In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: August 27, 2025
Refiled as Redacted: November 12, 2025

R.P. and T.P., on behalf of their minor son, L.L.P.,	*	PUBLISHED
Petitioners,	*	No. 20-544V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Table Injury; Influenza ("Flu") Vaccine; Guillain-Barré Syndrome ("GBS").
Respondent.	*	

Michael Firestone, Marvin Firestone, MD, JD & Associates, San Mateo, CA, for Petitioners.
Ryan Nelson, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

I. INTRODUCTION

On May 1, 2020, R.P. and T.P. ("Petitioners"), on behalf of their minor son, L.L.P., filed a petition under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioners allege L.L.P. suffered a Table Guillain-Barré Syndrome ("GBS") injury as a result of an influenza ("flu") vaccination

[1] When this Ruling was originally filed, I advised my intent 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). In accordance with Vaccine Rule 18(b), Petitioners filed a timely motion to redact certain information. This Ruling is being reissued with Petitioners' initials. Except for those changes and this footnote, no other substantive changes have been made. This Ruling will be posted on the court's website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, with no further opportunity to move for redaction.

[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 Ruling to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

administered on October 12, 2017. Petition at 1 (ECF No. 1). In the alternative, Petitioners allege L.L.P.'s injuries were caused-in-fact by the flu vaccination administered to L.L.P. on October 12, 2017. Id. at 1-2. Respondent argued "compensation is not appropriate in this case." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 16).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[3] the undersigned finds Respondent failed to provide preponderant evidence that factors unrelated to the administration of the vaccine caused Petitioner's GBS, and thus, failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005).[4] The undersigned finds Petitioners have proved by preponderant evidence the criteria required to establish that L.L.P. suffered a Table injury for GBS post-flu vaccination. Therefore, Petitioners are entitled to compensation.

II. ISSUES TO BE DECIDED

The parties agree L.L.P., at nine years of age, received a flu vaccine on October 12, 2017. Joint Rept. ("Joint Sub."), dated July 16, 2024, at 1-2 (ECF No. 131). The parties stipulate the vaccine was administered in the United States and is recognized on the Vaccine Injury Table. Id. at 2. Additionally, the parties agree L.L.P. was diagnosed with GBS in November 2017. Id. at 1-2.

With regard to Petitioners' Table claim, the parties agree "Petitioners have met the elements of a *prima facie* Table claim for [flu] vaccination and GBS." Joint Sub. at 2.

However, "Respondent contests entitlement based on the theory of alternative causation," specifically "[w]hether an intervening viral illness was the actual cause of L.L.P.'s GBS instead of the [flu] vaccination." Joint Sub. at 3. Consequently, the parties disagree about whether L.L.P.'s "[onset] timeline supports vaccine-related causation or an alternative cause" and "whether the initial symptoms observed (such as respiratory issues and gastrointestinal complaints) are consistent with the onset of GBS or indicative of a viral illness." Id. at 2-3.

[3] 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.'" (citation omitted)), aff'd, 601 F. App'x 982 (Fed. Cir. 2015).

[4] Because the undersigned finds Petitioners have proven a Table claim for GBS following flu vaccination, the undersigned does not discuss or analyze the experts' opinions that relate to Petitioners' causation-in-fact claim.

Lastly, the parties agree "[t]he burden is on Respondent to show by preponderant evidence whether L.L.P.'s 'illness, disability, injury, condition, or death described in the petition is due to factors unrelated to the administration of the vaccine described in the petition.'" Joint Sub. at 2 (quoting § 13(a)(1)(B)).

III. BACKGROUND

A. Procedural History

Petitioners filed a petition on May 1, 2020, followed by medical records on May 13, 2020.[5] Petition; Petitioners' Exhibits ("Pet. Exs.") 1-18. The case was assigned to the undersigned on May 27, 2020. Notice of Reassignment dated May 27, 2020 (ECF No. 10). Respondent filed his Rule 4(c) report, arguing against compensation, on September 14, 2020. Resp. Rept. at 1.

On December 1, 2020, Petitioner filed an expert report from Dr. Lawrence Steinman. Pet. Ex. 21. Respondent filed an expert report from Dr. Leslie Benson on April 5, 2021. Resp. Ex. A.

In May 2021, the parties began engaging in settlement discussions. Joint Status Rept., filed May 5, 2021 (ECF No. 36). This matter was put on a dual track, with an entitlement hearing set for August 2024 and an outside mediator retained in August 2023. Prehearing Order dated Apr. 25, 2023 (ECF No. 90); Joint Status Rept., filed Aug. 14, 2023 (ECF No. 93). During informal settlement discussions, Petitioner filed two expert reports from Dr. Safwan Jaradeh and a supplemental expert report from Dr. Steinman and Respondent filed a supplemental expert report from Dr. Benson. Pet. Exs. 50, 113-14; Resp. Ex. C.

The parties were unable to informally resolve the matter and required a hearing to resolve entitlement. Joint Status Rept., filed Aug. 9, 2024 (ECF No. 142). An entitlement hearing was held August 13 to August 14, 2024. Order dated Aug. 14, 2025 (ECF No. 143). Petitioners, Ms. T.P. and Mr. R.P., Dr. Benson, Dr. Jaradeh, and Dr. Steinman testified. Transcript ("Tr.") 3, 224.

Following the entitlement hearing, both parties filed additional documents as well as post-hearing briefs from August 2024 to November 2024. Pet. Exs. 119-22; Resp. Ex. E; Pet. Post Hearing Brief ("Br."), filed Sept. 30, 2024 (ECF No. 152); Resp. Post-Hearing Br., filed Oct. 23, 2024 (ECF No. 153); Pet. Reply to Resp. Post-Hearing Br. ("Pet. Post Hearing Reply Br."), filed Nov. 13, 2024 (ECF No. 154).

This matter is now ripe for adjudication.

[5] Medical records were filed throughout litigation.

B. Factual History

1. Summary of Relevant Medical Records

a. Pre-Vaccination

L.L.P. had a history of wheezing and asthma for which albuterol frequently helped in his first two years. Pet. Ex. 8 at 33-34. At six years of age, he was rarely using albuterol and noted to be "growing out of [his] asthma." Id. at 22.

L.L.P. presented for a routine eye exam on July 28, 2017. Pet. Ex. 105 at 1. His diagnoses included bilateral paresis of accommodation, astigmatism of the right eye, myopia (nearsightedness) of the left eye, and presbyopia (gradual loss of ability to focus on nearby objects). Id. at 5. The ophthalmologist recommended a prescription for reading glasses and bifocals for school. Id.

On October 4, 2017, L.L.P. complained to the school nurse that the right side of his jaw/teeth hurt. Pet. Ex. 93 at 13. He stated that it hurt to open and close his mouth and bite down. Id. L.L.P. denied any injury and all teeth were noted to be healthy and intact with no bleeding in his mouth. Id. He was given over-the-counter pain medication. Id. at 14. L.L.P.'s mother was called, and she reported that she thought one of L.L.P.'s back teeth was loose and causing the pain. Id.

On October 6, 2017, L.L.P. complained to the school nurse that his stomach hurt. Pet. Ex. 93 at 15-16. L.L.P. did not have a fever. Id. He denied the need to throw up or go to the bathroom. Id. at 16. L.L.P.'s mother picked him up from school early. Id. at 15.

b. Vaccination and Post-Vaccination

L.L.P., at the age of nine, received a flu vaccination in his right deltoid on October 12, 2017. Pet. Ex. 3 at 8; Pet. Ex. 8 at 14.

On November 15, 2017, L.L.P. saw the school nurse complaining of a stomachache and overall not feeling well. Pet. Ex. 93 at 17. L.L.P. "[s]tate[d] that he has been absent this whole week because he was sick with a 'stomach flu'" and throwing up. Id. "He felt a little better this morning so [he] wanted to come to school. Now, at lunch, his stomach has started to hurt and [he] [felt] like he need[ed] to throw up." Id. L.L.P. did not have a fever. Id. The school nurse indicated L.L.P. looked "slightly pale." Id. L.L.P.'s mother picked him up from school early. Id.

On November 16, 2017, L.L.P. presented to his primary care physician ("PCP"), Dr. Deborah S. Presken,[6] for nausea, vomiting (emesis) for a "couple of days," and abdominal pain. Pet. Ex. 17 at 12. L.L.P. was noted to be vomiting intermittently over the past six days. Id. at 13. The vomiting resolved the day before (November 15), so he went to school; however, he

[6] This was L.L.P.'s first visit to Dr. Presken. Tr. 25, 27, 46.

went home early because he did not feel well. Id. He also had a headache, jaw pain, and chest tightness. Id. L.L.P. had no other illnesses or recent travel other than a camping trip the prior month. Id. On examination, Dr. Presken noted L.L.P. appeared "acutely ill" and pale. Id. L.L.P. had decreased lung/breath sounds, injected eyes (redness), and lymphadenopathy (enlarged lymph nodes) in the neck. Id. L.L.P. was also fatigued and exhibited pain when sitting straight. Id. He had normal bowel sounds, and no tenderness or masses. Id. Dr. Presken administered three nebulizer treatments, which resolved his symptoms. Id. L.L.P.'s diagnoses were vomiting, viral syndrome, and dyspnea (shortness of breath). Id. at 14. Dr. Presken's assessment indicated "[l]ikely viral illness" with an "unclear" etiology. Id. at 13-14. L.L.P.'s treatment plan included steroids, albuterol, and anti-nausea medication (Zofran). Id. at 14.

At 10:00 a.m. on November 19, 2017, Petitioners brought L.L.P. to the emergency department ("ED") at Children's Hospital of Colorado ("CHCO"). Pet. Ex. 5 at 1. ED physicians Dr. John "Jay" Watson, Dr. Neil Desai, and Dr. Michael Distefano treated L.L.P. in the ED. Id. at 1-8. ED records documented L.L.P. "present[ed] with headache and vomiting," as well as "progressing weakness and refusal to ambulate." Id. at 3. History of present illness summarized L.L.P.'s clinical course:

> He was completely well and at baseline with no prior infectious illness symptoms or recent illness, until Saturday November 11, [eight] days prior to presentation. He felt fatigued that day and had [one] episode of emesis that evening. No fever at this time or throughout entirety of history. Then, he was fatigued with decreased appetite for the next two days, both nights waking up from sleeping with headache and emesis [one time], then back to sleep. He was unable to go about his routine activities, staying home from school, but he did go to Black Belt Candidacy class on Tuesday, where he was able to complete the class, but felt weaker than usual during it. He was able to go to school Wednesday, but was quickly sent home for appearing ill, despite no fever or focal symptoms. That evening, he began to have more headache, jaw pain, and for the first time developed mild rhinorrhea and cough. He also began to have chest pain and some difficulty breathing, and full body aches. Presented to his primary doctor, Dr. [Presken] (actually his first time to go to this MD), where he threw up, complained of some difficulty breathing and was sent home with albuterol, prednisone, and [Z]ofran. This helped with his breathing some, but did not help his other symptoms. He progressively developed more weakness and more difficulty with coordination throughout the weekend, leading into his presentation today.

Id. at 3-4. Dr. Watson noted L.L.P.'s mid-October camping trip and noted no known bites or lesions. Id. at 4. L.L.P. also had no recent trauma, ingestion, or other abnormal events. Id. Review of systems was positive for cough, rhinorrhea, vomiting, body aches, weakness, and behavior changes. Id. at 5. L.L.P. denied congestion, eye drainage, fever, diarrhea, rashes, joint pain, weight loss, bleeding, and bruising. Id.

On physical examination, L.L.P. was positive for pallor (appeared pale). Pet. Ex. 5 at 5. His nose was patent without discharge, and his mucous membranes were pink, moist, and

without lesions. Id. Lymphadenopathy was not noted. See id. Respiratory examination documented "normal [work of breathing], lungs clear to auscultation bilaterally, with good air movement." Id. Neurological examination revealed "4-5/5 strength symmetric in all extremities, somewhat effort dependent," and "shaking uncoordinated movements on finger-nose-finger and heel-to-shin, but able to effectively touch finger." Id. Dr. Watson documented reflexes were 2+ symmetric but noted that the "[n]eurology team felt that lower extremity reflexes were not present and that movements . . . were voluntary." Id. Gait was not tested as "[L.L.P.] refus[ed] to walk." Id. at 6.

Dr. Watson determined there was "[n]o obvious etiology for constellation of symptoms." Pet. Ex. 5 at 6. A computed tomography ("CT") was ordered and showed no intracranial mass. Id. at 3, 6-7. Bloodwork was ordered and revealed "normal inflammatory markers" and an elevated white blood cell ("WBC") count. Id. at 3, 6-7, 10-11. Erythrocyte sedimentation rate ("ESR") and C-reactive protein levels were normal, as were L.L.P.'s chest X-ray and electrocardiogram ("EKG"). Id. at 7-8, 10-11. Flu infection was ruled out. Id. at 3, 11-12. Dr. Watson noted L.L.P.'s leukocytosis (elevated WBCs) "could at least partially be accredited to" L.L.P.'s prednisone as prescribed by his PCP, "but reportedly toxic granulation and vacuous [polymorphonuclear leukocytes ("PMNs")] was present which would not be expected in corticosteroid-induced leukocytosis, though are not specific to bacterial infection." Id. at 3. Dr. Watson concluded that "although many diagnoses have been considered and many ruled out, it is unclear at this time what is leading to [L.L.P.'s] weakness and other symptoms. It is possible that this is generalized weakness secondary to a severe systemic viral illness, and that with supportive care including IV hydration, he may improve." Id. Given the lack of diagnostic clarity and L.L.P.'s refusal to ambulate, L.L.P. was admitted with diagnoses of headache and generalized weakness. Id. at 3, 13.

