at 5. Further, Respondent agrees that Petitioner has met the statutory jurisdictional requirements. Id.

Lastly, causation is also in dispute, including all three prongs of Althen. Joint Submission at 5.

II. BACKGROUND

A. Procedural History

Petitioner filed her petition on March 23, 2020. Petition. The early procedural history through April 2021 is summarized above and was set forth in the Chief Special Master's Fact Finding and will not be repeated here. See Findings of Fact at 2-3.

This case was reassigned to the undersigned in May 2021. Notice of Reassignment dated May 4, 2021 (ECF No. 33). From September 2021 to October 2022, Petitioner filed expert reports from Dr. Omid Akbari and Dr. Joseph Jeret and Respondent filed expert reports from Dr. Andrew MacGinnitie and Dr. Harold Moses. Petitioner's Exhibits ("Pet. Exs.") 22, 27, 39; Resp. Exs. A, C, J, Q.

A Rule 5 conference was held on January 10, 2023. Rule 5 Order dated Jan. 11, 2023 (ECF No. 80). The undersigned noted that she agreed with the Chief Special Master's finding that onset was more than 10 weeks (74 days). Id. at 1. However, she was unable to provide any preliminary findings or opinions as to a temporal association under Althen prong three. Id. at 2. Thereafter, Respondent indicated he was not interested in settlement discussions. Joint Status Rept., filed Apr. 21, 2023 (ECF No. 86).

Pursuant to Petitioner's request, this case was set for an entitlement hearing in January 2025. Order dated May 12, 2023 (ECF No. 87); Prehearing Order dated June 15, 2023 (ECF No. 90). However, in October 2024, the parties indicated their preference to now resolve entitlement through a ruling on the record, and a briefing schedule was set. Joint Status Rept., filed Oct. 26, 2024 (ECF No. 95); Ruling on the Record Order dated Oct. 28, 2024 (ECF No. 96).

Petitioner filed her motion for a ruling on the record on December 20, 2024. Pet. Motion for Ruling on the Record ("Pet. Mot."), filed Dec. 20, 2024 (ECF No. 101). Respondent filed his response on February 4, 2025, and Petitioner filed a reply on February 26, 2025. Resp. Response to Pet. Mot. and Cross-Mot. for Ruling on the Record Denying Entitlement ("Resp. Response"), filed Feb. 4, 2025 (ECF No. 104); Pet. Reply Brief in Support of Pet. Mot. ("Pet. Reply Br."), filed Feb. 26, 2025 (ECF No. 108).

This matter is now ripe for adjudication.

B. Factual History

1. Stipulated Facts[5]

On October 8 or 9, 2018, Petitioner, at forty-four years of age, received a flu vaccination at a public health department.[6] Pet. Ex. 1 at 1. The parties concur that whether the vaccination was administered on October 8 or 9 does not materially affect the parties' respective positions with regard to Althen's third prong.

On December 23, 2018, Petitioner went to the Mercy Health emergency department ("ED") for "bilateral upper and lower [extremity] tingling and numbness since yesterday." Pet. Ex. 3 at 143. On December 24, 2018, Petitioner went to Mercy Health urgent care, reporting that she had been to the ED the previous night and was directed to follow-up with her primary provider, but her provider was not in the office that day. Id. at 140. Symptomatology of "[t]ingling sensation in hands and feet [for] [one] day" was noted. Id. In a December 24, 2018 consultation with Gretchen Goltz, D.O. (family medicine), the following history was recorded: "On [December 22, 2018], [Petitioner] developed paresthesias in her feet. Th[at] spread gradually to her hands. The next day, she noted some weakness in her legs and worse this morning . . . ." Id. at 142. The impression was GBS, and admission was recommended. Id. at

[5] The parties agreed to the following stipulated medical history in their Joint Submission. See Joint Submission at 1-4. While the stipulated medical history is taken from the parties' joint submission, the undersigned has made minor edits for style.

[6] Petitioner dated the vaccination form October 8, 2018. Pet. Ex. 1 at 1. The petition alleges that vaccination occurred on October 9, 2018. It is possible that the administrator dated the vaccine form for October 9, but the notation is unclear to Respondent. Id. The undersigned agrees that whether the vaccination was administered on October 8 or 9 is not material to the issues in dispute. Therefore, the undersigned makes no finding as to the date of vaccination.

142-43; see also Pet. Ex. 4 at 11 (same history included in December 24, 2018 neurology consultation with Kevin Kellogg, M.D.).

Petitioner was hospitalized from December 24, 2018 to January 9, 2019 at Mercy Health, Muskegon. Pet. Ex. 4 at 21. On December 24, 2018, Petitioner reported to ED physician Jerry Evans, M.D., in part:

> She states this started Saturday morning [December 22] when she woke up. Yesterday she went to the [ED] at Mercy Hospital and they did lab tests. Nothing was found at that time and she was discharged. She presents today stating that the numbness and tingling is moving up her legs and now her legs feel weak.

Id. Petitioner also reported running three miles the previous week "without difficulty;" that she had "never experienced anything like this before;" and that "this all started Saturday morning and has gotten progressively worse." Id. Also on December 24, 2018, Petitioner reported to Kelly M. Thelen, P.A., the following in part: "She denie[d] any recent illnesses but states that she did have some fatigue [two] days last week." Id. Attending physician William Hughes, D.O., further noted: Petitioner reports "that [symptomatology] was sudden onset in nature just the other day. [She does] report some fatigue for the last couple weeks, but no recent sick contact exposures or fever/chills." Id. at 8. Following treatment, including five days of IVIG therapy, Petitioner was discharged to inpatient rehabilitation, still unable to move her legs. Pet. Ex. 4 at 4-5 (also noting only slight finger movements).

From January 9 to April 1, 2019, Petitioner was in inpatient rehabilitation at Mercy Health, Muskegon. Pet. Ex. 4 at 96-98. Upon discharge, she still had significant weakness in her bilateral lower extremities, and the following was written:

> After consultation with neurology, it was determined that it would be most appropriate to treat her like a CIDP patient. IVIG was reinitiated weekly times [four] weeks and then every other week for [four] weeks. She received her first [two] doses while she was on the inpatient rehab unit. She did notice an increase in neuropathic pain in bilateral lower extremities which previously proceeded the return of her function of the upper extremities.

Id. at 98 (also noting, "[electromyography ("EMG")] done twice during her stay. Severe demyelination in bilateral upper and lower extremities."). Petitioner was discharged to "Mary Free bed subacute rehabilitation." Id.

Petitioner remained in inpatient rehabilitation at Mary Free Bed Rehabilitation Hospital until May 31, 2019, with home therapy services planned upon discharge. Pet. Ex. 6 at 52-53.

By August 20, 2019, in neurology follow-up with Rachel Balkema, PA-C, with Patrick Pavwoski, D.O., attending, Petitioner was still on IVIG infusions with the following history written:

> [Petitioner] presents for follow up regarding her GBS. She states [that upon] the lapse of her [IVIG] infusions she noticed increased weakness. After her infusion in August she noticed better strength, able to stand and walk with PT. Her eating and taste has improved. She does not feel like the dexterity and rigidity in her hands has [] fully improved yet. Due to her insurance being approved late July her first infusion was not until August 1. . . . She has another infusion on Thursday, so she is scheduled every three weeks.

Pet. Ex. 9 at 8. Petitioner ambulated with a walker, and her review of symptoms included: "numbness, tingling, lightheadedness, weakness, muscle twitching, cramps, headaches, difficulty with gait or walking, imbalance or falling, double vision, tremor, incoordination, and sleep difficulty," as well as "muscle weakness, arthralgias/joint pain, back pain, and swelling in [her] extremities." Id. In follow-up on January 9, 2020, Physician Assistant Balkema noted in part: Petitioner "is doing well with her infusions every [three] weeks. She states that a few days before her next [infusion, she experiences increasing] fatigue, muscle cramping – especially in the abdomen, her thumbs get 'stuck.' She states that her [physical therapist] even notices when the next infusion is approaching." Id. at 5.

On February 1, 2023, Petitioner had a "[m]arkedly abnormal" EMG. Pet. Ex. 44 at 6. The impression included, "There are electrodiagnostic findings most consistent with a diffuse, primarily demyelinating polyradiculoneuropathy." Id.

2. Petitioner's Affidavit

Petitioner executed an affidavit on March 9, 2020. Pet. Ex. 2 at 7.

Petitioner received the flu vaccine at issue on October 9, 2018. Pet. Ex. 2 at ¶ 9. She averred that between vaccination (October 9, 2018) and her hospital admission (December 24, 2018), she "had no illnesses, sicknesses, viral infections[,] or bacterial infections." Id. at ¶ 10.

Petitioner explained that approximately one month post-vaccination, "or sometime between early November and mid-November, [she] started to experience tingling and numbness in [her] hands and wrists" as well as frequent weakness in her legs. Pet. Ex. 2 at ¶ 11. She was actively playing volleyball and thought her symptoms were related to a pinched nerve, so she did not seek medical attention. Id. These symptoms persisted until the week before Christmas when the symptoms worsened and spread. Id. at ¶ 13. On December 22, her symptoms "significantly worsen[ed];" she had increased tingling and numbness in her fingers, hands, toes, and feet and new-onset tongue numbness. Id. at ¶ 14. And on December 23, the numbness spread to her arms and feet, prompting her to visit the ED. Id. at ¶ 15. She returned to the ED the following day where she was admitted for testing and treatment. Id. at ¶ 16.

