In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 10, 2026

COLLEEN ALTHAUS,	*	PUBLISHED
Petitioner,	*	No. 18-1868V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Downbeat Nystagmus; One Day Onset.
Respondent.	*	

Mark T. Sadaka, Sadaka Associates LLC, Englewood, NJ, for Petitioner.
Eleanor A. Hanson, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

I. INTRODUCTION

On December 6, 2018, Colleen Althaus ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that she developed "vaccine-induced vertigo, autoimmune cerebellar visual disturbance, cerebellar visual disorder/atrophy, post vaccination downbeat nystagmus, and post vaccinal encephalitis caused-in-fact, . . . or in the

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

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

alternative, significantly aggravated by"[3] an influenza ("flu") vaccination administered on December 8, 2015. Petition at Preamble (ECF No. 1). Respondent argued against compensation and requested the "petition be dismissed for insufficient proof." Respondent's Report ("Resp. Rept.") at 1-2 (ECF No. 19).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[4] the undersigned finds that Petitioner has failed to provide preponderant evidence that the flu vaccination caused her illness because onset of her symptoms occurred within 24 hours, which was too soon to implicate vaccination given the proffered theory of molecular mimicry. Thus, Petitioner has failed to satisfy her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is not entitled to compensation.

II. ISSUES TO BE DECIDED

First, the parties dispute the "scope of [P]etitioner's diagnosis." Joint Sub. at 1. While they "agree she has downbeat nystagmus[5] . . . they disagree as to the scope of [her] diagnosis . . . and whether her alleged injury is auto-immune mediated." Id. Next, the parties dispute the

[3] In their joint submission, the parties did not identify significant aggravation as a relevant issue. See Joint Pre-Hearing Submission ("Joint Sub."), filed Oct. 9, 2024 (ECF No. 97). Further, the medical records do not show a factual basis for any claim based on significant aggravation, and the parties' experts do not opine that Petitioner had a pre-existing condition that was aggravated by the vaccination at issue. Therefore, the undersigned does not discuss the initial allegation of significant aggravation raised in the Petition.

[4] While the undersigned has reviewed all of 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 he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[5] Nystagmus is a congenital or acquired condition that involves "spontaneous involuntary eye movements" that may cause "oscillopsia (a subjective sense of visual motion) and/or reduction in visual acuity and clarity." Resp. Exhibit ("Ex.") A, Tab 1 at 2 (Janet C. Rucker, Nystagmus and Saccadic Intrusions, 25 Continuum 1376 (2019)). One of the most common forms of acquired central nystagmus is downbeat nystagmus. Id. at 15. "Downbeat nystagmus, in most cases, represents cerebellar dysfunction, typically with lesions involving the vestibulocerebellum." Id. "Downbeat nystagmus . . . is seen in a wide variety of cerebellar diseases, including degenerative, paraneoplastic, metabolic-toxic, and ischemic etiologies. A large percentage of cases are idiopathic." Id. at 15-16.

onset of Petitioner's injuries.[6] Id. Lastly, the parties dispute causation. Id. at 2. Specifically, they dispute whether Petitioner has satisfied all three Althen prongs required to establish entitlement to causation. Id.

III. BACKGROUND

A. Procedural History

On December 6, 2018, Petitioner filed a petition requesting compensation followed by medical records.[7] Petition; Petitioner's ("Pet.") Exs. 1-9. The case was assigned to now-Chief Special Master Corcoran. Notice of Assignment dated Dec. 6, 2018 (ECF No. 4). The case was then reassigned to the undersigned. Notice of Reassignment dated Oct. 1, 2019 (ECF No. 16). Respondent filed a Rule 4(c) report on December 3, 2019, arguing against compensation. Resp. Rept. at 1-2.

On October 5, 2020, Petitioner filed an expert report from Dr. Alberto Martinez-Arizala. Pet. Ex. 10. On June 7, 2021, Respondent filed expert reports from Dr. Marc Bouffard and Dr. James Moy. Resp. Exs. A, C.

The undersigned held a Rule 5 conference on August 10, 2021. Order dated Aug. 13, 2021 (ECF No. 41). While she noted that molecular mimicry was "a sound and reliable mechanism of causation," she advised the parties that Petitioner's "presentation of symptoms so short in time when the mechanism is molecular mimicry" was "unusual." Id. at 4. The undersigned also noted that she had "questions regarding onset." Id. She recommended the parties consider settlement. Id.

On November 29, 2021, Respondent advised he was not interested in settlement and the undersigned ordered additional expert reports. Order dated Nov. 30, 2021 (ECF No. 47). Petitioner filed a supplemental expert report from Dr. Martinez-Arizala on August 1, 2022. Pet. Ex. 23. On December 27, 2022, Respondent filed supplemental expert reports from Dr. Bouffard and Dr. Moy. Resp. Exs. E-F. Petitioner declined to file a responsive expert report, and the parties agreed to resolve the case through an entitlement hearing. Joint Status Rept., filed Feb. 27, 2023 (ECF No. 64).

On June 9, 2023, an entitlement hearing was set for November 2024. Prehearing Order dated June 9, 2023 (ECF No. 71).

Two months before the scheduled entitlement hearing, Petitioner requested to bifurcate the hearing in order to obtain an expert report from an immunologist. Pet. Status Rept., filed Sept. 10, 2024 (ECF No. 87). Respondent objected to bifurcation of the hearing and any new

[6] The undersigned's ruling on onset is set forth in the causation analysis regarding Althen prong three.

[7] Petitioner continued to file medical records throughout litigation.

expert reports from Petitioner. Resp. Response to Pet. Status Rept., filed Sept. 11, 2024 (ECF No. 89). The undersigned denied Petitioner's request to bifurcate the entitlement hearing. Order dated Sept. 17, 2024 (ECF No. 90). The undersigned noted that Petitioner had been on notice of any immunological issues since 2021. Id. She advised Petitioner that she could either proceed with the entitlement hearing or obtain a new expert report and have the case resolved via a ruling on the record. Id. Petitioner elected to proceed with the entitlement hearing. Pet. Status Rept., filed Sept. 27, 2024 (ECF No. 93).

On October 24, 2024, the entitlement hearing was cancelled due to the unavailability of Petitioner's expert witness. Order dated Oct. 24, 2024 (ECF No. 102). In lieu of a hearing, the case would be resolved by a ruling on the record. Id.

Petitioner filed his motion for a ruling on the record on December 23, 2024. Pet. Motion for a Ruling on the Record ("Pet. Mot."), filed Dec. 23, 2024 (ECF No. 109). Respondent filed his responsive brief on February 21, 2025, and Petitioner filed a reply on March 24, 2025. Resp. Response to Pet. Mot ("Resp. Response"), filed Feb. 21, 2025 (ECF No. 110); Pet. Reply to Resp. Response ("Pet. Reply"), filed Mar. 24, 2025 (ECF No. 111).

This matter is now ripe for adjudication.

B. Factual History[8]

Petitioner's pre-vaccination medical history was significant for anemia and tympanostomy/tympanoplasty.[9] Pet. Ex. 1 at 36, 61, 76. On December 8, 2015, at age forty-five, Petitioner received the flu vaccine (Fluarix) in her right deltoid. Pet. Ex. 2 at 5. At the time of vaccination, Petitioner worked as a medical secretary. Pet. Ex. 1 at 77.

On December 16, 2015, Petitioner presented to ophthalmologist Huiyi Chen, M.D., at Mercy Eye Specialists, for complaints of double vision/distorted vision in her left eye for one week. Pet. Ex. 3 at 3-5. Petitioner reported receiving a flu vaccine the prior Tuesday (December 8), and she started noticing visual disturbances the next day, Wednesday (December 9). Id. at 4. Specifically, Petitioner complained of seeing crossed or "wavy" images when she opened both eyes, though she had no visual disturbance when she covered either eye. Id. The problem was worse in the morning and improved in the evening. Id. She stated that her eyes felt "very 'tired'" and that she used the computer and reviewed documents for her work. Id. In addition to her vision issues, Petitioner also complained of "inner ear issues." Id. Review of symptoms was positive for migraines. Id. Petitioner had a normal eye examination, and no etiology was

[8] This summary of medical records is taken from Respondent's Response, as the undersigned finds it is an accurate representation of the medical records. See Resp. Response at 3-11. The undersigned has edited the summary and included additional relevant information.

[9] Tympanoplasty is the "surgical reconstruction of the hearing mechanism of the middle ear." Tympanoplasty, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=51586 (last visited Jan. 21, 2026).

identified. Id. at 4-7. She was diagnosed with subjective visual disturbance. Id. at 4-5. Dr. Chen thought that Petitioner could have "some symptoms of presbyopia" (aging sight) and recommended low strength reading glasses for extended periods of computer work or reading. Id. Dr. Chen advised Petitioner to return in one year, and to follow up with her primary care physician ("PCP") if her symptoms worsened. Id. at 5.

The following day, December 17, 2015, Petitioner called her PCP and requested azithromycin for "frequent sinus problems;" she also reported that her nasal drainage was now yellow. Pet. Ex. 1 at 59-60, 63.

On December 29, 2015, Petitioner presented to her PCP with complaints of "ear pain in both ears," "congestion, post nasal drip, coryza,[10] [and] sinus pressure" for two weeks. Pet. Ex. 1 at 66-67. Physical examination revealed purulent nasal discharge, "moderate congestion, turbinates red, sinus tenderness bilateral," and normal tympanic membranes. Id. at 67. She was diagnosed with acute sinusitis and prescribed amoxicillin and Mucinex. Id.

Petitioner called her PCP on January 12, 2016, reporting that she had been seen in December with a bilateral ear infection, and that she had completed her antibiotics the previous Friday. Pet. Ex. 1 at 75. She further reported her ears began hurting two days later (Sunday), and she was also having dizziness, congestion, pressure in her head, and problems focusing her left eye. Id. Petitioner was advised to make an appointment. Id. Petitioner noted that her insurance had changed so she had to follow up "on that first" and would call back "if necessary." Id. at 76. On January 14, 2016, Petitioner again called her PCP. Id. She "wanted [her doctor] to know she has been reading about her ear problem, thinks she may have labyrinthitis."[11] Id.

The following day, January 15, 2016, Petitioner returned to her PCP with ongoing complaints of congestion and sinus and bilateral ear pain since December 29, 2015 (for five weeks), with nasal congestion for the past five days, and "trouble focusing with her [left] eye." Pet. Ex. 1 at 81-82. Petitioner also reported "pain with left eye movements." Id. at 82. Her examination was normal, including "eye movements intact without nystagmus." Id. She was diagnosed with chronic sinusitis and prescribed prednisone, fluticasone nasal spray, and Mucinex. Id.