On the night of November 19, 2017, at approximately 7:30 p.m., L.L.P. was seen by family Medicine Resident Dr. Susan G. Hoyum. Pet. Ex. 5 at 28. Dr. Hoyum detailed L.L.P.'s history of present illness beginning on November 11, when "multiple symptoms" began. Id.

> On Saturday (11/11), [L.L.P.] woke up feeling normal, but by late morning he felt fatigued and had one episode of [non-bloody, non-bilious] emesis in the evening. That night he woke up at midnight from sleep with a headache and emesis. He continued to feel fatigued for the next two days along with decreased appetite. He also woke up from sleep both nights with headache and emesis but was able to return to sleep. [L.L.P.] also report[ed] food taste[d] "funny," but was not able to give a description, not metallic tasting.
>
> On Tuesday (11/14), he did not go to school, but did attend his Black Belt Candidacy class which he was able to complete, but felt weaker than his norm. He did get hit in the head during class, but was wearing his helmet and his opponent was wearing gloves. Headache was resolved by Tuesday AM.
>
> On Wednesday (11/15), [L.L.P.] did attend school, but after lunch was sent home [due to] ill appearing, "pale, sunken eyes." That evening he started to

> have jaw pain, return of his headache, mild rhinorrhea, and cough at his Cub Scout meeting.
>
> On Thursday (11/16), [L.L.P.] presented to his PCP and was placed on albuterol, prednisone, and [Z]ofran [due to] complaint of difficulty breathing. The albuterol improved his chest pain for awhile, but returned when coughing. He also started to report troubles with coordination and weakness to the exten[t] he needed to use a cane and need assistance to the bathroom.
>
> Friday to Sunday (day of admission), [L.L.P.] has had progressive weakness and increased difficulties with coordination, continued headache, jaw pain. His cough ha[d] progressed to a "weak" productive cough. He [was] also reporting [bilateral] ankle pain starting today. He felt better on Saturday night, but his symptoms returned early Sunday morning therefore presented to CHCO ED today.

Id. L.L.P. did not have a fever, although he was taking acetaminophen/ibuprofen "for jaw, back, [and] ankle pain." Id. Review of systems noted decreased activity and appetite, increased fatigue, congestion, rhinorrhea, voice change, blurry vision, headaches, cough, wheezing, chest pain, constipation, nausea, vomiting, decreased urine output, bilateral costovertebral angle tenderness, arthralgias, back pain, myalgias, and numbness in right toes. Id. at 28-29.

Dr. Hoyum's physical examination noted L.L.P. appeared lethargic and distressed. Pet. Ex. 5 at 32. His mucous membranes were dry, tympanic membrane had no erythema or bulging, and he had no cervical lymphadenopathy. Id. at 32-33. L.L.P.'s upper extremity and grip strength was 5/5 and symmetric while his lower extremity strength was 4/5 bilaterally. Id. at 32. L.L.P. also exhibited lower extremity tenderness, shuffling wide gait, point tenderness to approximately L3/L4, bilateral cervical tenderness (but no cervical adenopathy). Id. at 32-33. He required assistance to go to bathroom and to move from laying to sitting. Id. at 32. Finger-nose-finger test showed mild dysmetria[7] bilaterally. Id. at 33. Dr. Hoyum was unable to elicit deep tendon reflexes. Id. Lastly, L.L.P. has no sensory defects on testing and no loss of bowel or bladder. Id. Under plan, diagnoses included an upper respiratory infection ("URI") (due to L.L.P.'s "worsening weak cough"), arthralgia, headache, ataxia (with GBS in the differential), leukocytosis, transaminitis (elevated liver enzymes), costochondritis,[8] and elevated blood pressure. Id. at 33-34.

[7] Dysmetria is "a condition in which there is improper estimation of distance in muscular acts, with disturbance of the power to control the range of muscular movement, often resulting in overreaching." Dysmetria, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15236 (last visited Aug. 12, 2025).

[8] Costochondritis is "inflammation of the cartilaginous junction between a rib or ribs and the sternum." Costochondritis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=11357 (last visited Aug. 12, 2025).

Senior Pediatrics Resident Dr. Kristin Kalita also examined L.L.P. and added an "Addendum/Escalation Note." Pet. Ex. 5 at 34-35. Her examination also revealed "weakly spitting oral secretions which seemed to be pooling in his mouth," jaw pain, inability to open mouth fully, and weak cough. Id. at 35. Neurologic motor examination documented "4/5 upper extremity strength, 4/5 strength at [bilateral] ankles, 3-4/5 strength at [bilateral] hips and knees, [and] sitting up from laying position require[d] significant help." Id. Dr. Kalita was "unable to obtain patellar and [A]chilles reflexes," but noted "1+ biceps reflex in [right upper extremity] (unable to obtain [left] because of PIV)." Id. L.L.P. also had "some dysmetria with [finger-nose-finger] on [right] side and unable to perform on [left] because of PIV." Id. Dr. Kalita's assessment documented that L.L.P. presented with "vague symptoms of weakness, vomiting, headache, back/ankle pain, now with rapidly progressing decline in strength with associated weak cough. Concern at this time is for [GBS] vs another acute demyelinating process." Id. at 36. Infectious causes were also being considered, and a Respiratory Pathogen Panel was ordered. Id. Dr. Kalita requested a re-evaluation from neurology, who "agreed that exam[ination] had progressed" and that "his pooling of secretions was concerning" and recommended lumbar puncture and magnetic resonance imaging ("MRI") of brain and entire spine. Id. L.L.P. was transferred to the pediatric intensive care unit ("PICU"). Id.

Neurologists Dr. Andra Dingman and Dr. Timothy M. Luebbert consulted at 8:30 p.m. on November 19, 2017. Pet. Ex. 5 at 42-43. Neurology's assessment noted L.L.P.'s

> elevated [WBC] count suggest[ed] an inflammatory process, while his elevated liver enzymes would be consistent with a viral process. . . . Reflexes in lower extremities [were] absent, though it [was] not clear whether or not this might be his baseline or represent a new peripheral nervous system problem such as [GBS]. There [was] significant effort-dependence to his neurologic examination, though when he gives good effort, his strength is 5/5 throughout (arguing against [GBS]). Systemic viral infection seems most likely. His apparent ataxia is more likely effort-dependent rather than dysmetria, though if persistent, could represent cerebellar or proprioceptive disease.

Id. at 42. Lumbar puncture was ordered, as was an MRI of the brain "if movement difficulties worsen." Id.

PICU attending, Dr. Ryan John Good, admitted L.L.P. to the PICU that evening. Pet. Ex. 5 at 91-95. Impression documented

> [one] week of fatigue, [headache], emesis, and back pain, also with joint pain, weak cough[,] and weak voice. [Three] days ago noted to have worsening weakness, now having progressive respiratory distress secondary to weakness. No known unifying etiology at this time. High concern for primary [central nervous system] disease. Has been afebrile, making infection less likely.

Id. at 93.

Respiratory Pathogen Panel collected the night of November 19, 2017 was positive for rhinovirus/enterovirus. Pet. Ex. 5 at 98-99. The panel did not detect adenovirus, human metapneumovirus, flu A virus and its 2009 H1 and H3 subtypes, flu B virus; parainfluenza viruses 1-4; respiratory syncytial virus ("RSV"), coronaviruses, *Mycoplasma pneumoniae*, *Chlamydophilia pneumoniae*, or Bordetella pertussis. Id.

A lumbar puncture was done the morning of November 20. Pet. Ex. 5 at 81. L.L.P.'s cerebrospinal fluid ("CSF") was normal. Id. at 99. CSF bacterial cultures did not detect any organisms. Id. at 100-01. A meningitis encephalitis panel, testing for numerous bacteria and viruses, was also conducted and no organisms were detected. Id. at 99-100. L.L.P. also tested negative for mononucleosis ("mono")[9] and his serum anti-ganglioside panel (Asialo-GM1, GM1, GM2, GD1a, GD1b, and GQ1b Antibodies, IgG/IgM) and Myasthenia Gravis antibody panel were negative. Id. at 627, 643-44.

In the morning of November 20, L.L.P. required emergency intubation due to worsening respiratory weakness. Pet. Ex. 5 at 82, 93. The PICU attending noted that morning that although "[w]ork up [was] in progress," L.L.P. "likely [had] GBS." Id. at 102. L.L.P. underwent an MRI of his brain on November 20 that was normal. Id. at 611-12. An MRI of his entire spine, also conducted on November 20, showed "[a]bnormal nerve root enhancement consistent with an acute polyradiculoneuropathy." Id.

That evening, on November 20, 2017, neurologist Dr. Craig A. Press provided a consultation. Pet. Ex. 5 at 1049. Dr. Press explained,

> [L.L.P.] presented with diffuse[] weakness and loss of reflexes that has progressed rapidly to involve his bulbar muscles. Although his CSF was normal, his history and exam[ination] (weakness and areflexia),[10] along with nerve root enhancement seen on MRI is most consistent with an immune-mediated polyneuropathy, likely Fisher Miller given bulbar involvement. CSF can be normal earlier in the course of the disease.

Id. L.L.P. was treated with plasmapheresis and IVIG. Id. at 22, 1049. L.L.P. was improving and was extubated to BiPAP[11] on November 27. Id. at 15.

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[9] "The Mono Screen test detects a non-specific (heterophile) antibody commonly induced after infection of older children and adults with Epstein Barr virus [("EBV")]." Pet. Ex. 5 at 627.

[10] Areflexia refers to the "absence of reflexes." Areflexia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4035 (last visited Aug. 12, 2025).

[11] BiPAP, or bilevel positive airway pressure, is a noninvasive ventilation machine that assists with breathing. BiPAP, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=137503 (last visited Aug. 12, 2025); BiPAP, Cleveland Clinic, https://my.clevelandclinic.org/health/treatments/24970-bipap (last visited Aug. 12, 2025).

L.L.P. had an EMG on December 5, 2017, which showed "electrophysiologic findings [] most suggestive of a subacute, moderately severe, predominantly demyelinating sensorimotor polyneuropathy." Pet. Ex. 5 at 88-89.

L.L.P. was discharged on December 15, 2017 with a diagnosis of GBS, Miller Fischer variant. Pet. Ex. 5 at 15, 22, 680. Discharge summary documented that L.L.P. had areflexia, progressive weakness requiring intubation, and MRI findings consistent with GBS. Id. at 15-22.

Following discharge, from 2018 to 2024, L.L.P. was seen various providers for his residual symptoms of GBS. Pet. Pre-Hearing Br., filed June 25, 2024, at 30-43 (ECF No. 122); Resp. Prehearing Br., filed July 16, 2024, at 10-18 (ECF No. 132).

2. Declaration and Hearing Testimony

a. T.P., Mother of L.L.P.

Ms. P. is the mother of L.L.P. and submitted a declaration[12] and testified at the entitlement hearing. Pet. Ex. 1 at ¶ 1; Tr. 3.

Prior to his flu vaccination in October 2017, L.L.P. was healthy and active. Tr. 7-9; Pet. Ex. 1 at ¶ 4. On October 12, 2017, L.L.P. received a flu vaccination. Tr. 9; Pet. Ex. 1 at ¶ 5. Ms. P. recalled L.L.P.'s "only real complaint right after[]" vaccination was of a sore shoulder. Tr. 10; see also Pet. Ex. 1 at ¶ 6.

Ms. P. testified that beginning on approximately October 19, "and for several weeks thereafter," until approximately November 11, L.L.P. "had random complaints of joint pain, back pain, shortness of breath, tight chest, [and other] weird stuff [that she] largely disregarded at the time." Tr. 18-19; see also Pet. Ex. 1 at ¶ 7. During the hearing, Ms. P. described photographs of L.L.P. during this period of time that showed he was "healthy" and doing his normal activities. Tr. 15-18 (citing Pet. Ex. 117 at 5-8, 11-13).

On November 11, 2017, L.L.P. participated in a food drive, distributing flyers, for Cub Scouts beginning at approximately 9:00 a.m. Tr. 19; Pet. Ex. 1 at ¶ 8. By 10:30 a.m., L.L.P. began complaining that he was tired, and he went home by 11:30 a.m. Tr. 20; Pet. Ex. 1 at ¶ 8. When Ms. P. arrived home that afternoon, L.L.P. was on the couch and cold. Tr. 20; Pet. Ex. 1 at ¶ 8. He awoke around 4:00 or 4:30 p.m. and vomited. Tr. 20; Pet. Ex. 1 at ¶ 8. L.L.P. was still eating and did not have a fever. Tr. 20.

The following day, November 12, L.L.P. did not vomit, but "laid low" and rested the entire day. Tr. 21; Pet. Ex. 1 at ¶ 9. He did not have a fever or a cough. Pet. Ex. 1 at ¶ 9. That night, around midnight, L.L.P. woke up and vomited. Tr. 21; Pet. Ex. 1 at ¶ 10.