C. Expert Reports

1. Petitioner's Expert, Joseph S. Jeret, M.D.[7]

a. Background and Qualifications

Dr. Jeret is a board-certified neurologist. Pet. Ex. 39 at 1; Pet. Ex. 40 at 1. He received his M.D. from SUNY Downstate Medical Center. Pet. Ex. 40 at 1. He did a general internal medicine internship at Maimonides Medical Center followed by a residency in neurology and a fellowship in clinical neurophysiology at SUNY Downstate. Id. at 1; Pet. Ex. 39 at 1. Dr. Jeret is a practicing neurologist. Pet. Ex. 39 at 1-2. He works as a physician at the Icahn School of Medicine and maintains an active neurology practice in Long Island, NY. Id. at 1. He routinely cares for and diagnoses patients with various neurological illness including GBS and CIDP. Id. at 2. Dr. Jeret has authored or co-authored publications in many areas of neurology reflecting his general neurology practice. Id. at 1-2; Pet. Ex. 40 at 2-7.

b. Opinion

Dr. Jeret focused his opinions on the diagnosis of GBS. Pet. Ex. 39 at 1. He explained the history related to the evolution of the criteria required for a diagnosis of GBS, and the primary requirements, including subacute development of flaccid paralysis, bilateral weakness that is usually symmetrical, absent or decreased reflexes, and exclusion of other causes of the flaccid paralysis. Id. at 2. Asbury & Cornblath[8] noted that "[p]rogressive motor weakness must involve more than one limb" and be "accompanied by areflexia or distal areflexia with hyporeflexia at biceps and knees." Id. at 3 (citing Pet. Ex. 41(w)). Additional support for diagnosis includes "progressive motor weakness, relative symmetry, mild sensory symptoms, cranial nerve involvement, recovery, and autonomic involvement." Id. Lumbar puncture with cerebrospinal fluid ("CSF") analysis and EMG/nerve conduction study ("NCS") findings may also be characteristic of the illness. Id. at 2.

GBS has axonal subtypes,[9] but Dr. Jeret explained that Petitioner had acute inflammatory demyelinating polyneuropathy ("AIDP") and not an axonal subtype. Pet. Ex. 39 at 3. The disease is an inflammatory immune mediating illness and is "not genetic, degenerative, infectious, or traumatic." Id. The word demyelinating means dysfunction of or injury to the

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[7] Dr. Jeret submitted one expert report. Pet. Ex. 39.

[8] Arthur K. Asbury & David R. Cornblath, Assessment of Current Diagnostic Criteria for Guillain-Barré Syndrome, 27 Ann. Neurol. S21 (1990).

[9] The axonal subtypes of GBS are acute motor axonal neuropathy ("AMAN") and acute motor and sensory neuropathy ("AMSAN"). 42 C.F.R. § 100.3(c)(15)(ii). AMAN "is generally seen in other parts of the world[,] [] is predominated by axonal damage that primarily affects motor nerves," and "lacks features of demyelination." Id. AMSAN is an "axonal form of GBS that is similar to AMAN, but also affects the sensory nerves and roots." Id.

myelin sheath, the covering of peripheral nerves. Id. Lastly, the word polyneuropathy indicates the illness "affects multiple nerves." Id. at 4.

After providing a thorough medical chronology based on Petitioner's medical records, Dr. Jeret opined Petitioner's diagnosis is GBS. Pet. Ex. 39 at 9. She "had ascending numbness and weakness, elevated CSF protein with normal cells, areflexia, autonomic instability (manifest as tachycardia due to vagal nerve demyelination), and facial weakness (indicating cranial nerve involvement)." Id.

Dr. Jeret also opined that Petitioner did not have any recent stomach or intestinal symptoms to suggest an infectious etiology such as *Campylobacter* ("*C.*") *jejuni*.[10] Pet. Ex. 39 at 9-10. He also noted Petitioner was "never proven to have *C. jejuni*." Id. at 9. Other reasons Dr. Jeret concluded that Petitioner's GBS was not caused by *C. jejuni* include the fact that it causes an axonal form of GBS most often in China in the summer, none of which was present in Petitioner. Id.

Regarding onset, Dr. Jeret opined that Petitioner's symptoms began on December 22, 2018, the day before she presented to Mercy Health ED complaining of tingling in her hands and feet that began the previous day. Pet. Ex. 39 at 9 (citing Pet. Ex. 4 at 66-67). Petitioner received the flu vaccination on October 9, 2018, and onset was December 22, resulting in "an interval of 74 days." Id. (citing Pet. Ex. 1).

Dr. Jeret acknowledged that the Vaccine Injury Table provides an onset time frame that is up to 42 days after vaccination,[11] however, he cited several articles that he purported supported onset of up to ten weeks, and even up to "a few months and even years." Pet. Ex. 39 at 9 (quoting Pet. Ex. 41(e) at 4).[12] He concluded Petitioner's GBS 74 days post-vaccination was "still within the range reported in the medical literature." Id. at 10.

Dr. Jeret's reference to "a few months and years" is taken from Israeli et al. Pet. Ex. 39 at 9 (citing Pet. Ex. 41(e) at 4). There are two onsets that fit this statement: one related to the hepatitis B vaccine (referencing onset of three days to nine months) and the second related to the

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[10] *C. jejuni* is "is a common cause of enteric campylobacteriosis in humans." Campylobacter jejuni, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=62516 (last visited Dec. 1, 2025). *C. jejuni* has also "been found to stimulate cross-reactive antibodies that can result in GBS, particularly AMAN." Pet. Ex. 41(d) at 3 (Penina Haber et al., Vaccines and Guillain-Barré Syndrome, 32 Drug Safety 309 (2009)).

[11] Dr. Jeret misstated the onset period as five to 42 days in the Vaccine Injury Table. Pet. Ex. 39 at 9. The Vaccine Injury Table provides that for a Table claim of GBS following the flu vaccine, onset must fall within three and 42 days of vaccination. 42 C.F.R. § 100.3(a).

[12] Eitan Israeli et al., Guillain-Barré Syndrome—A Classical Autoimmune Disease Triggered by Infection or Vaccination, 42 Clinic. Rev. Allerg. Immunol. 121 (2012). This article was also cited as Resp. Ex. E.

measles-mumps-rubella vaccine (referencing onset of 80 days to years).[13] Pet. Ex. 41(e) at 5 tbl.2. Neither reference referred to the flu vaccination. Israeli et al.'s table on "[c]orrelation between vaccinations and GBS" does not include the flu vaccination. See id. Instead, the authors summarized articles related to the flu vaccination separately. Id. at 4.

Turning to GBS after flu vaccination (related primarily to the "swine flu" vaccination program in 1976), Israeli et al. relied upon Schonberger et al.[14] and stated "[t]he period of increased risk was concentrated primarily within the 5-week period after vaccination, although it lasted for approximately 9 or 10 weeks." Pet. Ex. 41(e) at 4 (quoting Pet. Ex. 41(q) at 1). Schonberger et al. found the peak relative risk of GBS "occurred in weeks 2 and 3 after vaccination." Pet. Ex. 41(q) at 8. "[P]rior to the 10th week after vaccination[,] all relative risks were significantly greater than 1, $p < .05$. From the 10th week on, the relative risks no longer remained significantly different from 1."[15] Id. Thus, the data showed the relative risk of GBS was not significant at 10 weeks post-vaccination and later.

Principi and Esposito[16] similarly summarized studies examining the risk of GBS post-flu vaccination. Pet. Ex. 18 at 4-5. Principi and Esposito cited the 2012 Institute of Medicine ("IOM") report[17] and noted the IOM undertook "a rigorous evaluation" of the casual association between the flu vaccination and GBS and determined that "[t]he period of highest risk was within the first 6 weeks after vaccine administration, although cases were detected even 9-10 weeks postvaccination." Id. at 4. Principi and Esposito stated that since then, there have been continued studies of the association with "conflicting results." Id. Although most studies have

[13] These papers were not filed in this case.

[14] Lawrence B. Schonberger et al., Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977, 110 Am. J. Epidemiol. 105 (1979). This article was also cited as Resp. Ex. A, Tab 15.

[15] "The *p*-value is the probability of getting a value of the test outcome equal to or more extreme than the result observed, given that the null hypothesis is true. The letter *p*, followed by the abbreviation "n.s." (not significant) means that $p > .05$ and that the association was not statistically significant at the .05 level of significance. The statement "$p < .05$" means that *p* is less than 5%, and, by convention, the result is deemed statistically significant. Other significance levels can be adopted, such as .01 or .1. The lower the *p*-value, the less likely that random error would have produced the observed relative risk if the true relative risk is 1." Michael D. Green et al., Reference Guide on Epidemiology, in Reference Manual on Scientific Evidence 594, 626 (3d ed. 2011), https://www.fjc.gov/content/reference-manual-scientific-evidence-third-edition-1.

[16] Nicola Principi & Susanna Esposito, Vaccine-Preventable Diseases, Vaccines and Guillain-Barré Syndrome, 37 Vaccine 5544 (2018).

[17] Inst. of Med., Adverse Effects of Vaccines: Evidence and Causality (Kathleen Stratton et al. eds., 2012).

not shown any association, at least two studies[18] showed that the risk of GBS was increased in the six weeks following vaccination. Id.

Dr. Jeret cited additional studies which he asserted supported his opinion that a 74-day onset of GBS is medically acceptable. Overall, these studies support a peak onset of GBS at two to three weeks post-vaccination, with a decline to baseline by six weeks of vaccination.

For example, he cited Souayah et al.,[19] a study examining the incidence of GBS after vaccination in the US from 1990 to 2005 using data from the Vaccine Adverse Event Reporting System ("VAERS"). Pet. Ex. 39 at 9 (citing Pet. Ex. 41(r) at 1). Dr. Jeret stated the authors "found that 8.4% of GBS cases associated with [flu] vaccination occurred [more than six] weeks after vaccination." Id. (citing Pet. Ex. 41(r) at 2 tbl.1). However, Souayah et al. found "the distribution of postvaccination GBS according to the date of onset of the disease follows a pattern with a peak occurrence within the first [two] weeks postvaccination followed by declining occurrence starting in the third week to reach a baseline in the fourth to sixth week."[20] Pet. Ex. 41(r) at 2 tbl.1, 4.

Dr. Jeret also cited a study from Stowe et al.[21] that he opined showed "the risk of GBS after flu vaccine was highest 61-90 days post-vaccination." Pet. Ex. 39 at 9 (citing Pet. Ex. 41(s)). Stowe et al. investigated the temporal association between GBS with both the flu vaccine and flu-like infectious illnesses using the United Kingdom General Practice Research Database of three million active patients for 1990 to 2005. Pet. Ex. 41(s) at 2. "The self-controlled case series method [] was used to test the hypothesis of an increased risk of [GBS] in the [three] risk periods of 0-30 days, 31-60 days, and 61-90 days after vaccination or [flu-like] illness." Id. Dr. Jeret did not acknowledge the fact that the authors "found no evidence of an association between [flu] vaccination and [GBS]." Id. at 4, 4 tbl.2.

[18] These studies (from Juurlink et al. and Lasky et al.) were not filed by the parties.

[19] Nizar Souayah et al., Guillain-Barré Syndrome After Vaccination in United States: Data from the Centers for Disease Control and Prevention/Good and Drug Administration Vaccine Adverse Event Reporting System (1990-2005), 11 J. Clin. Neuromuscul. Dis. 1 (2009).