On March 8, 2016, Petitioner presented to otolaryngologist Karen Boone, M.D., for a three-month history of dizziness with "some difficulty with vision." Pet. Ex. 4 at 3. Petitioner's

[10] Coryza, also known as acute rhinitis, is "an acute congestion of the mucous membrane of the nose, marked by dryness, followed by increased mucous secretion from the membrane, impeded respiration through the nose, and pain." Acute Rhinitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=103973 (last visited Jan. 23, 2026).

[11] Labyrinthitis refers to "inflammation of the internal ear." Labyrinthitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=27360 (last visited Jan. 23, 2026).

visual issues were described as "abnormal saccadic movements,[12] blurred vision, not quite double vision but 'overlay.'" Id. Dr. Boone noted Petitioner's history of "chronic ear issues" and that Petitioner's PCP thought there was an ear infection and prescribed antibiotics, which did not improve her symptoms. Id. An examination of Petitioner's ears, nose, and throat revealed evidence of a left tympanoplasty, but was otherwise normal, and her eye examination was unremarkable. Id. at 4. Dr. Boone's impression was abnormal central saccadic movements, and she did not believe Petitioner's issue was vestibular ("not clear vestibular").[13] Id. Dr. Boone ordered a brain magnetic resonance imaging ("MRI"), electronystagmography ("ENG"), and videonystagmography ("VNG") for further evaluation. Id.

Petitioner's March 17, 2016 brain MRI was unremarkable. Pet. Ex. 5 at 1. On April 4, 2016, Petitioner saw Mark Wallace, M.D., for ENG and VNG testing. Pet. Ex. 6 at 7-8. At the visit, Petitioner "reported that she had a flu shot on December 8, 2016[,] and the next day she started having visual disturbances. She describe[d] it as blurry vision, double vision, a feeling that her eyes were jumping." Id. at 7; Pet. Ex. 4 at 7. Petitioner completed a patient questionnaire prior to this visit, on March 28, 2016, wherein she wrote her problem first occurred on December 9, 2015. Pet. Ex. 6 at 1, 5. In the patient questionnaire, Petitioner specifically noted that she had "bouncing eyes" since December 9, 2015. Id. at 5.

The ENG and VNG tests revealed abnormal spontaneous nystagmus, abnormal smooth pursuit, abnormal vertical gaze, abnormal post headshake test, and abnormal Dix-Hallpike maneuver.[14] Pet. Ex. 6 at 7-8. Impression following testing was normal saccade testing and abnormal vertical and horizontal smooth pursuit testing with saccadic intrusions, which could "be age related, medication induced, or indicative of a central deficit." Id. at 8. Gaze testing revealed "spontaneous Leftbeat nystagmus." Id. "There was direction changing nystagmus throughout testing, downbeat nystagmus, and no subjective reports of vertigo, all of which are consistent with a central deficit." Id. (emphasis omitted). Petitioner was advised to follow up with Dr. Boone. Id.

On April 7, 2016, Petitioner had a follow-up appointment with Dr. Boone. Pet. Ex. 4 at 1-2. At that time, Petitioner had an unremarkable eye examination. Id. at 2. Based on the ENG/VNG results, Dr. Boone referred Petitioner to a neurologist or a neuro-ophthalmologist. Id.

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[12] Saccadic movement is "the quick movement of the eye in going from one fixation point to another." Saccadic Movement, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=89719 (last visited Jan. 23, 2026).

[13] The vestibular system is "the bodily structures connected with receiving and processing sensations of the sense of equilibrium." Vestibular System, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111913 (last visited Jan. 23, 2026).

[14] Dix-Hallpike maneuver is "a test for benign positional vertigo . . . the examiner observes for positional nystagmus, which is indicative of benign positional vertigo." Dix-Hallpike Maneuver, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87854 (last visited Jan. 23, 2026).

Petitioner presented to neurologist Sudhir Batchu, M.D., on May 16, 2016. Pet. Ex. 7 at 2-7. Petitioner reported dizziness and visual disturbances since receiving the flu vaccine, which started with "diplopia and skewed vision on extreme gaze" and progressed to include problems with balance. Id. at 3. She reported that her symptoms initially progressed, but later plateaued, although she continued to have dizziness and blurred vision. Id. On examination, Dr. Batchu found "[n]ystagmus in all directions." Id. at 5. Dr. Batchu's impression was "vertigo and nystagmus," "possible post vaccinal encephalitis," and "chronic ear problems and history of tympanoplasty." Id. at 6. Dr. Batchu ordered additional testing, including a lumbar puncture, and prescribed prednisone and memantine.[15] Id. at 6-7. Laboratory work ordered included testing for Lyme disease and Herpes simplex. Id. Diagnoses for the physician orders was vertigo and vestibular neuronitis, bilateral. Id. at 8-14. Lyme disease testing at an outside lab returned negative results.[16] Id. at 39.

On June 6, 2016, Petitioner returned to Dr. Batchu for follow-up. Pet. Ex. 7 at 21-23. She reported she did "not have the lumbar puncture due to [] insurance denial." Id. at 21. By the time the insurance company agreed to cover the procedure, she was feeling better after starting on steroids, so she decided not to have it. Id. Also, she did not feel she could take time off work for the procedure. Id. On examination, nystagmus were present in horizontal pursuit, coarse and greater in the left than in the right. Id. at 22. Dr. Batchu's impression was nystagmus, improving vertigo, and "possible post vaccinal inflammatory condition." Id. at 23.

Petitioner sought a consultation with neurologist Laurence Kinsella, M.D., at St. Claire's Neurology on July 19, 2016. Pet. Ex. 8 at 2-8. Petitioner told Dr. Kinsella that "she received a flu shot on [December 8, 2015] and when she woke up the next day she had [double vision]" on her left side. Id. at 2. She complained of having a hard time focusing and problems with balance and movement. Id. She had some improvement with prednisone, but it caused weight gain of 25 pounds. Id. She denied nausea associated with her double vision. Id. Petitioner also described "skew deviation looking to the right" and that "[l]ooking left induces sense of movement up and down." Id. Petitioner's neurological examination revealed "constant vertical, downbeat nystagmus in primary gaze, increased looking up[,] and end gaze horizontal nystagmus." Id. at 5. Dr. Kinsella's impression was a normal MRI of the brain and post-vaccination downbeat nystagmus. Id. at 6-7, 11-12. Dr. Kinsella noted that despite the nystagmus and diplopia, Petitioner was remarkably unaffected, she worked two jobs and was active with outdoor activities. Id. at 7. Because there was no evidence of Miller-Fisher syndrome, multiple sclerosis, or post-infectious leukoencephalopathy, Dr. Kinsella referred Petitioner to neuro-ophthalmologist Sophia Chung, M.D. Id.

[15] Memantine hydrochloride is a "an N-methyl-d-aspartate . . . antagonist" that "protects the brain's nerve cells against glutamate." Memantine Hydrochloride, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30289 (last visited Jan. 29, 2026). Memantine is used in the treatment of nystagmus. See Resp. Ex. A, Tab 1 at 16.

[16] Results for herpes simplex testing do not appear to be included in Dr. Batchu's records.

Petitioner was evaluated by neuro-ophthalmologist Dr. Chung, a Professor of Ophthalmology, Neurology, and Neurosurgery at St. Louis University School of Medicine, on September 13, 2016. Pet. Ex. 9 at 2-9. Petitioner again "report[ed] that she had a flu shot on [December 8, 2015] and the next day she woke up with binocular double vision in left gaze." Id. at 2. Dr. Chung agreed with Dr. Kinsella's diagnosis of primary position downbeat nystagmus with skew deviation in right gaze. Id. at 8. Dr. Chung stated,

> While flu vaccinations are associated with neurologic sequelae, the usual time of onset is [one to four] weeks. It would be highly unusual to have onset the following day given that it takes time to develop an autoimmune response. However, she does report that her symptoms progressed over the ensuing couple/few weeks. Therefore[,] I think it is reasonable to do some other tests to be sure there are not alternate causes although the history of improvement in response to steroids (albeit [four] months after onset) supports an inflammatory cause.

Id. at 9.

Dr. Chung ordered additional diagnostic testing for antibodies and noted that, pending the test results, Petitioner could consider treatment with baclofen, gabapentin, or antiepileptics. Pet. Ex. 9 at 9, 17-26. The antibody testing revealed positive glutamic acid decarboxylase ("GAD") 65 antibodies,[17] insulinoma-associated-2 ("IA-2") antibodies, and insulin auto-antibodies.[18] Id. at 15. Petitioner's GAD65 was greater than 250 IU/ml (reference range is less than 5 IU/ml). Id. Other diagnostic lab tests were normal/negative. See id. at 21-25. Dr. Chung made Petitioner aware of the positive GAD65 antibodies and explained the association between GAD65 antibodies with type I diabetes and cerebellar syndrome. Id. at 19. Dr. Chung suggested that Petitioner return to see Dr. Kinsella for treatment of the nystagmus given the discovery of GAD65 antibody and made Dr. Kinsella aware of the testing. Id.

Petitioner returned for follow up with Dr. Kinsella on October 11, 2016. Pet. Ex. 8 at 13-22. Dr. Kinsella noted Petitioner had been seen by Dr. Chung and had been referred back for treatment. Id. at 14. Dr. Kinsella's diagnosis was post-vaccination downbeat nystagmus,

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[17] "GAD antibodies are autoantibodies against the enzyme [GAD], which is involved in the production of the neuro-transmitter gamma-aminobutryic acid." Resp. Ex. C, Tab 1 at 1 (Anitra D.M. Koopman et al., The Association Between GAD65 Antibody Levels and Incident Type 2 Diabetes Mellitus in an Adult Population: A Meta-Analysis, 95 Metabolism 1 (2019)). GAD67 and GAD65 is "needed throughout the development of normal cellar functioning in the brain . . . [and] the regulation of the . . . pancreas." Id. GAD65 is "strongly associated" with type I diabetes and "latent autoimmune diabetes." Id.

[18] Insulin autoantibodies, IA-2, and GAD antibodies are used to screen for type I diabetes as their presence "precede onset of clinical disease." Erika F. Brutsaert, Type 1 Diabetes Mellitus, Merck Manual, https://www.merckmanuals.com/professional/endocrine-and-metabolic-disorders/diabetes-mellitus-and-hypoglycemia/type-1-diabetes-mellitus (last visited Feb. 3, 2026).

autoimmune cerebellar visual syndrome, and "[positive] Anti GAD and IA-2 antibodies." Id. at 15. Dr. Kinsella noted that Petitioner's condition was "indicative of an autoimmune process, limited to the visual cerebellar pathways, sparing limb and axial cerebellar pathways. It is unclear how it is related to the flu shot since symptoms were immediately following. She doesn't find these symptoms disabling." Id. Petitioner wished to hold off on treatment until she did additional research. Id. at 16.