[12] Although titled an affidavit, it is not notarized. The declaration was executed in April 2020. Pet. Ex. 1 at 28. Ms. P. relied upon text messages and handwritten notes in preparing her declaration. Tr. 10-11; see Pet. Exs. 119-20.

L.L.P. stayed home from school on November 13 per school policy. Tr. 21; Pet. Ex. 1 at ¶ 10. Ms. P. recalled that L.L.P. "didn't seem all that ill," but "he just laid around the house lethargically." Tr. 21; see also Pet. Ex. 1 at ¶ 10 (averring "he was moderately lethargic"). L.L.P. had no diarrhea, "no fever, no cough, no runny nose, no congestion, [and] nothing else seemed to be wrong," although "maybe his appetite started to wane a little bit." Tr. 22; see also Pet. Ex. 1 at ¶ 10. When he went to bed that night, everything was fine, but around midnight, he vomited again. Tr. 22; Pet. Ex. 1 at ¶¶ 10-11.

L.L.P. again stayed home from school on November 14. Tr. 22; Pet. Ex. 1 at ¶ 11. "Throughout the second day home from school, again, he was lethargic, stayed hydrated, had no fever and no cough, but still no appetite." Pet. Ex. 1 at ¶ 11. That night, at 7:30 p.m., L.L.P. attended his last candidacy class for his black belt. Tr. 22-23; Pet. Ex. 1 at ¶ 11. After the class, L.L.P. indicated he was tired, which Ms. P. thought was "not particular surprising" given the fact that "[h]e had eaten little for several days." Pet. Ex. 1 at ¶ 11.

On November 15, 2017. L.L.P. went to school. Tr. 23; Pet. Ex. 1 at ¶ 12. Ms. P. received a call from the school nurse around mid-day stating L.L.P. "did not feel well and was tired." Pet. Ex. 1 at ¶ 12; see also Tr. 23. The school nurse indicated he "did not look right," although he had no fever, no specific complaints, and had not vomited. Pet. Ex. 1 at ¶ 12; see also Tr. 23. Ms. P. took L.L.P. home for the remainder of the day. Tr. 23; Pet. Ex. 1 at ¶ 12. Because he seemed to feel better, L.L.P. attended a Cub Scout Pack meeting that evening. Tr. 24; Pet. Ex. 1 at ¶ 12. She testified that L.L.P. was engaged and running around outside with the other children. Tr. 24; see also Pet. Ex. 1 at ¶ 12. However, upon leaving, L.L.P. indicated he had trouble breathing and complained of chest, eye, jaw, and back pain as well as a headache and fatigue. Tr. 24; Pet. Ex. 1 at ¶ 12. Ms. P. noticed L.L.P. started coughing that night, and she described the cough as deep, hoarse, and non-productive. Tr. 25; Pet. Ex. 1 at ¶ 12. L.L.P. took Tylenol before bed. Tr. 25; Pet. Ex. 1 at ¶ 12. However, in the middle of the night, he awoke in pain and was unable to sleep. Tr. 26; Pet. Ex. 1 at ¶ 12. Ms. P. administered more pain relievers and gave him ice packs. Tr. 26; Pet. Ex. 1 at ¶ 12.

The following morning, November 16, Ms. P. called Dr. Presken's office, who was able to see him later that day. Tr. 27; Pet. Ex. 1 at ¶ 13. L.L.P. retrieved a cane from a closet and used it to ambulate that day due to difficulty walking and pain. Tr. 27-28; Pet. Ex. 1 at ¶ 13. L.L.P.'s cough was intermittent, he had no appetite, and he complained of a headache, back pain, and jaw pain. Pet. Ex. 1 at ¶ 13. His eyes were red and intermittently sensitive to light. Id. His fluid intake was also diminishing. Id. On arrival at Dr. Presken's office, L.L.P. vomited. Tr. 28; Pet. Ex. 1 at ¶ 13. Albuterol treatment was administered and after three treatments, L.L.P. appeared "perkier" and "[h]is color looked a little better." Tr. 28-29. L.L.P. was prescribed Zofran, albuterol treatments, and prednisone. Tr. 29; Pet. Ex. 1 at ¶ 13. L.L.P. did not receive a "definitive diagnosis," and Ms. P. stated the "treatment protocol seemed appropriate for the symptoms that had presented." Pet. Ex. 1 at ¶ 13.

On November 17, Ms. P. testified that L.L.P.'s "appetite was waning." Tr. 29. He was drinking, doing albuterol treatments, and taking prednisone. Tr. 29. He was not vomiting so she did not administer Zofran. Id. L.L.P. was unable to walk, stand, or get to the restroom without assistance, which Ms. P. thought was due to his lack of eating. Tr. 29-30; Pet. Ex. 1 at ¶ 14.

L.L.P. also fell getting into the bathtub. Tr. 31; Pet. Ex. 1 at ¶ 14. He had a headache, absent or intermittent cough, light sensitivity, joint pain, back pain, chest pain with difficulty breathing, and jaw pain that made opening and closing his mouth difficult. Tr. 30-31; Pet. Ex. 1 at ¶ 14. L.L.P. did not complain of nausea, abdominal pain, or diarrhea. Tr. 31-32. L.L.P.'s father, Mr. P. returned home from business that evening. Tr. 29; Pet. Ex. 1 at ¶ 14.

L.L.P.'s condition did not improve on November 18. Tr. 32-33; Pet. Ex. 1 at ¶ 14. He was having difficulty breathing, which Ms. P. would treat with albuterol. Tr. 32. He also had a headache, weakness, and pain in his eyes and jaw. Tr. 32-33. Overall, L.L.P.'s symptoms waxed and waned over this week, November 11 to November 18. Tr. 53-54.

The following morning, November 19, Petitioners took L.L.P. to CHCO ED after speaking with Dr. Presken. Tr. 33-35; Pet. Ex. 1 at ¶ 15. L.L.P. was admitted that day until December 15, when he was discharged. Pet. Ex. 1 at ¶¶ 16-32.

Ms. P. testified that prior to his hospitalization beginning November 19, L.L.P. did not have a fever, diarrhea, or gastrointestinal issues (other than intermittent vomiting). Tr. 37. He had issues getting to the bathroom, but did not have difficulty going to the bathroom. Tr. 49-50. Additionally, no one in the household was sick in the two months prior to his hospitalization. Tr. 50-51. Nor were there any sicknesses reported after his Cub Scouts camping trip in mid-October. Tr. 51.

On cross-examination, Ms. P. was questioned about a note from November 19, 2017 that indicated L.L.P. had a cough for two months. Tr. 40-41 (citing Pet. Ex. 4 at 80). Ms. P. did not recall this statement being made. Id. Ms. P. noted that if L.L.P. were to have had a cough for that long, she would have likely brought him in to see a doctor sooner. Tr. 52. To the best of her knowledge, L.L.P.'s cough began the night of Wednesday, November 15. Tr. 53.

Ms. P. also did not recall L.L.P. complaining of a headache the night of November 11. Tr. 43-44. She believed his headache did not start until later that week. Tr. 44.

b. R.P., Father of L.L.P.

Mr. P. is the father of L.L.P. and is husband of Ms. P. and he testified at the entitlement hearing. Tr. 56.

Prior to L.L.P.'s flu vaccination on October 12, 2017, L.L.P. was happy, well adjusted, and "had a lot of desire and energy to be outdoors" and "engage with his friends." Tr. 56. He was very active, in boy scouts, camping, Taekwondo, and swim team. Id. Mr. P. did not recall any coughs or other illnesses within the month prior to vaccination, or any issues the week after vaccination. Tr. 57.

From October 12, 2017 (date of vaccination) until November 17, Mr. P. did not notice any signs or symptoms of illness in L.L.P. Tr. 59. Prior to his business trip beginning November 13, he went camping with L.L.P. in mid-October and did not notice any signs or symptoms of illness. Tr. 60-61.

Mr. P. was out of town on business the week of November 13 for five days and returned home on November 17. Tr. 57. When he returned home, he noticed L.L.P. "did not look like himself at all;" L.L.P.'s pallor and muscle tone were "off," he "he had this propensity to want to use a cane," he was unable to go up and down stairs easily, he looked ill, and he had no appetite. Tr. 58. L.L.P.'s "continence was off;" he was "just [] kind of flat, not his normal self, not smiling." Id. The following day, November 18, L.L.P.'s condition was deteriorating to the point Mr. P had to carry L.L.P. up and down the stairs. Tr. 64.

C. Experts Reports[13]

The presentation of expert opinions begins with Respondent's expert since Respondent has the burden of proof with respect to proving L.L.P.'s GBS was due to an alternative cause, or factor unrelated to vaccination. See Joint Sub. at 2 (citing § 13(a)(1)(B)).

1. Respondent's Expert, Dr. Leslie Benson, M.D.[14]

a. Qualifications

Dr. Benson is a board-certified pediatric neurologist. Resp. Ex. A at 1. She received a B.S. in biological sciences from Colorado State University and an M.D. from University of Colorado Health Sciences Center. Resp. Ex. E at 1. Following her education, she completed two years of a pediatrics residency at Massachusetts General Hospital, three years of a pediatric neurology residency at Boston Children's Hospital, and a fellowship in neuro-inflammatory diseases at Boston Children's Hospital. Id. Since 2013, she has taught neurology at Harvard Medical School and has held hospital appointments at three hospitals in the Boston area. Id. at 2. Since 2013 she has also been the assistant director of the neuroimmunology program at Boston Children's Hospital. Tr. 71. Dr. Benson "treat[s] children with neuroinflammatory disease in both the inpatient and outpatient settings," including patients with GBS. Resp. Ex. A at 1; see also Tr. 72. She has authored or co-authored over 50 publications throughout her career. Resp. Ex. A at 1; Resp. Ex. E at 16-26.

b. Opinions

Dr. Benson concluded, "more likely than not[,] that an infection in November 2017 triggered [L.L.P.'s] GBS . . . unrelated to the seasonal inactivated [flu] vaccine . . . as supported by the human scientific literature showing a much stronger association between infections and GBS than [flu] vaccinations and GBS." Resp. Ex. A at 11. At the entitlement hearing, she opined, "more likely than not[,] [] an infectious illness contributed to the onset of [L.L.P.'s] GBS." Tr. 133; see also Tr. 75, 77, 94 (testifying that "there's a reasonable degree of medical certainty that there was an infection in [the] preceding days to [L.L.P.'s] weakness that

[13] For reasons previously explained, the undersigned does not discuss the experts' opinions related to Petitioner's causation-in-fact claim of GBS. See supra note 4.

[14] Dr. Benson submitted two expert reports and testified at the hearing. Resp. Exs. A, C; Tr. 3.

contributed to his case"); Resp. Ex. A at 6 ("[L.L.P.'s] GBS cannot be clearly attributed to his vaccination due to the long time interval between vaccination and onset of GBS . . . and [his] preceding URI that is much more likely the trigger, backed [b]y scientific data.").

i. Althen Prong One

Dr. Benson described GBS as "an autoimmune condition where the body's own immune system . . . starts to fight against the peripheral nervous system tissue," causing injury. Tr. 75. Classic cases of GBS begin with neuropathic pain or paresthesias in the lower extremities that progresses upwards. Id. "[C]ore features of GBS are weakness with some component of . . . neuropathic pain, maybe sensory symptoms," although, there can be variability in the presentation of symptoms. Tr. 95, 97, 102. The timing from symptom onset to nadir (the worst point of symptoms) is an average of eight days, with the majority of patients hitting nadir within two to four weeks, with rare cases reaching up to six weeks post-onset. Tr. 76, 98.

"[T]he majority of [GBS] cases are associated with an infectious illness, although not always specified. Many cases don't have an obvious preceding association[.] [] [R]are cases have been reported following vaccination . . . ." Tr. 75; see also Resp. Ex. A at 8; Resp. Ex. A, Tab 12 at 2 (noting that "with rare exceptions, associations between vaccines and GBS have been only temporal" with "little evidence to support a causal association with most vaccines").[15] "[I]nfections are much more common triggers than vaccines, evident in around [two-thirds] of cases." Resp. Ex. A at 8; see also Resp. Ex. A, Tab 11 at 1, 6, 6 tbl.2 (finding "the association between GBS and antecedent infection was strongly elevated" and "no evidence for an elevated GBS risk following" flu vaccination);[16] Resp. Ex. A, Tab 12 at 3; Resp. Ex. A, Tab 14 (classifying GBS as a "typical post-infectious disorder," yet acknowledging cases of GBS have been reported post-vaccination).[17]

In Greene et al., the authors found "no evidence for an elevated GBS risk" post-flu vaccination but found "the association between GBS and antecedent infection was strongly elevated." Resp. Ex. A, Tab 11 at 1. The authors found 18 patients who were vaccinated in the risk interval period prior to GBS onset; seven (39%) had symptoms of a respiratory infection documented in the medical records, three of which were medically-attended and diagnosed with "[a]cute [URIs] of unspecified site." Id. at 5, 6 tbl.2. However, no patients were categorized as having an "[u]nspecified viral infection" and flu vaccination within 42 days of GBS onset. Id. at 6 tbl.2.

