# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

Filed: June 17, 2025

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| SHELLE JOHNSON, | * | |
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| Petitioner, | * | No. 18-410V |
| | * | |
| v. | * | Special Master Young |
| | * | |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| | * | |
| Respondent. | * | |

*Courtney Christine Jorgenson*, Siri & Glimstad, LLP, Phoenix, AZ, for Petitioner.
*Nina Ren*, United States Department of Justice, Washington, DC, for Respondent.

## DECISION ON ENTITLEMENT[1]

On March 19, 2018, Shelle Johnson ("Petitioner") filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program")[2] alleging that the influenza ("flu") vaccine Petitioner received on September 28, 2016, caused her to suffer from transverse myelitis ("TM") and subsequent "medical issues as a result of her [TM], including lack of mobility, tingling in her feet bilaterally, and weakness of her lower extremity." Pet. at 1, ECF No. 1. After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards,[3] I find that Petitioner has failed to provide

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

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

[3] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the decision will be discussed. *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.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding

preponderant evidence that the flu vaccine Petitioner received on September 28, 2016, caused her to develop transverse myelitis or any other medical issues. Accordingly, Petitioner is not entitled to compensation.

## I. Procedural History

Petitioner filed her petition on March 19, 2018. Pet. On April 5, 2018, Petitioner filed medical records and a statement of completion. Pet'r's Exs. 1–7, ECF No. 8; ECF No. 9

Respondent filed his Rule 4(c) Report, recommending that compensation be denied on January 28, 2019. Resp't's Report, ECF No. 17. Petitioner filed an expert report from Peter-Brian Andersson, M.D., PhD as well as his curriculum vitae ("CV") on November 2, 2020. Pet'r's Exs. 10–11, ECF No. 40. On November 6, 2020, and November 17, 2020, Petitioner filed vaccine exemption documents. Pet'r's Exs. 12–13, ECF Nos. 41–42. Supporting medical literature was submitted by Petitioner on December 3, 2020. Pet'r's Exs. 14–30, ECF Nos. 43–44.

Respondent filed expert reports from Dara Jamieson, M.D., and Thomas Forsthuber, M.D., along with their CVs and accompanying medical literature on March 3, 2021. Resp't's Ex. A, Tabs 1–21, ECF No. 46; Resp't's Ex. B, ECF No. 47; Resp't's Ex. C, Tabs 1–15, ECF No. 48; Resp't's Ex. D, ECF No. 49

On July 19, 2021, Petitioner filed a supplemental expert report from Dr. Andersson. Pet'r's Ex. 31, ECF No. 52. Respondent filed responsive supplemental expert reports from Drs. Jamieson and Forsthuber on September 19, 2021. Resp't's Exs. E, Tabs 1–10, F, Tab 1, ECF No. 54. Petitioner submitted additional medical records on April 5, 2022, June 7, 2022, and May 18, 2023. Pet'r's Exs. 32–35, ECF Nos. 55–56, 59.

On May 26, 2023, I issued an order for an entitlement hearing scheduled for February 6–7, 2024. ECF No. 60. After informal communications with the parties, a status conference was held on July 19, 2023. Min. Entry, docketed July 19, 2023. Respondent stated that his expert had a work conflict during the scheduled hearing dates. *Id.*; ECF No. 61. The parties determined that in lieu of rescheduling the hearing, they would agree to a ruling on the record. ECF No. 61. An order cancelling the entitlement hearing issued on August 10, 2023. *Id.* On August 21, 2023, Petitioner filed supplemental medical records. Pet'r's Ex. 36, ECF No. 62.

On November 14, 2024, Petitioner filed a motion for ruling on the record. Pet'r's Mot., ECF No. 64. Respondent filed his response on December 27, 2024; Petitioner replied on January 2, 2025. Resp't's Response, ECF No. 65; Pet'r's Reply, ECF No. 66. Petitioner filed letters from Nurse Practitioner Anne Beighley and Dr. Brian Crum on March 14, 2025. Pet'r's Exs. 37–38, ECF No. 71.

This matter is now ripe for consideration.

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certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

## II. Factual Background

### A. Medical Records

Petitioner was 52 years old when she received her flu vaccine on September 28, 2016. Pet'r's. Ex. 3 at 1. Her medical history included a vertebrobasilar vascular insufficiency in 2015. Pet'r's Ex. 5 at 27. Two days post vaccination, on September 30, 2016, Petitioner presented to the emergency department ("ED") with complaints of "some 'fatigue' feeling in back of her legs" that occurred the previous night at about 9:00 pm. Pet'r's Ex. 4 at 1. She was unable to stand in the morning and had since developed a numb feeling in the back of her left leg. *Id.* Petitioner also reported a little pain in her right mid-back. *Id.* Preliminary differential diagnoses included back pain, sciatica, spinal stenosis, cauda equine syndrome, and anxiety. *Id.* at 3.

On September 30, 2016, Petitioner was seen at the Mayo Clinic with a chief complaint of leg weakness. Pet'r's Ex. 5 at 1. She stated that she did not believe she had the "strength to stand and walk." *Id.* at 5. She also reported "a sense of numbness that radiates down the posterior aspect of both legs." *Id.* Petitioner denied trauma and recent infectious symptoms. *Id.* On examination, she had tenderness at the T11 area and weakness on standing in both legs. *Id.* Her patellar reflexes were brisk. *Id.* Differential diagnoses included TM, epidural hematoma, abscess, mass, or early Guillain-Barré syndrome ("GBS").[4] *Id.* A neurologic examination revealed "an upper motor neuron pattern of weakness in the bilateral lower extremities, more pronounced in the right lower extremity than the left, with distal greater than proximal flexor weakness." *Id.* at 11. Petitioner had back allodynia below the T5-7 level and "decreased sensation in the left lower extremity compared to the right lower extremity with pinprick." *Id.* Her examination was concerning for thoracic myelopathy[5] and magnetic resonance imaging ("MRI") of the thoracic spine was ordered. *Id.* at 12. The MRI did not reveal an obvious lesion, and the weakness was still present on the right lower side, although less convincing on reexamination. *Id.* Petitioner was able to squat on both legs, but could not squat on the right leg alone, and she had an antalgic gait. *Id.* The diagnosis was uncertain, and Petitioner was admitted for further evaluation. *Id.* Differentials included query spinal cord infarction[6] ("SCI") versus thoracic myelopathy and paraparesis,[7] right greater than left. *Id.* at 15.

A repeat MRI done on October 3, 2016, showed a single change in the thoracic cord,

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[4] GBS is a "rapidly progressive ascending motor neuron paralysis of unknown etiology, frequently seen after an enteric or respiratory infection." *Guillain-Barré Syndrome*, DORLAND'S MED. DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=110689 (last visited June 11, 2025). "It begins with paresthesias of the feet, followed by flaccid paralysis of the entire lower limbs, ascending to the trunk, upper limbs, and face; other characteristics include slight fever, bulbar palsy, absent or lessened tendon reflexes, and increased protein in the cerebrospinal fluid without a corresponding increase in cells." *Id.*

[5] Myelopathy refers to "any of various functional disturbances or pathologic changes in the spinal cord, often referring to nonspecific lesions in contrast to the inflammatory lesions of myelitis." *Myelopathy*, DORLAND'S MED. DICTIONARY ONLINE.

[6] Infarction is "an area of coagulation necrosis in a tissue due to local ischemia resulting from obstruction of circulation to the area, most commonly by a thrombus or embolus." *Infarct*, DORLAND'S MED. DICTIONARY ONLINE.

[7] Paraparesis is "partial paralysis of the lower limbs." *Paraparesis*, DORLAND'S MED. DICTIONARY ONLINE.

consistent with either an inflammatory/immune-mediated response or possible ischemia.[8] Pet'r's Ex. 5 at 39. No structural cause for Petitioner's symptoms was found and there was "no abnormal cord signal or compression." *Id.* at 36. The next day, Dr. Lyell K. Jones, a neurologist, "personally met with and examined [Petitioner] after a detailed review of the record with the Inpatient Neurology Team." *Id.* at 42. Dr. Jones concluded Petitioner "likely ha[d] a thoracic [SCI] to account for her asymmetric paraparesis." *Id.* He noted that "a workup looking for autoinflammatory or coagulopathic mechanisms for her infarction" would be completed. *Id.* Dr. Jones further ordered that Petitioner "complete three days of IV methylprednisolone and pursue continued aggressive rehabilitative therapy." *Id.*

A transesophageal echocardiogram conducted on October 4, 2016, was normal. Pet'r's Ex. 5 at 48. Her neurology hospital service records at discharge listed probable thoracic SCI and noted "[r]egarding her thoracic myelopathy, likely reflecting a [SCI]." *Id.* at 51. Additionally, a "repeated thoracic MRI . . . show[ed] T2 hyperintensity at the T8 level. This likely represent[ed] a thoracic cord infarction." *Id.* at 56. Petitioner was discharged for rehabilitation on October 5, 2016. *Id.* at 48–49. Her final primary diagnosis was "acute paraparesis, right greater than left – suspect [SCI]." *Id.* at 62. Rehabilitation facility admission records listed Petitioner's assessment as acute thoracic myelopathy. *Id.* at 72. Petitioner's course of treatment included physical and occupational therapy to allow her to "ascend and descend three stairs to enter her home as well as manage in-home mobility independently." *Id.* Her treaters agreed that inpatient treatment was needed but added that she would "only need a relatively brief inpatient rehabilitation stay." *Id.*