Dr. Kinsella's notes referenced an article[19] discussing a case report of autoimmune cerebellar ataxia related to GAD-Ab, which was noted to typically affect women with late onset type I diabetes[20] or autoimmune "polyglandular failure." Pet. Ex. 8 at 16. Per Dr. Kinsella, the article concluded that "[a]uto-immune cerebellar ataxia related to GAD-Ab is a rare condition that typically affects women with late-onset type [I] diabetes or other auto-immune disorders, including auto-immune polyendocrinopathy." Id. Symptoms improved with immunoglobulin therapy. Id.

Approximately seven months later, on May 22, 2017, Petitioner returned to Dr. Kinsella. Pet. Ex. 8 at 23-31. Petitioner's condition remained unchanged since her last visit, and she still had complaints of imbalance and that her vision was "off." Id. at 24. Her diagnoses were the same as well. Id. Treatment options were discussed but Petitioner wished to wait to start any therapy. Id. The record also noted that "[Petitioner] would like to have you contact her lawyer as she is filing a petition regarding the vaccine she received and had SE[21] from it." Id. Dr. Kinsella's visit notes contained the following statements: "The story is [consistent with] a vaccine associated autoimmune neurologic disorder. I placed a call to her attorney," and "[i]t is unclear how it is related to the flu shot since symptoms were immediately following." Id. at 24, 26.

On June 19, 2017, Petitioner returned to her PCP. Pet. Ex. 1 at 101-10. She complained of unintentional weight loss of 20 pounds in eight months. Id. at 101. She reported being on prednisone in the summer of the prior year and gained some weight at that time. Id. Once the prednisone was discontinued, she lost weight. Id. She also reported "thinning hair, excess thirst, [and] itchiness." Id. The record also noted that Petitioner had an underlying autoimmune condition. Id. She had not yet started gabapentin. Id. at 102. The physical examination, including her eye examination, was normal. Id. at 102-03.

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[19] C. Bayreuther et al., Auto-Immune Cerebellar Ataxia with Anti-GAD Antibodies Accompanied by De Novo Late-Onset Type 1 Diabetes Mellitus, 34 Diabetes & Metab. 386 (2008). This article was not filed.

[20] Type I diabetes is "an autoimmune disease that involves destruction of the insulin-secreting pancreatic beta cells, leading to impaired insulin secretion, hyperglycemia, and . . . peripheral insulin resistance." Brutsaert, supra note 18.

[21] SE may be an abbreviation for "side effect."

On June 30, 2017, Petitioner again presented to her PCP for new-onset type II diabetes.[22] Pet. Ex. 1 at 123. She declined angiotensin-converting enzyme ("ACE") inhibitor and statin prescriptions, which were offered. Id. at 122. She also declined the Hepatitis B and pneumococcal vaccines. Id. at 123. Her hemoglobin A1C was high at 12.1 and blood glucose was 404 mg/dL. Id. at 124. Her physical examination was unremarkable. Id. at 123-24. Petitioner was diagnosed with uncontrolled type II diabetes, and the plan included medication and referral to a nutritionist. Id. at 124-25. Petitioner's PCP also reminded her to get a yearly retinal exam. Id. at 125.

On February 2, 2018, Petitioner followed up with her PCP for her diabetes. Pet. Ex. 1 at 158. She was taking metformin and exercising irregularly. Id. Her morning blood sugar averages were 115-120, but she had not checked it "in a while." Id. Her hemoglobin A1C was still high at 12.7. Id. Her physical examination was unremarkable. Id. at 159. The assessment remained the same, and she was prescribed glimepiride (Amaryl).[23] Id. at 160.

Between December 2018 and October 2020, Petitioner had eight medical encounters related to her uncontrolled type II diabetes. See Pet. Ex. 26 at 3-4, 41; Pet. Ex. 28 at 208-10; Pet. Ex. 29 at 12-15, 31-35, 73-74, 96-98, 117. Petitioner struggled to control her blood sugar during this period. See Pet. Ex. 29 at 44-45, 96-98, 117. As of October 23, 2020, Petitioner's hemoglobin A1C was still high at 12.0. Pet. Ex. 26 at 41.

An entry in Petitioner's medical records from December 7, 2020 noted that Petitioner was allergic to the flu vaccine. Pet. Ex. 26 at 45. Specifically, it noted that Petitioner had suffered post-vaccination downbeat nystagmus, autoimmune cerebellar visual syndrome, and positive Anti GAD and IA-2 antibodies. Id.

On January 11, 2021, Petitioner was seen by her PCP. Pet. Ex. 26 at 52. At that visit, her PCP recorded that Petitioner has started seeing an endocrinologist.[24] Id. In the assessment and

[22] Type II diabetes "is characterized by insulin resistance and inadequate insulin secretion relative to needs." Erika F. Brutsaert, Type 2 Diabetes Mellitus, Merck Manual, https://www.merckmanuals.com/professional/endocrine-and-metabolic-disorders/diabetes-mellitus-and-hypoglycemia/type-2-diabetes-mellitus (last visited Feb. 5, 2026).

[23] Glimepiride, the generic form of Amaryl, is a "a sulfonylurea compound used . . . in the treatment of type [II] diabetes mellitus." Glimepiride, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=20328 (last visited Jan. 23, 2026).

[24] Petitioner did not file medical records from her endocrinologist.

plan for the visit, Petitioner was noted to have "latent autoimmune diabetes in adults"[25] that was "managed as" type I diabetes. Id. at 53. Petitioner's "active problem list" included latent autoimmune diabetes as diagnosed by Dr. Matthews, an endocrinologist, in December 2020. Id. at 54. Petitioner was noted to have "insulin autoantibodies [and] GAD antibodies." Id.

At visits on March 6, 2022, and June 13, 2023, Petitioner's PCP again listed latent autoimmune diabetes managed as type I diabetes as an "active problem" and noted Petitioner was followed by an endocrinologist for this condition. Pet. Ex. 26 at 124, 126, 176, 179.

In a letter dated May 11, 2023, Petitioner asked Dr. Kinsella for an excusal letter from jury duty due to "the brain injury [she] suffered." Pet. Ex. 27 at 2. Dr. Kinsella's office wrote back explaining that the office could not issue such a letter because she had not been seen in six years, and per office policy she was no longer an established patient of the clinic. Id. at 3. Petitioner then sought a letter from her PCP. Pet. Ex. 26 at 159-60. Petitioner's PCP noted that she had not been seen in that office in over a year and was unsure whether the office could write such a letter. Id.

No additional relevant medical records were provided.

C. Expert Reports

1. Petitioner's Expert, Dr. Alberto A. Martinez-Arizala[26]

a. Background and Qualifications

Dr. Martinez-Arizala is a Professor of Clinical Neurology, Neurosurgery, and Physical Medicine and Rehabilitation at the University of Miami School of Medicine, where he has practiced more than 31 years. Pet. Ex. 10 at 1. After receiving his M.D. from University of Miami School of Medicine, he completed his internship and residency in US Army Medical Centers, at Walter Reed Army Medical Center in Washington, D.C. and Letterman Army Medical Center in San Francisco, California. Pet. Ex. 11 at 1. He achieved the rank of Major in 1985, and he served in the US Army Medical Corp from 1979 until 1994. Id. at 2-3. Dr. Martinez-Arizala is board certified in neurology, neurorehabilitation, and spinal cord injury medicine. Id. at 2. He is a member of various professional societies and has extensively published, presented, and lectured on neurological disorders and spinal cord injuries. Id. at 3-12.

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[25] Latent autoimmune diabetes "is a variant [of diabetes] that develops in adulthood in which one or more autoantibodies is present. It is more slowly progressive than classic type [I] diabetes . . . [and] is commonly misdiagnosed as type [II] diabetes due to its slower progression and overlapping clinical features." Erika F. Brutsaert, Overview of Diabetes Mellitus, Merck Manual, https://www.merckmanuals.com/professional/endocrine-and-metabolic-disorders/diabetes-mellitus-and-hypoglycemia/overview-of-diabetes-mellitus (last visited Feb. 9, 2026).

[26] Petitioner submitted two expert reports from Dr. Martinez-Arizala. Pet. Exs. 10, 23.

He has also received numerous honors and awards. Id. at 11-12. Dr. Martinez-Arizala was Chief of the Spinal Cord Injury service at the Miami Veteran's Affairs Health Care system from 2003 to 2016, where he remained a staff physician until 2019. Id. at 2. Since 2019, he has engaged in a full-time practice in the University of Miami Department of Neurology as Chief of the Spine Division. Id. at 1.

b. Opinions

i. Diagnosis

Dr. Martinez-Arizala opined that Petitioner's diagnosis is downbeat nystagmus and autoimmune cerebellar visual syndrome, which was caused by the flu vaccine she received on December 8, 2015. Pet. Ex. 10 at 4. Dr. Martinez-Arizala noted the presence of GAD65 antibodies and opined these GAD65 antibodies "implicated her disease was related to an autoimmune process." Id.

ii. Althen Prong One

Dr. Martinez-Arizala opined that the causal theory connecting Petitioner's injury and vaccination was "based on molecular mimicry where components of the vaccine resemble antigens in the peripheral nerve and trigger the inflammatory response that damaged the cerebellar pathways." Pet. Ex. 10 at 6. He did not explain how homology with antigens in peripheral nerves could induce damage to cerebellar pathways. See id. at 4-6.

Several papers referenced by Dr. Martinez-Arizala describe the theory of molecular mimicry. For example, Segal and Shoenfeld[27] explained that "[m]olecular mimicry refers to a significant similarity between certain pathogenic elements contained in the vaccine and specific human proteins . . . [which] may lead to immune cross reactivity . . . causing autoimmune disease." Pet. Ex. 12 at 1.

To explain how the flu vaccine can cause downbeat nystagmus and autoimmune cerebellar visual syndrome, Dr. Martinez-Arizala stated that in this case, testing revealed "vestibular dysfunction was from a central deficit." Pet. Ex. 10 at 4. He further explained that the "development of an autoimmune disorder that affects the nervous system after vaccination is based on the concept of molecular mimicry, where components of the vaccine possess sequence similarities [with] . . . specific human proteins that results in the cross-activation of autoreactive T or B cells." Id. This cross reactivity "causes a reaction of the immune system against the pathogenic antigens that may harm the similar human proteins, [] causing autoimmune disease." Id.

[27] Yahel Segal & Yehuda Shoenfeld, Vaccine-Induced Autoimmunity: The Role of Molecular Mimicry and Immune Crossreaction, 15 Cell. & Mol. Immunol. 586 (2018).