When questioned about her theory during the hearing, specifically how an unspecified infectious illness can cause GBS, Dr. Benson relied upon molecular mimicry and

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[15] Penina Haber et al., Vaccines and Guillain-Barré Syndrome, 32 Drug Safety 309 (2009).

[16] Sharon K. Greene et al., Guillain-Barré Syndrome, Influenza Vaccination, and Antecedent Respiratory and Gastrointestinal Infections: A Case-Centered Analysis in the Vaccine Safety Datalink, 2009–2011, 8 PLoS ONE e67185 (2013).

[17] Hugh J. Willison et al., Guillain-Barré Syndrome, 388 Lancet 717 (2016).

epidemiological data, including the fact that infections are common triggers of GBS. Tr. 136-37. She explained "the association [between infection and GBS] is there and the data is there even if we don't have a perfect molecular pathway to connect every infectious agent with GBS." Tr. 137; see also Resp. Ex. A at 11. She did not further extrapolate on this data or theory.

Here, the only positive testing showed rhinovirus/enterovirus. Pet. Ex. 5 at 98-99. Dr. Benson opined that "[she] [was] not proposing that rhinovirus causes GBS" as "[t]here's not a strong association there." Tr. 93. While she suspected she might have been able to find a case report of GBS after rhinovirus if she had looked, she does not find case reports "meaningful." Tr. 121.

As to enterovirus, Dr. Benson noted "[s]ome studies have linked enteroviruses with GBS;" however, "the association is not as strong as" other infections and GBS. Resp. Ex. A at 9. She acknowledged there are case reports depicting an association between enterovirus and GBS. Tr. 93. But again, she testified case reports are not "meaningful." Tr. 121.

ii. Althen Prongs Two and Three

Dr. Benson summarized that following a flu vaccination on October 12, 2017, L.L.P. was diagnosed with GBS in November 2017, nine to 10 days after onset of vomiting, fatigue, intermittent headache, runny nose, and two days of cough with shortness of breath. Resp. Ex. A at 1-2. Dr. Benson agreed with the diagnosis of GBS, opining L.L.P.'s symptoms, treatment response, and recovery were consistent with the diagnosis of GBS with features of the Miller Fisher variant. Id. at 6.

Dr. Benson opined L.L.P.'s vaccination did not cause his GBS. Tr. 75. She opined, "more likely than not," "an infectious illness [] contributed to the development of [his] GBS." Tr. 77, 117, 133; see also Resp. Ex. A at 11 (opining "more likely than not [] an infection in November 2017 triggered [L.L.P.'s] GBS"); Resp. Ex. C at 2. She believed "L.L.P. had a nonspecific [infectious] illness that was not prov[en] to be due to a specific bug that preceded his GBS symptoms." Tr. 131-32. When asked on cross-examination whether she was able to place this "nonspecific infectious illness" to any particular system (e.g., respiratory, gastrointestinal, etc.), Dr. Benson stated L.L.P.'s illness was "more of a multisystem generalized illness." Tr. 132. She also opined it was "extremely unlikely" that L.L.P.'s GBS was due to multiple factors (i.e., vaccination and infection). Tr. 134; see also Resp. Ex. A at 11.

Dr. Benson acknowledged an infectious agent was not identified in L.L.P.'s case. Tr. 79, 117. However, she emphasized that that "doesn't mean he could not have had an infection." Tr. 79; see also Tr. 129 ("I do not agree that a positive test is required in order to make a diagnosis of an infection."). "If you have symptoms of an infection, then you very well may have an infection." Tr. 94.

She asserted that "most of the common infections associated with GBS weren't tested and were not ruled out." Tr. 93; see also Resp. Ex. A at 8-9. And even those that were tested "cannot rule out all possible viral infections." Resp. Ex. A at 8. "There are [too many] numerous viruses to be tested thoroughly in each case of viral symptoms, and thus the work up

for [L.L.P.] is typical but does not rule out a preceding infection." Id. (emphasis omitted).

Dr. Benson then discussed limitations of the tests conducted in L.L.P.'s case. First, L.L.P.'s testing for EBV/mono was "quite limited" and did not test for titers of EBV to rule out EBV infection. Tr. 78-79, 92-93. Because mono is a "fatigue predominant illness," she opined it is "still a very reasonable possibility." Tr. 79; see also Resp. Ex. A at 8.

She testified that *Mycoplasma pneumoniae* and *Campylobacter jejuni*, which are associated with GBS, were also not tested in L.L.P. Tr. 79, 92, 121-22. However, on cross-examination, she acknowledged that the nasal testing did test for *Mycoplasma pneumoniae* and it was negative. Tr. 123; see also Resp. Ex. A at 5 (documenting he tested negative for *Mycoplasma pneumoniae*). Thus, she agreed that "[i]n a reasonable manner," *Mycoplasma pneumoniae* infection was ruled out, or "off the table." Tr. 123-24. As for *Campylobacter jejuni*, she opined it is "possible" that L.L.P.'s symptoms the week leading up to hospitalization were due to *Campylobacter jejuni*. Tr. 139.

Although ganglioside antibody testing, which may indicate GBS, was negative, these antibody panels in Dr. Benson's experience are not "highly utilized or helpful in clinical management of [GBS]." Tr. 126-28. In her expert report, she noted L.L.P. had features of the Miller Fisher variant, which she described as a form of GBS "where anti-GQ1b antibodies are often, but not always detected." Resp. Ex. A at 6. L.L.P. was tested for anti-GQ1b antibodies in his anti-ganglioside panel and the results were negative. Id. at 5; Pet. Ex. 5 at 644.

L.L.P. had lumbar puncture and CSF testing on November 20, 2017, which was extensively tested ("pretty extensive infectious testing"), and all tests were normal, and no nervous system infection was found. Tr. 91-92. She explained that CSF testing does not show a preceding virus that may have triggered GBS, or whether there was an infection prior to the lumbar puncture; instead, it would only rule out a direct infection. Tr. 92. And for GBS, markers of infection in the CSF are not expected. Tr. 119.

Dr. Benson acknowledged that L.L.P.'s only positive test was for enterovirus[18] or rhinovirus via nasal swab testing. Tr. 93. She explained that this is a "nonspecific" test that only indicates that one of these viruses, both of which "are relatively common," is present. Tr. 93, 118. The test does not specify which virus is present, or if both are present. Id. She noted rhinorrhea (runny nose) can be associated with an infection, seasonal allergies, and "possib[ly]"

[18] L.L.P.'s CSF was negative for enterovirus meningitis, which Dr. Benson contended does not rule out "a systemic enteroviral infection." Tr. 118-19.

dysautonomia.[19] Tr. 104. However, again she did "not propos[e] that rhinovirus causes GBS." Tr. 93. Dr. Benson opined that a positive rhinovirus/enterovirus nasal swab does not explain the cause of L.L.P.'s GBS. Tr. 121.

Dr. Benson testified that her "best guess" for L.L.P.'s GBS onset was November 16 or November 17 when he began showing signs and symptoms that were "potentially consistent with GBS." Tr. 77-78, 108. On November 16, L.L.P. was in "a lot of pain . . . [and] that may have been related to GBS." Tr. 114. He also began having difficulty ambulating, using a cane for support, which "may be [the] initial manifestation[] of [GBS]." Tr. 108-10.

She opined that L.L.P.'s symptoms prior to November 16 were "nonspecific," "common" to a variety of infectious illnesses, and waxed and waned, all of which she asserted were "most suggestive of infection." Tr. 78; see also Resp. Ex. A at 6 (opining "the symptoms between [October 19 and November 15, 2017] cannot be attributed to GBS").

As to the symptoms beginning November 11, Dr. Benson opined these were symptoms of a "non-specific illness most attributable to infection" and predated L.L.P.'s "clearly GBS related symptoms of weakness and toe numbness." Resp. Ex. A at 6.

Overall, Dr. Benson opined "[L.L.P.'s] clinical presentation has many findings supportive of a concurrent infection." Resp. Ex. A at 8. For support, she discussed each of L.L.P.'s symptoms between vaccination and hospitalization and explained why she believed they were not symptoms of GBS and instead supported a finding that L.L.P. had a viral illness.

First, Dr. Benson opined it was "extremely—exceedingly unlikely" that the symptoms beginning approximately October 19 were the initial symptoms of GBS because "GBS does not involve vastly fluctuating nonspecific symptoms lasting 28 to 31 days before more classic symptoms." Tr. 84; see also Resp. Ex. A at 7; Resp. Ex. C at 1-2.

Dr. Benson agreed pain is common in approximation with weakness in GBS; however, she did not believe L.L.P.'s complaints of pain in October, as noted in Ms. P.'s affidavit, were

[19] Dysautonomia is the "malfunction of the autonomic nervous system," which is "the portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glandular epithelium" and is a component of the peripheral nervous system. Dysautonomia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15146 (last visited Aug. 12, 2025); Autonomic Nervous System, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111779 (last visited Aug. 12, 2025); Divisio Autonomica Systematis Nervosi Peripherici, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=71403 (last visited Aug. 12, 2025). "[A]utonomic nervous system dysfunction may be prominent during the course of [GBS]." Resp. Ex. C, Tab 4 at 2 (Francis J. DiMario & Carrie Edwards, Autonomic Dysfunction in Childhood Guillain-Barré Syndrome, 27 J. Child Neurology 581 (2012)).

related to GBS. Tr. 89; Resp. Ex. A at 8. She cited Yao et al.[20] who found less than 5% of patients had "pain more than two weeks before their weakness and clear GBS symptoms." Tr. 85, 90 (citing Resp. Ex. C, Tab 11). Thus, "to propose that [L.L.P.] had pain dating a month prior would put him into a group . . . of [approximately] [four] percent of patients studied that have pain in the setting of GBS," which is "very rare." Tr. 89. Dr. Benson testified that "L.L.P. had significant pain that started within days of when he got [] weak and was falling and ended up in the hospital," which was mid-November, not October, 2017. Tr. 85-89.

As to the chest pain and shortness of breath described by L.L.P.'s mother, Ms. P., in October, she opined these symptoms "related to a known condition," his "reactive airway disease history," which was "the most obvious" reason. Tr. 85-86. She acknowledged that "shortness of breath is not specific to one condition" and "[y]ou can get shortness of breath from all sorts of things," including reactive airway disease and GBS. Tr. 86. But if the chest pain in October was due to GBS, it would be due to weakness in the diaphragm muscles, which was "implausible" because L.L.P.'s respiratory function at hospital admission in November was good, signaling to Dr. Benson that "he did not have, in October, respiratory muscle weakness." Tr. 85-86. And his quick respiratory function deterioration once at the hospital was a "timeline [that] just does not make sense to [Dr. Benson] for [GBS]," as "four weeks of respiratory symptoms prior to focal weakness is not consistent with GBS evolution." Tr. 86; Resp. Ex. C at 2.

She added that "[s]hortness of breath and chest pain are not common signs and symptoms of dysautonomia," which she agreed that L.L.P. had, and which she acknowledged is associated with GBS. Tr. 87-88, 101; see Resp. Ex. A at 6-8. However, she did not believe L.L.P.'s dysautonomia preceded his weakness by weeks. Resp. Ex. A at 6-8.

Overall, Dr. Benson opined that any symptoms in October, even if associated with GBS generally, were not associated with L.L.P.'s GBS because these symptoms "are not typically preceding weakness by long stretches of time." Tr. 87-88; see also Resp. Ex. A at 6-8.

Next, Dr. Benson addressed L.L.P.'s symptoms that began the week of November 11. For fatigue, she opined that it was a "nonspecific" symptom that is "common" to a variety of infectious illnesses. Tr. 78. She agreed he showed signs of fatigue on November 11, but asserted "fatigue itself does not indicate weakness per se." Tr. 105. She would have expected focal weakness, not a generalized fatigue, with GBS. Id. She opined that fatigue is not one of the "predominant GBS onset symptoms" and is instead "associated with recovery and disease course." Tr. 115.

L.L.P. reported taste disturbances on November 11, which Dr. Benson opined was an "extremely unlikely" initial symptom of GBS for two reasons. Tr. 79-80. First, taste disturbances were not reported after November 11 and would have persisted if due to nerve damage. Tr. 80. And second, taste disturbances are "an extremely rare symptom of GBS." Tr. 80.

[20] Shaoli Yao et al., Pain During the Acute Phase of Guillain-Barré Syndrome, 97 Medicine 1 (2018).

On cross-examination, Dr. Benson opined some patients may experience loss of taste "associated with facial weakness." Tr. 95-96. However, when questioned about a patient reported in Combarros et al.,[21] who lost his sense of taste without experiencing weakness of his facial muscles, she agreed this patient's loss of taste was not associated with facial weakness. Tr. 96-97 (citing Pet. Ex. 59). Yet, she maintained the cranial nerves involved with taste and facial weakness are both implicated anatomically. Tr. 97.