Dr. Jeffrey A. Strommen, a physiatrist with the Mayo Clinic, examined Petitioner on October 6, 2016. Pet'r's Ex. 5 at 88. He noted her history included a "comprehensive neurologic evaluation, including spinal imaging. It was felt that she likely had a spinal cord infarct versus myelitis." *Id.* Dr. Strommen agreed that the etiology was uncertain, but saw Petitioner's "substantial improvement with steroids," as evidence that her condition was "more of a [TM]-type picture." *Id.* On October 7, 2016, Petitioner started on a low dose of gabapentin at night to ease burning and aching in her right lower limb. *Id.* at 97. She was discharged on October 12, 2016. *Id.* at 109–14. The discharge summary noted that her antinuclear antibodies ("ANA") were mildly elevated at 3.1 and that her cerebrospinal fluid ("CSF") was normal. *Id.* at 64. A thrombophilia workup was pending at the time of discharge. *Id.* The final primary diagnosis was listed as "spinal cord injury, non-traumatic, paraplegia, incomplete." *Id.* at 109. Additional diagnoses included thoracis myelopathy; paraparesis, right greater than left, lower extremity only; probable thoracic SCI versus inflammation; urinary retention; transient diplopia[9] (approximately one year ago); and anxiety. *Id.*

On October 27, 2016, Petitioner saw her primary care physician ("PCP"), Dr. Melanie McKendrick Johnson, for a checkup "after being hospitalized for myelitis of the spinal cord." Pet'r's Ex. 4 at 4. She complained of right leg spasticity at night, which caused her difficulty sleeping. *Id.* Petitioner requested an increase in her gabapentin dosage to alleviate her continued symptoms. *Id.* She noted that she was doing well in physical therapy and planned to return to work

[8] Ischemia is a "deficiency of blood in a part, usually due to functional constriction or actual obstruction of a blood vessel." *Ischemia,* DORLAND'S MED. DICTIONARY ONLINE.

[9] Diplopia is "the perception of two images of a single object." *Diplopia,* DORLAND'S MED. DICTIONARY ONLINE.

the following day. *Id.* Petitioner reported that she still had decreased sensation to temperature in her left leg and foot and weakness in the quadriceps. *Id.* An examination revealed hyperactive reflexes in her right leg, and normal reflexes on the left. *Id.* Her strength was normal and symmetric in both legs. *Id.* The impression was myelitis with the note that "there [was] some thought that this may be secondary to the flu shot and we . . . added that to allergies." *Id.* at 6. Dr. McKendrick Johnson also noted positive ANA test results. *Id.* Petitioner's gabapentin was increased to 600 mg at bedtime. *Id.*

Petitioner had a follow up visit with the Mayo Clinic Physical Medicine and Rehabilitation Department on November 14, 2016. Pet'r's Ex. 5 at 125. She reported that she was continuing to improve and had returned to work. *Id.* Petitioner felt about 95% better but still had some deficits. *Id.* She "continue[d] on a daily aspirin for potential infarction," and tramadol and gabapentin for neuropathic pain. *Id.*

On December 12, 2016, Petitioner saw her PCP for a general examination. Pet'r's Ex. 4 at 7. She reported that she had restless leg syndrome and that she thought she may have shingles. *Id.* Her neurologic and musculoskeletal examinations were normal. *Id.* at 9.

On Wednesday, December 21, 2016, Petitioner called the Mayo Clinic Neurology Department and reported left lower extremity numbness and difficulty walking since Monday. Pet'r's Ex. 5 at 127. When the call was returned, she reported that she had improved somewhat. *Id.* Dr. M.W. Ruff expressed concern because "the etiology of her prior paraparesis was felt to be a probable [SCI], but this was indeterminate." *Id.* She was advised to schedule an appointment for follow-up or come to the ED as soon as possible. *Id.* Petitioner was seen in the ED the next day for left leg numbness. *Id.* at 128. The ED record noted flu virus vaccines under allergies/adverse reactions with a note that "[a]llergies above current as of Thursday, 22-Dec-2016 at 18:01." *Id.* It was noted that Petitioner was told to come in for a repeat thoracic spine MRI due to a recent increase in left lower extremity weakness. *Id.* at 129. During her neurology consultation, Petitioner reported that roughly ten days earlier (December 11, 2016), she had an outbreak of shingles on her right leg. *Id.* at 136. Three days before that, she developed sudden onset left lower extremity numbness, which she described as a loss of feeling with an occasional pins and needles sensation. *Id.* Petitioner did not report weakness but did describe difficulty walking because of the feeling of heaviness. *Id.* She noted improvement over the last two days. *Id.*

On examination, Petitioner had near normal strength, brisk reflexes, and upgoing toes bilaterally. Pet'r's Ex. 5 at 138. She had decreased sensation to temperature, and pinprick on the left, and some impairment of proprioception and vibratory sense (distal greater than proximal). *Id.* There was a minimal level change to pinprick at around T9-10. *Id.* The MRI showed a T2 hyperintense lesion without mass effect or enhancement involving the right lateral aspect of the thoracic cord at the T8 level. *Id.* "A demyelinating or inflammatory focus remain[ed] the primary considerations." *Id.* at 122. Dr. K.N. Kreck noted the "finding remain[ed] nonspecific, but suspicious for focal cord infarction." *Id.* at 123.

On March 17, 2017, Petitioner called the Mayo Neurology Department to report slight weakness of her legs, numbness in her left leg, lack of sensation to temperature, walking with a

hitch, restless legs at night, and difficulty urinating. Pet'r's Ex. 5 at 142. During a follow-up call on March 23, 2017, she was advised to schedule an appointment. *Id.* at 144.

On April 17, 2017, Petitioner saw neurologist Dr. Brian Crum and reported that her symptoms had been stable since late December, with no worsening but not much improvement either. Pet'r's Ex. 5 at 146. She reported some trouble initiating urination and some trouble with constipation, numbness in her left leg, and an inability to run. *Id.* Examination revealed a very mild upper motor neuron pattern of weakness of the right leg and no weakness of the left leg. *Id.* at 147. She had decreased sensation to pinprick up to about T10 on the left. *Id.* Her gait showed some right lower extremity spasticity. *Id.* New imaging showed "improvement in the hyperintense area in the mid-thoracic spine when you look at her previous studies compared to the one in December." *Id.* Petitioner was advised to follow up with rehabilitation to see if additional therapies might help her. *Id.* She was also assured that she had improved and that nothing she did "would significantly harm her or set her back or cause any increasing neurological attacks to occur of her central nervous system." *Id.* Dr. Crum told Petitioner that "[w]e may not have a good explanation for what happened to her in September but at this point six months out, she is definitely improved and has imaging studies which are improved as well, which is all reassuring." *Id.* His listed diagnoses were modified slightly from previous records and listed thoracic myelopathy, myelitis versus cord infarction, and lower extremity spasticity. *Id* at 147.

An MRI scan completed on September 14, 2017, revealed "no change compared to the MRI scans done in the past with a small area of T2 hyperintensity in the right spinal cord at T8. There are no other abnormalities seen in the thoracic spine." Pet'r's Ex. 34 at 35. Petitioner's neurological examinations remained unchanged through August of 2018. *Id.* at 29.

Petitioner was seen at Olmsted Medical Center on November 9, 2020, with a chief complaint of TM and spasticity. Pet'r's Ex. 33 at 222. She reported a four-year history of myelopathy of presumed autoimmune cause following her flu shot. *Id.* Dr. Keith Gonzalez noted that Petitioner had "a history of acute onset of myelopathy most consistent with TM. The etiology of which is unclear." *Id.* at 225. Petitioner's diagnosis "suggest[ed] the possibility of demyelinating disease either as clinically isolated syndrome or with risk of developing multiple sclerosis." *Id.* Dr. Gonzalez ordered "an MRI of the brain [and] cervical and thoracic spine with and without contrast." *Id.* He also noted that "it [did] not appear that there [was] a clear medical solution." *Id.* Results from a December 10, 2020 MRI revealed "[n]o abnormal enhancement." *Id.* at 231. Dr. Gonzalez did note "areas of [non-enhancing] high T2 signal in the cord [were] concerning for demyelination." *Id.* A medical record dated December 21, 2020, documented Dr. Gonzalez's comparison of Petitioner's brain CT dated July 29, 2015, and her MRI from December 10, 2020. *Id.* at 241. His impression was that "[n]onspecific foci of T2/FLAIR signal hyperintensity within the subcortical white matter regions bilaterally[, but t]hese may be related to chronic small vessel ischemic changes." *Id.* at 242. Dr. Gonzalez also noted that a demyelinating process cannot be entirely excluded." *Id.*

No other relevant medical records were filed.

### B. Vaccine Exemption Letters

Nurse Practitioner Anne Beighley submitted a letter dated October 28, 2020, that detailed how Petitioner was advised "not to get a seasonal [flu] vaccination" because of her "chronic medical condition." Pet'r's Ex. 12. Dr. Crum submitted a letter dated November 5, 2020, that noted Petitioner had been his patient "since 2016 when she developed a myelitis (spinal cord inflammation) following a flu shot." Pet'r's Ex. 13. Dr. Crum continued that Petitioner's condition, "which has been noted to happen after flu shots," led him to recommend "that she not receive a flu vaccination again due to risk of another, or worse, reaction to it." *Id.*

### C. Petitioner's Affidavit

Petitioner submitted an affidavit that briefly noted her receipt of the flu vaccine on September 28, 2016, her symptom onset of bilateral leg numbness "shortly after receiving the . . . vaccination," and her subsequent hospitalization. Pet'r's Ex. 2 at 1. She added that she "was eventually diagnosed with [TM] and was told this was more than likely from the flu vaccination." *Id.* Petitioner stated that she "believe[s her] diagnosis of [TM] occurred as a result of the flu vaccination." *Id.* at 2.