Dr. Martinez-Arizala cited an article by Kanduc[28] describing the similarity of proteins between viruses and bacteria and humans, along with the role of cross-reactions which can cause autoimmune diseases. Pet. Ex. 10 at 4-5 (citing Pet. Ex. 14 at 3). Dr. Martinez-Arizala gave the example of encephalitis cases responsive to steroids, which have been reported after flu vaccination, as evidence that Petitioner's condition is autoimmune since she also was also responsive to steroids. Id. at 5 (citing Pet. Ex. 17).[29] And Dr. Martinez-Arizala noted reports of encephalitis following vaccination. Id. (citing Pet. Ex. 16).[30]

Next, Dr. Martinez-Arizala referenced case reports of acute cerebellitis[31] after flu vaccination. Pet. Ex. 10 at 5. Willi et al.[32] described a four-year-old child vaccinated against H1N1, who developed fever, seizure, and encephalopathy following vaccination. Pet. Ex. 15 at 1. The child developed a fever the day after vaccination and 40 hours after vaccination suffered a seizure, followed by another seizure the following day. Id. The child received treatment with antibiotics and immunoglobulin. Id. at 2. By nine months, the child was much improved but had continued deficits in motor and language development. Id. While sepsis was thought to be a possible cause, the authors agreed the diagnosis was vaccine-induced encephalitis following a thorough workup. Id. The authors did not discuss a causal mechanism.

Another case of post-vaccination acute cerebellitis was reported by Park et al.[33] in a 66-year-old man following flu vaccination. Pet. Ex. 19 at 1. The patient presented with "limb and gait ataxia" three weeks after vaccination. Id. Brain MRI showed no abnormality, however, computed tomography ("CT") showed "markedly cerebellar asymmetry, suggesting hypoperfusion in the right cerebellum." Id. Although the authors explained the cause of acute cerebellitis "remains uncertain," the "direct invasion of an etiologic agent or autoimmune demyelinating process[es]" have "been suggested." Id. at 2. Although a "definite etiology" was not found, the authors suggested vaccination as a cause of the patient's acute cerebellitis due to the temporal association between vaccination and onset. Id. at 3. The authors described the

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[28] Darja Kanduc, Peptide Cross-Reactivity: The Original Sin of Vaccines, 4 Front. Biosci. 1393 (2012).

[29] Recai Turkoglu & Erdem Tuzan, Brainstem Encephalitis Following Vaccination: Favorable Response to Steroid Treatment, 27 Vaccine 7253 (2009).

[30] Kengo Maeda & Ryo Idehara, Acute Disseminated Encephalomyelitis Following 2009 H1N1 Influenza Vaccination, 51 Intern. Med. 1931 (2012).

[31] Cerebellitis is "inflammation of the cerebellum." Cerebellitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=8722 (last visited Jan. 23, 2026).

[32] Bettina Willi et al., Encephalitis After Vaccination Against H1N1 Influenza Virus, 15 Eur. J. Paediatr. Neurol. 279 (2011).

[33] Kang Park et al., An Elderly Case of Acute Cerebellitis After Alleged Vaccination, 5 J. Mov. Disord. 21 (2012).

patient's finding of hypoperfusion in the cerebellum, however, they did not explain how vaccination could lead to such hypoperfusion. See id. Molecular mimicry was not discussed.

Dr. Martinez-Arizala also cited to a paper by Lessa et al.[34] describing neurological complications in four patients following H1N1 flu vaccination. Pet. Ex. 10 at 5 (citing Pet. Ex. 21). The four cases included cerebellar ataxia and hydrocephalus occurring 12 days post-vaccination, facial palsy nine days after vaccination, altered mental status with abnormal findings in the right fronto-parietal on brain MRI with onset 15 days after vaccination, and myelitis with C6-7 spinal cord expansion with symptoms beginning four days post-vaccination. Pet. Ex. 21 at 2-3. There was very limited discussion of causal mechanisms. See id. at 3-4.

Lastly, Dr. Martinez-Arizala cited to a case report about bilateral deafness with onset occurring two days after flu vaccination authored by Kolarov et al.[35] Pet. Ex. 10 at 5 (citing Pet. Ex. 22). The authors concluded that the patient's bilateral deafness "speculatively may be related to [flu] vaccination." Pet. Ex. 22 at 2. The authors did not discuss molecular mimicry as a causal mechanism. See id. at 1-2.

Additional articles related to molecular mimicry were filed but not discussed by Dr. Martinez-Arizala. A paper about autoimmune diseases authored by Steinman[36] provided an overview of autoimmune illnesses and molecular mimicry. Pet. Ex. 31 at 3. The focus of the paper was multiple sclerosis, and the visual problems at issue here were not discussed. See id. at 1-3. Gautam et al.[37] discussed the structural requirements for amino acid sequences or peptides to induce experimental autoimmune encephalomyelitis. See Pet. Ex. 32 at 1. While this is an important paper describing induction of disease in animals which is used to study human disease, it did not discuss the condition at issue here nor vaccine-induced autoimmunity. The same is true of the paper by Fujinami and Oldstone[38] which explained their studies of cross-reactivity between viruses and hosts that implicated molecular mimicry. See Pet. Ex. 33 at 1.

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[34] Ronaldo Lessa et al., Neurological Complications After H1N1 Influenza Vaccination: Magnetic Resonance Imaging Findings, 72 Arq. Neuropsiquiatr. 496 (2014). This article was also filed as Petitioner's Exhibit 24.

[35] Claudia Kolarov et al., Bilateral Deafness Two Days Following Influenza Vaccination: A Case Report, 15 Hum. Vaccin. & Immunother. 107 (2019).

[36] Lawerence Steinman, Autoimmune Disease, 296 Sci. Am. 106 (1993).

[37] Anand M. Gautam et al., A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis, 176 J. Exp. Med. 605 (1992).

[38] Robert S. Fujinami & Michael B. Oldstone, Amino Acid Homology Between the Encephalitogenic Site of Myelin Basic Protein and Virus: Mechanism for Autoimmunity, 230 Science 1043 (1985).

Two articles filed by Petitioner, but not discussed by Dr. Martinez-Arizala, described case reports of cerebellitis associated with flu infections, not vaccinations. The first of these is a case report by Sfeir and Najem[39] about a woman who developed slurred speech and ataxia after being ill for four days with fever, fatigue, headache, cough, and vomiting. Pet. Ex. 38 at 1. Brain MRI was abnormal with enlargement of the cerebellar hemispheres and other findings. Id. at 2. Additional diagnostic testing revealed positive nasopharyngeal polymerase chain reaction ("PCR") testing for H1N1 flu. Id. Pathogenesis was described as a direct infection of the central nervous system ("CNS") by the flu virus or "more typically, a delayed autoimmune demyelinating post-viral encephalopathy." Id.

The second case report was by Hackett et al.[40] and described a six-year-old who had been ill two weeks before onset with cough and fever (viral upper respiratory tract infection). Pet. Ex. 39 at 1. She presented with acute headache, dysarthria, and ataxia. Id. Examination revealed abnormal coordination, truncal ataxia, and sustained horizontal nystagmus. Id. at 1. Testing for both flu A and B was positive. Id. Her MRI was consistent with cerebellitis. Id. She was treated with an antiviral and her symptoms resolved after one week. Id.

The other articles filed by Petitioner but not referenced by Dr. Martinez-Arizala discussed post-vaccination encephalomyelitis.[41] The first is a case report from Shoamanesh and Traboulsee[42] describing a case of acute disseminated encephalomyelitis ("ADEM")[43] after the flu vaccination. Pet. Ex. 34 at 1. MRI of the brain and spinal cord showed significant abnormalities, including brainstem involvement and lesions of the entire spinal cord resulting in quadriplegia. Id. at 1-2. The authors noted that the flu vaccine may retain their "natural occurring antigens . . . . [which] could potentially serve as epitopes, eliciting autoimmunity" like live virus infections. Id. However, the authors did not discuss the condition at issue here. Id. The authors identified reported cases of ADEM from the literature from 1982 until 2011, noting

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[39] Maroun M. Sfeir & Catherine E. Najem, Cerebellitis Associated with Influenza A(H1N1)pdm09, United States, 2013, 20 Emerg. Infect. Dis. 1578 (2014).

[40] I. Hackett et al., Acute Cerebellitis Associated with Dual Influenza A (H1N1) and B Infection, 106 Ir. Med. J. 87 (2013).

[41] Encephalomyelitis is "inflammation involving both the brain and the spinal cord." Encephalomyelitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=8722 (last visited Jan. 29, 2026).

[42] A. Shoamanesh & A. Traboulsee, Acute Disseminated Encephalomyelitis Following Influenza Vaccination, 29 Vaccine 8182 (2011).

[43] ADEM is "an acute or subacute encephalomyelitis . . . characterized by . . . demyelination" that "occurs most often after an acute viral infection . . . and is believed to be a manifestation of an autoimmune attack on the myelin of the [CNS]." Acute Disseminated Encephalomyelitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=73033 (last visited Jan. 29, 2026).

onset ranged from two days (confusion and fluctuating level of consciousness) to 33 days. Id. at 3 tbl.1. An onset of one day was not identified. See id. Another case report of ADEM described a young woman with onset five days after receipt of the H1N1 flu vaccination. Pet. Ex. 36 at 1.[44] The authors concluded the cause "might have been related" to vaccination. Id.

Finally, an article about ADEM from Huynh et al.[45] explained that the "presumptive mechanism [of ADEM] is immune-mediated demyelination although immune-complex mediated vasculopathy has also been postulated." Pet. Ex. 35 at 1-2. The authors describe molecular mimicry, explaining that because there is homology (similarity) between proteins in the vaccine and host proteins, the "pathogen is not recognized as 'foreign' for elimination, nor 'self' for immune tolerance." Id. at 4. "The local reactivation by antigen presenting cells subsequently culminates in a destructive autoimmune process in the CNS." Id. In Huynh et al., the authors noted that "[d]epending on the inciting agent, the onset of symptoms may vary slightly" with onset ranging "from [one] to 14 days with non-neural vaccines." Id. at 5. The authors did not provide further discussion of onset; however, they did provide a "illustrative case report" of a 61-year-old male presenting with symptoms (bilateral visual blurriness and eye pain) three weeks after a flu vaccine. Id. at 7.

iii. Althen Prong Two

Dr. Martinez-Arizala opined components of the flu vaccine "contain a protein that is cross-reactive with peripheral nerve components." Pet. Ex. 10 at 6. He did not identify or describe these components in the flu vaccination administered to Petitioner nor identify what components of the peripheral nerve were involved. See id. He did not explain what peripheral nerve was involved in Petitioner's illness or how molecular mimicry affecting a peripheral nerve component would affect Petitioner's cerebellar pathways. See id.

Next, Dr. Martinez-Arizala explained there was no evidence that Petitioner had a neurological condition prior to her flu vaccination. Pet. Ex. 10 at 5. He reviewed Petitioner's medical records and noted the "contemporaneous medical records are consistent in pinning the start of her neurological symptoms after the vaccination." Id.