In response to L.L.P.'s intermittent vomiting beginning November 11, Dr. Benson testified it was "possible" that the vomiting was due to GBS, but it was also a "nonspecific" symptom that was "most likely" due to an infection. Tr. 78, 86-88. She opined vomiting was "less likely" due to GBS "because it was associated with [] headaches and [] severe fatigue, which would be [an] uncommon . . . preceding symptom[]" of GBS. Tr. 87. She added that vomiting would be "very convincing for an infection" if it was associated with "florid diarrhea," "florid constipation[,] or sensations of being full." Id. However, L.L.P. did not have these symptoms. See Tr. 88; Resp. Ex. C at 3. She also opined it was "extremely unlikely" that the vomiting during this timeframe was consistent with early autonomic parasympathetic vagus nerve dysfunction (related to GBS). Tr. 87.

Dr. Benson opined L.L.P.'s "lack of diarrhea is not super specific." Tr. 88. According to Dr. Benson, "infectious illnesses [can have] a variety of symptoms" and "present themselves in different ways." Id. She testified that it would "not [be] impossible" to have vomiting without diarrhea and "it is very much plausible to have vomiting without diarrhea associated with an infection." Id. Further, she suggested that L.L.P.'s lack of constipation or diarrhea could be due to his lack of intake. Resp. Ex. C at 3.

Next, Dr. Benson acknowledged that joint pain and back pain are common early symptoms of GBS. Tr. 102; Resp. Ex. A at 8. She testified that a nine-year-old "might" describe GBS-related symptoms as jaw pain and full body aches. Tr. 102-04. Although she had not seen jaw pain associated with GBS, she testified that "it's conceivable that it's part of the neuropathic manifestations or a cranial neuropathy manifestation of [GBS] but certainly not the most common." Tr. 104. She also "suspect[ed] it's possible" that L.L.P.'s jaw pain that limited him from opening his mouth completely was indicative of GBS. Tr. 114. On redirect, she indicated that with the information provided from the records, there was no way to determine whether L.L.P.'s jaw pain on October 4, 2017, pre-vaccination, and L.L.P.'s jaw pain on November 15 was the same kind of pain. Tr. 141-42.

Dr. Benson testified that "[v]ision can be impacted by GBS in multiple different ways." Tr. 107. "Eye misalignment is a common manifestation of the Miller Fisher variant of [GBS], [and it] can be an early symptom" that would lead to double vision, not blurry vision. Tr. 103. She asserted that any complaints of blurred vision prior to hospitalization was not due to GBS, and instead L.L.P. needed to wear his glasses. Tr. 107; see Resp. Ex. C at 3 (categorizing L.L.P.'s complaints of blurry vision as "non-specific" and without objective evidence of its

[21] O. Combarros et al., Taste Loss as an Initial Symptom of Guillain-Barré Syndrome, 47 Neurology 1604 (1996).

cause prior to hospitalization). However, she clarified that it is "very possible" later in his GBS course he could have developed blurred vision. Tr. 107. "The autonomic component of dilated or abnormal pupillary responses or asymmetric pupils fits into that autonomic group of symptoms when they are part of GBS, as they can be, more part of the kind of acute weak patient phase of [GBS]." Tr. 103.

As to headaches, Dr. Benson noted headaches can present with blurred vision outside the context of GBS. Tr. 141; Resp. Ex. C at 3. Also, because headaches were associated with vomiting as well, she opined GBS was "less likely" the cause. Tr. 87.

L.L.P. did not have a fever the week of November 11 leading up to his hospitalization, and Dr. Benson explained that because he had been taking Tylenol and ibuprofen, it was "possible" the medication masked a fever. Tr. 93-94.

Moving forward to November 16, when L.L.P. saw Dr. Presken, Dr. Benson explained that L.L.P.'s symptom improvement with three nebulizer treatments[22] "suggest[ed] to [Dr. Benson] that [there was] bronchoconstriction[,] meaning that . . . the airways within the lungs [were] constricting, causing decreased air flow." Tr. 82. This process occurs with reactive airway disease, which "could be triggered by infection," as well as exercise, cold weather, and allergies, which were not pertinent to L.L.P.'s clinical picture. Id. Dr. Presken's examination documented lymphadenopathy (enlarged lymph nodes) in the neck, which Dr. Benson opined "is most often a reaction to an illness, often a virus." Tr. 83.

Despite Dr. Presken's physical examination not documenting sensory symptoms often seen in GBS, Dr. Benson testified L.L.P. was showing signs and symptoms "potentially consistent with GBS" on November 16. Tr. 83, 108. According to Ms. P., L.L.P. used a cane to ambulate on November 16 and Dr. Benson believed L.L.P.'s "use of a cane and difficulty walking may be [the] initial manifestation[] of [GBS]." Tr. 110. She maintained "that does not mean that there aren't ongoing viral symptoms or infectious systems that overlap with the onset of [GBS]" as "[t]here is no requirement that the infectious trigger be completely resolved before the onset of [GBS]." Id.

Overall, Dr. Benson agreed that L.L.P. had rare symptoms of GBS. Tr. 95, 97. She also agreed in rare cases a patient with GBS can reach nadir up to six weeks after onset. Tr. 98. However, she maintained L.L.P. showed signs and symptoms consistent with a "nonspecific infectious illness" that caused his GBS. Tr. 77-78, 131-33.

[22] L.L.P.'s hospitalization records documented a cough for two months. Tr. 112. Dr. Benson testified that could indicate mild asthma, which would also respond to albuterol, as well as allergies with post-nasal drip or intermittent infections. Tr. 112; see also Resp. Ex. C at 2.

2. Petitioners' Expert, Dr. Safwan Jaradeh, M.D.[23]

a. Qualifications

Dr. Jaradeh is a board-certified neurologist with specialty fellowship training in electrodiagnostic and neuromuscular medicine, autonomic medicine, and clinical neurophysiology. Pet. Ex. 50 at 1. He completed his medical education at the University of Damascus in Syria, residencies in neurology and internal medicine in France, a neurology residency at the University of Cincinnati, and neuromuscular fellowships at Mayo Clinic in Minnesota and at the University of Michigan. Pet. Ex. 121 at 1-2. Dr. Jaradeh has held various faculty appointments since 1983, and he has held hospital and clinical appointments in Wisconsin and California since 1989. Id. at 2-3. Since 2011, he has taught at Standford University and has been the Director of the Stanford Autonomic Disorders Program. Id. at 2, 4; Pet. Ex. 50 at 1. He has authored or co-authored over 150 publications throughout his career. Pet. Ex. 121 at 45-60.

He has an active clinical practice in autonomic disorders and neuromuscular disorders; "[he] [is] actively involved in patient care and evaluate[s] more than 900 patients per year. Over the past 30 years, [his] practice has cared for hundreds of adults and children with various forms of neuromuscular and autonomic disorders, including [GBS]." Pet. Ex. 50 at 1. He estimated that he has seen a dozen cases of GBS with associated autonomic issues in his career. Tr. 240-41. He also estimated that one-third of his pediatric patients are under ten. Tr. 227.

b. Opinions

i. Althen Prong One

Dr. Jaradeh focused his opinions on the disputed issues of whether the flu vaccine or an unknown infectious illness caused L.L.P.'s GBS as well as the onset of L.L.P.'s GBS symptoms. Therefore, he did not specifically address Althen prong one as it relates to Respondent's alternative causation argument.

In his first expert report, however, he stated "[r]hinovirus infections cause common cold[s] and are among the least likely culprits to produce [GBS]." Pet. Ex. 50 at 4.

ii. Althen Prongs Two and Three

Dr. Jaradeh opined, "to a reasonable degree of medical certainty," L.L.P. developed GBS due to his flu vaccination on October 12, 2017. Pet. Ex. 50 at 1; see also Tr. 202-03.

Dr. Jaradeh explained L.L.P.'s presentation, albeit atypical, was consistent with GBS. Tr. 158-61; Pet. Ex. 50 at 1; Pet. Ex. 113 at 1. Typically, GBS presents as numbness, tingling, problems with balance, weakness, and loss of reflexes, although the presentation of symptoms can be varied. Tr. 159. L.L.P.'s GBS, according to Dr. Jaradeh, presented with "pain and

[23] Dr. Jaradeh submitted two expert reports and testified at the hearing. Pet. Exs. 50, 113; Tr. 3.

digestive symptoms indicative of earlier involvement of the small nerve and autonomic nerve fibers,[24] before the process generalized to other large nerve fiber functions, causing weakness, paresthesia[,] and loss of reflexes." Pet. Ex. 50 at 1; see also Tr. 161. "And because [this presentation] is not well recognized and described in the literature, it is considered by many as atypical." Tr. 161. In his experience and practice, L.L.P.'s symptom evolution was consistent with GBS. Tr. 203; see also Pet. Ex. 113 at 2 (explaining that "in his experience as an autonomic specialist who evaluates and perform[s] autonomic testing on adults and children presenting with various postinfectious autonomic symptoms, such atypical presentations are rather common, even when they do not evolve into progressive weakness and paralysis"); Tr. 207-13 (describing literature documenting autonomic dysfunction manifestations as the presenting symptoms in GBS) (citing Pet. Ex. 52;[25] Pet. Ex. 53;[26] Pet. Ex. 54).[27]

In response to Dr. Benson's argument that this atypical presentation of GBS is seen in less than 5% of patients and therefore would be rare for L.L.P., Dr. Jaradeh opined the percentage of these patients "is significantly higher because many clinical symptoms and signs of GBS are over-shadowed by the patient's weakness, reducing the reported frequency of other, non-sensory motor symptoms." Pet. Ex. 113 at 2. He cited Demuth et al.,[28] which documented atypical presentations with arthralgias and myalgias preceding motor weakness by several days to weeks, with paralysis presenting up to 60 days after the onset of atypical nociceptive pain symptoms. Pet. Ex. 113 at 2 (citing Pet. Ex. 51); Tr. 206-07. On cross-examination, he acknowledged none of the patients in Demuth et al. had recent vaccinations. Tr. 228 (citing Pet. Ex. 51 at 2). Dr. Jaradeh also addressed Yao et al., cited by Respondent, and argued that the study did not quantify the pattern of pain in patients in relation to their weakness. Tr. 204 (citing Resp. Ex. C, Tab 11 at 5).

To support his opinion that L.L.P.'s GBS presented with autonomic symptoms such as myalgias, arthralgia, and shortness of breath and was triggered by the flu vaccine, Dr. Jaradeh detailed L.L.P.'s clinical course, pre-vaccination to hospitalization.

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[24] Autonomic nerve fibers are "peripheral nerve fibers that innervate smooth muscle, cardiac muscle, and glandular epithelium, or synapse in autonomic ganglia." Autonomic Nerve Fibers, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92553 (last visited Aug. 12, 2025).

[25] Tia Chakraborty et al., Dysautonomia in Guillain-Barré Syndrome: Prevalence, Clinical Spectrum, and Outcomes, 32 Neurocritical Care 113 (2020). This is also cited as Pet. Ex. 26.

[26] Alicia S. Ferraro-Herrera et al., Autonomic Dysfunction as the Presenting Symptom in Guillain-Barré Syndrome, 78 Archives Physical Med & Rehabilitation 777 (1997). This is also cited as Pet. Ex. 27.

[27] Douglas W. Zochodne, Autonomic Involvement in Guillain-Barré Syndrome: A Review, 17 Muscle & Nerve 1145 (1994). This is also cited as Pet. Ex. 28.

[28] Stanislas Demuth et al., Rheumatic Presentations of Guillain-Barré Syndrome as a Diagnostic Challenge: A Case Series, 88 Joint Bone Spine 105144 (2021).

As to L.L.P.'s pre-vaccination medical history, Dr. Jaradeh opined there was nothing in L.L.P.'s records prior to the flu vaccination on October 12, 2017 to indicate a more likely cause for L.L.P.'s GBS. Tr. 161. Nor did Dr. Jaradeh find any evidence in the record of an infectious illness at the time of vaccination. Tr. 162.

Seven days following L.L.P.'s flu vaccination at issue, on approximately October 19, 2017, L.L.P. developed myalgias, arthralgia, and shortness of breath, and Dr. Jaradeh opined this was the onset of L.L.P.'s GBS. Pet. Ex. 50 at 1, 4; Tr. 162-63. These symptoms then progressed over the following weeks, "consistent with nociceptive prodromal onset of his GBS." Pet. Ex. 50 at 1 (citing Pet. Ex. 51).

Dr. Jaradeh next addressed L.L.P.'s set of symptoms that began November 11, 2017. Tr. 164. He opined L.L.P.'s fatigue that week was an early symptom of his GBS. Tr. 164-65. He agreed fatigue is not thought to be a typical symptom of GBS, however, he asserted it is in his experience. Tr. 164. He explained that fatigue can fluctuate, with individuals having more energy later in the day, and for L.L.P., he did have fluctuating fatigue as evidenced by his ability to complete certain tasks, such as his black belt candidacy class, later in the day. Tr. 167-70; Pet. Ex. 50 at 2. However, if this fatigue was neuromuscular weakness (e.g., GBS), then Dr. Jaradeh expects L.L.P. would not have been able to complete physical activities such as pushups, lunges, and running, although adrenaline could have assisted in the completion of such exercises. Tr. 239, 241-42.