[20] Souayah et al. noted that similar results were found in studies from Lasky et al. and Haber et al. Pet. Ex. 41(r) at 4. These studies were not filed.

[21] Julia Stowe et al., Investigation of the Temporal Association of Guillain-Barré Syndrome with Influenza Vaccine and Influenzalike Illness Using the United Kingdom General Practice Research Database, 169 Am. J. Epidemiol. 382 (2009).

Moving to Polakowski et al.,[22] Dr. Jeret noted the study "used 126 days as the cut-off for GBS onset after vaccines." Pet. Ex. 39 at 9 (citing Pet. Ex. 41(l)). Polakowski et al. studied post-vaccination GBS cases using medical records of hospitalized Medicare beneficiaries during the 2009-2010 flu season and focused on patients with GBS who were hospitalized within 126 days of receiving the flu vaccine. Pet. Ex. 41(l) at 1-2. Seventeen cases occurred in the risk period of one to 42 days and 12 occurred during the comparison period of 50 to 119 days. Id. at 4. The authors' analysis "found a slightly increased statistically significant risk of GBS within the [six]-week period." Id. at 7. Additionally, Polakowski et al. noted the "GBS risk was higher 8-21 days after vaccination," or one to three weeks post-vaccination. Id. at 8. No increased risk was reported after six weeks.

Lastly, Dr. Jeret cited Verboon et al.[23] for the proposition that "gradual GBS onset from first symptom to nadir can be up to [eight] weeks in 3% of patients." Pet. Ex. 39 at 9 (citing Pet. Ex. 41(u) at 2). Verboon et al. focused on the progression or worsening of symptoms after onset as it relates to optimal treatment; they did not discuss vaccine causation or the time interval of onset. See Pet. Ex. 41(u). The issue here is not the length of time from onset to nadir. Thus, Verboon et al. does not appear to be relevant.

Dr. Jeret also cited other literature that undersigned finds informative. Haber et al. studied the association between vaccination and GBS by reviewing peer-reviewed published literature from 1950 and 2008. Pet. Ex. 41(d) at 1. As background information the authors noted that "two-thirds of GBS cases occur several days or weeks after an apparent infectious illness . . . or upper respiratory tract infection." Id. at 1, 3. Potential biological mechanisms identified included molecular mimicry along with a genetic predisposition. Id. at 4. A thorough history of the association of the seasonal flu vaccine and GBS was provided, noting the risk period of six to eight weeks after swine flu vaccination (Schonberger et al.) and more recent studies showing an increased risk of "less than one additional case of GBS per million persons vaccinated" within six weeks of vaccination.[24] Id. at 5.

Petitioner also cited to excerpts of the 1994 and 2012 Institute of Medicine ("IOM") reports. See Pet. Exs. 47-50. One excerpt discussed the latency period between antigen exposure and peak adaptive immune response (for B and T cells), which is relevant to Petitioner's theory of molecular mimicry. Pet. Ex. 48 at 19-20. There are three phases of the immune response (lag, logarithmic, and plateau) and these phases occur with variability in time based on route of exposure, the antigen, whether it is a primary or secondary response. Id. Depending on the variables involved, "the development of the primary antibody response is 7 to

[22] Laura L. Polakowski et al., Chart-Confirmed Guillain-Barré Syndrome After 2009 H1N1 Influenza Vaccination among the Medicare Population, 2009-2010, 178 Am. J. Epidemiol. 962 (2013). This article was also cited as Pet. Ex. 19 and Pet. Ex. 26(a).

[23] Christine Verboon et al., Treatment Dilemmas in Guillain-Barré Syndrome, 88 J. Neurol. Neurosurg. & Psychiatry 346 (2017).

[24] These studies (from Lasky et al. and Varricchio et al.) were not filed by the parties.

10 days." Id. The IOM does not discuss a vaccine-induced immune response (innate or adaptive) where onset was ten weeks or longer.

In summary, Dr. Jeret opined that Petitioner met the diagnostic criteria for GBS, and that her symptoms began at 74 days after vaccination, which he maintained was "still within the range reported in the medical literature." Pet. Ex. 39 at 10. He further opined that the cause of Petitioner's GBS was her flu vaccine. Id.

Of note, Dr. Jeret did not discuss CIDP, other than to state that he has experience diagnosing patients with both GBS and CIDP. See Pet. Ex. 39.

2. Petitioner's Expert, Omid Akbari, Ph.D.[25]

a. Background and Qualifications

Dr. Akbari is a professor of immunology and professor of medicine at the University of Southern California, Keck School of Medicine. Pet. Ex. 22 at 2. He received a Ph.D. in cellular and molecular immunology at the National Institute for Medical Research in London, United Kingdom. Pet. Ex. 23 at 1. Thereafter, he completed a postdoctoral fellowship at Stanford University. Id. Dr. Akbari's research activities focus on the "[r]ole of [i]mmune cells in health and disease," including "[i]mmune tolerance and immune dysregulation" and the "[r]ole of [i]mmune cells in viral and demyelinating disease." Id. at 5-7. Dr. Akbari serves as an associate editor and reviewer on several journals. Id. at 4-5. He has authored or co-authored numerous publications. Id. at 9-16.

b. Opinion

i. Althen Prong One

Dr. Akbari generally advanced the theory of molecular mimicry as the causal mechanism relevant here and gave three examples of how molecular mimicry can cause "an immune stimulated response to the flu vaccination" to "trigger demyelinating disease such as GBS/CIDP." Pet. Ex. 22 at 17. The first is potential molecular mimicry between hemagglutinin

[25] Dr. Akbari submitted two expert reports. Pet. Exs. 22, 27. In portions of Dr. Akbari's reports, he discussed issues not related to the question of whether the flu vaccine can cause GBS/CIDP, onset, or the Althen causation prongs. For example, in his first report, he discussed the heterogenicity of CIDP, the concept of immune tolerance, and host susceptibility. See Pet. Ex. 22 at 4-6, 16. In his second report, he discussed the issue of denominators representing "significance" in rare diseases, the role of IL-17 in CIDP, and the roles of genetic and environmental factors in host susceptibility. See Pet. Ex. 27 at 8-13. As these matters do not directly relate to the issues in dispute, these sections are not summarized.

(a protein in the flu vaccine) and myelin basic protein.[26] Id. at 7-8. He asserted that the amino acid sequence "fyknli" has homology with myelin basic protein sequence "ffkniv." Id. Dr. Akbari opined that disease inducing homology can occur when there are five homologous amino acids within a 12 amino acid sequence. Id. at 8. Dr. Akbari's second example of homology was based on the flu A New Jersey "swine flu" vaccination in 1976 and its association with GBS. Id. at 12. And Dr. Akbari's third example of homology included "[*C.*] *jejuni* antigens that mimic human gangliosides capable of inducing an anti-GM1 antibody response" associated with demyelinating disease. Id.

In addition to offering examples of molecular mimicry, Dr. Akbari discussed his opinion regarding delayed onset of autoimmunity after flu vaccination. Pet. Ex. 22 at 14-15. He stated that many regulatory mechanisms maintain "peripheral immune tolerance after infection and vaccination." Id. at 14. Regulatory T cells are charged with maintaining immune tolerance, but there are occasions when they are unable to do so, "particularly if new autoreactive antibodies and immune cells keep forming in the recipients for a long period of time." Id.

In support of his opinion that immune cells can form antibodies for a long period of time, Dr. Akbari cited a study by Toplak et al.[27] that evaluated autoimmune responses in a group of healthy adults (92) following flu vaccination. Pet. Ex. 22 at 14-15 (citing Pet. Ex. 17 at 1). In the 92 heathy adults, increased or new levels of autoantibodies[28] were present in 15% at one month after vaccination and in 13% at six months after vaccination. Id. at 1, 5. Toplak et al. concluded that the changes in autoantibodies were attributable to the flu vaccination. Id. at 4. Molecular mimicry was described as a "[p]ossible" explanation for the study findings. Id. The authors stated that a "[p]rolonged autoimmune response" after the flu vaccination could not be excluded. Id. at 5. None of the study participants developed any autoimmune illnesses. Id. at 4. The authors did not suggest that the findings supported a delayed onset of an autoimmune illness after vaccination. See id. at 1-5.

Based on the findings in Toplak et al., that the flu vaccination can "transiently or persistently" elevate autoantibodies, Dr. Akbari opined its ability to "generate autoimmunity is significantly higher." Pet. Ex. 22 at 15. He asserted the findings support "many cases [of] delayed onset of GBS/CIDP" post-vaccination and cited three additional studies in support. Id.

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[26] Myelin basic protein "constitutes about 30 per cent of myelin proteins; elevated levels of [myelin basic protein] occur in acute exacerbation of multiple sclerosis and acute cerebral infarction. Immunization of laboratory animals with [myelin basic protein] produces encephalomyelitis by inducing T-cell activity that leads to demyelination and lymphoid infiltration." Myelin Basic Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100535 (last visited Dec. 1, 2025).

[27] N. Toplak et al., Autoimmune Response Following Annual Influenza Vaccination in 92 Apparently Healthy Adults, 8 Autoimmun. Revs. 134 (2008). Petitioner also cited this article as Pet. Ex. 25(z).

[28] The autoantibodies studied included antinuclear antibodies ("ANA"), anticardiolipin antibodies ("aCL"), lupus anticoagulant ("LA"), and others. Pet. Ex. 17 at 1, 3 tbl.1.

The first was Polakowski et al., also cited by Dr. Jeret. See Pet. Ex. 41(l). To reiterate, Polakowski et al. focused on patients with GBS who were hospitalized within 126 days of receiving the flu vaccine. Id. at 1-2. Their analysis "found a slightly increased statistically significant risk of GBS within the [six]-week period," with a higher risk of GBS within 8-21 days (one to three weeks) post-vaccination. Id. at 7-8. The authors made no reference to the concept of delayed onset of GBS attributable to vaccination.

Two other studies cited by Dr. Akbari as examples of "delayed onset" reported similar results. Perez-Vilar et al.[29] used a risk window of one to 42 days after vaccination with a control window of 43 to 84 days post-vaccination to study Medicare patients and Vaccine Safety Datalink data for the 2018-2019 flu season. Pet. Ex. 26(b) at 1-2. Like Polakowski et al., Perez-Vilar et al. "found slightly elevated borderline statistically significant [odds ratios] in the 8- to 21-day risk window." Id. at 6. The authors did not discuss the notion of delayed onset of GBS.