Lastly, he opined that Petitioner had a thorough diagnostic workup and there was no infection, illness, or other condition to explain her autoimmune cerebellar visual syndrome other than vaccination. Pet. Ex. 10 at 5. He agreed that Petitioner had "congestion, post nasal drip, coryza, sinus pressure, [and] otalgia for [two] weeks" which was diagnosed as sinusitis on December 29, 2015. Pet. Ex. 23 at 1. However, he opined "uncomplicated sinusitis . . . should not have caused visual disturbances." Id. He concluded that Petitioner's "sinusitis was not related to her downbeat nystagmus and autoimmune cerebellar syndrome." Id.

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[44] Isabelle Van Ussel et al., Encephalitis Related to a H1N1 Vaccination: Case Report and Review of the Literature, 124 Clin. Neurol. & Neurosurg. 8 (2014).

[45] William Huynh et al., Post-Vaccination Encephalomyelitis: Literature Review and Illustrative Case, 15 J. Clin. Neurosci. 1315 (2008).

iv. Althen Prong Three

Dr. Martinez-Arizala agreed "that the time of onset was atypical," but opined "this does not totally exclude [vaccination] as the cause." Pet. Ex. 23 at 1. Dr. Martinez-Arizala acknowledged that the flu vaccine was administered on December 8, 2015, and that onset of Petitioner's neurological symptoms began the "following day and progressed for several weeks." Pet. Ex. 10 at 6. He noted "[i]n neurological disorders the onset of symptom can precede the finding of abnormalities in the neurological examination." Pet. Ex. 23 at 1. Dr. Martinez-Arizala also opined the onset "was well within the expected time boundaries of the vaccine administration." Pet. Ex. 10 at 5. He noted that both Dr. Chung and Dr. Kinsella documented Petitioner's symptoms began after vaccination and "progressed for weeks following the vaccination," which was "quite consistent with the literature" he cited. Id. Finally, Dr. Martinez-Arizala asserted the paper by Lessa et al., where neurological complications were reported four days after flu vaccination, supported his opinion that Petitioner's onset (the day following vaccination) implicates the flu vaccine. Pet. Ex. 23 at 1-2 (citing Pet. Ex. 21).

2. Respondent's Expert, Dr. Marc A. Bouffard[46]

a. Background and Qualifications

Dr. Bouffard is an Assistant Professor of Neurology at the Harvard Medical School. Resp. Ex. I at 1. He is a staff physician at Massachusetts General Brigham and Beth Israel Deaconess Medical Center and a neuro-ophthalmologist at Massachusetts Eye and Ear. Id. He obtained his M.D. from Tufts University School of Medicine, completed an internship in internal medicine, a residency in neurology, followed by a neuro-ophthalmology fellowship at Massachusetts Eye and Ear, as well as a fellowship in Advanced General and Autoimmune Neurology at Massachusetts General Hospital. Resp. Ex. A at 1. He has experience in the diagnosis and treatment of downbeat nystagmus in patients with GAD65 antibodies. Id. In addition to his clinical practice, he has been involved in research projects and teaches residents, clinical fellows, and research fellows. Resp. Ex. I at 3-7. Dr. Bouffard has authored or co-authored a number of publications. Id. at 7-10. He is a member of several professional societies and an ad hoc reviewer for several journals. Id. at 1-2.

b. Opinions

i. Diagnosis

Dr. Bouffard opined that Petitioner has two neurological problems, downbeat nystagmus and skew deviation. Resp. Ex. A at 3.

Dr. Bouffard explained nystagmus is "characterized by abnormal eye movements away from the eye position at which the eyes would be directed." Resp. Ex. A at 3. Petitioner had "downbeat nystagmus" where the "eyes unintentionally draft up and jerk or 'beat' down; this

[46] Respondent submitted two expert reports from Dr. Bouffard. Resp. Exs. A, E.

cycle continues incessantly[,] though in some positions of gaze it may be more or less conspicuous" as noted by her treating providers. Id. Given that the retina in the center of vision is "continuously in motion," patients with nystagmus may experience blurry vision or that the target of their vision is "'bouncing' up and down." Id. This bouncing effect is called oscillopsia. Id. Dr. Bouffard explained that "[d]ownbeat nystagmus reflects disruption of a complex feedback circuit within the cerebellum and brainstem that keeps the eyes in the neutral/staring ahead position."[47] Id.

According to Dr. Bouffard, the most common causes of downbeat nystagmus include "stroke, hemorrhage, tumor, [and] demyelinating lesions" and less common causes are "infectious encephalitis, autoimmune encephalitis, and nutritional deficiency." Resp. Ex. A at 3 (citing Resp. Ex. A, Tab 2).[48] In Wagner et al., the authors identify additional causes of nystagmus such as "[c]raniocervical malformations, cerebellar degeneration, vascular pathology, inflammatory disease[,] and intoxication with lithium or antiepileptic drugs." Resp. Ex. A, Tab 2 at 1. Wagner et al. studied the etiology of downbeat nystagmus in 117 patients and found that most common identifiable cause was "cerebellar degenerative disease," accounting for 20% of the patients studied, followed by vascular lesions and craniocervical malformations. Id. at 2. In 45 of the patients studied (38%), no cause was found. Id.

Dr. Bouffard opined that here, the cause of Petitioner's condition was the GAD65 autoantibody. Resp. Ex. A at 3. Petitioner was positive for the GAD65 antibody with "an extremely high titer" thought to be pathogenic by her neuro-ophthalmologist. Id.; Resp. Ex. E at 2 (citing Pet. Ex. 9 at 15). He explained that as GAD65 is "a sporadic autoimmune condition, no other cause or contributing factor needs to be involved to account for [P]etitioner's findings." Resp. Ex. A at 4.

Next, Dr. Bouffard opined that Petitioner's second problem is skew deviation, a "supranuclear vertical ocular misalignment" caused by dysfunction of nerve fibers ("or their relays within the brain") that control the muscles which move the eye and the vestibulo-ocular reflex that maintains "stable ocular position despite head movements." Resp. Ex. A at 3-4. Dr. Bouffard explained that "skew deviations, like downbeat nystagmus, often reflect cerebellar damage." Id. at 4. He further noted downbeat nystagmus and skew deviation often occur together due to the proximity of the nerve fibers that serve these systems. Id. Finally, Dr. Bouffard opined "GAD65-associated autoimmunity targeting the cerebellum is a well-recognized cause of downbeat nystagmus and skew deviation." Resp. Ex. E at 2.

ii. Althen Prong One

Dr. Bouffard opined that the GAD65 autoantibody is the cause of Petitioner's condition and "there is no evidence to suggest that this antibody is generate by the flu vaccine." Resp. Ex.

[47] For more information, see Resp. Ex. A, Tabs 1 and 6 (Shin C. Beh et al., Cerebellar Control of Eye Movements, 37 J. Neuroophthalmol. 87 (2017)).

[48] J.N. Wagner et al., Downbeat Nystagmus: Aetiology and Comorbidity in 117 Patients, 79 J. Neurol. Neurosurg. & Psychiatry 672 (2008).

E at 2. He disagreed that the flu vaccine can cause downbeat nystagmus and skew deviation for two reasons—there is a lack of knowledge related to molecular mimicry and no evidence of homology as well as a lack of epidemiological or "real world" evidence. Id.; Resp. Ex. A at 4.

Dr. Bouffard opined Petitioner offered no evidence of homology between the flu vaccine and the GAD65 antibody. Resp. Ex. A at 4. Regarding Petitioner's reliance on articles about ADEM, Dr. Bouffard acknowledged that a causal association has been questioned between vaccines and ADEM. Id. However, he found no basis to extrapolate findings about ADEM to GAD65 syndromes because the two conditions have "different clinical phenotypes, imaging findings, natural history, and optimal treatments." Id.

The other reason for Dr. Bouffard's opinion was the lack of epidemiological evidence, or "lack of any real-world evidence associating [the flu] vaccine with GAD65 autoimmunity." Resp. Ex. A at 4; Resp. Ex. E at 2.

iii. Althen Prongs Two and Three

Dr. Bouffard disagreed with Dr. Martinez-Arizala's opinion that Petitioner's vaccination played a role in the development of her downbeat nystagmus and skew deviation. Resp. Ex. A at 4. He opined that the GAD65 autoantibody is the cause of her condition, and "there is no evidence to suggest that this antibody is generated by the flu vaccine." Resp. Ex. E at 2. Given Petitioner's GAD65 antibody, "no other cause or contributing factor needs to be invoked to account" for her illness. Resp. Ex. A at 4.

Regarding timing, Dr. Bouffard noted there are several places in the medical record "explicitly stating that the symptoms which define [Petitioner's] neurological illness began within a day of vaccination." Resp. Ex. E at 1. These include:

- Dr. Chen's note recording onset as December 9, 2015. Pet. Ex. 3 at 3-5.
- Dr. Wallace's history stating that Petitioner had a flu shot on December 8, and "the next day she started having visual disturbances. She describe[d] it as blurry vision, double vision, a feeling that her eyes were jumping." Pet. Ex. 4 at 7.
- Dr. Kinsella's note stating Petitioner received a flu shot on December 8, 2015, and "when she woke up the next day she had [double vision]." Pet. Ex. 8 at 3.
- Dr. Chung writing Petitioner "had a flu shot on [December 8, 2015] and the next day she woke up with binocular double vision in left gaze." Pet. Ex. 9 at 2.

Id. at 1-2.

Dr. Bouffard opined that this "extremely short interval" between vaccine exposure and onset of disease "undermines the applicability of a theory of molecular mimicry." Resp. Ex. A at 5. He opined that "molecular mimicry requires at least several days for an exposure to generate a relevant immune response." Id. at 4. Here, the interval between "exposure and outcome" based on onset the day following vaccination was "unusually brief." Id.

In support of his opinion, Dr. Bouffard cited Karussis and Petrou,[49] who noted that the mean duration between vaccination and onset of CNS demyelination was 14.2 days. Resp. Ex. A at 4 (citing Resp. Ex. A, Tab 7 at 2). Dr. Bouffard added that Karussis and Petrou stated that onset would not occur earlier than a "few days" after vaccination.[50] Id. (quoting Resp. Ex. A, Tab 7 at 2).

In addition to the medical literature cited, Dr. Bouffard referenced statements by Petitioner's treating physicians to the effect that onset here was too short to support vaccine causation. Resp. Ex. A at 4. Dr. Bouffard cited a note by Dr. Kinsella that stated, "it is unclear how it [nystagmus, skew] is related to the flu shot since symptoms were immediately following." Id. (quoting Pet. Ex. 8 at 15). Similarly, Dr. Chung noted that although flu vaccinations have been associated with neurological complications, "the usual time of onset is [one to four] weeks and it would be highly unusual to have onset the following day given that it takes time to develop an autoimmune response." Id. (quoting Pet. Ex. 9 at 9).