Dr. Jaradeh also opined L.L.P.'s intermittent vomiting, along with his constipation, beginning November 11 was an early symptom of his GBS. Tr. 165-67; see also Pet. Ex. 50 at 2 (noting the intermittent vomiting was "consistent with an early autonomic parasympathetic vagus nerve dysfunction"). "While the presence of gastrointestinal symptoms such as constipation and vomiting suggest a hypothetical enteroviral illness, [Dr. Jaradeh asserted] it [was] far more likely in L.L.P.'s case that they reflect his autonomic and small fiber nerve impairment rather than a hypothetical new enteroviral illness." Pet. Ex. 50 at 4. Dr. Jaradeh noted "there [was] no evidence at any time that L.L.P. had a new enteroviral illness" and it would be "unusual" to have an enteroviral illness without a fever, which L.L.P. never had. Id. He acknowledged on cross-examination that limited food intake can contribute to constipation or lack of voiding. Tr. 231-32.

L.L.P. then developed blurred vision and jaw pain, which suggested to Dr. Jaradeh that these symptoms were also early signs of GBS. Tr. 170-71; Pet. Ex. 50 at 2. He opined L.L.P.'s blurred vision "was probably a reflection of a slow reaction of his pupils" and "pupil innervation is entirely autonomic." Tr. 170. If the parasympathetic and sympathetic systems are "unbalanced," the pupils do no constrict as they should, leading to blurred vision. Tr. 170-71. On cross-examination, he acknowledged L.L.P.'s pupil examination on hospital admission was normal. Tr. 237. However, he explained that "the only way really to test the pupils accurately is to make sure you are in a dark room," which is not likely in the ED, and thus, is "frequently missed." Tr. 242-45.

And the jaw pain suggested to Dr. Jaradeh that L.L.P. had "partial trigeminal sensory small fiber nerve involvement." Pet. Ex. 50 at 2; see also Tr. 180-81. He also opined that L.L.P.'s jaw pain pre-vaccination differed from his post-vaccination jaw pain, which was "of an entirely different nature." Tr. 247. The pre-vaccination jaw pain was localized to the right side and associated with loose teeth, while the post-vaccination jaw pain was bilateral, not associated with dental pathology, and presented as "significant tightness in the jaw muscles." Tr. 247-48. Thus, the post-vaccination jaw pain "was more of a nerve or neuropathic pattern rather than due to a dental etiology," and the pre-vaccination jaw pain was unrelated to L.L.P.'s GBS and post-vaccination jaw pain. Tr. 248-50.

Dr. Jaradeh cited a case report described by Sklirou et al.,[29] noting its similarities to L.L.P.'s presentation. Tr. 214 (citing Pet. Ex. 55). Sklirou et al. described a six-and-one-half year old girl who had bilateral temporomandibular joint pain for three days, prior to the development of generalized arthralgias, inability to walk, and absent deep tendon reflexes. Pet. Ex. 55 at 1. She had joint pain in upper and lower extremities and could hardly open her mouth. Id. Examination of the oral cavity was unremarkable. Id. She was diagnosed with GBS. Id. at 2. The authors noted she had a diarrheal illness three weeks prior on onset and an enteroviral infection was documented serologically. Id. at 3. Before their patient developed "typical" GBS symptoms, her initial symptoms of jaw pain, arthralgias, and myalgias were attributed to a viral infection. Id. at 3. They concluded this was a case of an unusual presentation of GBS. Id.

As to L.L.P.'s headaches, Dr. Jaradeh opined "headaches are common in children" and "[t]hey may or may not occur with vomiting." Tr. 173.

L.L.P. also reported taste disturbances, and taking everything together, Dr. Jaradeh opined that L.L.P.'s taste impairment "suggested that the taste modality with [L.L.P.] was just not working [] very well, and that is mediated strictly by the autonomic fibers that carry information about the taste back . . . to the brain and allow its interpretation." Tr. 174. To Dr. Jaradeh, "there was no question . . . that the taste impairment . . . indicated autonomic problems[,] . . . coincid[ing] with everything else that was affecting [L.L.P.'s] small nerve fiber function." Tr. 175. And L.L.P. subsequently developed issues with his facial nerves, which also related to his GBS. Tr. 176-77.

He explained that issues with taste can occur simultaneously with or precede facial nerve issues. Tr. 174-75. He agreed with Dr. Benson that taste impairment usually coincides with facial weakness, but he asserted "there are always exceptions." Tr. 175. For support, he again cited Combarros et al., who described a patient whose initial symptom of GBS was taste impairment without any facial weakness. Tr. 175-76 (citing Pet. Ex. 59 at 1-2). However, the reported patient had tongue numbness associated with his taste disturbance, which was not documented for L.L.P. Tr. 228 (citing Pet. Ex. 59 at 1-2). The authors wrote, "Facial numbness is usually present in a distal distribution corresponding to the longest trigeminal nerve territories and therefore involving the lips, tip of the nose, and tip of the tongue;" however, their patient's "sensation was affected in the distribution of both lingual nerves." Pet Ex. 59 at 2. Dr. Benson

[29] Evgenia Sklirou et al., A Girl with Bilateral Temporomandibular Joint Pain, Generalized Arthralgias, and Inability to Walk, 49 Clinical Pediatrics 400 (2010).

also cited Nishijima et al.[30] for support. Tr. 176 (citing Pet. Ex. 60). In Nishijima et al., the authors reported six of 10 GBS patients had taste impairment. Pet. Ex. 60 at 1. The authors concluded "[f]acial palsy and facial hypesthesia appeared not to be related to taste impairment" and "taste may be more frequently involved in patients with GBS than expected." Id.

Dr. Jaradeh opined that L.L.P.'s above-mentioned symptoms and presentation up to November 15, 2017 did not indicate a viral infection or illness and instead "[were] more likely part of [L.L.P.'s] autonomic aura." Tr. 177-78. L.L.P. did not have a fever or other significant systemic findings, which would be "pretty odd" if L.L.P. had an infection. Tr. 178.

Dr. Jaradeh next addressed L.L.P.'s rhinorrhea (runny nose) that developed on November 15. Dr. Jaradeh opined rhinorrhea can be a symptom of various ailments, including a virus, allergies, and autonomic symptoms in the absence of an infection. Tr. 178-80, 201; Pet. Ex. 50 at 4. Yet, rhinovirus infections "are among the least likely culprits to produce [GBS]." Pet. Ex. 50 at 4.

Dr. Jaradeh acknowledged that "[L.L.P.'s] congestion and rhinorrhea might suggest another intercurrent viral illness, [but he asserted] they can also reflect early autonomic cranial dysfunction." Pet. Ex. 50 at 4. L.L.P. had a runny nose as well as other cranial autonomic symptoms, including blurred vision secondary to pupillary impairment, impaired taste, hoarse voice, and jaw pain, which Dr. Jaradeh opined were indicative of "autonomic vasomotor rhinitis, rather than [] of an independent, new viral infection." Id.

Moving forward to November 16, the date L.L.P. presented to Dr. Presken, Dr. Jaradeh detailed and analyzed records from this visit. Tr. 181; Pet. Ex. 50 at 2; Pet. Ex. 113 at 1. First, L.L.P. used a cane that day, which Dr. Jaradeh opined was an indication of "muscle weakness or imbalance[,] or both." Tr. 181.

Dr. Presken documented L.L.P.'s complaints of chest pain. Tr. 182. Dr. Jaradeh opined chest pain could be due to a flare up of his airway disease or a manifestation of muscle pain (myalgia), which is seen in patients with neuropathic diseases. Tr. 182-83.

Regarding L.L.P.'s complaint of pain while sitting up straight, Dr. Jaradeh opined this could be due to pain in back (paraspinal) muscles; if there is inflammation in the nerve roots going into these muscles, pain can occur with change of position. Tr. 188-89. Dr. Jaradeh acknowledged the records were not clear as to where the pain was located. Tr. 189. He explained that if the pain "in the upper body, particularly behind the neck, the upper shoulder down to the scapula is common in patients with autonomic disorders" due to the drop in blood pressure when sitting up. Tr. 189. If the pain was in his back, Dr. Jaradeh opined he "probably" had irritation to the nerve roots that innervate his muscle; however, if the pain was specifically only with sitting up, then Dr. Jaradeh opined it would be due to a drop in blood pressure. Tr. 189.

[30] Haruo Nishijima et al., Taste Impairment in Guillain-Barré Syndrome: More Frequent Than Thought?, 16 J. Peripheral Nervous Sys. 270 (2011).

Dr. Presken's examination indicated L.L.P.'s eyes were injected (red), which Dr. Jaradeh noted could be due to various reasons. Tr. 183. L.L.P. was also documented to have cervical lymphadenopathy on examination, which Dr. Jaradeh opined was "probably [why] both Dr. Presken as well as Dr. Benson felt that this was [] solid evidence that there was some type of an infection." Id. Dr. Jaradeh opined, however, that "there was no quantitation of the lymphadenopathy," and at admission a few days later, L.L.P.'s lymph nodes were not enlarged. Tr. 183-84; see Pet. Ex. 5 at 5, 33. He testified that he "[could not] say that lymph node enlargement is [] autonomic." Tr. 184.

Dr. Presken also noted normal bowel sounds with auscultation of the abdomen, which Dr. Jaradeh opined "does not rule out at all an autonomic issue." Tr. 190. He asserted this examination was a "snapshot in time," and they "[did] not know how his bowel sounds were when he was vomiting[,] [] shortly before he vomited[,] or shortly afterwards." Tr. 190; see also Tr. 233.

Examination of L.L.P.'s lungs on November 16 documented findings that were not consistent with an active infection or inflammation according to Dr. Jaradeh. Tr. 184-86 (noting no rales, no rhonchi, and negative percussion). L.L.P. did have decreased breath sounds, which Dr. Jaradeh opined could have been due to either issues air moving freely or issues with lung tissue expansion. Tr. 185-86. "[I]f there was a slight lack of the elasticity of the airway, the air will not flow in and out as smoothly as it should, and that will lead to secondary decrease in the breath sounds." Tr. 186. L.L.P.'s decreased breath sounds coupled with his lack of wheezing, according to Dr. Jaradeh, "argue[d] against acute asthma exacerbation, and in retrospect suggest[ed] early restrictive respiratory hypofunction rather than obstructive airway exacerbation." Pet. Ex. 50 at 2. Dr. Jaradeh opined that L.L.P.'s lack of signs of infection on lung examination coupled with the lack of nasal discharge made an URI "much less likely." Tr. 187.

L.L.P.'s improvement after three nebulizer treatments, which dilated the bronchial tree, could be due to "a couple of possibilities" according to Dr. Jaradeh. Tr. 187. First, L.L.P. had a history of asthma or bronchial active airway and "it [was] possible that he had a flare-up" that was "not necessarily . . . infectious" and could be autonomic. Id. Dr. Jaradeh believed that despite L.L.P.'s wheezing on auscultation, "it is very likely that [Dr. Presken] considered a flare up of [L.L.P.'s] pre-existing asthma as etiology, because she also discharged him with a short course of oral prednisone," which can treat an asthma flare-up. Pet. Ex. 113 at 1; see also Tr. 190. Various factors including exercise, cold weather, and environmental allergies can cause respiratory exacerbations in asthmatics like that seen in L.L.P. Pet. Ex. 113 at 1. He opined "the therapeutic response to inhaler therapy is most consistent with a flare up of his bronchoreactive airway disease, even in the absence of wheezing." Id.

Second, L.L.P. could have felt better because the medication stimulates the adrenaline system and indirectly improves muscle function. Tr. 188. And third, "[i]t could be just he was breathing a little better[,] . . .oxygenating better," which "[made] him feel better without necessarily curing the problem." Id. Dr. Jaradeh agreed that at admission, negative inspiratory force test was normal. Tr. 231.

On November 17, L.L.P. was exhibiting weakness and incoordination, further symptoms of his GBS. Tr. 192-93; Pet. Ex. 50 at 3. Additionally, other symptoms he was experiencing progressively worsened. Tr. 193.

Moving forward to admission, L.L.P. reported ankle pain, which Dr. Jaradeh opined "could be . . . a joint-related issue because of the muscle overexertion" or could be due to his GBS. Tr. 193-94. And the jaw pain was evidence of nerve irritation. Tr. 194. L.L.P. also had difficulty swallowing (oropharyngeal dysphagia), cough, and hoarseness of his voice, which suggested to Dr. Jaradeh that L.L.P. had laryngeal nerve impairment due to GBS. Pet. Ex. 50 at 5; Tr. 101-02, 249.

Dr. Jaradeh next addressed testing completed during L.L.P.'s hospitalization. Dr. Jaradeh opined L.L.P.'s elevated WBC count, with a shift in neutrophils, was "transient," subsiding within two days and presenting with no other systemic signs, making infection less likely. Tr. 200; see also Pet. Ex. 50 at 5. He explained that in GBS, "it's not unusual to find transient increase in the [WBCs] with[] [] [a] shift to neutrophils" due to "activity in the immune system." Tr. 200; see also Pet. Ex. 50 at 5. He cited studies that examined both WBC and neutrophil counts and found total neutrophil ratios and counts significantly higher in patients with GBS compared to healthy controls. Pet. Ex. 50 at 5 (citing Pet. Ex. 67;[31] Pet. Ex. 68).[32] And this finding, he asserted, corresponds with autonomic symptomatology and rapid progression. Tr. 200. Additionally, prednisone, prescribed by Dr. Presken, can lead to a transient elevation of WBCs and neutrophils. Tr. 201; Pet. Ex. 50 at 5. Lastly, he maintained L.L.P.'s rapid recovery of WBCs "would be unexpected in a new hypothetical enteroviral infection leading to severe GBS." Pet. Ex. 50 at 5. Thus, for these reasons, he concluded L.L.P.'s elevation of WBCs was not a sign of an infectious illness. Tr. 201; Pet. Ex. 50 at 5.