Salmon et al.[30] also used a "self-controlled risk-interval design" with an exposure or risk window of one to 42 days post-vaccination and a comparison or control period of 50 to 91 days post-vaccination. Pet. Ex. 26(c) at 4. Like Polakowski et al., the authors used a one-week washout period during days 43 to 49. Id. They found a "small increased risk of [GBS]" in the exposure period of one to 42 days. Id. at 5. The authors did not discuss the concept of delayed onset.

In his second expert report, Dr. Akbari responded to Dr. MacGinnitie's criticism of his reliance on Toplak et al. to support the idea of a delayed immune response ("second peak of autoimmunity"). Pet. Ex. 27 at 9, 14-15. Dr. Akbari explained that Toplak et al. was "simply presented as a proof of concept." Id. at 14 (emphasis omitted). Dr. Akbari also responded by opining that molecular mimicry was "only part of the equation;" "pathogenic T cells, [regulatory T cells ("Tregs")], [and] environmental factors" are "often essential" and "explain how molecular mimicry can facilitate both immune tolerance and immune autoreactivity." Id. at 8. He did not explain how these other factors could cause delayed onset after vaccination.

Additionally, Dr. Akbari, in his second expert report, generally discussed inflammasome activation and the presence of cytokines (IL-1, IL6, and others) found in patients with GBS/CIDP and concluded that "studies support the notion that induction of inflammasome [] along with T cell stimulation[] after administration of [flu] vaccine is able to trigger pathogenic T cells and cause GBS and CIDP." Pet. Ex. 27 at 1-6. He did not opine that his theory of inflammasome activation would explain an onset of 74 days.

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[29] Silvia Perez-Vilar et al., Guillain-Barré Syndrome After High-Dose Influenza Vaccine Administration in the United States, 2018-2019 Season, 223 J. Infect. Dis. 416 (2021). Petitioner also cited this article as Pet. Ex. 20.

[30] Daniel A. Salmon et al., Association Between Guillain-Barré Syndrome and Influenza A (H1N1) 2009 Monovalent Inactivated Vaccines in the USA: A Meta-Analysis, 381 Lancet 1461 (2013). Petitioner also cited this article as Pet. Ex. 21.

Petitioner's medical literature shows that the flu vaccination increases levels of proinflammatory cytokines, which may be associated with local and systemic responses that occur shortly after vaccination. Christian et al.[31] studied post-flu vaccination proinflammatory cytokine responses. Pet. Ex. 28(c) at 1. Serum cytokines were tested at days one through three after vaccination. Id. Injection site soreness and increases in two cytokines tested were elevated at one day post-vaccination. Id. Illness-like symptoms and serum cytokines were elevated at two days post-vaccination. Id. at 1-2. There was no mention of delayed onset of these effects.

ii. Althen Prongs Two and Three

Regarding Althen prong two, Dr. Akbari opined that Petitioner's onset of symptoms support "the most logical sequence of cause and effect" that the flu vaccine caused her "demyelinating disease and CIDP." Pet. Ex. 22 at 17.

For Althen prong three, Dr. Akbari concluded that "although the timing between receipt of the vaccine and development of demyelinating disease in [Petitioner] was slightly longer than usual, many recent reports support the fact that adverse effects and autoantibodies may be detected at [a] later time after flu vaccination." Pet. Ex. 22 at 18. He opined "this might be due to the late generation of autoantibodies and dysregulated immune responses." Id.

3. Respondent's Expert, Harold Moses, Jr., M.D.[32]

a. Background and Qualifications

Dr. Moses is a board-certified neurologist. Resp. Ex. C at 1; Resp. Ex. D at 2. He is an associate professor of neurology in the division of neuroimmunology and MS at Vanderbilt Medical Center. Resp. Ex. C at 1. He received his M.D. from the University of North Carolina, Chapel Hill. Resp. Ex. D at 1. He then completed a neurology residency at the Mayo Clinic followed by a fellowship at Vanderbilt Medical Center. Id. In his clinical neurology practice, he sees approximately 1600 patients. Resp. Ex. A at 1. Ninety percent of his patients have MS. Id. Dr. Moses has also been "involved in several clinical trials in MS and [has] been for more than 25 years." Id. He has also "seen patients with [GBS] in the past five years in a hospital setting." Id. In addition to his clinical practice, he has "published on both the clinical and immune aspects of MS." Id.; see also Resp. Ex. D at 8-10.

[31] Lisa M. Christian et al., Proinflammatory Cytokine Responses Correspond with Subjective Side Effects After Influenza Virus Vaccination, 33 Vaccine 3360 (2015).

[32] Dr. Moses submitted two expert reports. Resp. Exs. C, Q.

b. Opinion

Dr. Moses agreed with Dr. Jeret that onset of Petitioner's symptoms began December 22, 2018. Resp. Ex. C at 2. Dr. Moses opined that this onset period of 75 days[33] "between [flu] vaccination and development of symptoms falls outside an acceptable hypothetical time frame for an association." Id. at 5.

Regarding diagnosis, Dr. Moses agreed Petitioner's diagnoses was GBS ("presumed GBS"). Resp. Ex. C at 2-3. He further agreed that her CSF study showed an elevated protein level in the absence of an abnormal white blood cell count indicating cytoalbuminologic dissociation, "a hallmark feature of inflammatory peripheral neuropathies." Id. at 2. While he did not refute the diagnosis of GBS, Dr. Moses noted that over time, Petitioner's diagnosis changed from GBS to CIDP. Id. at 3. He attributed this to Petitioner's "evolving weakness despite IVIG therapy and likely also to justify treating her with IVIG over time." Id. Because no EMG/NCS reports were available for his review, Dr. Moses opined that Petitioner's pathology and diagnosis is not clear. Id. Further, because she did not improve on IVIG treatment, he suspected Petitioner may have had axonal injury. Id. He conceded, however, that there is a rare type of CIDP characterized by progressive weakness as mentioned in Dr. Akbari's report. Id. (citing Pet. Ex. 22 at 4). Regardless, Dr. Moses opined that Petitioner "did not have typical CIDP" since her presentation included "an acute and discrete onset of [] symptoms." Id. at 4.

As for the etiology of Petitioner's GBS/CIDP, Dr. Moses quoted a reference from her hospital admission history and physical where she reported no recent illness, "but [] she did have some fatigue [two] days last week." Resp. Ex. C at 2 (quoting Pet. Ex. 4 at 8). Petitioner also reported that "a viral [gastrointestinal] bug did go through her office," but she denied having any symptoms. Id. (quoting Pet. Ex. 4 at 8). Dr. Moses explained that in approximately one third of GBS cases, symptoms begin "a few days or weeks after . . . symptoms of a respiratory or gastrointestinal microbial infection." Id. at 2, 4. He agreed, however, that the medical records do not support a finding that Petitioner's GBS was due to a *C. jejuni* infection. Resp. Ex. Q at 1. Dr. Moses further agreed that in rare cases, "surgery or vaccinations may trigger [GBS]." Resp. Ex. C at 4.

In conclusion, Dr. Moses opined that Petitioner's onset was outside the time frame for an association with vaccination. Resp. Ex. C at 5. Therefore, he opined that the "[flu] vaccination had no role in the subsequent development of [P]etitioner's GBS or CIDP." Resp. Ex. Q at 2.

[33] Respondent's experts use October 8 instead of October 9 as Petitioner's date of vaccination, resulting in an onset of 75 days instead of 74.

4. Respondent's Expert, Andrew MacGinnitie, M.D., Ph.D.[34]

a. Background and Qualifications

Dr. MacGinnitie is board certified in allergy/immunology and pediatrics. Resp. Ex. A at 2; Resp. Ex. B at 10-11. He received his M.D. from University of Chicago in 1998, after which he completed a pediatric residency at Boston Children's Hospital and Boston Medical Center, an allergy/immunology fellowship at Boston Children's Hospital, and a pediatric clinical fellowship at Harvard Medical School from 1998 to 2004. Resp. Ex. B at 1. Since 2004, Dr. MacGinnitie has held academic appointments as well as hospital appointments. Id. at 1-2. He "maintain[s] an active clinical practice seeing more than 1600 patients annually[] and ha[s] extensive experience in caring for children and adults with a variety of immunologic diseases including reactions to vaccines." Resp. Ex. A at 2. He "also perform[s] research and [has] published articles in a number of areas related to Allergy/Immunology including food allergy, vaccine reactions, and primary immunodeficiency." Id. He is a member of professional societies, holds editorial positions on journals, and has published in the field of immunology. Resp. Ex. B at 3-4, 12-16.

b. Opinion

Dr. MacGinnitie focused on question of whether the flu vaccine can cause GBS/CIDP given a "substantial delay[] after vaccination." Resp. Ex. A at 5, 10; see also Resp. Ex. J at 1. He concluded that onset here was "outside the time interval that would typically be accepted for an immune stimulus triggering an autoimmune disease." Resp. Ex. J at 1.

The risk period for flu vaccination to be causally associated with GBS, as defined by the Vaccine Injury Table, is three to 42 days. Resp. Ex. A at 11 (citing 42 C.F.R. § 100.3(a)). Dr. MacGinnitie described how this risk period was established, citing Schonberger et al. and the swine flu vaccine experience of 1976-1977. Id. As previously noted, Schonberger et al. determined the risk period of GBS post-flu vaccine was six weeks but suggested it could be as long as nine to 10 weeks. Id. (citing Pet Ex. 41(q)). Follow-up studies found the increased risk period was six weeks, after which the rate of GBS decreased to the baseline rate. Id. Some of the studies showed no increased risk of GBS following receipt of the flu vaccination. Id.

Before turning to his specific opinions regarding timing, Dr. MacGinnitie criticized Dr. Akbari's proffered causal theories. Dr. MacGinnitie explained that Dr. Akbari's opinion about molecular mimicry based on proposed homology between hemagglutinin protein in the vaccine and myelin basic protein is from a study about multiple sclerosis. Resp. Ex. A at 7. Multiple sclerosis is not the disease at issue here and myelin basic protein "is not a known target of autoreactive T-cells or antibodies in GBS."[35] Id. at 7-8 (emphasis omitted).

[34] Dr. MacGinnitie submitted two expert reports. Resp. Exs. A, J.