## III. Experts

### A. Expert Backgrounds and Qualifications

#### 1. Petitioner's Expert, Peter-Brian Andersson, M.D., Ph.D

Dr. Andersson is a board-certified clinical neurologist and professor and at the University of California, Los Angeles. Pet'r's Ex. 10 at 1. Dr. Andersson received his medical degree from the University of Cape Town in South Africa. Pet'r's Ex. 11 at 1. He completed his internship, residency, and chief residency at the University of California, San Francisco. Pet'r's Ex. 10 at 1. Dr. Andersson then completed two fellowships at Stanford University in Neuroimmunology & Multiple Sclerosis and Neuromuscular Disease & Electrodiagnostic Medicine. *Id.* He also has a Ph.D in neuroimmunology. *Id.* at 2. Dr. Andersson has done research on the central nervous system ("CNS") immune response and published original research in clinical neurology. *Id.* Over the course of his 24-year clinical practice, including managed care neurology, Dr. Andersson has "performed thousands of inpatient and outpatient consultations for weakness, numbness and gait difficulty, and have examined and/or treated at least 100-200 cases of [TM]." *Id.*

#### 2. Respondent's Expert, Dara G. Jamieson, M.D.

Dr. Jamieson was a board-certified practicing neurologist for 32 years before transitioning to a teaching appointment. Resp't's Ex. A at 1. She received her medical degree from the University of Pennsylvania School of Medicine, followed by a neurology residency and a cerebrovascular fellowship at the Hospital of the University of Pennsylvania. *Id.*; Resp't's Ex. B at 1. She is currently a Clinical Associate Professor of Neurology at Weill Cornell Medicine where she teaches neurology courses and clinical inpatient clerkships. Resp't's Ex. A at 1. Dr. Jamieson

has authored or co-authored several publications on multiple neurological topics. *Id.* at 2; *see* Resp't's Ex. B at 11–17.

#### 3. Respondent's Expert, Thomas Forsthuber, M.D.

Dr. Forsthuber is board certified in anatomical and clinical pathology. Resp't's Ex. C at 1. He received his medical degree from the University of Tübingen in Germany and completed a residency in pathology at the University Hospitals in Cleveland. Resp't's Ex. D at 2. He is currently a Professor of Immunology at the University of Texas at San Antonio and Adjunct Professor of Pathology and Microbiology & Immunology at the University of Texas Health Sciences Center, San Antonio. Resp't's Ex. C at 1. He has "over 25 years of experience in immunology, in particular in autoimmune disease research and T cell immunology." *Id.* Dr. Forsthuber has numerous publications in the fields of T cell immunology and autoimmune diseases including multiple sclerosis ("MS"), autoimmune diabetes, autoimmune heart disease, and their respective animal models. *Id.*; *see* Resp't's Ex. D at 21–30.

### B. Expert Opinions

#### 1. Dr. Andersson's First Report

In his first expert report, Dr. Andersson defined TM as a "a heterogenous, acquired, immune-mediated disorder causing injury to spinal cord neurons and oligodendrocytes which results in weakness, numbness and autonomic dysfunction." Pet'r's Ex. 10 at 9. He explained that TM has been associated with CNS and systemic autoimmune disorders; viral, bacterial, and fungal infections; and parasites. *Id.*at 10. TM is also associated with small cell lung carcinoma or can be idiopathic in many cases. *Id.* Dr. Andersson noted that TM was described as a complication of smallpox and rabies vaccines in 1922 and 1923, but "[a] direct causal relationship by vaccination to TM has still not been definitively established." *Id.* He cited to the National Institute of Health's, National Institute of Neurological Disorders and Stroke ("NINDS") Transverse Myelitis Fact Sheet, which stated that the condition can be caused by "post-infectious or post-vaccine autoimmune phenomenon, in which the body's immune system mistakenly attacks the body's own tissue while responding to the infection or, less commonly, a vaccine." *Id.* (citing Pet'r's Ex. 14 at 2).[10]

In identifying molecular mimicry as the biological mechanism for vaccine-caused TM, Dr. Andersson asserted that "[t]he current understanding of the etiology of vaccination-induced TM is the same as for vaccination induced [GBS]." Pet'r's Ex. 10 at 10. He described the immune system's response to the flu vaccine as a "misdirected humeral and/or cell-mediated attack on the host's spinal cord because of antigens possessed both by the triggering agent and the host's spinal cord tissues, causing a 'friendly fire' injury." *Id.* at 11. As evidence to support a molecular mimicry process, Dr. Andersson noted that in TM cases, there can be "histopathological evidence of a cellular and humoral immune attack on the nerves and oligodendrocytes associated with microgial

[10] *Transverse Myelitis Fact Sheet*, NINDS, http://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Transverse-Myelitis-FactSheet (last reviewed Oct. 25, 2020).

and astrocyte activation." *Id.* Dr. Andersson also named neuromyelitis optica[11] and MOG-Antibody disorder as comparable myelin-related conditions with "identified pathogenetic antibodies" susceptible to molecular mimicry. *Id.* He identified immune complex depositions as a "mechanism proposed to explain recurrent attacks in a patient with high circulating anti-hepatitis B surface antigen in whom these antibodies were also found in the CSF," and described plasma exchange as "a beneficial therapy" for TM consistent with the presence of these autoantibodies. *Id.* He also referred to animal models where encephalomyelitis was induced by injecting T-cells from vaccinated subjects and mentioned an increased distribution of TM cases within six weeks of vaccination according to Vaccine Adverse Event Reporting System ("VAERS") data. *Id.*

Following his discussion of molecular mimicry, Dr. Andersson referenced a notation from Petitioner's PCP that the flu vaccine was a potential cause of her myelitis. Pet'r's Ex. 10 at 12 (citing Pet'r's 4 at 6). Based on multiple medical records, Dr. Andersson placed the initial onset of Petitioner's relevant symptoms—leg fatigue—at 9:00 pm the day following her vaccination. *Id.* Dr. Andersson then opined that the proper diagnosis for Petitioner is TM, and the cause was her flu vaccine. *Id.* at 13.

Dr. Andersson acknowledged that "[r]esolving between [TM] and [SCI] is frequently difficult because of their similar clinical and radiologic features." Pet'r's Ex. 10 at 13. He provided the definition of TM from the National Institute of Neurological Disorders and Stroke—"an inflammation of the spinal cord;" and the definition of SCI from the American Heart Association—"cell death attributable to ischemia, based on . . . evidence of spinal cord focal ischemic injury in a defined vascular distribution." *Id.* Dr. Andersson noted that severe back pain is a symptom of infarction, but asserted that "Petitioner's pain was not severe" and not limited in location. *Id.* He argued that her symptoms do, however, satisfy three of the six most critical inclusion criteria articulated by the Transverse Myelitis Consortium Working Group ("TMCWG"). *Id.* at 14 (citing Pet'r's Ex. 25).[12] These three criteria are "[s]ensory, motor or autonomic dysfunction attributable to the spinal cord, T2 hyperintense signal change on MRI and no evidence of compressive cord lesion." *Id.* Dr. Andersson argued that TMCWG's full list of six inclusion and six exclusion criteria were for research purposes and relied on one medical reference source, *UpToDate*, that reported "not all [criteria] are necessarily required to make the diagnosis in clinical practice." *Id.* (citing Pet'r's Ex. 26).[13] Dr. Andersson asserted that Petitioner "satisfie[d] these critical three criteria easily." *Id.* Specifically, Dr. Andersson noted the following:

> To wit, she had asymmetric numbness and weakness in both legs and urinary retention satisfying the first criterion, a T2 spinal cord hyperintense lesion on MRI thoracic spine imaging satisfying the second and no cord compression on the October 2, October 3 and December 22 scans satisfying the third critical criterion.

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[11] Neuromyelitis optica is the "combined, but not usually clinically simultaneous, demyelination of the optic nerve and the spinal cord; it is marked by diminution of vision and possibly blindness, flaccid paralysis of the extremities, and sensory and genitourinary disturbances." *Neuromyelitis Optica*, DORLAND'S MED. DICTIONARY ONLINE.

[12] Transverse Myelitis Consortium Working Group, *Proposed Diagnostic Criteria and Nosology of Acute Transverse Myelitis*, 59 NEUROLOGY 499 (2002).

[13] Chitra Krishnan & Benjamin Greenberg, *Transverse Myelitis*, UPTODATE (May 28, 2020), http://www.uptodate.com/contents/transverse-myelitis/print.

*Id.* Dr. Andersson further asserted that Petitioner "satisfie[d] [five] of the [six] inclusion criteria and all [six] of the exclusion criteria." *Id.* The only criterion that Petitioner did not meet was "[i]nflammation defined by [CSF] pleocytosis, elevated immunoglobin G index or gadolinium enhancement on MRI." *Id.* This is inconsequential according to Dr. Andersson "because an absence of gadolinium enhancement, an absence of CSF pleocytosis[,] and an absence of CSF oligoclonal bands is commonly found in idiopathic [TM], specifically in 32%- 62%, 43-80% and 55-83% of cases." *Id.* Additionally, he relied on submitted medical literature to opine that "the absence of inflammatory markers does not rule out TM," in individuals "with a clinical pattern that otherwise resembles TM." *Id.* at 15 (citing Pet'r's Ex. 26).