As to L.L.P.'s negative antibody panels, Dr. Jaradeh opined such testing is "helpful only if positive. If negative, they do not rule out subtypes of GBS." Pet. Ex. 50 at 5. He also agreed that L.L.P.'s negative mono test cannot rule out EBV. Pet. Ex. 113 at 1. However, there was no testing for EBV serology. Id.

Although some of L.L.P.'s treating physicians, including Dr. Presken, suspected a viral illness, Dr. Jaradeh opined an infection with an unknown, hypothetical virus was "unlikely" given L.L.P.'s presentation and lack of evidence of infection on testing. Pet. Ex. 113 at 1. "[E]ven if we assume that L.L.P. had an independent rhinoviral/enteroviral illness four weeks after his vaccination, the onset of his [GBS] within such a short period after this presumed additional infection is less likely than for a post-vaccin[]al case that preceded the neurologic symptoms within [three] weeks of his vaccination." Pet. Ex. 50 at 4.

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[31] Kaixi Ren et al., Association Between Serum Low-Density Neutrophils and Acute-Onset and Recurrent Guillain-Barré Syndrome, 12 Brain and Behavior 1 (2022).

[32] Yuanyuan Huang et al., The Clinical Significance of Neutrophil-to-Lymphocyte Ratio and Monocyte-to-Lymphocyte Ratio in Guillain-Barré Syndrome, 128 Int'l J. Neurosci. 729 (2018).

Overall, Dr. Jaradeh opined that even though L.L.P.'s presentation was atypical, that did not make it any less likely that the flu vaccine triggered GBS. Tr. 216. Dr. Jaradeh "couldn't find any other trigger but the vaccine" and opined to "a reasonable degree of medical certainty" the flu vaccination caused L.L.P.'s GBS. Tr. 203. He simplified, "[t]he vaccination etiology [was] probable, [while] the presumed viral etiology [was] possible." Pet. Ex. 113 at 2.

3. Petitioners' Expert, Dr. Lawrence Steinman, M.D.[33]

a. Qualifications

Dr. Steinman is board certified in neurology and has practiced neurology at Stanford University for over 40 years. Pet. Ex. 21 at 2; Pet. Ex. 101 at 1-2. He received his B.A. from Dartmouth College in 1968 and his M.D. from Harvard University in 1973. Pet. Ex. 101 at 1. Thereafter, he completed a surgery internship, pediatrics residency, and pediatric and adult neurology residency at Stanford University Hospital, as well as three fellowships, including one in clinical immunology. Id. Dr. Steinman is currently a Professor at Stanford University. Id. Dr. Steinman "is actively involved in patient care" and "ha[s] cared for hundreds of adults and children with various forms of neuroinflammatory diseases including optic neuritis, [GBS], chronic inflammatory encephalomyelitis (CIDP), transverse myelitis, inflammatory neuropathy, acute disseminated encephalomyelitis (ADEM), neuromyelitis optica (NMO) [,] and multiple sclerosis (MS)." Pet. Ex. 21 at 2. He has authored or co-authored over 600 publications. Pet. Ex. 101 at 5-51. One of Dr. Steinman's specialties is in the area of MS, and he has received a Charcot Prize for Lifetime Achievement due to his research in MS. Pet. Ex. 21 at 2. In 2015, he was elected to the National Academy of Sciences. Id. at 3.

b. Opinions

Dr. Steinman concluded "by a preponderance of the evidence, . . . the [flu] vaccine on October 12, 2017 triggered [L.L.P.'s] GBS." Pet. Ex. 21 at 25; Pet. Ex. 114 at 2-3; see also Tr. 271.

Dr. Steinman did not dispute that certain infections can cause GBS via molecular mimicry. Tr. 261-62 (citing Pet. Ex. 115).[34] Nor did he dispute that a vast majority of GBS patients have an antecedent infection. Tr. 280; see also Pet. Ex. 21 at 9 ("Usually [GBS] occurs a few days or weeks after the patient has had symptoms of a respiratory or gastrointestinal viral infection."). However, he "opposed" finding L.L.P.'s GBS was caused by "unknown infection," explaining that "[he] cannot accept an unknown infection as being more important than a known vaccine." Tr. 262, 271, 275-76. And therefore, he opined the flu vaccine triggered L.L.P.'s GBS by a preponderance of the evidence. Tr. 271.

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[33] Dr. Steinman submitted two expert reports and testified at the hearing. Pet. Exs. 21, 114; Tr. 224. Dr. Steinman also wrote a letter regarding L.L.P.'s long-term care that is not discussed herein as it does not pertain to the issue of entitlement. See Pet. Ex. 95.

[34] Hans-Peter Hartung, Infections and the Guillain-Barré Syndrome, 66 J. Neurology Neurosurgery & Psychiatry 277 (1999).

He cited Hartung, who wrote, "[m]any diverse infectious agents have been incriminated as triggers of [GBS] . . . . Based on [the] evidence, infections with the gram negative enteropathogen *Campylobacter jejuni*, cytomegalovirus (CMV), [EBV], and *Mycoplasma pneumoniae* are precipitants of GBS whereas other infections occur no more often in [GBS] than in controls." Pet. Ex. 115 at 1. Here, L.L.P. was not found to have any of these infections. Tr. 261-63, 283.

On cross-examination, he acknowledged that these infections, including *Campylobacter jejuni*, were not definitively ruled out in L.L.P.'s case because they were either not tested or complete testing was not done (i.e., lack of PCR testing or serology testing for EBV). Tr. 280-83, 302-03. He noted, however, that L.L.P. did not have diarrhea or elevated anti-ganglioside antibodies, which are common in most patients with *Campylobacter jejuni*. Tr. 296, 299-300. Nor did L.L.P. have findings consistent with CMV. Tr. 296-97.

L.L.P. was tested for a number of viruses and bacteria, and the only positive test result was that for rhinovirus/enterovirus via a nasal swab. Tr. 265-67; Pet. Ex. 21 at 10. And Dr. Steinman agreed with Dr. Benson that rhinovirus/enterovirus infections are not associated with GBS. Tr. 271, 304; Pet. Ex. 21 at 10. Dr. Steinman concluded "there was no specific diagnosis of an infectious disease" or "infectious cause that Respondent can say was a definitive alternate cause." Tr. 266, 270.

Next, Dr. Steinman discussed additional reasons why he opined L.L.P. did not have an infection that caused his GBS. He agreed L.L.P.'s use of acetaminophen/Tylenol could have masked his fever, but because of L.L.P.'s lack of fever and lack of positive testing of infection, Dr Steinman opined "[he] [did] not think there was an explanation that one could give with specificity and granularity that it was some microbial infection." Tr. 271-72.

With regard to the bloodwork taken on November 19, the date of admission, Dr. Steinman noted L.L.P.'s blood results did show signs of inflammation. Tr. 273. But he asserted that such results do not mean the inflammation was due to an infection. Id. He believed L.L.P. showed "sterile inflammation," which is when "the body [] react[s] to its own tissues." Tr. 273-74.

As to onset, Dr. Steinman wrote in his first expert report that "[t]he first symptoms of [GBS] include varying degrees of weakness or tingling sensations in the legs." Pet. Ex. 21 at 8. He also cited studies that found autonomic symptoms, including vomiting, may be the presenting symptoms of GBS. Id. at 8-9, 24 (citing Pet. Ex. 25 at 2 (reporting a case of GBS that presented with nausea and vomiting);[35] Pet. Ex. 52; Pet. Ex. 53 at 1 (reporting a case of GBS with autonomic dysfunction (chest pain and hypertension) as the presenting symptom); Pet. Ex. 54).

Given these studies, he opined onset began seven days after vaccination, on October 19, with L.L.P. having autonomic symptoms, including "multiple and iterative complaints of

[35] Robert Spitzer, Guillain Barre Syndrome Presenting as Nausea and Vomiting, 100 Am. J. Gastroenterology S263 (2005). The full article was not filed.

shortness of breath, pain in his chest, and random instances of severe joint (ankles, knees, elbows) and back pain severe enough that he would not want to go to his Taekwondo classes or be active." Pet. Ex. 21 at 10 (quoting Pet. Ex. 1 at ¶ 7).

During the hearing, Dr. Steinman clarified that if onset occurred on November 16, his opinions would not change. Tr. 303-04. He maintained that the flu vaccine triggered L.L.P.'s GBS. Id. And that although "[a]n infection could cause [L.L.P.'s GBS]," there was no "proof of what infection." Tr. 305.

IV. LEGAL FRAMEWORK AND 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).

Petitioners' 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).

To receive compensation through the Program, Petitioners must prove either (1) that L.L.P. suffered a "Table injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that L.L.P. suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). There is a statutorily prescribed presumption of causation for a Table injury. § 14(a).

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). "[F]actors unrelated to the administration of the vaccine" do not include "any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness, or condition." § 13(a)(2)(A). The Vaccine Act further provides that "factors unrelated to the administration of the vaccine" are those "which are shown to have been the agent . . . principally responsible for causing the petitioner's illness, disability, injury, condition or death." § 13(a)(2)(B). To prove the factor unrelated is "principally responsible," Respondent must "present[] sufficient evidence to establish that [the factor unrelated] was the sole substantial factor in bringing about the injury." de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1354 (Fed. Cir. 2008).

Therefore, in this case, although the parties agree "Petitioners have met the elements of a *prima facie* Table claim for [flu] vaccination and GBS," Petitioners are not entitled to compensation if Respondent has proven by preponderant evidence that a factor unrelated to L.L.P.'s flu vaccination caused L.L.P.'s GBS. Joint Sub. at 2; see 42 C.F.R. § 100.3.

Where Respondent claims a factor unrelated to the administration of the vaccine is causal, the same standards apply to Respondent as to petitioners in a causation-in-fact off-Table claim. See Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d. 543, 549 (Fed. Cir. 1994). Thus, Respondent must establish,

> by preponderant evidence[,] that the [factor unrelated] brought about [L.L.P.'s] injury by providing: (1) a medical theory causally connecting the [factor unrelated] and the injury; (2) a logical sequence of cause and effect showing that the [factor unrelated] was the reason for the injury; and (3) a showing of a proximate temporal relationship between [factor unrelated] and injury.

Althen, 418 F.3d at 1278.

In reviewing the evidence, medical records, specifically contemporaneous medical records, are presumed to be accurate and generally "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). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a party may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

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.

V. FACTOR UNRELATED ANALYSIS

Petitioners allege L.L.P. suffered a Table injury of GBS as a result of a flu vaccination administered on October 12, 2017. Diagnosis is not in dispute; the parties agree L.L.P.'s diagnosis is GBS. Joint Sub. at 1-2. The parties also agree "Petitioners have met the elements of a *prima facie* Table claim for [flu] vaccination and GBS." Id. at 2. At dispute is whether Respondent provided preponderant evidence that L.L.P.'s GBS was caused by a factor unrelated to the administration of the flu vaccine—here, a "nonspecific infectious illness," as Dr. Benson described. Id. at 2-3; Tr. 131-32.

After fully reviewing all of the evidence in this matter, the undersigned concludes that Respondent has not met his burden of demonstrating, "by preponderant evidence[,] [that] L.L.P.'s 'illness, disability, injury, condition, or death described in the petition is due to factors unrelated to the administration of the vaccine described in the petition.'" Joint Sub. at 2 (quoting § 13(a)(1)(B)).

A. Althen Prong One

Under Althen prong one, Respondent must set forth a medical theory explaining how the factor unrelated, an "nonspecific infectious illness," could have caused L.L.P.'s GBS. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1375 (Fed. Cir. 2009); Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355-56 (Fed. Cir. 2006). The 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 Respondent 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))).

The experts agree many cases of GBS are preceded by infections, namely respiratory and gastrointestinal. And they agreed that cases of GBS after vaccination have been reported.

Respondent's expert, Dr. Benson opined L.L.P. had an unidentified nonspecific infectious illness that caused him to develop GBS via molecular mimicry. The experts do not dispute the theory of molecular mimicry generally. At issue is whether an unidentified nonspecific infectious illness can cause GBS via molecular mimicry.

Dr. Benson asserted the theory of molecular mimicry was at play but did not provide any additional explanation, opinion, or evidence in support of this theory. She did not discuss how an unknown infectious illness can trigger molecular mimicry and GBS. Nor did she

discuss or mention what proteins or antigens may be involved, or potential homologies.

Respondent need not make a specific type of evidentiary showing or require identification of a specific antigenic trigger for an immune-mediated pathology to prove that a theory is sound and reliable by preponderant evidence. Given the state of current scientific knowledge, there is no way to satisfy such a requirement. Requiring proof of the identify of a specific antigen to prove causation would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program").