3. Respondent's Expert, Dr. James N. Moy[51]

a. Background and Qualifications

Dr. Moy is an Associate Professor of Immunology/Microbiology as well as the Vice-Chair for Research for the Department of Internal Medicine at Rush University Medical Center. Resp. Ex. C at 1; Resp. Ex. H at 2. He received his M.D. from University of Illinois. Resp. Ex. C at 1; Resp. Ex. H at 1. He completed a residency in pediatrics at the University of Minnesota followed by a fellowship in allergy/immunology at Rush-Presbyterian-St. Luke's Medical Center. Resp. Ex. H at 1. Dr. Moy is board certified in allergy and immunology as well as pediatrics. Id. at 2. In his clinical practice, he has "evaluated and managed over 300 pediatric, adolescent and young adult patients with autoimmune diseases and disorders." Resp. Ex. C at 1. He has published on topics of "allergic reactions, medication side-effects[,] and immunodeficiency diseases." Id.; see Resp. Ex. H at 8-14. Dr. Moy is a member of various professional societies and committees, and he has served on the manuscript review board of several journals. Resp. Ex. H at 4.

[49] Dimitrios Karussis & Panayiota Petrou, The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes, 13 Autoimmun. Rev. 215 (2014).

[50] Karussis and Petrou provide a list of cases reports of post-vaccination demyelinating syndromes. Resp. Ex. A, Tab 7 at 4-5 tbl. 2. The range of onset was three days to five months (with the exception of three cases of optic neuritis involving the measles and/or rubella vaccines, which contain live viruses, where onset was noted to within a few hours). Id.

[51] Respondent submitted two expert reports from Dr. Moy. Resp. Exs. C, F.

b. Opinions

Dr. Moy deferred to the experts in neurology and ophthalmology regarding Petitioner's diagnosis. Resp. Ex. C at 5. He limited his opinion to whether the flu vaccine Petitioner received on December 12, 2015 "caused her to develop an autoimmune disease(s)." Id.

i. Althen Prong One

Dr. Moy opined that Petitioner's expert, Dr. Martinez-Arizala, did not offer a persuasive causal mechanism to explain how the flu vaccine could cause Petitioner's neurological symptoms. Resp. Ex. C at 7. While Dr. Moy generally agreed that molecular mimicry is a relevant theory that explains how infections can cause autoimmune diseases, he did not believe the theory of molecular mimicry can be extended to scenarios involving vaccination. Id. at 6-7. He explained that infection and vaccination "are by no means equal." Id. at 6. Infections cause "extensive damage to the human cells that are infected." Id. When the infected cells undergo cell death (apoptosis), "antigens inside the cells are released, including possible self-antigens." Id. This process can lead to "an amplified response" by host T and B cells and activate "self-reactive T cells" in those who are genetically predisposed to develop autoimmune illnesses. Id. at 7; see also Resp. Ex. C, Tab 3 at 1.[52] Dr. Moy explained this infectious process contrasts with vaccines because the antigens in vaccines "do not cause damage to human cells." Resp. Ex. C at 7. Since vaccination does not result in damaged infectious cells, there is no release of self-antigens from these injured cells. Id.

Moreover, Dr. Moy noted that Petitioner's expert opined that the flu vaccine contains "components that triggered the autoimmune cerebellar visual syndrome." Resp. Ex. C at 5 (citing Pet. Ex. 10 at 5). However, these "components" were not identified. Id. Dr. Moy explained that the flu virus "contains eight genes that encode 11 different proteins," yet Petitioner's expert did not identify any specific components suspected of causing Petitioner's illness. Id. Dr. Moy opined that the specific components should be identified. Id.

In conclusion, Dr. Moy opined that Petitioner's expert did not offer "a persuasive biological mechanism to show how the [flu] vaccine could have caused [Petitioner's] eye and neurological symptoms." Resp. Ex. C at 7.

ii. Althen Prong Two

Regarding the finding that Petitioner had GAD65 antibodies, Dr. Moy explained that these antibodies are "found in type I diabetes and type II adult diabetes." Resp. Ex. C at 5. Additionally, Petitioner had IA-2 antibodies and insulin auto-antibodies which are also present in individuals with diabetes. Id. Further, in June 2017, Petitioner's hemoglobin A1C was 12.1 (normal range < 6.4) and blood glucose was 404 mg/dL (normal range < 107 mg/dL). Id. She was diagnosed with adult-onset diabetes (type II) and prescribed metformin. Id. Based on these

[52] Connie C. Qiu et al., Triggers of Autoimmunity: The Role of Bacterial Infections in the Extracellular Exposure of Lupus Nuclear Autoantigens, 10 Front. Immunol. 2608 (2019).

facts, Dr. Moy opined that the flu vaccine "had nothing to do with" the development of Petitioner's GAD65 autoantibodies; instead, Petitioner's GAD65 antibodies were "associated with her diabetes." Id.

In support of this opinion, Dr. Moy cited a study published by Koopman et al. describing the associated between GAD65 and adults with type II diabetes. Resp. Ex. C at 5 (citing Resp. Ex. C, Tab 1). Koopman et al. found "a strong association was observed between GAD65 antibody positivity" and type II diabetes mellitus in a non-diabetic adult population. Resp. Ex. C, Tab 1 at 6. Although the presence of GAD65 antibodies had previously been shown to be "strongly associated" with type I diabetes, this finding had not been extended to type II adult-onset diabetes. Id. Koopman et al. showed GAD65 antibody seropositive study participants had a three-fold increased risk of developing type II diabetes when compared to controls who did not have GAD65 antibodies. Id.

An additional article filed by Respondent, but not cited by Dr. Moy, discussed GAD65 antibodies in the context of autoimmune vestibulocerebellar syndromes. Resp. Ex. G at 1, 6, 10-11.[53] The authors explained GAD65 antibodies account for 35% of patients with autoimmune cerebellar ataxia. Id. at 10. About 80 to 90 percent of cases occurred in women, with onset usually in 50- and 60-year-olds. Id. "Oculomotor abnormalities are [] characteristic in this disorder," and may include downbeat nystagmus. Id. The pathogenesis of the condition is thought to involve "disruption of [gamma-aminobutyric acid] transmission in the vestibulocerebellar pathways." Id. at 11. While "[a]utopsy studies and animal experiments suggest a cytotoxic T-cell mediated-phenomenon," the causal mechanism has not been identified. Id.

iii. Althen Prong Three

Dr. Moy opined that onset of double vision the day after vaccination "is not consistent with autoantibodies induced by the [flu] vaccine since antibodies to the [flu] vaccine are not elevated until five days after vaccination." Resp. Ex. C at 7.

In support of his opinion, Dr. Moy cited a study done by Sun et al.[54] describing the timeline for immune protection following vaccination with the 2009 A/H1N1 flu vaccine. Resp. Ex. C at 7 (citing Resp. Ex. C, Tab 4).[55] In Sun et al., blood serum was sampled from volunteers at days three, five, 10, 14, and so forth, to determine when vaccinees showed an antibody

[53] Ram N. Narayan et al., Autoimmune Vestibulocerebellar Syndromes, 40 Semin. Neurol. 97 (2020).

[54] Yizhuo Sun et al., Immune Protection Induced on Day 10 Following Administration of the 2009 A/H1N1 Pandemic Influenza Vaccine, 5 PLoS ONE e14270 (2010).

[55] Sun et al. was identified as Resp. Ex. C, Tab 4 in Respondent's notice of filing; however, the document is incorrectly labeled as Resp. Ex. C-1. The undersigned will refer to the article as Resp. Ex. C, Tab 4.

response sufficient to provide protection against infection.[56] Resp. Ex. C, Tab 4 at 1. The research showed that significant antibody production did not began to occur until five days post-vaccination. Id. at 3, 4 fig. 1. By day 10, the rates of antibody production reached levels sufficient to provide protection against infection. Id. at 3.

Responding to Dr. Martinez-Arizala's reliance on Lessa et al., Dr. Moy noted that the four cases presented had onset time frames of four to 15 days after vaccination. Resp. Ex. F at 1 (citing Pet. Ex. 21). Dr. Moy also noted that Lessa et al. also discussed pathogenesis, stating that while the cause is not known, post-vaccination neurological conditions are thought to be due to "immune-mediated alteration resulting in the production of antibodies." Id. (quoting Pet. Ex. 21 at 3-4). Citing the study by Sun et al., Dr. Moy again noted that the production of any significant number of antibodies takes about ten days after vaccination. Id. at 2. Dr. Moy opined that one day was "too soon for a significant number of antibodies to be produced." Id.

IV. DISCUSSION

A. Standards for Adjudication

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

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

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

[56] Seroprotection was defined as hemagglutination inhibition ("HI") titer of 1:40 or more. Resp. Ex. C, Tab 4 at 3.

unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

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

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

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

testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

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

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

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

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

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and

noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

V. ANALYSIS

A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury[,]" determining facts relating to the claimed injury can be significant in a case where diagnosis is not clear. Id. Here, the parties dispute the scope of Petitioner's diagnosis, and so it is appropriate to first resolve that issue.

The undersigned finds that Petitioner's diagnosis is downbeat nystagmus and skew deviation associated with GAD65 antibodies, "indicative of an autoimmune process, limited to the visual cerebellar pathways, sparing limb and axial cerebellar pathways." Pet. Ex. 8 at 15. This finding is based on the records of Dr. Chung, Dr. Kinsella, as well as the parties' experts, who agree on diagnosis. The parties also "agree[d] she has downbeat nystagmus" in their joint submission. Joint Sub. at 1. With regard to the "scope" of Petitioner's diagnosis, the experts also agree that Petitioner's condition is characterized by GAD65 antibodies and is autoimmune in nature. See, e.g., Pet. Ex. 10 at 4 (noting the presence of GAD65 antibodies "implicated her disease was related to an autoimmune process"); Resp. Ex. E at 2 (recognizing "GAD65-associated autoimmunity" as a cause of both downbeat nystagmus and skew deviation).

B. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support her 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, 618 F.3d at 1347 ("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))).