Petitioner filed the TMCWG proposed diagnostic criteria of acute TM ("ATM"). Pet'r's Ex. 25. The authors expressed a need for uniform diagnostic criteria to "ensure a common language of classification, reduce diagnostic confusion, and lay the groundwork necessary for multicenter clinical trials." *Id.* at 1. They noted that "[a]cute transverse myelopathy (which includes noninflammatory causes) and ATM have often been used interchangeably throughout published literature." *Id.* at 1–2. After several diagnostic iterations, "cases of ATM were classified as parainfectious, related to MS, spinal cord ischemia, or idiopathic." *Id.* at 2. Furthermore, "acute noncompressive myelopathies were classified according to an etiologic scheme: [to include] 5) spinal cord infarct, and 6) idiopathic myelopathy." *Id.*

The TMCWG paper stated that "[a] diagnosis of idiopathic ATM should require that all of the inclusion criteria and none of the exclusion criteria are fulfilled." Pet'r's Ex. 25 at 2. The criteria table appears below:

| Inclusion criteria | Exclusion criteria |
|---|---|
| Development of sensory, motor, or autonomic dysfunction attributable to the spinal cord | History of previous radiation to the spine within the last 10 y |
| Bilateral signs and/or symptoms (though not necessarily symmetric) | Clear arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery |
| Clearly defined sensory level | Abnormal flow voids on the surface of the spinal cord c/w AVM |
| Exclusion of extra-axial compressive etiology by neuroimaging (MRI or myelography; CT of spine not adequate) | Serologic or clinical evidence of connective tissue disease (sarcoidosis, Behc¸et's disease, Sjo¨gren's syndrome, SLE, mixed connective tissue disorder, etc.)* |
| Inflammation within the spinal cord demonstrated by CSF pleocytosis *or* elevated IgG index *or* gadolinium enhancement. If none of the inflammatory criteria is met at symptom onset, repeat MRI and lumbar puncture evaluation between 2 and 7 d following symptom onset meet criteria | CNS manifestations of syphilis, Lyme disease, HIV, HTLV-1, *Mycoplasma*, other viral infection (e.g. HSV-1, HSV-2, VZV, EBV, CMV, HHV-6, enteroviruses)* |
| Progression to nadir between 4 h and 21 d following the onset of symptoms (if patient awakens with symptoms, symptoms must become more pronounced from point of awakening) | Brain MRI abnormalities suggestive of MS* |
| | History of clinically apparent optic neuritis* |

*Do not exclude disease-associated acute transverse myelitis.

*Id.* at 2 tbl. 1. Further, they noted "ATM represents a subset of acute myelopathies," and that "[a] diagnosis of ATM requires evidence of inflammation within the spinal cord." *Id.* at 2. Therefore, "[e]nhanced spinal MRI and a lumbar puncture are mandatory in the evaluation of suspected ATM." *Id.* In cases where a "spinal MRI shows an appropriately located high signal intensity lesion on T2-weighted sequences but no clear-cut enhancement," and CSF testing is normal, "then a diagnosis of ATM would not be possible." *Id.* at 3. Indeed, "cases that fulfill all of the proposed criteria with the exception of objective documentation of inflammation within the spinal cord"

may be best labeled as "possible ATM." *Id.* The "interval between symptom onset and maximal deficit" is also a noteworthy criterion that can be used to distinguish "ATM from a rapidly evolving vascular myelopathy [from] a slowly progressive or stuttering hereditary myelopathy." *Id.*

The authors caution that "[i]f these diagnostic criteria are going to be used to identify patients for prospective therapeutic trials, it will be critical to exclude patients with ischemic myelopathies for whom anti-inflammatory strategies may not be indicated." Pet'r's Ex. 25 at 3. For patient workup and evaluation, "a lumbar puncture should be performed to distinguish an inflammatory from a noninflammatory myelopathy." *Id.* at 4. Spinal cord ischemia "should be considered" in cases where "no gadolinium enhancement is seen on spinal cord MRI and the CSF does not show pleocytosis or increased IgG index." *Id.* "Alternatively, individuals who have mono-focal demyelination in the spinal cord (evoked potential and brain MRI do not show demyelination) and meet the criteria set forth above are defined as having ATM." *Id.*

The Krishnan & Greenberg article is a TM primer pulled directly from the *UpToDate* website. Pet'r's Ex. 26. The authors defined the condition and identified the following categories of causes: CNS inflammatory demyelinating disorders, systemic inflammatory disorders, infectious causes, paraneoplastic syndromes, and deficiency syndromes. *Id.* at 9–11. They also noted case reports that have associated TM with vaccination; however, they found that in "[c]omparing each TM case with all matched subjects in the exposure interval who received the same vaccination, there was no association of TM with prior vaccination." *Id.* at 2. Molecular mimicry was discussed "as [a] potential mechanism of autoimmunity" causing TM, following a pinworm infection. *Id.* The authors also stated that "some autoantibodies initiate a direct and selective injury of neurons or glia that express antigens that cross-react with antibodies directed against infectious pathogens." *Id.* at 3. In addition to focusing on the most critical inclusionary features Dr. Andersson mentioned, the authors noted that a TM diagnosis "requires exclusion of a compressive cord lesion, usually by MRI." *Id.* at 7, They also recommended a gadolinium-enhanced MRI or lumbar puncture. *Id.* at 8. Anterior spinal artery infarction is identified as a noninflammatory condition that may mimic TM. *Id.* at 12.

The medical literature filed with Dr. Andersson's report also included an article by de Seze et al.[14] Pet'r's Ex. 27. This article identified the "main etiologies of [ATM as] systemic diseases, spinal cord infarct, parainfectious events, and, more rarely, multiple sclerosis." *Id.* at 1. The authors conducted a retrospective study of ATM patients in the context of the TMCWG's criteria. *Id.* The authors described the criteria for idiopathic TM as stringent, but without the support of patient studies. *Id.* Study results showed that the criteria "lead to a relatively homogenous group in terms of clinical and MRI data but that . . . the outcome remain[ed] unpredictable." *Id.* at 3. The authors noted one third of the study's patients were designated with possible idiopathic ATM because "of the lack of inflammatory signs in CSF or on MRI," and consequent inability to meet all of the criteria. *Id.* Ultimately, the study did not reveal any "difference between the definite and possible idiopathic ATM groups, especially regarding the outcome." *Id.*

Dr. Andersson rejected Petitioner's differential diagnosis of SCI because of the length of her temporal course. Pet'r's Ex. 10 at 15. He explained that "[a] subacute presentation suggests an

[14] J. de Seze et al., *Idiopathic Acute Transverse Myelitis: Application of the Recent Diagnostic Criteria*, 65 NEUROLOGY 1950 (2005).

inflammatory etiology . . . while a hyperacute presentation suggests a spinal cord ischemic stroke." *Id.* The "largest cohort study in the literature" distinguishing TM and infarction cases showed that 90% of [infarction] patients had a nadir to maximum deficits within 24 hours. *Id.* (citing Pet'r's Ex. 28 at 5).[15] Additionally, in the remaining 10% of cases, "all had a stuttering and stepwise decline and all severe deficits had occurred by 12 hours." *Id.* Petitioner's onset to nadir lasted at a minimum of 39 hours and 47 minutes, excluding an infarction diagnosis. *Id.* Dr. Andersson also opined that the success of Petitioner's intravenous methylprednisolone is indicative of TM. *Id.* at 16. This treatment is not "not indicated for [SCI] but [does] reduce[] the severity and duration of attacks of demyelinating disease and [TM]." *Id.* Petitioner's "symptoms improved 50 percent after the first dose." *Id.*

The Zalewski et al. article, which proposed diagnostic criteria for spontaneous SCI, was heavily relied on by Petitioner. Pet'r's Ex. 28 at 5. The authors noted that SCI patients "are often misdiagnosed as having [TM]," and they aim "[t]o describe the characteristics of spontaneous SCI and propose diagnostic criteria." *Id.* at 1. The following table was proposed for definite, probable, and possible SCI:

**Proposed Spinal Cord Infarction (SCI) Diagnostic Criteria**

Criteria

1. Acute nontraumatic myelopathy (no preceding progressive myelopathy)

   Onset to nadir severe deficits[a] 12 h or less

   If stuttering course is more than 12 h, severe deficits[a] rapidly develop 12 h or less

2. Magnetic resonance imaging

   A. No spinal cord compression

   B. Supportive: Intramedullary T2-hyperintense spinal cord lesion (eBox 2 in the Supplement)

   C. Specific (1 of): diffusion-weighted imaging/apparent diffusion coefficient restriction, associated vertebral body infarction, arterial dissection/occlusion adjacent to lesion

3. Cerebrospinal fluid

   Noninflammatory (normal cell count, IgG index and no oligoclonal bands)

4. Alternative diagnoses

   Alternative diagnosis is not more likely (eBox 1 in the Supplement)

Types of SCI

Definite spontaneous SCI (1, 2A, 2B, 2C, 4)

Probable spontaneous SCI (1, 2A, 2B, 3, 4)

Possible spontaneous SCI (1, 4)

Definite periprocedural SCI (1, 2A, 2B, 4)

Probable periprocedural SCI (1, 4)

[a] A severe acute deficit (motor and/or sensory) typically consists of loss of antigravity strength or worse, severe objective sensory loss impairing function (eg, severe sensory ataxia).

*Id.* at 5.

[15] Nicholas L. Zalewski et al., *Characteristics of Spontaneous Spinal Cord Infarction and Proposed Diagnostic Criteria*, 76 JAMA NEUROLOGY 56 (2019).

The authors stressed that because infarction "is underrecognized and misdiagnosed," there is "a need for diagnostic criteria that can be broadly applied to patients with spontaneous and periprocedural SCI." Pet'r's Ex. 28 at 6. They went on to identify "[t]he most critical component [as] the rapid accumulation of severe deficits within 12 hours because more gradual worsening favors alternative etiologies." *Id.* Common features that are "often considered to be atypical [include] >4 hours to nadir, MRI and clinical evidence outside the anterior spinal artery territory, and gadolinium enhancement." *Id.* The authors cautioned against physicians ruling out SCI if one or more of these is present. *Id.* They also studied the use of different therapies and found that "[e]mpirical intravenous methylprednisolone treatment is reasonable when concern remains for a possible inflammatory myelopathy." *Id.* at 7.