However, simply asserting a causal theory without identification of the trigger (here, an infection) is insufficient. Some infections have been associated with GBS, including *Mycoplasma pneumoniae*, *Campylobacter jejuni*, CMV, and EBV. But many infections have not been shown to have an association with GBS. To show that an infection can cause GBS, the type of infection must be known, or the infection should be identified as one associated with GBS. But where the specific infectious organism has not been identified, and the infection is not characterized by symptoms like diarrhea (which is associated with *Campylobacter jejuni* and GBS) it is speculative to presume a causal association.

Although molecular mimicry is an accepted scientific mechanism, generally opining that molecular mimicry is a causal theory, without more, is insufficient to reach the level of preponderance. See, e.g., Loyd ex rel. C.L. v. Sec'y of Health & Hum. Servs., No. 16-811V, 2021 WL 2708941, at *31 (Fed. Cl. Spec. Mstr. May 20, 2021) ("[T]hough molecular mimicry is a generally accepted scientific concept, and is frequently invoked in Program cases, the mere mention of it does not constitute satisfaction of the preponderant evidentiary standard. Rather, it must be shown that the mechanism likely does link the vaccine in question [or the purported alternative cause] to the relevant injury." (internal citations omitted)); McKown v. Sec'y of Health & Hum. Servs., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (explaining that "merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or vaccine [or alternative cause] in question" (emphasis omitted)); Sheets v. Sec'y of Health & Hum. Servs., No. 16-1173V, 2019 WL 2296212, at *17 (Fed. Cl. Spec. Mstr. Apr. 30, 2019) (determining Althen prong one was not satisfied when the theory of molecular mimicry was not related to the cause/trigger or the injury).

Dr. Benson acknowledged that the only positive viral test in this case was for rhinovirus/enterovirus. However, she rejected this virus as a cause of GBS, noting there was not a strong association to support rhinovirus as a cause of GBS. Tr. 93. Dr. Jaradeh agreed rhinovirus infections are unlikely to cause GBS. Pet. Ex. 50 at 4. And Dr. Steinman agreed rhinovirus infections are not associated with GBS. Tr. 271, 304; Pet. Ex. 21 at 10. The same is true for enteroviruses. Dr. Benson opined that the association between enterovirus and GBS "is not as strong as" other infections and GBS. Resp. Ex. A at 9. Petitioner's expert, Dr. Steinman, agreed with Dr. Benson, noting enterovirus infections are not associated with GBS. Tr. 271, 304; Pet. Ex. 21 at 10.

The undersigned finds Respondent did not provide preponderant evidence of a sound and reliable theory to explain how a "nonspecific infectious illness" can cause GBS. Moreover, Respondent failed to show by preponderant evidence that rhinovirus and/or enterovirus can cause GBS via molecular mimicry, as the evidence shows these viruses are not generally associated with GBS.

Thus, the undersigned finds Respondent has failed to provide preponderant evidence with respect to the first Althen prong.

B. Althen Prong Two

Under Althen prong two, Respondent must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect" showing the nonspecific infectious illness was the reason for the injury. Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). Respondent must show that the nonspecific infectious illness "was the 'but for' cause of the harm . . . or in other words, that [it] 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 [or an alternative cause] 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. Respondent 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, Respondent may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Since Respondent failed to prove Althen prong one, it follows that he cannot prove Althen prong two. However, even if Respondent had proven Althen prong one, the undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause-and-effect showing a nonspecific infectious illness was the cause of L.L.P.'s GBS. See Althen, 418 F.3d at 1278.

First, the Vaccine Act limits the scope of unrelated factors by excluding any "idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness, or condition." § 13(a)(2)(A). "In other words, alternative causes that are 'idiopathic, unexplained, unknown, hypothetical, or undocumentable' cannot overcome a petitioner's prima facie case." Doe v. Sec'y of Health & Hum. Servs., 601 F.3d 1349, 1357 (Fed. Cir. 2010) (quoting § 13(a)(2)(A)).

Here, Respondent's expert Dr. Benson described the cause as a "nonspecific infectious illness." Using "nonspecific" to describe the alleged infectious illness is akin to one that is "unexplained, unknown, hypothetical, or undocumentable." Using the word "nonspecific" does not take away from the fact that Respondent's alleged "factor unrelated" is not known or identified. Thus, based on the language of the Vaccine Act alone, Respondent failed to provide preponderant evidence that a factor unrelated to L.L.P.'s vaccination caused his GBS.

Furthermore, the Vaccine Act provides that "factors unrelated to the administration of the vaccine" are those "which are shown to have been the agent . . . principally responsible for causing the petitioner's illness, disability, injury, condition or death." § 13(a)(2)(B). Here, Respondent has not shown that the "nonspecific infectious illness" was "principally responsible for causing [L.L.P.'s] [GBS]." Thus, Respondent's argument of a "factor unrelated" further fails.

Second, the undersigned finds L.L.P.'s clinical course does not support Respondent's factors unrelated argument, specifically that L.L.P. had a nonspecific infectious illness prior to the onset of GBS that caused his GBS. Respondent's expert, Dr. Benson opines that L.L.P.'s symptoms prior to November 16 were "nonspecific" and "common" to a variety of infectious illnesses. Yet, she agreed that some of these "nonspecific" complaints can also be seen with GBS. She was unable to place the nonspecific illness to any particular system. Instead, she called it a "multisystem generalized illness." Tr. 132. However, L.L.P. was never diagnosed with a multisystem infection.

Further, the alleged nonspecific infectious illness here was not attributed to a virus or bacteria known to cause GBS. Dr. Benson did not opine that the "nonspecific infectious illness" was one that is commonly associated with GBS. Two infections associated with GBS include *Mycoplasma pneumoniae* and *Campylobacter jejuni*. Dr. Benson agreed that L.L.P. tested negative for *Mycoplasma pneumoniae*, and she opined that *Campylobacter jejuni* was only "possible." However, opinions expressed as possibilities are not sufficient to establish causation. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that possibilities does not equate to proof of causation by a preponderance of the evidence).

Here, Respondent and Dr. Benson's arguments are conclusory and lack supportive evidence. Special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). Nor do special mastery rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315).

Third, the only known infection was rhinovirus/enterovirus, and Respondent did not implicate rhinovirus/enterovirus as the cause of L.L.P.'s GBS. Dr. Benson testified that "[she] [was] not proposing that rhinovirus causes GBS" as "[t]here's not a strong association there." Tr. 93. She also opined that the association between enterovirus and GBS "is not as strong as"

other infections and GBS. Resp. Ex. A at 9; see also Tr. 121. Dr. Jaradeh agreed rhinovirus infections "are among the least likely culprits to produce [GBS]." Pet. Ex. 50 at 4. And Dr. Steinman opined rhinovirus and enterovirus infections are not associated with GBS. Tr. 271, 304; Pet. Ex. 21 at 10.

Fourth, although some of L.L.P.'s treating physicians questioned whether L.L.P. had a viral illness, after an extensive workup, they did not find any obvious or known etiology for L.L.P.'s GBS. Dr. Watson, on November 19, opined, "it is unclear at this time what is leading to [L.L.P.'s] weakness and other symptoms. It is possible that this is generalized weakness secondary to a severe systemic viral illness." Pet. Ex. 5 at 3. However, opinions expressed as possibilities are not sufficient to establish causation. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that possibilities does not equate to proof of causation by a preponderance of the evidence). Thus, the undersigned finds Dr. Watson's statement based on a possibility falls short of preponderant evidence.

On the night of November 19, 2017, at approximately 7:30 p.m., L.L.P. was seen by family Medicine Resident Dr. Hoyum, who listed a diagnosis of URI due to L.L.P.'s "worsening weak cough" and ataxia (with GBS in the differential). Pet. Ex. 5 at 33-34. However, the worsening of L.L.P.'s respiratory condition was determined to be secondary to his GBS requiring intubation and ventilation. Further, the diagnosis of URI was dropped after the diagnosis of GBS was confirmed (which required intubation).

Senior Pediatrics Resident Dr. Kristin Kalita's assessment on November 19 indicated "[c]oncern at this time is for [GBS] vs another acute demyelinating process." Pet. Ex. 5 at 36. Neurologists Dr. Dingman and Dr. Luebbert, on November 19, indicated that L.L.P.'s "elevated [WBC] count suggest[ed] an inflammatory process, while his elevated liver enzymes would be consistent with a viral process." Id. at 42. When L.L.P. exerted effort on neurologic examination, "his strength [was] 5/5 throughout (arguing against [GBS])," and thus, they opined "[s]ystemic viral infection seems most likely." Id. However, later that evening, PICU attending, Dr. Good, wrote, L.L.P. "[h]as been afebrile, making infection less likely." Id. at 93.

Generally, treating physician statements are "favored" 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.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280).

Taking these treating physicians' statements together, the undersigned finds that some treating physicians questioned whether L.L.P. had an infectious process, but they did not diagnosis him with a nonspecific infectious illness or conclude that, more likely than not, an infectious illness was the cause of his GBS.

Further, Dr. Jaradeh provided persuasive evidence that instead of a nonspecific infectious illness, L.L.P.'s presentation was consistent with autonomic dysfunction due to GBS. L.L.P. had stomach and abdominal pain and digestive symptoms indicative of involvement of the small nerve and autonomic nerve fibers. L.L.P. also had arthralgias and myalgias, and shortness of breath, also consistent autonomic symptoms triggered by the flu

vaccine. Dr. Jaradeh opined that L.L.P.'s intermittent vomiting, along with constipation, was consistent with early autonomic parasympathetic vagus nerve dysfunction. L.L.P. also developed blurred vision and jaw pain, which Dr. Jaradeh explained was probably due to a slow reaction of his pupils, again due to autonomic dysfunction. And the jaw pain suggested to Dr. Jaradeh that L.L.P. had trigeminal sensory small fiber nerve involvement. L.L.P. also reported taste disturbances, which also indicated autonomic problems. L.L.P. also had difficulty swallowing (oropharyngeal dysphagia), cough, and hoarseness of his voice, which Dr. Jaradeh explained was laryngeal nerve impairment due to GBS.

Dr. Jaradeh opined that L.L.P.'s above-mentioned clinical course was not caused by a nonspecific infectious illness but instead caused by autonomic dysfunction consistent with his GBS presentation. Dr. Jaradeh has special expertise in this subject matter, he cited supportive medical literature, and these opinions explain L.L.P.'s overall clinical course, accounting for the entire presentation. For these reasons, the undersigned agrees with Dr. Jaradeh's opinion that as of November 11,[36] many of the signs and symptoms initially attributed to an infectious illness were, more likely than not, due to autonomic dysfunction. Further, the undersigned finds that there is not preponderant evidence that L.L.P.'s GBS was caused by a "nonspecific infectious illness."

Moreover, the undersigned's finding is consistent with case law. In Castenada, the special master found in favor of entitlement where a petitioner proved her Table claim for flu/GBS by preponderant evidence and Respondent failed to identify a specific infectious pathogen to support his position that a preceding viral illness caused the petitioner's GBS. Castenada ex rel. S.E.C. v. Sec'y of Health & Hum. Servs., No. 18-1958V, 2021 WL 2547911 (Fed. Cl. Spec. Mstr. June 1, 2021). A similar result occurred in Taylor, where the medical records did not allow the Chief Special Master to conclude that a CMV infection was more likely than not the cause of the petitioner's GBS. Taylor v. Sec'y of Health & Hum. Servs., No. 22-335V, 2025 WL 1234906 (Fed. Cl. Spec. Mstr. Mar. 25, 2025).

Accordingly, the undersigned finds that Respondent has not satisfied his burden under Althen prong two.

C. Althen Prong Three

Althen prong three requires Respondent to establish a "proximate temporal relationship" between the nonspecific infectious illness and L.L.P.'s GBS. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. Respondent 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 be consistent 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.

[36] Regarding L.L.P.'s complaints in October 2017, as reflected in Ms. P.'s affidavit and testimony, the undersigned finds them to be too vague to suggest either an infection or autonomic nervous system manifestations of GBS.

Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358.

L.L.P. received a flu vaccination on October 12, 2017. The experts disagree as to whether the symptoms and complaints leading up to November 16 are consistent with GBS or indicative of an infectious illness. Yet, the experts agree L.L.P. exhibited weakness on November 16, 2017, 35 days post-flu vaccination. Further, the parties stipulate that L.L.P.'s onset was within three and 42 days of vaccination as required for a Table claim of GBS following the flu vaccine. 42 C.F.R. § 100.3(a).

Because the undersigned finds Respondent did not provide preponderant evidence that L.L.P. had a nonspecific infectious illness during the period between October 12, 2017 (date of vaccination) and November 16, 2017 (agreed upon date of onset of weakness), the undersigned finds L.L.P.'s onset timeline does not support an alternative cause of an infectious illness.

Therefore, the undersigned finds Respondent failed to provide preponderant evidence in support of Althen prong three. Even if Respondent provided preponderant evidence satisfying Althen prong three, Respondent cannot prevail since he failed to provide preponderant evidence to prove Althen prongs one and two and a temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury.").

VI. CONCLUSION

For all of the reasons stated herein, Respondent has failed to meet his burden, by preponderant evidence, to establish that L.L.P.'s GBS was due to an alternative cause or "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). Therefore, because Petitioners have proven by preponderant evidence that L.L.P. suffered from the Table injury of GBS caused by the flu vaccination that he received on October 12, 2017, Petitioners have established entitlement to compensation.

A separate damages order shall issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master