[35] For more on this subject, and the back and forth between Dr. Akbari and Dr. MacGinnitie related to proteins other than hemagglutinin, see Resp. Ex. J at 7.

Dr. MacGinnitie also noted Dr. Akbari cited to a study where complete Freund's adjuvant was used to induce experimental autoimmune encephalitis in animals, and he explained that this adjuvant is toxic and not used in vaccines. Resp. Ex. A at 7. Furthermore, the vaccine here did not include an adjuvant. Id. Similarly, Dr. Akbari discussed the role of alum as an adjuvant, and again, this is not relevant, since alum was not present in the vaccine given to Petitioner. Id. at 5, 7, 9-10.

As for Dr. Akbari's principal theory of molecular mimicry, that the flu vaccine "triggered development of autoreactive T cells and/or antibodies" that caused GBS/CIDP, epidemiologic evidence shows this immune response occurs within 42 days of vaccination. Resp. Ex. A at 12. Dr. MacGinnitie noted that an immune response of up to 42 days is consistent with the understanding that T-cell responses take about one week to develop after infection. Id. at 12-13. Antibody responses take longer because B cells require T cells to mount effective antibody levels, and thus, B cell responses occur at two to four weeks. Id. at 13. Therefore, Dr. MacGinnitie opined an onset of more than ten weeks (75 days) is too long to attribute causation to vaccination. Id.

Dr. Akbari equated infections to vaccinations as evidence of his theory of delayed onset of autoimmunity as well as support of his proffered theory related to T cell responses in CIDP. Pet. Ex. 22 at 5, 14. He opined the timing here is "consistent with what has been published about onset of CIDP resulting from infections and/or vaccinations." Id. at 17.

Responding to Dr. Akbari's reliance on infections in support of onset, Dr. MacGinnitie observed that the most well-established risk factor for GBS is infection with *C. jejuni*, and the risk interval for it is one to three weeks. Resp. Ex. A at 12; see, e.g., Resp. Ex. A, Tab 7 at 11-12 (the IOM noting "[a]pproximately one fourth of patients with GBS have had [*C.*] *jejuni* infection in the preceding few weeks"); Resp. Ex. A, Tab 8 at 1 ("Almost 25%-40% of GBS patients worldwide suffer from *C. jejuni* infection [one to three] weeks prior to the illness.");[36] Resp. Ex. A, Tab 9 at 2 ("Based on the time course from onset of [*C. jejuni*] enteritis to the onset of neurologic symptoms ([one] to [three] weeks), this temporal relationship suggested that humoral immunopathogenic mechanisms were operative.").[37] This was true for other infections as well. See, e.g., Resp. Ex. A, Tab 17 at 4 ("The time frame from [flu] infection to GBS onset varies, but is generally between [three] days and 30 days, with an average of about 15 days. This time to onset is similar to that reported in GBS after other infections.").[38] The literature he cited also explained that some natural infections, including *C. jejuni* and flu virus, are thought to cause GBS via molecular mimicry. See, e.g., Resp. Ex. A, Tab 9 at 7; Resp. Ex. A, Tab 17

[36] Kishan Kumar Nyati & Roopanshi Nyati, Role of *Campylobacter jejuni* Infection in the Pathogenesis of Guillain-Barré Syndrome: An Update, 2013 BioMed. Rsch. Int'l 1.

[37] Irving Nachamkin et al., *Campylobacter* Species and Guillain-Barré Syndrome, 11 Clin. Microbiol. Rev. 555 (1998).

[38] Helmar C. Lehmann et al., Guillain-Barré Syndrome After Exposure to Influenza Virus, 10 Lancet Infect. Dis. 643 (2010).

("Molecular mimicry is the leading pathogenetic concept in postinfectious GBS . . . ."). Thus, using data regarding infections to support the theory of molecular mimicry as applied to vaccines supports an onset consistent with what is seen in post-infectious GBS. Dr. MacGinnitie concluded that "the 75 days gap between vaccination and onset of symptoms is too delayed to reasonably be explained by an adaptive immune response to the vaccine." Resp. Ex. A at 13.

Specific to the flu virus as well as flu vaccination, Cox et al.[39] described the time frame of the immune response. Resp. Ex. P at 5. "The humoral immune system produces antibodies against [] [flu] antigens . . . ." Id. During a primary or initial flu infection, "the three major Ig classes can be detected within 10-14 days." Id. As compared to the humoral immune system, cellular immune responses assist in recovery from infection and may prevent complications of infection. Id. "The primary cytotoxic response is detectable in blood after 6-14 days and disappears by day 21 . . . ." Id. Cox et al. next described time frames relative to the immune response to the trivalent inactivated flu vaccine. Id. at 8. They explained that antibodies can be detected within two to six days, and within two weeks, most vaccinees have protective antibody titers. Id. The antibody response "peaks [two to three] weeks postvaccination . . . and then wanes over time." Id.

Dr. MacGinnitie observed that Dr. Akbari presented two arguments. First, Dr. Akbari asserted that in general, the "[flu] vaccine is associated with delayed onset of autoimmunity." Resp. Ex. A at 10. Second, Dr. Akbari argued that the "[flu] vaccine has been shown to trigger GBS [] with substantial delays after vaccination." Id. Dr. MacGinnitie disagreed with both assertions.

In support of his delayed onset opinions, Dr. Akbari relied on Toplak et al., and Dr. MacGinnitie took issue with Dr. Akbari's application of the study. Resp. Ex. A at 10-11. First, most of the study participants (52 of 92) had autoantibodies before vaccination. Id. at 10 (citing Pet. Ex. 25(z) at 3 tbl.1). Next, the level of autoantibodies was stable in the majority (53 participants) at one month and at six months (54 participants), with some variation noted. Id. Third, the authors concluded that "our study demonstrated no statistically significant difference in the percentage of participants with positive autoantibodies before, [one] and [six] months after the [flu] vaccination." Id. (quoting Pet. Ex. 25(z) at 4). Fourth, regarding methodology, Dr. MacGinnitie observed that the study did not include a control group of participants who did not receive the flu vaccine. Id. at 10-11. And fifth, "the study did not examine autoantibodies against nerve cells such as those against gangliosides often seen in GBS." Id. at 11. For these reasons, Dr. MacGinnitie opined the study did not support Dr. Akbari's opinions. Id.

Dr. MacGinnitie disagreed with Dr. Akbari's opinion that the "[flu] vaccine has been shown to trigger GBS [] with substantial delays after vaccination," as well as the purported supportive literature. Resp. Ex. A at 10-12. Specific to Petitioner, Dr. MacGinnitie agreed her

[39] R.J. Cox et al., Influenza Virus: Immunity and Vaccination Strategies. Comparison of the Immune Response to Inactivated and Live, Attenuated Influenza Vaccines, 59 Scand. J. Immunol. 1 (2003).

onset was December 22, 2018, 75 days post-vaccination. Id. at 11-12. But none of the literature cited by Petitioner's experts support such a long onset period. Id.; Resp. Ex. J at 1-6; see, e.g., Pet. Exs. 26(a), 26(b), 26(c). Acknowledging Dr. Akbari proposed several mechanisms to explain how the flu vaccine can cause GBS/CIDP, Dr. MacGinnitie explained that all the proffered mechanisms cause immune effects that "occur soon after vaccination." Resp. Ex. J at 1.

Starting with Dr. Akbari's opinions related to inflammasomes, Dr. MacGinnitie reviewed articles cited by Dr. Akbari showing inflammasome induction occurred within 24 hours of vaccination. Resp. Ex. J at 1-2 (citing Pet. Ex. 51 at 4 ("[Flu] vaccination did not appear to have an immediate effect on macrophage inflammasome activity, although IL-1β levels were slightly elevated in unstimulated macrophages 24 h[ours] post-vaccination compared to baseline.");[40] Pet. Ex. 28(c) at 1-2, 11 fig.1 (noting "a modest increase in IL-1β [was shown] in patients with significant arm soreness after vaccination, but not [in] those without arm soreness," and "this increase was only present for a single day and Il-1β levels [] returned to baseline by [two] days after vaccination")). Although Dr. MacGinnitie agreed the flu vaccination can "trigger cytokine production" that causes mild adverse side effects, such effects "are brief and self-limited." Id. at 2. Dr. MacGinnitie concluded that Dr. Akbari did not offer evidence that inflammasome-induced cytokines could trigger an immune response with the first symptoms occurring over 10 weeks post-vaccination. Id.

Next, Dr. MacGinnitie reviewed the studies cited by both Dr. Akbari and Dr. Jeret which supposedly evidenced a "second peak" or delayed onset of GBS/CIDP following flu vaccination. Resp. Ex. A at 11-12; Resp. Ex. J at 2-4. Dr. MacGinnitie explained that Petitioner's experts' explanations of these studies were misplaced and ignored the baseline rate of GBS in the population. Resp. Ex. A at 11-12; Resp. Ex. J at 2-4. Dr. MacGinnitie opined that "the mere presence of cases in a certain time interval does not implicate vaccination." Resp. Ex. A at 11. To implicate vaccination, evidence that the rate of GBS was increased during the time period would be required, and such evidence was not provided. Id. And even though the studies utilized control periods that were more than 42 days (six weeks), the studies did not find an increased rate of GBS past six weeks. Id. at 11-12. For example, Salmon et al. "examine[d] the baseline rate of GBS in the population and show[ed] the rate seen in the control period between 50 and 91 days after vaccination was not increased compared to baseline."[41] Id. at 12 (citing Pet. Ex. 26(c)). Dr. MacGinnitie concluded "Dr. Akbari has chosen to only present studies which support his hypothesis and ignored those . . . that do not" and has created data "post-hoc for the sole purpose of supporting his argument." Resp. Ex. J at 4.

[40] Stephen N. Crooke et al., Inflammasome Activity in Response to Influenza Vaccination Is Maintained in Monocyte-Derived Peripheral Blood Macrophages in Older Adults, 2 Front. Aging 1 (2021).

[41] For the applicable baseline rate, see Resp. Ex. A at 12 n.1.

For additional support, Dr. MacGinnitie cited literature that does not support an increased risk of GBS after six to eight weeks post-vaccination. In 1991, Safranek et al.[42] reported no increased risk of GBS after six weeks post-vaccination. Resp. Ex. A, Tab 16 at 1. And in 1984, Langmuir et al.[43] found that the increased risk of post-vaccination GBS "lasted for at least six weeks and possibly for eight weeks but not longer." Resp. Ex. K at 1, 22 (noting "it is probable that the observed rates during the seventh and eighth 7-day intervals [days 43-56 post-vaccination] are significantly elevated but the observation is a borderline one" and after the eighth 7-day interval, the incidence rate falls below baseline).