Finally, Dr. Andersson noted that Petitioner has no history to explain her symptoms, nor has there been any evidence of trauma, rash, systemic illness, infectious or rheumatologic cause. Pet'r's Ex. 10 at 16. Petitioner's symptoms developed two days following her vaccination, and Dr. Andersson could identify "no competing precipitating causes." *Id.* He reasoned that although this time frame for TM "is shorter than any case [series], it is within hours and consistent with their short interval to onset and the activation of a memory response." *Id.* at 17. Dr. Andersson cited the Van Ussel et al.[16] paper to provide examples of "acute, even dramatic demyelination of the central nervous system and encephalitis [that] can occur after viral i.e. [flu] A/H1N1 vaccination or infection." Pet'r's Ex. 20 at 1. The authors reviewed 22 patients and found the time lapse from vaccination/infection to symptom onset ranged from one day in the case of a 77-year-old patient who was also suffering from rectal cancer and developed TM post vaccination, to one month in a case involving a 44-year-old that developed TM post vaccination with no comorbidities. *Id.* at 4. Two other patients included in the study, a five-year-old and six-year-old, both with no comorbidities, each had two-day onsets from vaccination/infection respectively, to development of acute disseminated encephalomyelitis ("ADEM"). *Id.* There was one patient listed in the study that developed TM four days post vaccination without any relevant comorbidities, but no age was provided. *Id.* The article noted that "ADEM following the administration of an inactivated component or live vaccine may be temporally associated with, but is not necessarily the result of, the administration of a vaccine." *Id.* at 6. Likewise, the authors noted an association seen in some patients between vaccination and acute transverse myelitis, but they do not conclude the relationships to be causal. *Id.* Their "case report and the review of the literature indicate that both infection with or vaccination for [flu] A/H1N1 virus may be linked rarely to central nervous system demyelination," including TM and ADEM. *Id.* at 7. Dr. Andersson argued that Petitioner's 17-year vaccination history is evidence of a "a primary immune response and this rapid onset of [TM]." *Id.*

**2. Dr. Jamieson's First Report**

Dr. Jamieson provided a very detailed recount of Petitioner's medical records beginning with her vaccination on September 28, 2016. Resp't's Ex. A at 2. She noted Petitioner's first neurological symptom was lower extremity fatigue that occurred "within a very short period of time" following vaccination, inconsistent with an immunological cause. *Id.* at 11. Dr. Jamieson also noted Petitioner's laboratory tests did not reveal any inflammatory cause for her weakness as

[16] Isabelle Van Ussel et al., *Encephalitis Related to a H1N1 Vaccination: Case Report and Review of the Literature*, 124 CLINICAL NEUROLOGY & NEUROSURGERY 8 (2014).

her CSF was bland and the very subtle spinal cord lesion was non-enhancing. *Id.* "Thoracic spine imaging was [also] originally unrevealing." *Id.* Identifying SCI as the appropriate diagnosis for Petitioner, Dr. Jamieson noted that 20%-40% of cases "remain cryptogenic despite extensive diagnostic investigation." *Id.* at 12. Dr. Jamieson also discussed the Zalewski et al. article's SCI criteria in the context of Petitioner's symptoms. *Id.* at 13. She noted that symptom onset is variable and may be abrupt or may result in "a decline over a few to several hours." *Id.* She also noted one case series wherein "the time from symptom onset to nadir deficit was more than four hours in 44 percent of patients, and greater than 12 hours in 23 percent." *Id.* (citing Pet'r's Ex. 28). Petitioner's "symptoms of right sided leg weakness with left sided decrease in pain and temperature are consistent with an anatomic pattern of a spinal cord infarct." *Id.* at 13. Dr. Jamieson noted that "[a] hemi-spinal cord (Brown-Séquard) infarct of the thoracic spinal cord produces ipsilateral unilateral leg weakness hemiparesis; contralateral loss of pain/temperature beginning two dermatomes below the lesion; and ipsilateral loss of light touch, proprioception, and vibration below the lesion." *Id.*

In comparison, Dr. Jamieson explained that "[p]atients initially suspected as having idiopathic [TM] often are given another diagnosis after completion of an appropriate diagnostic evaluation." Resp't's Ex. A at 14. TM is defined by

> acute or subacute spinal cord dysfunction resulting in weakness and sensory loss below the level of the lesion and impairment in autonomic functioning, with the symptoms due to disruption of descending (motor) and ascending (sensory) neural pathways causing a disconnection between the spinal cord below the level of the lesion and the brain.

*Id.* at 13. After presenting with a "loss of sensation to sensory modalities such as pain," an MRI of the spinal cord will localize the offending lesion and spinal cord final analysis will reveal inflammation or elevated protein levels. *Id.* at 14. One study of Mayo Clinic patients revealed that out of 226 patients initially suspected of having TM, ultimately 18.1% of them met the diagnostic criteria. *Id.* (citing Resp't's Ex. A, Tab 5).[17] The preferred treatment initially is generally high dose intravenous corticosteroids, that "[vary] with extent of return of function dependent on etiology of the [TM] and the extent of the spinal cord damage." *Id.* at 14–15.

TM is a syndrome, Dr. Jamieson further explained, and "rather than a specific disease entity, there may be variable and non-specific supposed linkages to environmental triggers, including to vaccinations." Resp't's Ex. A at 15. However, she added that the "[t]he linkage to the injected inactivated [flu] vaccination however is particularly weak, with a case-centered analysis showing no association between [TM] and prior immunization." *Id.* The specific vaccines used are often unknown, and she also questioned whether these cases relied on to make this argument "would be classified as [TM] today using the 2002 [TMCWG] diagnostic criteria." *Id.* Dr. Jamieson discussed several case studies, arguing that in two articles "the authors mischaracterized these [four] cases as isolated myelitis occurring after [flu] vaccination, when in fact they were not isolated and did not occur de novo." *Id.* at 16 (viting Resp't's Ex. A, Tab 18;[18] Resp't's Ex. A, Tab

[17] Nicholas L. Zalewski et al., *Evaluation of Idiopathic Transverse Myelitis Revealing Specific Myelopathy Diagnoses*, 90 NEUROLOGY e96 (2018).

[18] Naoko Nakamura et al., *Neurologic Complications Associated with Influenza Vaccination: Two Adult Cases*, 42 INTERNAL MED. 191 (2003).

19).[19] According to Dr. Jamieson, these cases were then "incorrectly classified as isolated myelitis related to [flu] vaccination by Karussis and Petrou; and they do not advance the hypothesis that injected [flu] vaccination causes transverse myelitis." *Id.* at 16 (citing Resp't's Ex. A, Tab 17).[20] Dr. Jamieson argued that these cases have been repeatedly used by subsequent researchers, "giv[ing] the false impression that there are more instances of [TM] following [flu] vaccinations than actually exist in the medical literature." *Id.* She then identified a case of TM following a flu vaccination where, unlike Petitioner, "this young woman showed an extensive lesion extending from C3 to the upper thoracic level spinal cord and the CSF was abnormal with an elevated white blood cell count and protein level." *Id.* at 17 (citing Resp't's Ex. A, Tab 21).[21]

In summary, Dr. Jamieson argued that Petitioner did not have TM because: 1) her MRI did not reveal a lesion "extending for more than two spinal column segments and involving more than two thirds of the transverse extent of the spinal cord;" 2) there was no evidence of inflammation; and 3) her "hemi spinal cord symptoms with unilateral crossed motor and sensory deficits [were] not consistent with a transverse lesion . . . of the spinal cord." Resp't's Ex. A at 18.

### 3. Dr. Forsthuber's First Report

Dr. Forsthuber's expert report included a summary of relevant medical history and background information on TM and molecular mimicry before his assessment of Petitioner's case. *See* Resp't's Ex. C at 3–5. He defined TM as "a monofocal inflammatory myelopathy of the spinal cord in which an immune-mediated process causes rapid onset of varying degrees of weakness (paralysis), sensory alterations, and autonomic dysfunction." *Id.* at 3. Dr. Forsthuber continued that TM's pathogenesis is not fully understood, but that "a wide range of infectious pathogens have been implicated in post-infectious TM." *Id.* Pathological evidence collected during autopsies and biopsies reveals "that TM patients show focal monocytic and lymphocytic infiltrates in spinal cord tissue and perivascular spaces, astroglial and microglial activation, and demyelination and axonal injury." *Id.* Dr. Forsthuber also noted the "[CSF] pleocytosis and blood-brain-barrier breakdown within a focal area of the spinal cord, consistent with an (auto)immune-mediated mechanism," in TM patients. *Id.* He identified a number of potential mechanisms, "such as autoimmune disease pathology induced by molecular mimicry, microbial superantigen-mediated inflammation, or humoral (antibody)-mediated derangements." *Id.* at 4.

Dr. Forsthuber explained molecular mimicry generally as "the induction of T cells, B cells, and/or antibodies that cross-react between antigens on microorganisms and host self-antigens, and this cross-reactivity may lead to autoimmune disease pathology." Resp't's Ex. C at 4. "Specifically, T cells recognize epitopes of generally [eight to] 15 amino acid length presented by MHC1 class I or II molecules on antigen presenting cells." *Id.* They "are highly specific for a particular antigen," compared to antibodies and B cells that "can bind to linear and/or conformational epitopes and to a wide variety of molecules including proteins, lipids,

[19] Sabrina Ravaglia et al., *Post-Infectious and Post-Vaccinal Acute Disseminated Encephalomyelitis Occurring in the Same Patients*, 251 J. NEUROLOGY 1147 (2004).
[20] Dimitrios Karussis & Panayiota Petrou, *The Spectrum of Post-Vaccination Inflammatory CNS Demyelinating Syndromes*, 13 AUTOIMMUNITY REVS. 215 (2014).
[21] Rohit Bakshi & John C. Mazziotta, *Acute Transverse Myelitis After Influenza Vaccination: Magnetic Resonance Imaging Findings*, 6 J. NEUROIMAGING 248 (1996).

polysaccharides, and chemicals." *Id.* Dr. Forsthuber noted studies that "have focused on the analysis of sequence similarities between linear epitopes on microorganisms and self-antigens," but given the "enormous number" of sequence matches, he doubted that molecular mimicry is a principal cause of autoimmune disease as once postulated. *Id.*