Petitioner's expert, Dr. Martinez-Arizala, offers the causal theory of molecular mimicry to explain how the flu vaccine can cause GAD65 antibody autoimmune associated nystagmus and skew deviation, suggesting that vaccine components have homology with antigens "in the peripheral nerve" that triggered an inflammatory response that damaged cerebellar pathways. Pet. Ex. 10 at 6. Respondent's expert Dr. Bouffard did not generally dispute the theory of molecular mimicry, or that it was a sound and reliable mechanism to explain an autoimmune cerebellar injury. Instead, Dr. Bouffard questioned the application of molecular mimicry in this context, where there was no evidence of homology and a lack of evidence that the flu vaccination has been associated with GAD65 autoimmunity. Respondent's immunologist, Dr. Moy, agreed that molecular mimicry was a relevant theory to explain how infections can cause autoimmune illnesses, but he did not agree it explains how a vaccine can cause autoimmune diseases. Additionally, like Dr. Bouffard, Dr. Moy disagreed there was evidence that the flu vaccine contained any proteins that could cause the injury in question via molecular mimicry.

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

While the undersigned is not making a finding as to whether Petitioner has provided preponderant evidence of Althen prong one, the undersigned notes that a one-day onset is not compatible with the theory of molecular mimicry. See, e.g., Forrest v. Sec'y of Health & Hum. Servs., No. 14-1046V, 2019 WL 925495, at *6, *8 (Fed. Cl. Spec. Mstr. Jan. 28, 2019) (finding "a preponderance of the evidence shows that molecular mimicry is not likely to happen within 36 hours, even for a recall response"); Martinez v. Sec'y of Health & Human Servs., No. 16-736V, 2022 WL 4884923, at *27 (Fed. Cl. Spec. Mstr. Sept. 9, 2022) (finding "a relative short onset" of one day "not medically acceptable" in an "an autoimmune reaction involving antibodies or other immune cells associated with the adaptive[] [] immune response in reaction to antigenic exposure" (emphasis omitted)); see infra Part V.B.3 (analyzing Althen prong three).

2. Althen Prong Two

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

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

The undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause-and-effect showing the December 8, 2015 flu vaccine to be the cause of Petitioner's autoimmune condition. See Althen, 418 F.3d at 1278. There are several reasons for this finding.

First, in determining whether Petitioner has established a prima facia case, and as related to Althen prong two, the undersigned finds it relevant to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379. "In asserting an off-Table injury, [Petitioner] need[s] to show, by preponderant evidence, that his vaccination was a substantial factor" in causing her alleged vaccine injury. Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 962 (Fed. Cir. 2023). Petitioner "[does] not need to show that [she] did not suffer from an infection, or that said infection did not contribute to [her alleged injury]. Nor [does] [she] have to show that the vaccination was the only cause of [her] [vaccine-related illness]." Id.; see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding a petitioner does not bear the burden of eliminating alternative independent potential causes). Thus, the undersigned considers evidence relating to the association between GAD65 antibodies and Petitioner's alleged vaccine-related illness, as "[s]uch contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law." Winkler, 88 F.4th at 963; see also Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case.").

Petitioner's treating physicians, Dr. Chung and Dr. Kinsella, documented an association between GAD65 antibodies, type I diabetes, and cerebellar syndrome. Dr. Chung informed

Petitioner of her positive GAD65 antibodies and explained the association between these antibodies with type I diabetes and cerebellar syndrome. After learning Petitioner was positive for GAD65 and IA-2 antibodies, Dr. Kinsella's diagnosis was post-vaccination downbeat nystagmus, autoimmune cerebellar visual syndrome, and included that she was positive for GAD and IA-2 antibodies. In his records, Dr. Kinsella referenced a case report of cerebellar ataxia related to GAD antibodies and the relationship to diabetes. Petitioner's PCP initially diagnosed Petitioner with type II diabetes. She was later diagnosed with latent autoimmune diabetes (a variant of type I diabetes characterized by adult onset) by an endocrinologist who noted the presence of GAD antibodies and insulin autoantibodies.

Dr. Bouffard disagreed with Dr. Martinez-Arizala that Petitioner's vaccination played a role in the development of her illness and instead opined that the GAD65 autoantibody was causal. He further opined that there was no evidence that the flu vaccine induced the GAD65 antibodies. Given Petitioner's GAD65 antibodies, Dr. Bouffard opined that no other cause need be invoked as the cause of her illness.

Drs. Chung and Kinsella documented a relationship between the Petitioner's positive GAD antibodies and type I diabetes generally. Pet. Ex. 9 at 19 (informing Petitioner of the association between GAD65 antibodies and type I diabetes); Pet. Ex. 8 at 16 (discussing a case report that reported an association between autoimmune cerebellar ataxia related to GAD and noting GAD "typically affects women with late-onset type [I] diabetes or other auto-immune disorders."). An endocrinologist later diagnosed Petitioner with latent autoimmune diabetes, a variant of type I diabetes. Petitioner was specifically noted to have positive GAD antibodies.

Moreover, Dr. Moy provided evidence extending the association of GAD antibodies to type II diabetes, the variant of diabetes that Petitioner's PCP diagnosed. In support of his opinion, Dr. Moy cited Koopman et al. who reported a strong association between positive GAD65 antibodies and type II diabetes mellitus. In another paper, Narayan et al. found that GAD65 antibodies account for 35% of patients with autoimmune cerebellar ataxia, and of these, 80 to 90% of cases occurred in women, with onset usually in 50- and 60-year-olds. Narayan et al. further reported that oculomotor abnormalities are characteristic in the condition, which can include downbeat nystagmus. The pathogenesis of the condition is thought to involve "disruption of [gamma-aminobutyric acid] transmission in the vestibulocerebellar pathways." Resp. Ex. G at 11.

Regardless of whether Petitioner's diabetes was a type I or type II variant, the discussion of GAD65 antibodies by Petitioner's treating physicians, medical literature, and Respondent's experts' opinions provide evidence of other possible sources of Petitioner's injury.

In addition to the above evidence of another cause of Petitioner's condition, there are two more reasons there is not preponderant evidence of a logical sequence of cause and effect. These include the undersigned's finding that Petitioner's onset was too soon to implicate the flu vaccine and Petitioner's treating physicians' statements that do not support causation. Both reasons relate to the temporal association between vaccination and onset and are discussed below in the analysis of Althen prong three.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also 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. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358.

Petitioner received the flu vaccine at issue on December 8, 2015. The medical records consistently document that she reported that her visual abnormalities began the following day, December 9, 2015. Below are examples of some of these entries in the medical records:

- On December 16, 2015, Petitioner reported to Dr. Chen that she noticed visual disturbances on Wednesday, December 9. Pet. Ex. 3 at 3-5.
- In a questionnaire which appears to have been executed by Petitioner on March 28, 2016, she wrote that her problem first occurred December 9, 2015. Pet. Ex. 6 at 1, 5.
- Dr. Wallace's history dated April 4, 2016 documents that Petitioner reported she had a flu vaccine on December 8, 2015, and "the next day she started having visual disturbances. She describes it as blurry vision, double vision, a feeling that her eyes were jumping." Pet. Ex. 4 at 7.
- Dr. Kinsella's history on July 19, 2016 states that Petitioner received a flu vaccine on December 8, and "when she woke up the next day she had [double vision]." Pet. Ex. 8 at 3.
- When seen by Dr. Chung on September 13, 2016, Petitioner reported she "had a flu shot on [December 8, 2015] and the next day she woke up with binocular double vision in left gaze." Pet. Ex. 9 at 2.

While there is mention in the records that Petitioner's condition progressed from the date of onset, this does not change the fact that the first manifestation of visual abnormality which heralded the onset of her condition occurred on December 9, 2015. Petitioner did not offer evidence to suggest that progression of her autoimmune condition altered or negated December 9, 2015 as the onset date as it relates to the mechanism of molecular mimicry. As stated by Petitioner's expert, Dr. Martinez-Arizala, "[i]n neurological disorders the onset of symptom can precede the finding of abnormalities in the neurological examination." Pet. Ex. 23 at 1. This statement, however, does not explain how molecular mimicry could result in onset of double vision in the span of one day.

In summary, Petitioner consistently reported her history which was documented, also consistently, by four different physicians. She also completed a questionnaire that noted the

same history. All these records document onset on December 9, the day following vaccination. Thus, the undersigned finds by preponderant evidence that onset occurred December 9, 2015.

The next question is whether an onset of one day is an appropriate temporal interval given the proffered causal mechanism of molecular mimicry. The undersigned finds it is not.

Overall, the experts' opinions weigh in favor of a finding that one day is too soon for molecular mimicry to create a cross reaction resulting in autoantibodies that would attack host structures and cause injury. Petitioner's expert, Dr. Martinez-Arizala acknowledged that onset was "atypical." See Pet. Ex. 23 at 1. After acknowledging that onset was "atypical," Dr. Martinez-Arizala added that "this does not totally exclude [vaccination] as the cause." Id. However, Dr. Martinez-Arizala did not provide medical literature or other evidence to support a finding that molecular mimicry can occur within one day and cause injury manifesting as double vision.

Respondent's expert, Dr. Bouffard, opined that an onset of one day undermined the applicability of molecular mimicry, explaining that it requires several days for an exposure to generate a relevant immune response. He also cited Karussis and Petrou, who noted that the mean duration between vaccination and onset of demyelinating illnesses was 14.2 days.

Similarly, Dr. Moy agreed onset was too short to explain an autoimmune disease. He referenced the study by Sun et al. who studied how long it took for antibodies to be produced following the flu vaccine. The study showed that significant antibody numbers were not generated until five days post-vaccination. Moreover, the case reports described by Leesa et al. described neurological complications after H1N1 vaccination described onset periods of four days, nine days, 12 days, and 15 days. The authors stated that the pathogenesis of encephalitis after vaccination is thought to be immune-mediated "resulting in the production of antibodies." Pet. Ex. 24 at 3-4. This description suggests the theory of molecular mimicry.

In addition to the experts' opinions, the undersigned finds the weight of the evidence presented in the medical literature filed by the parties does not support causation within 24 hours of vaccination.

For example, Park et al. described a case of acute cerebellitis with onset three weeks after vaccination. The authors explained that although the mechanism was "uncertain," an autoimmune demyelinating process, like the process thought to cause ADEM, was suspected.[57] Pet. Ex. 19 at 2; see also Pet Ex. 39 (discussing cerebellitis onset four days following flu infection and noting pathogenesis was "typically, a delayed autoimmune demyelinating post viral encephalopathy").

Several papers describing case reports of ADEM were filed, with onsets of nine and 15 days. Turkoglu and Tuzun describe a 44-year-old male who developed left-sided hemiparesis,

[57] In Park et al., the authors also mentioned the theory of direct infection. That theory has not been offered here, and a theory of direct infection would not be appropriate given that the flu vaccine is not a live-virus vaccine.

with symptom onset nine days after vaccination. The suggested mechanism was CNS vasculitis, but the pathogenesis of the vasculitis was not identified. Maeda et al. described a 33-year-old who, 15 days after an H1N1 flu vaccination, developed numbness of her legs, which progressed to sensory loss. Brain and thoracic cord MRIs were abnormal showing hyperintense lesions consistent with demyelination. A review of published ADEM cases from 1982 to 2011, conducted by Shoamanesh and Traboulsee, reported onset ranged from two days to 33 days after vaccination.