Regarding Dr. Jeret's reliance on Israeli et al., who suggested that GBS can be triggered years after vaccination, Dr. MacGinnitie opined there is no evidence to support this assertion, and thus, he does not find it credible. Resp. Ex. J at 5.

In conclusion, Dr. MacGinnitie opined that the onset of Petitioner's GBS/CIDP occurred December 22, 2018, "even more than 10 weeks after vaccination." Resp. Ex. J at 8. However, the studies cited by Petitioner's experts show an increased risk of GBS "confined to [six] to possibly [eight] weeks after vaccination." Id. Thus, he concluded Petitioner's illness was not caused by vaccination. Id.

III. DISCUSSION

A. Standards for Adjudication

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

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum.

[42] Thomas J. Safranek et al., Reassessment of the Association between Guillain-Barré Syndrome and Receipt of Swine Influenza Vaccine in 1976-1977: Results of a Two-State Study, 133 Am. J. Epidemiol. 940 (1991).

[43] Alexander D. Langmuir et al., An Epidemiologic and Clinical Evaluation of Guillain-Barré Syndrome Reported in Association with the Administration of Swine Influenza Vaccines, 119 Am. J. Epidemiol. 841 (1984).

Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 13(b)(1)(A).

Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical

conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

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

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

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner 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." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine he received caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v.

Sec'y of Health & Hum. Servs., 35 F.3d. 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether a petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

IV. ANALYSIS

A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury," determining facts relating to the claimed injury can be significant in a case like this, where the parties dispute Petitioner's diagnosis. Id.

The facts stipulated to by the parties taken from Petitioner's medical records establish that Petitioner was diagnosed with GBS ("impression was GBS") on December 24, 2018, after she presented to Mercy Health ER. Joint Submission at 1-2; Pet. Ex. 3 at 142-43; Pet. Ex. 4 at 21. The experts agreed. Dr. Jeret opined that Petitioner's diagnosis is GBS. He explained that she had ascending numbness and weakness, CSF characteristic of GBS, areflexia, autonomic dysfunction, and facial weakness. Respondent's expert neurologist, Dr. Moses, did not disagree

with the diagnosis of presumed GBS.[44] Dr. Moses explained that over time, Petitioner's diagnosis evolved from GBS to CIDP. He attributed this change to Petitioner's continued weakness even after treatment with IVIG. Dr. Moses also suggested the diagnosis of CIDP was needed for the physicians to justify continued treatment with IVIG.

The medical literature suggests that CIDP is considered to be the chronic form of GBS. Dalakas et al.[45] stated "[d]espite ongoing clinical challenges with the diagnosis and definition, CIDP can be practically viewed as the chronic counterpart of [GBS] owing to various electrophysiological, histological[,] and immune similarities." Pet. Ex. 15 at 1. Donofrio et al.[46] explained that "[o]ver time, the question of CIDP arises [in a GBS patient] if the patient has a long protracted course of weakness." Pet. Ex. 41(b) at 13.

The undersigned finds that there is preponderant evidence that Petitioner's initial diagnosis following vaccination was GBS. This finding is based on her clinical course, diagnostic tests, the opinions of Petitioner's treating physicians, and the opinions of the experts. Further, the undersigned finds that given the facts and circumstances of this case, Petitioner was subsequently diagnosed with CIDP due to the chronicity of her illness; she did not initially present with CIDP. Further, Petitioner's diagnosis of CIDP did not indicate that she did not initially have GBS. Instead, the diagnosis of CIDP was used by Petitioner's physicians to acknowledge her continued weakness and to justify the continued need for IVIG treatment.

Therefore, as to the issue of diagnosis, the undersigned agrees with Petitioner and finds that her condition is "best described as GBS/CIDP." Joint Submission at 4.

This finding is also consistent with case law. The Federal Circuit has made clear that "identifying [the Petitioner's] injury is a prerequisite" to the Althen analysis. Broekelschen, 618 F.3d at 1346. But it is not necessary to diagnose an exact condition. Astle v. Sec'y of Health & Hum. Servs., No. 14-369V, 2018 WL 2682974, at *19 (Fed. Cl. Spec. Mstr. May 15, 2018). In Lombardi, the Federal Circuit explained that "[t]he function of a special master is not to diagnose vaccine-related injuries, but instead to determine based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused the [P]etitioner's injury." Lombardi v. Sec'y of Health & Hum. Servs., 656 F.3d 1343, 1351 (Fed. Cir. 2011) (internal quotation marks omitted) (quoting Andreu, 569 F.3d at 1382); see also Broekelschen, 618 F.3d at 1346 (citing Kelley v. Sec'y of Health & Hum. Servs., 68 Fed. Cl. 84, 100-01 (2005) for the proposition that "the [P]etitioner [is] not required to categorize his injury where the two possible diagnoses [are] 'variants of the same disorder'"). Furthermore, neither the Vaccine Act nor Althen burdens Petitioner with establishing a specific diagnosis. See

[44] Although Dr. Moses questioned whether Petitioner had the axonal form of GBS, while Dr. Jeret opined that Petitioner had the demyelinating form and did not have axonal injury, they both agreed her initial diagnosis was GBS.

[45] Marinos C. Dalakas, Advances in the Diagnosis, Pathogenesis and Treatment of CIDP, 8 Nat. Revs. Neurol. 507 (2011).

[46] Peter D. Donofrio et al., Guillaine-Barré Syndrome, 23 Continuum 1295 (2017).

Kelley, 68 Fed. Cl. at 100 ("The Vaccine Act does not require [P]etitioners coming under the non-Table injury provision to categorize their injury; they are merely required to show that the vaccine in question caused them injury—regardless of the ultimate diagnosis.").

Therefore, undersigned finds that Petitioner has proven by preponderant evidence that she suffered GBS. Her illness was subsequently diagnosed as CIDP, but that fact does not invalidate her original diagnosis of GBS.

B. Onset

Chief Special Master Corcoran previously found that the onset of Petitioner's GBS "more likely than not began on December 22, 2018." Findings of Fact at 5. The undersigned has reviewed all of the medical records, Petitioner's affidavit, and the expert reports and also finds that onset occurred December 22, 2018. The facts that form the basis of the undersigned's finding are the same facts relied on by Chief Special Master Corcoran, and the undersigned agrees with the reasons set forth in his ruling.

Specifically, as noted in the fact finding from Chief Special Master Corcoran, the medical records document an onset of December 22, 2018. Findings of Fact at 4-5. To reiterate, on December 23, 2018, at the ED, Petitioner was "seen in the ED because of bilateral upper and lower extremities tingling and numbness since yesterday." Pet. Ex. 3 at 143. Further history recorded that day also documented her "complaint of numbness/tingling in bilateral feet to ankle and both hands that started yesterday." Id. at 145. The next day, December 24, 2018, Petitioner "present[ed] to urgent care with complaints of paresthesia of hands and legs" that "started yesterday" and prompted an ED visit. Id. at 140-41. An ED note from December 24, 2018 indicated Petitioner reported her "numbness and tingling in her hands and feet . . . started Saturday [December 22] when she woke up." Id. at 138. And at a consult that same day, December 24, 2018, chief complaint documented "12/22/2018, the patient developed parenthesis in her feet." Id. at 142.

Further, the undersigned agrees with Chief Special Master Corcoran's finding that Petitioner's contemporaneous medical records are not consistent with Petitioner's account in her affidavit, which was executed two years after the events described. The contemporaneous records are silent as to the earlier signs or symptoms described by Petitioner in her affidavit.

Therefore, the undersigned does not find Petitioner's affidavit persuasive regarding the events that occurred after vaccination which are inconsistent with the medical records. See Vergara, 2014 WL 2795491, at *4 ("[T]estimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." (quoting Murphy, 23 Cl. Ct. at 733)); see also Doe/70, 95 Fed. Cl. at 608 ("Given the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law.").

Moreover, the experts, Dr. Jeret, Dr. Moses, and Dr. MacGinnitie, agree that Petitioner's symptoms of GBS began on December 22, 2018. Dr. Akbari did not commit to a specific date

for onset but did not refute the opinions of the other experts that December 22, 2018 represented the onset of symptoms. See generally Pet. Exs. 22, 27.

Considering the medical records, Petitioner's affidavit, expert opinions, case law, and the Findings of Fact, the undersigned finds by preponderant evidence that onset of Petitioner's symptoms of GBS occurred December 22, 2018.

C. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Here, the experts do not dispute the theory of molecular mimicry advanced by Dr. Akbari, nor do they dispute it as a sound and reliable theory that can cause disease or GBS in certain instances. However, they dispute whether the flu vaccine can cause GBS/CIDP when onset occurs 10 weeks (74 days) after vaccination.

Assuming that Petitioner has proven a sound and reliable causal mechanism under Althen prong one, the undersigned finds Petitioner did not provide preponderant evidence of a logical sequence of cause and effect or a proximate temporal relationship between the flu vaccination and Petitioner's GBS/CIDP. Due to the facts and circumstances of this case, specifically the fact that Petitioner developed symptoms of GBS 74 days after receipt of a flu vaccine, the undersigned's determination as to causation turns on an analysis of Althen prongs two and three, and thus, the undersigned focuses on Althen prongs two and three. See Vaughan ex rel. A.H. v. Sec'y of Health & Hum. Servs., 107 Fed. Cl. 212, 221-22 (2012) (finding the special master's failure to rule on Althen prong one not fatal to his decision because Althen prong two was fatal to Petitioner's case); Hibbard v. Sec'y of Health & Hum. Servs., 698 F.3d 1355, 1364 (Fed. Cir. 2012) ("discern[ing] no error in the manner in which the special master chose to address the Althen [prongs]" when he focused on Althen prong two after "assuming the medical viability of [the] theory of causation").

While the undersigned is not making a finding as to whether Petitioner has provided preponderant evidence of Althen prong one, the undersigned notes that a 74-day onset, which exceeds eight weeks, is not compatible with the theory of molecular mimicry. See, e.g., Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) ("[S]pecial masters have never gone beyond a two-month (meaning eight weeks) interval in holding that a vaccination caused a demyelinating illness."). Petitioner offered other causal mechanisms, but did not show by preponderant evidence that they could cause GBS/CIDP more than eight weeks after vaccination.

2. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

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

The undersigned finds Petitioner failed to provide preponderant evidence that there is a logical sequence of cause and effect showing Petitioner's flu vaccine caused her GBS/CIDP.

First, Petitioner's clinical course was not consistent with the theory of molecular mimicry, or any other theory offered due to the long onset period of 74 days, which is more than eight weeks. See, e.g., Barone, 2014 WL 6834557, at *13 (noting eight weeks/two months is the longest reasonable timeframe for a flu/GBS injury); De La Cruz v. Sec'y of Health & Hum. Servs., No. 17-783V, 2018 WL 945834, at *1 (Fed. Cl. Spec. Mstr. Jan. 23, 2013) (finding an onset of GBS more than two months after flu vaccination not compensable); Chinea v. Sec'y of Health & Human Servs., No. 15-095V, 2019 WL 1873322, at *32-33 (Fed. Cl. Spec. Mstr. Mar. 15, 2019) (finding a GBS onset of eleven to twelve weeks after vaccination was beyond the six- to eight-week medically appropriate timeframe for the occurrence of vaccine-induced GBS), mot. for rev. denied, 144 Fed. Cl. 378 (2019); Aguayo v. Sec'y of Health & Hum. Servs., No. 12-563V, 2013 WL 441013, at *3 (Fed. Cl. Spec. Mstr. Jan. 15, 2013) (rejecting an onset of three-and-one-half months in a flu/GBS case); Corder v. Sec'y of Health & Hum. Servs., No. 08-

228V, 2011 WL 2469736, at *27-29 (Fed. Cl. Spec. Mstr. May 31, 2011) (finding petitioner failed to prove that the flu vaccine can cause GBS four months after vaccination).

Second, Petitioner's treating physicians did not offer opinions in her medical records associating her GBS with her flu vaccination. In cases with such evidence, it can be considered in an analysis of Althen prong two. See Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)).

Accordingly, the undersigned finds that Petitioner failed to satisfy her burden under Althen prong two.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see also Pafford, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. See, e.g., Veryzer, 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that [P]etitioner must posit a medical theory causally connecting the vaccine and injury").

The Chief Special Master and the undersigned have both found onset to be December 22, 2018, 74 days or more than 10 weeks post-vaccination. The undersigned finds this time frame too long for Petitioner's GBS/CIDP to be causally associated with her October 2018 flu vaccination for several reasons.

First, the medical literature does not support this time frame. Petitioner asserts she has "provided scientific literature that supports a secondary peak window of 60-90 days with medical literature language that the 'period of increased risk . . . lasted approximately 9 or 10 weeks.'" Pet. Mot. at 3 (citing Pet. Ex. 41 (e) at 4). However, Petitioner and her experts mischaracterize the medical literature to support their contentions. See Pet. Mot. at 3.

Petitioner's reference to "9 or 10 weeks" is taken from Israeli et al., who cited to Schonberger et al. Pet. Ex. 41(e) at 4 (citing Pet. Ex. 41(q)). Schonberger et al. reported the peak relative risk of GBS "occurred in weeks 2 and 3 after vaccination" and an "increased risk [] concentrated primarily within the 5-week period after vaccination." Pet. Ex. 41(q) at 1, 8. Schonberger et al. also reported that "[p]rior to the 10th week after vaccination[,] all relative

risks were significantly greater than 1, $p < .05$ []. From the 10th week on, the relative risks no longer remained significantly different from 1." Id. at 8. Thus, the source relied upon by Israeli et al. did not find an increased risk at ten weeks or after ten weeks.

Further, Safranek et al. reassessed the Schonberger et al. data and determined there was "[n]o increase in relative risk for [GBS] . . . beyond [six] weeks after vaccination." Resp. Ex. A, Tab 16 at 1. Similarly, Langmiur et al. examined the Schonberger et al. data and found an increased risk for six weeks, a "borderline" risk in the seventh to the eighth week (up to day 56), followed by incidence rate below baseline after the eighth week (after day 56). Resp. Ex. K at 22.

In her Motion, Petitioner references Souayah et al. and Dr. Jeret's statement that the authors "found that 8.4% of GBS cases associated with [flu] vaccination occurred [more than six] weeks after vaccination." Pet. Mot. at 3; Pet. Ex. 39 at 9 (citing Pet. Ex. 41(r) at 1, 2 tbl.1). A review of the Souayah et al. data shows 511 (81%) of GBS cases occurred within six weeks after vaccination and 53 (8.4%) occurred after six weeks.[47] They reported the "the distribution of postvaccination GBS according to the date of onset of the disease follows a pattern with a peak occurrence within the first [two] weeks postvaccination followed by declining occurrence starting in the third week to reach a baseline in the fourth to sixth week," as seen in the below figure. Pet. Ex. 41(r) at 4, 4 fig.1.

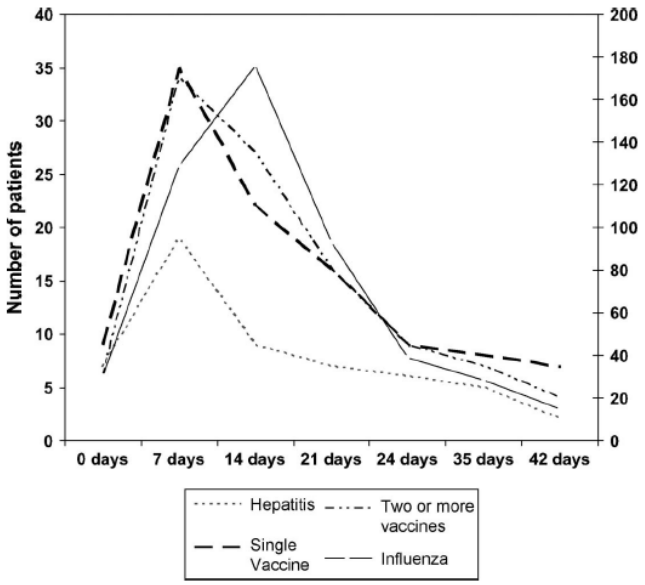

FIGURE 1. Guillain-Barré syndrome reported after vaccination, 1990 to 2005; distribution of cases in the first 42 days after vaccination.

Next, Petitioner cites to Stowe et al. asserting "the risk of GBS after flu vaccine was highest 61-90 days post vaccination." Pet. Mot. at 3 (citing Pet. Ex. 41(s)); Pet. Ex. 39 at 9. Although Petitioner and Dr. Jeret accurately noted the data, they failed to acknowledge the fact that the authors "found no evidence of an association between [flu] vaccination and [GBS]." See

[47] The remaining 68 (11%) cases did not have a known onset interval. Pet. Ex. 41(r) at 2 tbl.1.

Pet. Ex. 41(s) at 4, 4 tbl.2. Petitioner's expert, Dr. Jeret, did not account for the baseline rate of GBS as persuasively explained by Dr. MacGinnitie.

Petitioner also refers to Perez-Vilar et al., noting "the early-season analyses for all seasonal vaccines combined . . . identified 52 GBS claims in the secondary risk window and 42 in the control window." Pet. Mot. at 53 (citing Pet. Ex. 26(b) at 6 fig.2). Perez-Vilar et al.'s use of "Secondary Risk Window" refers to GBS cases in days 1-42 post-vaccination.[48] See Pet. Ex. 26(b) at 3, 5. There is no indication that the phrase was meant to indicate that cases occurring in this window were delayed in onset.

Petitioner argues Perez-Vilar et al. shows there is no "statistically significant drop off in GBS cases 70-80 days after vaccination." Pet. Mot. at 53. However, the authors "analyses did not find [any] statistically significant increased GBS risk[] for [the flu vaccine studied] during the 2018-2019 season." Pet. Ex. 26(b) at 6.

Petitioner also cited to Salmon et al., noting "many [GBS cases] were seen in the 68-to-92-day window after vaccination." Pet. Mot. at 54 (citing Pet. Ex. 26(c)). Salmon et al. found 54 cases in the "exposure period" (days 1-42) and 23 cases in the "comparison period" (days 50-91). Id. (citing Pet. Ex. 26(c) at 5 fig.2). Like Perez-Vilar et al., Salmon et al. did not conclude that the cases of GBS that occurred in the "comparison period" (days 50-91) were caused by vaccination.

Dr. Akbari stated, "many recent reports support the fact that adverse effects . . . may be detected at [a] later time after flu vaccination," citing Polakowski et al., Perez-Vilar et al., and Salmon et al. Pet. Ex. 22 at 18. However, as persuasively explained by Dr. MacGinnitie, these case-controlled studies may show GBS occurred during a control or comparison risk interval but the incidence did not exceed the background rate[49] or the results were not statistically significant based on the methodology used for the study. The authors of these studies did not show that GBS cases occurring after eight weeks were caused by the flu vaccine. Moreover, Respondent's experts did not opine that the methodology of any of these studies was problematic, raise technical issues, or otherwise suggest that the studies showed a statistical significance when the findings failed to show an association between the flu vaccination and onset of GBS.

In summary, Petitioner's argument that GBS cases that occurred during comparison/control periods support Petitioner's claim that cases of these onset periods can be attributed to vaccination is not accurate or sound and reliable. GBS cases do occur after 42 days post-vaccination, but to attach a causal relationship to such an occurrence, there must be a statistically significant number of cases not accounted for due to seasonality or background rates of disease. As such, Petitioner's attempts to mischaracterize the data to assert that the flu vaccine can cause delayed onset of vaccine-related GBS falls short of its intended mark.

[48] The authors "used days 8-21 and 1-42 postvaccination as primary and secondary risk windows, respectively, and days 43-84 postvaccination as control window." Pet. Ex. 26(b) at 3.

[49] For information on incidence and seasonal variation, see Resp. Ex. A, Tab 9 at 2.

The second reason why the undersigned finds Petitioner failed to provide preponderant evidence in support of Althen prong three is because she did not offer studies showing that onset of GBS can exceed eight weeks after the flu vaccination and be medically appropriate given any mechanism of causation. Petitioner, through Dr. Akbari, offered molecular mimicry and several other theories of causation, and cited studies that purported to show delayed onset or a secondary window of increased risk that lasted beyond eight weeks.