Conversely, Dr. Forsthuber argued that "remarkable contradictions can be found in most situations where molecular mimicry was alleged as the cause of human autoimmune diseases." Resp't's Ex. C at 5. He referenced the initial hypothesis that molecular mimicry occurred between "a hepatitis B peptide and myelin basic protein, but injection of the hepatitis peptide failed to induce clinical [experimental autoimmune encephalomyelitis ("EAE")] even when injected in complete Freund's adjuvant." *Id.* In most cases, "failure to induce autoimmune diseases in animal models with molecular mimic antigens is the rule, not the exception," as in that case. *Id.* at 5. This inability to uncover "solid" evidence of molecular mimicry's role in autoimmune disease pathology "despite massive research efforts" dating back to the 1980s has led Dr. Forsthuber and others to believe that "molecular mimicry may be necessary, but not sufficient to induce human autoimmune pathology." *Id.* Other factors may include bystander activation, dual T cell receptors, regulatory T cell failure, individual genetic susceptibility, "as well as external and internal environmental factors." *Id.* He concluded for these reasons that "molecular mimicry is not generally accepted as the mechanism for the induction of autoimmune diseases, let alone for autoimmune diseases allegedly induced by vaccines." *Id.*

Dr. Forsthuber analyzed Petitioner's case by responding directly to contentions made by her expert, Dr. Andersson. Resp't's Ex. C at 6–19. He highlighted Dr. Crum's diagnoses of thoracic myelopathy, myelitis versus cord infarct, and lower elastic spasticity at the time of her October 2016 hospitalization, and how that did not change through to her last medical visit with Dr. Crum documented in the record on April 17, 2017. *Id.* at 6 (citing Pet'r's Ex. 5 at 146–47). He also noted that Dr. Crum "did not raise concerns about her [flu] vaccination." *Id.* Dr. Forsthuber acknowledged that Dr. Crum did write the November 5, 2020 letter recommending that Petitioner not receive the flu vaccine again. *Id.* However, Dr. Forsthuber focused on Dr. Crum's notes during the time of Petitioner treatment in 2016 and 2017, which did not identify Petitioner's vaccine as the casual factor for her condition. *Id.*

Dr. Andersson's reliance on "neurological complications after smallpox and rabies vaccines in the 1920s," was another point of contention for Dr. Forsthuber. Resp't's Ex. C at 7. Dr. Forsthuber explained that these vaccines "are different vaccines that have nothing in common with the [flu] vaccine used in the present case, including that the smallpox vaccine is a live virus vaccine, . . . [with] completely different virus proteins and are manufactured in completely different ways." *Id.* Similarly, Dr. Forsthuber objected to Dr. Andersson's use of vaccine-induced GBS as an analogy for TM via molecular mimicry. *Id.* at 8. Dr. Forsthuber asserted that GBS "is probably one of the most robust examples of molecular mimicry as the cause of human autoimmune diseases and reliable evidence from both animal models and human studies supports that immunological cross-reactivity." *Id.* By comparison, TM is an unrelated neurological condition, whose cause remains unknown. *Id.* Dr. Forsthuber argued that "Dr. Andersson has merely invoked a generic concept of molecular mimicry without providing any further arguments for molecular mimicry in general in TM, or with the [flu] vaccine and TM in the present case." *Id.*

The arguments that Dr. Andersson made to support his conclusion that vaccination can trigger TM are individually addressed by Dr. Forsthuber. Resp't's Ex. C at 8–13. Dr. Forsthuber asserted that evidence of inflammatory events resulting from an immune-mediated attack is not de facto evidence of a vaccine causation without a link. *Id.* at 8. He also argued that the presence of specific antibodies in TM patients has not revealed "an inciting infectious agent [], and certainly not a particular vaccine." *Id.* Dr. Forsthuber opined that "there is no reliable link between [flu] vaccination and autoantibodies in TM," and therefore, Dr. Andersson's "claims are speculative and not applicable to TM or the neurological condition in the present case." *Id.* at 9. Dr. Forsthuber also disagreed with Dr. Andersson's interpretation of the Matsui et al.[22] article to support his focus on pathogenic circulating antibodies and the premise that immune complex depositions could explain recurrent TM attacks. *Id.* at 9. The article noted that "[t]here were neither autoantibodies nor evidence of vasculitis" in the case study. Pet'r's Ex. 17 at 1. However, the authors opined that "circulating immune complexes composed of [hepatitis B surface] antigen disappeared after treatment, indicating that [virus immunity] played a role in the demyelinating lesion formation in the central nervous system." *Id.*

Dr. Forsthuber cautioned against Dr. Andersson's citation of publications that discussed the development of TM following rabies, hepatitis B, and oral polio vaccines. Resp't's Ex. C at 10. He argued that they are not analogous to the flu vaccine at issue here. *Id.* He further cautioned that many of the case studies Petitioner filed contained other potential issues, including the presence of comorbidities or imprecise diagnosis of TM, inappropriate temporal relationships between vaccination and disease onset, and clearly identified alternative causes besides vaccination. *Id.* at 10–11. Dr. Forsthuber included the Institute of Medicine's ("IOM")[23] assessment that "the mechanistic evidence of an association between [flu] vaccine and TM [is] weak," and based on "temporal association." *Id.* at 12. He also discussed "two large studies conducted since the publication of the 2012 IOM report." *Id.* The first study was retrospective observational cohort study using Vaccine Safety Datalink data of 75,906 pregnant women vaccinated with the trivalent inactivated flu vaccine and 147,992 unvaccinated women. Resp't's Ex, C, Tab 10.[24] Nordin et al. "found no cases of TM in the first 42 days after vaccination." Resp't's Ex. C at 12. A second VSD study conducted by Baxter et al.,[25] found no statistically significant increased risk of TM following vaccination in nearly 64 million inoculations. *Id.* (citing Resp't's Ex. C, Tab 11).

Following his discussion of Petitioner's general proposed mechanism, Dr. Forsthuber turned to the specifics of Petitioner's case. *See* Resp't's Ex. C at 13. He questioned "whether it is probable that within 1.5 days after vaccination of [Petitioner] meaningful autoimmune T cell, B cell, and autoantibody responses would be generated that could have resulted in her neurological condition." *Id.* Dr. Forsthuber answered this question in the negative by breaking down the process of autoimmune disease following a primary immune response and a secondary immune response.

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[22] Makoto Matsui et al., *Recurrent Demyelinating Transverse Myelitis in a High Titer HBs-Antigen Carrier*, 139 J. NEUROLOGICAL SCIS. 235 (1996).
[23] The IOM is now called the National Academy of Medicine.
[24] James D. Nordin et al., *Maternal Safety of Trivalent Inactivated Influenza Vaccine in Pregnant Women*, 121 OBSTETRICS & GYNECOLOGY 519 (2013).
[25] Roger Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case Centered Analysis*, 63 CLINICAL INFECTIOUS DISEASES 1456 (2016).

*Id.* at 13–17. He noted that "it takes at least two to three days for the initial T and B cell responses to become detectable in regional lymph nodes at the site of the infection or vaccine injection," in the case of an initial exposure. *Id.* at 13–14. This delay includes the time for the body to recognize the presence of an antigen and mount an acute, innate immune response, typically during the first 48 hours following introduction. *Id.* at 14.

> During this time and in order to properly activate cells of the adaptive immune system, specialized cells called dendritic cells need to migrate to the site of the infection/vaccination, find and capture the antigens/microbes, break them down into small fragments (peptides), and then transport these fragments to regional lymph nodes where they "present" these fragments to T and B cells in order to activate them.

*Id.* Following activation, these cells enter the next "phase of lymphocyte activation, proliferation, and acquisition of effector functions." *Id.* at 15. Eventually, IgM antibodies are produced, a process that "generally takes [two to three] days" to reach detectable levels. *Id.* at 16. This is followed by "[p]roduction of the much more powerful class of IgG antibodies [that] takes significantly longer." *Id.* Dr. Forsthuber explained that "IgG responses begin to peak approximately [two] weeks after infection/antigen encounter." *Id.* The period during a secondary immune response is shortened because once the body has encountered an antigen (via a primary response), memory T cells that were created initially, can "facilitate[] and accelerate[] initiation of immune responses after re-infection and curtail[] the severity of this infection." *Id.* "Nevertheless," Dr. Forsthuber argued, "it will still take longer than 1.5 days for a notable immune response to develop, because even memory T and B cells still have to become activated and expanded before they can generate an effective immune response." *Id.* Furthermore, "additional time is required after an autoimmune response has already formed on a cellular-only level," until symptoms develop and "disease can be detected by clinical tests." *Id.* Dr. Forsthuber explained that this process includes the proliferation of T cells to the CNS and spinal cord where damage is sustained, and the autoimmune disease manifests. *Id.* He noted that models show it can be nine to 11 days post immunization "until the animals develop the first neurological symptoms." *Id.* Dr. Forsthuber agreed with Dr. Andersson's concession that Petitioner's case "is shorter than any case" referenced. *Id.* at 17. Dr. Forsthuber noted that "the development of TM is typically observed [three to four] weeks after exposure." *Id.* at 18.

In response to Dr. Andersson's reliance on a memory response to explain the short onset time, Dr. Forsthuber argued that Petitioner received the flu vaccine for 15 years following her initial exposure with no incident. Resp't's Ex. C at 18. He found it improbable that this most recent vaccination would cause a memory response sufficient to cause her neurological condition. *Id.* Dr. Forsthuber argued that Dr. Andersson "has merely invoked the generic concept of memory responses without providing any reasoning of how the [flu] vaccination would cause TM in the present case via a memory response." *Id.* He concluded that 1.5 days was not an appropriate temporal relationship for vaccine-caused TM via a primary or secondary response. *Id.*

#### 4. Dr. Andersson's Supplemental Report

Dr. Andersson filed a supplemental report to respond to Respondent's experts. Pet'r's Ex. 31. His responses to Dr. Jamieson are reiterations of his previously asserted points. *See id.*

Specifically, Dr. Andersson characterized Dr. Jamieson's opinions as false and contrived. *Id.* at 1. He asserted that she "merely erected her own more stringent criteria than are used in clinical practice then applied them to find Petitioner fail[ed] to make the requirements for a TM diagnosis." *Id.* at 1. Despite Dr. Jamieson's concern that Petitioner did not have a clearly defined sensory level, Dr. Andersson argued that clear definition ensures that "it localizes to the spinal cord," and "Petitioner's doctors never doubt a myelopathy localization" to meet that criterion. *Id.* He reiterated that inflammation is not necessary for a diagnosis, that her doctors did not arrive at a definitive diagnosis, and that thoracic myelopathy/myelitis remained a differential. *Id.* at 3. Dr. Andersson did not dispute Dr. Jamieson's opinion on the strength of published literature linking flu vaccination to TM, however he described the evidence as "rare rather than weak" and argued that it "does not disprove linkage." *Id.* at 4.