One case report of encephalitis references a 40-hour onset in a five-year-old child who developed fever the day after vaccination, and the following night (40 hours after vaccination) suffered a seizure, followed by another seizure the following day. Several days post-vaccination, the child had an altered and "unstable mental state." Pet. Ex. 15 at 1. Differential diagnosis included post-vaccination ADEM, sepsis, and gastrointestinal etiologies. The clinical presentation of fever followed by seizure in a child makes this case quite different than the presentation here, and there was no discussion of causal mechanism in the article. Thus, the undersigned does not find that the article provides preponderant evidence that molecular mimicry could induce the onset of an autoimmune condition in one day.

A case report from Van Ussel et al. described an individual who developed ADEM five days after H1NI flu vaccination. The authors reviewed 21 other cases of CNS demyelination related to vaccination or infection with onset ranging from one day to one month. Of the reviewed cases, a single case involved the development of transverse myelitis ("TM") one day after vaccination. The authors did not provide further information on this case of TM with a one-day onset.

Likewise, Karussis and Petrou provided a list of cases reports of post-vaccination demyelinating syndromes. The range of onset was three days to five months with the exception of three cases of optic neuritis involving the measles and/or rubella vaccines, which contain live virus, where onset was noted to within a few hours. Petitioner did not receive a live virus vaccine.

The majority of the case reports describe onset periods of days to weeks, consistent with the expert opinions of Dr. Bouffard and Dr. Moy.

Petitioner contends the medical literature is supportive of a "rapid" initial onset. Pet. Mot. at 12. Specifically, Petitioner incorrectly asserts Lessa et al. reported a 24-hour onset[58] and notes several other articles that reference onset of neurological symptoms within one to two days of vaccination. Moreover, Petitioner argues that the medical literature disputes Respondent's

[58] This is incorrect. Lessa et al. report on four cases of neurological symptoms following vaccination with the earliest onset being four days post-vaccination. Case one in the article does reference "cerebellar ataxia, which developed over 24 hours;" however, the article clearly states that the patient had received a vaccination 12 days (rather than 24 hours) before the onset of symptoms. Pet. Ex. 24 at 2. The earliest onset of symptoms discussed in the article was four days after vaccination. Id. at 1.

contention that antibodies associated with an autoimmune response "typically take [five to 10] days to form."[59] Id. at 11, 11 n.4.

The undersigned disagrees with Petitioner's interpretation of the medical literature. As Respondent notes, of the thirty-nine articles filed by Petitioner, there are few reports of a one-day onset, and they are distinguishable from this case. As discussed above, the bulk of case reports describe onset periods of days to weeks consistent with the opinions of Respondent's experts. One of the articles relied on by Petitioner, Lessa et al., does not discuss a 24-hour onset. The other articles make passing reference to one-day onset for cases of ADEM or TM, conditions distinct from downbeat nystagmus and skew deviation and not at issue here. These references do not demonstrate that a one day onset is appropriate for downbeat nystagmus and skew deviation.

Additionally, in her brief, Petitioner also puts forward an inflammasome activation theory in support of a 24-hour onset. Pet. Mot. at 13. This theory was not developed by Petitioner's expert, nor did Petitioner provide medical literature to support an inflammasome theory.[60] Accordingly, Petitioner did not provide preponderant evidence that inflammasome activation could induce the onset of an autoimmune condition within one day of vaccination.

Further, the opinions of the treating physicians do not support vaccine causation, and two of the treating physicians specifically question the short temporal association.

Dr. Batchu stated that Petitioner had "possible post vaccinal encephalitis." Pet. Ex. 7 at 6, 23. 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 "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).

Dr. Kinsella documented his impression as "post-vaccination downbeat nystagmus." Pet. Ex. 8 at 6-7, 11-12, 25. He noted "[i]t is unclear how it is related to the flu shot since symptoms were immediately following." Id. at 15, 24. In the same visit documentation, he also wrote that he had spoken to Petitioner's lawyer, and that Petitioner's case was "c/w" (or consistent with) vaccine causation. Id. at 26. It is difficult to square these two statements. On one hand, Dr. Kinsella suggests he agrees that the vaccine may have been causal. On the other hand, he

[59] Petitioner also contends the "general knowledge of this experienced Court" supports her assertion that five-to-10-day antibody response is "incorrect." Pet. Mot. at 11 n.4. Petitioner provided no case law in support of this contention.

[60] Petitioner was given ample opportunity to develop this theory. Two months before the scheduled entitlement hearing, Petitioner asked to file an additional expert report from an immunologist addressing inflammasome activation. The undersigned noted that Petitioner had been on notice of the onset issues since her Rule 5 conference three years prior; nevertheless, she gave Petitioner the opportunity to cancel the entitlement hearing, obtain an expert report from an immunologist, and have the case resolved via a ruling on the record. Petitioner opted to not file an additional expert report and requested to proceed with the entitlement hearing. The entitlement hearing was later cancelled on unrelated grounds.

concludes that causation is unclear because onset was immediate. The statements seem contradictory.

Dr. Chung, a neuro-ophthalmologist, and thus the most specialized physician who evaluated Petitioner, wrote, "[w]hile flu vaccinations are associated with neurological sequela, the usual time of onset is [one to four] weeks. It would be highly unusual to have onset the following day given that it takes time to develop an autoimmune response." Pet. Ex. 9 at 9. This statement weighs against vaccine causation. Dr. Chung is clear in her opinion that an autoimmune response would usually not occur in one day.

Generally, treating physician statements are typically "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). However, no treating physician's views bind the special master, per se; rather, their views are carefully considered and evaluated. § 13(b)(1); Snyder ex. rel. Snyder v. Sec'y of Health & Hum. Servs., 88 Fed. Cl. 706, 746 n.67 (2009). "As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases." Welch v. Sec'y of Health & Hum. Servs., No. 18-494V, 2019 WL 3494360, at *8 (Fed. Cl. Spec. Mstr. July 2, 2019).

The undersigned finds Dr. Bachau's statement does not support causation due to his use of the word "possibility." Dr. Kinsella and Dr. Chung express doubt about causation based on the one-day onset. On the whole, this evidence, when combined with the expert opinions of Dr. Bouffard and Dr. Moy, weighs against a finding that a one-day onset is consistent with an autoimmune neurological injury.

Lastly, the undersigned finds that case law supports the finding that onset here was too soon given the proffered theory of molecular mimicry.

The undersigned has previously denied compensation in cases with 24-hour onset. See, e.g., True v. Sec'y of Health & Hum. Servs., No. 21-2110V, 2025 WL 1343027 (Fed. Cl. Spec. Mstr. Apr. 11, 2025); Brancheau v. Sec'y of Health & Hum. Servs., No. 21-1209V, 2024 WL 1619606 (Fed. Cl. Spec. Mstr. Mar. 21, 2024); see also O.M.V. v. Sec'y of Health & Hum. Servs., No. 16-1505V, 2021 WL 3183719, at *47-48 (Fed. Cl. Spec. Mstr. June 16, 2021) (finding the petitioner failed to establish Althen prong three in a flu/ADEM claim when onset was within 24 hours based on an innate immune response), mot. for rev. denied, 157 Fed. Cl. 376 (2021).

In True, the undersigned rejected a one-to-two-day onset of TM following human papillomavirus vaccination given the proposed theory of molecular mimicry. True, 2025 WL 1343027, at *29-31. The undersigned determined that Petitioner's expert failed to explain how a one- to two-day onset was appropriate given the latency period between antigen exposure and peak adaptive immune response. Id. Similarly, in Brancheau, the petitioner offered a causal theory of molecular mimicry to support her claim of vaccine-induced TM, but she failed to explain how a one-day onset was compatible with the latency period between vaccination and

manifestation of symptoms. Brancheau, 2024 WL 1619606, at *24 ("[A] latency period "is characterized by a lag phase, logarithmic phase, and plateau phase. Petitioner failed to explain how this would allow for an onset of one day." (internal citations omitted)); see also Forrest, 2019 WL 925495, at *6-7 (explaining, in detail, the latency period of adaptive immune response).

Other special masters have reached similar conclusions. For example, in Rowan, the special master rejected Petitioner's 30-to-36-hour onset of GBS following flu vaccination. Rowan v. Sec'y of Health & Hum. Servs., No. 17-760V, 2020 WL 2954954, at *16-18 (Fed. Cl. Apr. 28, 2020). The special master explained that the adaptive immune response timeframe, "which under a theory of molecular mimicry would occur," involves a sequential "lag and log phase." Id. at *17 (internal quotation marks omitted). "Under a theory of an autoimmune cross-reactive attack . . . the pathologic phase resulting in clinical symptoms could not begin until the end of the lag phase at the earliest, and likely would occur sometime within the log phase." Id. Accordingly, the special master determined a 36-hour onset was not temporally appropriate. Id. at *18.

And special masters have consistently rejected 24-hour onset in the context of molecular mimicry. See, e.g., Parker v. Sec'y of Health & Hum. Servs., No. 14-979V, 2019 WL 3425297 at *28-29 (Fed. Cl. Spec. Mstr. June 24, 2019) (finding 24 hours is an insufficient time for molecular mimicry); Henry v. Sec'y of Health & Hum. Servs., No. 17-721V, 2022 WL 2301321, at *35-36 (Fed. Cl. Spec. Mstr. May 2, 2022) (finding 20 hours too fast for an adaptive immune response due, in part, to the petitioner's expert stating that 24-48 hours is needed for a significant immunoglobin response); Forrest, 2019 WL 925495, at *6, *8 (finding "a preponderance of the evidence shows that molecular mimicry is not likely to happen within 36 hours, even for a recall response"); see also Martinez v. Sec'y of Health & Hum. Servs., 165 Fed. Cl. 76 (2023) (denying motion for review and stating that special master was not arbitrary in finding onset was approximately 24 hours after vaccination, and, therefore, too short).

While the above cases are not binding here, the undersigned agrees with the reasoning of other special masters as it relates to onset where the alleged causal mechanism is molecular mimicry. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

In summary, the undersigned finds by preponderant evidence that the onset of Petitioner's autoimmune neurological condition was December 9, 2015, one day after her flu vaccination. There is not preponderant evidence that an onset of one day is appropriate given the proposed mechanism of molecular mimicry.

Therefore, the undersigned finds the temporal association is not appropriate given the mechanism of injury. Petitioner has failed to satisfy the third Althen prong.

VI. CONCLUSION

The undersigned extends her sympathy to Petitioner for what she has suffered due to her neurological condition. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination she received caused her to develop the neurological condition at issue. Therefore, Petitioner is not entitled to compensation and the petition must be dismissed.

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

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master