However, Toplak et al. does not support the concept of delayed onset. The undersigned finds Dr. MacGinnitie's discussion of Toplak et al. more persuasive, especially since the authors did not find antibodies associated with GBS/CIDP. Further, Dr. Akbari failed to provide foundational evidence to bridge the gap between the results of the study (finding of antibodies in minority of participants at six months after vaccination with no symptoms of illness) and any nexus with pathology or onset of illness. "An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be . . . persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of Health & Hum. Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280). This is not a new issue to Dr. Akbari. See, e.g., Nieves v. Sec'y of Health & Hum. Servs., No. 18-1602V, 2023 WL 2580148, at *44-45 (Fed. Cl. Spec. Mstr. Apr. 17, 2023) ("[E]vidence that vaccines might stimulate production of some immune cells, or even interact with them, does not mean that the vaccine causes the injury . . . .").

Although a petitioner need not make a specific type of evidential showing (i.e., epidemiologic studies) to satisfy her burden, special masters shall still consider and weigh the evidence in the record, including the epidemiological studies filed. See § 13(b)(1) (indicating the special master shall consider all materials in the record); Capizzano, 440 F.3d at 1325-26; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1149 (Fed. Cir. 1992) (finding "epidemiological studies are probative medical evidence relevant to causation" and "considerable weight [is] due to epidemiological studies in the absence of direct evidence of actual causation"). And after weighing the submitted evidence, the undersigned finds the evidence does not preponderate in Petitioner's favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."). The undersigned finds no evidence supports Petitioner's "delayed onset" argument or an association between the flu vaccine and the development of GBS when onset exceeds eight weeks.

Overall, Dr. Akbari's explanations of references cited in support of "delayed onset" misrepresents the methodology and conclusions of the studies. None of the studies stand for the proposition that GBS cases occurring during a comparison or control window were caused by vaccination. Dr. Akbari did not explain how these studies supported his opinion of delayed onset. By suggesting the studies reported an association between flu vaccine and delayed onset GBS, Dr. Akbari mischaracterizes the findings, adversely affecting his credibility and the reliability of his opinions. This critique of Dr. Akbari is not new. See, e.g., Powell v. Sec'y of Health & Hum. Servs., No. 20-1726V, 2025 WL 3443590, at *32 (Fed. Cl. Spec. Mstr. Oct. 8, 2025); Williams v. Sec'y of Health & Hum. Servs., No. 19-1269V, 2024 WL 5040482, at *43

(Fed. Cl. Spec. Mstr. Nov. 13, 2024); Nieves, 2023 WL 2580148, at *44-45 (noting Dr. Akbari "attempted to spin medical evidence" and had "several erroneous foundational contentions").

The third reason why the undersigned finds Petitioner failed to provide preponderant evidence of Althen prong three is that the medical literature consistently reports eight weeks as the outside limit within which the flu vaccine can induce an immune response so as to cause a demyelinating illness like GBS/CIDP. See, e.g., Resp. Ex. K at 1, 22 (finding an increased risk of post-vaccination GBS "lasted for at least six weeks and possibly for eight weeks but not longer," and explaining "the observed rates during the seventh and eighth 7-day intervals [days 43-56 post-vaccination] are significantly elevated but the observation is a borderline one" and after the eighth 7-day interval (day 56), the incidence rate falls below baseline); Resp. Ex. A, Tab 8 at 8 ("Vaccine-induced GBS was first observed within 6-8 weeks of receiving the 'swine flu' vaccine during [the flu] vaccination program in 1976-1977."); Resp. Ex. A, Tab 17 at 4 (explaining that in determining "what period after vaccination is reasonable to suggest a causal association[,] [m]ost studies have used a period of [six] weeks" based on the 1976-1977 swine flu program).

A fourth reason for the undersigned's findings arises out of Dr. Akbari's use of the words "possibility" and "might" in support of the notion of delayed onset. In his first report, in describing the study by Toplak et al., Dr. Akbari stated, "a recent study evaluated the possibility of autoimmune responses following [the] annual [flu] vaccination." Pet. Ex. 22 at 14. And at the end of this expert report, Dr. Akbari concluded that the delay in Petitioner's GBS onset "might be due to the late generation of autoantibodies and dysregulated immune responses." Id. at 18. In her brief, Petitioner also uses the word possible and may several times. See, e.g., Pet. Mot. at 34, 38, 40, 49.

The Merriam-Webster dictionary defines "might" as a word "used to say something is possible." Might, Merriam-Webster Dictionary, https://www.merriam-webster.com/dictionary/might (last visited Dec. 1, 2025). Similarly, Cambridge Dictionary indicates "might" is "used to express the possibility that something will happen or be done, or that something is true although not very likely." Might, Cambridge Dictionary, https://dictionary.cambridge.org/us/dictionary/english/might (last visited Dec. 1, 2025).

Opinions based on possibilities, however, are insufficient to prove causation. See Waterman, 123 Fed. Cl. 564, 573-74; Moberly, 592 F.3d at 1322; de Bazan, 539 F.3d at 1351.

Lastly, the undersigned finds the case law does not support an onset of 74 days. See, e.g., Barone, 2014 WL 6834557, at *13 (noting eight weeks/two months is the longest reasonable timeframe for a flu/GBS injury); Kindle v. Sec'y of Health & Hum. Servs., 177 Fed. Cl. 689, 712 (2025) (finding "[t]he Chief Special Master properly concluded that Petitioner had not proven his off-Table claim because the nine-week delay from Petitioner's vaccination to his symptom onset [was] too lengthy to be considered medically acceptable to infer causation-in-fact" (internal quotations omitted)); Jones ex rel. Boon v. Sec'y of Health & Hum. Servs., No. 19-788V, 2025 WL 2045820, at *22 (Fed. Cl. Spec. Mstr. June 24, 2025) (finding "an onset of 94 days or over three months after flu vaccination [] wholly unsupported by the medical literature," regardless of whether the petitioner suffered GBS or CIDP); Chinea, 2019 WL 1873322, at *32-33 (finding a

GBS onset of eleven to twelve weeks post-vaccination not medically appropriate); De La Cruz, 2018 WL 945834, at *1 (finding an onset of GBS more than two months after flu vaccination not compensable); Strong v. Sec'y of Health & Hum. Servs., No. 15-1108V, 2018 WL 1125666, at *21 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (finding a four-month onset between flu vaccination and CIDP was too long); Aguayo, 2013 WL 441013, at *3 (rejecting an onset of three-and-one-half months in a flu/GBS case); Corder, 2011 WL 2469736, at *27-29 (finding petitioner failed to prove that the flu vaccine can cause GBS four months after vaccination).

Although decisions of other special masters are not binding, the undersigned finds these cases instructive. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

In Kindle, the Chief Special Master found the record supported a GBS onset no earlier than 65 days post-vaccination, which was "too lengthy" and "too remote" to be medically acceptable. Kindle v. Sec'y of Health & Hum. Servs., No. 20-1423V, 2025 WL 603690, at *8 (Fed. Cl. Spec. Mstr. Jan. 21, 2025). For support, the Chief Special Master noted the longest timeframe generally accepted for similar non-Table claims was eight weeks (two months). Id.

On review, the Court of Federal Claims found "[t]he Chief Special Master properly concluded that Petitioner had not proven his off-Table claim because the nine-week delay from Petitioner's vaccination to his symptom onset [was] too lengthy to be considered medically acceptable to infer causation-in-fact." Kindle, 177 Fed. Cl. at 712. Judge Roumel explained the Chief Special Master's conclusion was not arbitrary and capricious for numerous reasons. Id. at 712-16. First, Judge Roumel found it was "permissible and reasonable for the Chief Special Master to rely on his own 'accumulated expertise,' as well as that of other special masters" in declining a more than eight-week delay in onset supportive of causation. Id. at 713.

Judge Roumel also cited numerous cases to support eight weeks/two months as the "longest timeframe . . . generally accepted" for non-Table GBS cases and noted "[t]he Court is not aware of any case in which a special master recognized an onset of longer than 60 days post-vaccination." Kindle, 177 Fed. Cl. at 712-13, 713 n.23 (citing Redzepagic v. Sec'y of Health & Hum. Servs., No. 19-853V, 2025 WL 1147520, at *16 (Fed. Cl. Spec. Mstr. Mar. 19, 2025) ("Decisions in the Vaccine Program have gone out as far as two months or 60 days for vaccine-caused demyelinating illness."); Cooper v. Sec'y of Health & Hum. Servs., No. 18-1885V, 2024 WL 1522331, at *18-20 (Fed. Cl. Spec. Mstr. Mar. 12, 2024) (noting that a 60-day onset is "on the very edge of the entirely undisputed eight-week latency period")).

Along with relying on his expertise, Judge Roumel noted the Chief Special Master also relied upon the medical literature in concluding a nine-week onset was not medically acceptable, and none of the literature supported an onset of vaccine-induced GBS beyond the generally accepted eight-week interval. Kindle, 177 Fed. Cl. at 713-14.

Additionally, Judge Roumel determined "[t]he Chief Special Master did not impermissibly impose a "hard and fast deadline . . . between vaccination and the onset of clinically apparent symptoms of neurologic injury." Kindle, 177 Fed. Cl. at 714-15 (quoting

Paluck v. Sec'y of Health & Hum. Servs., 786 F.3d 1373, 1383 (Fed. Cir. 2015)). Judge Roumel explained that

> Petitioner failed to proffer any persuasive evidence—in the form of medical literature or otherwise—which could preponderantly prove that his GBS onset occurred within a medically acceptable timeframe to infer vaccine causation. Absent such preponderant evidence supporting that—based on Petitioner's specific circumstances—a longer timeframe is appropriate, it was reasonable for the Chief Special Master to conclude that the timing prong of Althen had not been met.

Id. at 715. Thus, Judge Roumel determined the Chief Special Master appropriately concluded that a nine-week (or 65-day) onset of GBS was not medically acceptable under Althen prong three, and therefore, the petitioner's failure to meet this prong was dispositive of his case. Id. at 715-16.

In summary, there is a lack of evidence to establish that an onset of GBS occurring 74 days after vaccination is medically appropriate, and an onset of 74 days is too long to be explained by any sound or reliable medical theory of causation. Therefore, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to Althen prong three.

V. CONCLUSION

The undersigned extends her sympathy to Petitioner for the pain and suffering that she experienced due to her illness. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that her flu vaccine caused her GBS/CIDP. Therefore, Petitioner is not entitled to compensation, and the petition must be dismissed.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master