Responding to Dr. Forsthuber, Dr. Andersson offered conclusions that can be drawn "to a reasonable medical probability," due to Drs. Crum and McKendrick Johnson instructing Petitioner to refrain from future flu vaccines. Pet'r's Ex. 31 at 5. He articulated:

> 1) both Dr Crum and Dr McKendrick Johnson diagnose[d] Petitioner with [TM], not spinal cord stroke. Their instruction [was] meaningless otherwise, and potentially harmful too.
> 2) both Dr Crum and Dr McKendrick Johnson believe[d] Petitioner's flu vaccination may have been responsible for her [TM]. There is no reason to deprive health benefits of a flu vaccine otherwise.
> 3) both Dr Crum and Dr McKendrick [Johnson] [made] their recommendation to a likelihood that is at least greater than the cumulated lost health benefits of an annual flu vaccination, for the rest of her life.

*Id.*

Dr Andersson then turned to Dr. Forsthuber's objection to discussion of rabies and smallpox vaccines causing TM. Pet'r's Ex. 31 at 5. He clarified that it is "self-evident" that these two vaccines differ from each other and flu vaccines. *Id.* However, he argued that "[t]he evidence of their association is offered as one of the lines of evidence for a sound reliable theory of how flu vaccination could cause [TM]." *Id.* Dr. Andersson noted that Dr. Forsthuber's discussion "does nothing to refute the published cases of flu vaccination associated [TM] in principle or in the specific." *Id.* at 6. Further, Dr. Andersson noted that the evidence presented is "limited by existing knowledge . . . . In the same way, the lack of an explicit step-by-step explanation by which vaccination can trigger this same disease fails as a reason for skepticism." *Id.* He asserted that he "offer[ed] a theory how flu vaccination could reasonably [cause TM], logically and plausibly and probably." *Id.* Dr. Andersson cited the NINDS TM Fact Sheet, *UpToDate*, Victor and Adams's Neurology and Harrison's Textbook of Medicine,[26] and the package insert for Fluvarix to make the point that "the association of [TM] with vaccination" is in line with "mainstream thinking." *Id.*

In a corrective note, Dr. Andersson acknowledged that immune complex deposition in the CNS was not shown in the Matsui et al. article. Pet'r's Ex. 31 at 7. However, he

[26] Petitioner did not file this literature.

argued "[w]hat was shown was evidence to support [TM] having an immune-mediated pathogenesis effected by molecular mimicry," consistent with his theory. *Id.* Due to the peptide homology between hepatitis B and rabbit myelin basic protein, "immunization of rabbits with a relevant peptide of [hepatitis B virus] polymerase actually induced [an autoimmune] response" via molecular mimicry. *Id.*

Dr. Andersson continued and addressed the significance of Petitioner's adverse reaction occurring after her 16th flu vaccination. Pet'r's Ex. 31 at 9–10. He submitted that it is probable "there were antigens in the 16th [flu] vaccination which activated a memory response against host antigens through molecular mimicry causing [TM]." *Id.* at 9. Lastly, Dr. Andersson objected to Dr. Forsthuber's criticism of his memory cell analysis. Dr. Anderson responded:

> Again, the specific antigens and step-by-step pathway of the pathogenesis is not known for [TM] as a disease, but its existence as a disease and its association with vaccination stands undiminished by this ignorance, and so too the theory [he] offered to explain all that is known, with what is currently known.

*Id.*

**5. Dr. Jamieson's Supplemental Report**

Dr. Jamieson also sought to clarify arguments previously made in her initial report. Resp't's Ex. F. She noted that TMCWG criteria, like all diagnostic criteria, "are used all the time by physicians to determine as accurate and specific a diagnosis as possible for patients' treatment and counseling." *Id.* at 1. Dr. Jamieson acknowledged that diagnostic certainty is not always possible, and indeed, here Petitioner was treated both for SCI and TM. *Id.* However, she argued that "discordance between the TMCWG criteria and her clinical presentation was the reason why [Petitioner] was evaluated and treated for [SCI], as well as for [TM]." *Id.*

Both Drs. Jamieson and Andersson agreed that Petitioner "had a myelopathy." Resp't's Ex. F at 1. Dr. Jamieson argued that "because [TM] is, by definition, an 'itis' or an inflammatory condition," the absence of inflammation in the spinal cord is not insignificant. *Id.* Further, Dr. Jamieson opined that Petitioner had one of the exclusion criteria: an arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery. *Id.* (citing Pet'r's Ex. 25 at 2).

According to Dr. Jamieson, Petitioner's "sensory examinations varied from being normal to showing unilateral subjective sensory findings on one leg or the other, without a level of sensory loss referable to a localized spinal cord lesion" Resp't's Ex. F at 2. She concluded that "there was no clearly defined sensory level to indicate a specific transverse demyelinating lesion, as opposed to a more diffuse ischemic lesion." *Id.* Alternatively, her variable sensation loss was "characteristic of an anterior spinal artery infarct with leg weakness with lateral spinothalamic tract involvement and preservation of posterior column function." *Id.* at 3.

Dr. Jamieson cited to the Goh et al.[27] article to support her premise that classic TM lesions extend over more than two spinal cord segments and involves more than two-thirds cross section of the cord. Resp't's Ex. F at 3 (citing Resp't's Ex. F, Tab 1 at 3). The article discussed clinical symptoms, diagnostic criteria and related features of TM. Resp't's Ex. F, Tab 1 at 1. The article also included diagnostic criteria noting "that evolution of symptoms to the maximal clinical severity must be between [four] hours and 21 days . . . to prevent cases of cord infarction, typically of abrupt onset, from being erroneously diagnosed as [TM]." *Id.* The authors noted "the heterogeneous pathogenesis and prognosis of [TM, and stated] it is vitally important that there be uniformly applied criteria for diagnosis and classification." *Id.* They identified confirmation of an active inflammatory process by presence of a cellular infiltrate and/or elevated protein" as a requirement for diagnosis. *Id.* The authors noted a study of transverse myelopathy patients and found they "could be grouped as 15.6% idiopathic, 20.5% systemic disease, 17.3% infectious or parainfectious, . . . with a further 18.8% found to have [SCI] instead of [TM]." *Id.* at 2. The article noted that "[p]ostvaccination [TM] seems to be a rare complication of vaccination." *Id.* at 12. The "vaccines implicated" included flu, and it was noted that "[t]he pathogenesis is likely similar to the proposed mechanisms of immune-mediated neural damage described in parainfectious [TM]." *Id.*

The timing of Petitioner's symptom onset was readdressed by Dr. Jamieson in response to Dr. Andersson. Rep't's Ex. F at 3. She maintained that Petitioner experienced "the onset of severe deficits within 12 hours of symptom [manifestation]." *Id.* Adding that "[t]he time over which symptoms of spinal cord infarcts evolve is variable," Dr. Jamieson opined that Petitioner's "clinical course [was] consistent with both the medical literature and [her] experience in making the diagnosis of spinal cord ischemia." *Id.* She also reasoned that Petitioner's inconsistent "neurological examinations may have been related to her varying participation in her examinations; [or] reflect the 'stuttering course' that Dr. Andersson believe[d] [was] necessary" for SCI diagnosis. *Id.*

Again noting that TM and SCI were incorporated into the treatment plan, Dr. Jamieson asserted that "[w]ith the benefit of reviewing the record retrospectively, the diagnosis is more likely than not SCI." Resp't's Ex. F at 3. The advice "offered in an abundance of caution, to avoid future [flu] vaccination [was not] persuasive in establishing the diagnosis of [TM] or as endorsing a causative linkage between the [flu] vaccine and [TM]." *Id.* at 4.

### 6. Dr. Forsthuber's Supplemental Report

Noting again that Dr. Crum did not diagnose Petitioner with TM, Dr. Forsthuber discounted the opinion of Dr. McKendrick Johnson, "a family medicine specialist." Resp't's Ex. E at 2. Dr. Forsthuber opined that as a neurologist, Dr. Crum's "impressions of [Petitioner's] medical condition seem[d] more reliable than that of Dr. McKendrick Johnson's." *Id.* As of April 2017, Dr. Crum had still not resolved Petitioner's diagnosis. *Id.* Dr. Forsthuber thought "it is unclear how Dr. Crum arrived at his diagnosis of 'myelitis' in 2020, given the fact that he previously did not have a definitive explanation for her medical condition." *Id.*

[27] Christine Goh et al., *Neuroimaging in Acute Transverse Myelitis*, 21 NEUROIMAGING CLINICS N. AM. 951 (2011).

Drs. Andersson and Forsthuber disagreed about the viability of the comparison between rabies vaccine-induced neuroinflammation and flu vaccine-induced TM. Resp't's Ex. E at 3. Dr. Forsthuber argued that unlike the rabies vaccine, the flu vaccine "is not contaminated with autoantigens, . . . does not contain an adjuvant," and "[i]mmunization of animals with the [flu] vaccine does not induce neuroinflammation." *Id.* According to Dr. Forsthuber, these differences are critical in illustrating why the former process cannot be used to explain the latter. *Id.*

The experts also disagreed on whether there is a medical community consensus on flu vaccine-induced TM. Resp't's Ex. E at 3–4. Dr. Forsthuber noted the NINDS TM Fact Sheet lists as potential causes of TM: parasites; viral, bacterial, and fungal infections; and immune system, vascular, and other inflammatory disorders. *Id.* at 4 (citing Pet'r's Ex. 14). Likewise, *UpToDate* and Victor and Adams's Neurology and Harrison's Textbook "do not specifically refer to [flu] vaccines." *Id.* The Fluarix vaccine insert package lists myelitis and encephalomyelitis, but not TM. *Id.* Dr. Forsthuber argued that this is not a medical document. *Id.* He noted that the package lists "all events observed in all studies of the vaccine including post-marketing studies, irrespective of whether or not these events are statistically significantly increased." *Id.* He continued "myelitis or TM have not been listed in the 'Warnings and Precautions' section of the 2016-2017 Fluarix vaccine insert, which lists adverse events based on scientific evidence." *Id.* Likewise, "[t]he VAERS database is useful as an early warning system to identify rare adverse events, but VAERS cannot be used to establish causality." *Id.* at 5.

Lastly, Dr. Forsthuber responded to Dr. Andersson's assertion that a memory response is responsible for Petitioner's molecular mimicry reaction to the flu vaccine after 15 previous exposures. Resp't's Ex. E at 5–9. He explained that a memory response only occurs when the adaptive immune system's T and B cells are "exposed to the same recall antigen for a second time." *Id.* at 6. Dr. Forsthuber noted that the flu vaccines that Petitioner received from 2014 to 2016 all "contained hemagglutinin derived from the identical vaccine strains A/California/7/2009 (H1N1)-like virus, B/Brisbane/60/2008-like (Victoria lineage), and B/Phuket/3073/2013-like (Yamagata lineage)." *Id.* Despite this, she did not develop TM following any of these vaccinations. *Id.* Further, antigen presentation and cell activation "reasonably takes an estimated one to two days, even for memory T cells, to be become activated by secondary immunization with their recall antigens." *Id.* Dr. Forsthuber argued that once that process is complete, additional time is needed for tissue damage to occur and "become detectable in [the] form of neurological deficits." *Id.* In the case of B cells, memory cells "do not produce antibodies," and need four to five days to "develop into antibody secreting cells." *Id.* at 6–7. Dr. Forsthuber asserted that in the Krammer et al. study,[28] individuals without previous exposure to SARS-CoV-2 mounted an antibody response "within [nine to] 12 days after vaccination," while "individuals with preexisting SARS-CoV-2 immunity showed an increase in IgG antibodies by [five to eight] days after vaccination, but not before that." *Id.* at 8 (citing Resp't's Ex. E, Tab 9). He concluded that "[e]ven after adoptive transfer of large numbers of pre-activated, autoimmune T cells, it takes approximately four to six days before the onset of clinical disease becomes notable." *Id.* This explanation, according to Dr. Forsthuber, is based on "reliable and scientific facts," and "soundly disprove what Dr. Andersson claim[ed] as a theory." *Id.* at 9.

[28] F. Krammer et al., *Antibody Responses in Seropositive Persons After a Single Dose of SARS-CoV-2 mRNA Vaccine*, 384 NEW ENG. J. MED. 1372 (2021).

## IV. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) that petitioner suffered an "off-Table injury," one not listed on the Table, as a result of his receiving a covered vaccine. *See* § 11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). In this case, Petitioner does not allege a Table injury and must prove that her injury was caused-in-fact by a Table vaccine.

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. § 13(a)(1)(A). A petitioner is required to prove 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 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of the Dept. of Health & Hum. Servs*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). In *Broekelschen v. Sec'y of Health and Hum. Servs.*, the Federal Circuit recognized that in some circumstances, the special master may "first determine which injury was best supported by the evidence in the record before applying the *Althen* test." 618 F.3d 1339, 1346 (Fed. Cir. 2010). This principle also means that a petitioner must establish that the vaccinee suffers the injury allegedly linked to the vaccination. *Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343, 1353–54 (Fed. Cir. 2011).

The *Althen* test requires petitioners to set forth: "(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." 418 F.3d at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano,* 440 F.3d at 1326.

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine . . . at issue cause the type of injury alleged?" *Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge . . . the presence of pathological markers or genetic disposition, or general acceptance in the scientific or

medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322 and *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357,1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*requires that courts determine the reliability of an expert opinion before it may be considered as evidence. 509 U.S. 579 (1993). However, in the Vaccine Program, the *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted."); *see also Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the

> factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

*Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors are "meant to be helpful, not definitive." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 151 (1999). The factors do not "constitute 'a definitive checklist or test'" and may be applied differently depending on the facts of a particular case. *Id.* at 150 (quoting *Daubert*, 509 U.S. at 593).

"In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Daubert*, 509 U.S. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Nothing requires the acceptance

of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *24 (Fed. Cl. Spec. Mstr. Nov. 28, 2016) (stating that the Vaccine Act "require[s] a chain of reliable propositions supporting [a] petitioner's theory").

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually caused the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove . . . both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. This is because "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.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not "binding on the special master or court." § 13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record." *Id.*

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due

to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Fed. Cl. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Hum. Servs*., 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that he suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen*, 35 F.3d at 547. In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Knudsen*, 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden then shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that the respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" § 13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

## V. Discussion

### A. Diagnosis

Petitioner alleged that she developed TM as a result of her flu vaccine. Respondent countered that Petitioner suffered from SCI unrelated to her vaccination. 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. *Id.* In this case, the medical records provide some evidence for both perspectives, but ultimately Petitioner's burden is to provide preponderant evidence that she suffered from TM. Petitioner failed to do so.

There is strong disagreement between the parties' experts, Drs. Andersson and Jamieson, on Petitioner's diagnosis, and they both discuss the significance of the presence or absence of certain criteria used to distinguish TM from SCI. These criteria are key supporting factors for each expert's respective opinion.

Dr. Andersson asserted that the proper diagnosis for Petitioner is TM based on the TMCWG's criteria. Dr. Andersson noted that Petitioner developed sensory, motor, or autonomic dysfunction attributable to her spinal cord that was bilateral with a clearly defined sensory level. Petitioner's neurological examination on September 30, 2016, revealed "an upper motor neuron pattern of weakness in the bilateral lower extremities." Pet'r's Ex. 5 at 11. She also exhibited allodynia starting at T5-T7. These symptoms are consistent with TM; however, Dr. Jamieson more strictly applied the inclusionary criteria of TM to Petitioner's case. She noted that the TMCWG's diagnostic criteria, according to the group's guidance, requires evidence of inflammation within the spinal cord. Without evidence of inflammation via gandolinium enhancement and abnormal CSF results, the TMCWG advised that "noninflammatory myelopathy should be considered." Pet'r's Ex. 25 at 4. Furthermore, Dr. Jamieson made the rudimentary point that myelitis by definition, indicates inflammation of the spinal cord. Conversely, Dorland's Medical Dictionary includes in the definition the disclaimer that "[i]n practice, the term is also used to denote noninflammatory lesions of the spinal cord." Myelitis, DORLAND'S MED. DICTIONARY ONLINE. Throughout her treatment, Petitioner's medical records maintained a diagnosis of thoracic myelopathy, but TM is not always listed. Her treaters also noted "[t]he abrupt onset of symptoms was initially concerning for ischemia to the cord." Pet'r's Ex. 5 at 12.

Dr. Andersson further argued that Petitioner suffered from TM because "Petitioner's pain was not severe and so not discriminatory, and at times not even midline in location." Pet'r's Ex. 10 at 13. He reasoned, supported by filed literature, that SCI patients more often have severe limb or back pain at onset. Petitioner's medical records from the September 30, 2016 Mayo Clinic ED record a complaint of "discomfort in her lower thoracic midline spine" that "worsened with movement," and could not be alleviated with over-the-counter pain medication. Pet'r's Ex. 5 at 5. Based on this account, combined with reported leg weakness and numbness, treaters initially considered TM and GBS. This is consistent with Dr. Andersson's opinion.

Dr. Jamieson argued that many patients who are initially suspected of suffering from TM are diagnosed with another type of myelopathy following further evaluation. In Petitioner's case, an MRI completed on September 30, 2016, revealed no obvious lesion or evidence of compression. Her treater also noted "right lower extremity weakness predominantly in an upper motor neuron pattern with generally brisk reflexes." Pet'r's Ex. 5 at 15. Petitioner's diagnosis was changed to SCI versus thoracic myelopathy due to a lack of inflammation or demyelination. Dr. Jamieson argued that Petitioner's ipsilateral unilateral leg weakness hemiparesis, contralateral loss of pain/temperature beginning two dermatomes below the lesion, and ipsilateral loss of light touch, proprioception, and vibration below the lesion are characteristic of SCI. *See* Resp't's Ex. A at 13.

Dr. Andersson argued that SCI is excluded as a possible diagnosis because Petitioner's temporal profile "at a minimum 39 hours and 47 minutes from onset to nadir," is too long. Pet'r's Ex. 10 at 15. He cited the Zalewski et al. article's findings to support his claim, which recorded maximum deficits in 90% of the study's patients by 24 hours, and that "the 10% with a nadir longer

than 24 hours all had a stuttering and stepwise decline and all had 'severe' deficits already by 12 hours." *Id.* Dr. Jamieson responded to Dr. Andersson's argument on this point over the course of two pages in her initial report. However, her explanation merely consisted of a general statement that Petitioner's symptoms started on September 29, 2016, and worsened the next day. *See* Respt's' A at 13. She noted that in 23% of patients, it can take over 12 hours from symptom onset to nadir but provided no additional information on longer periods more consistent with the time frame in Petitioner's case. Dr. Jamieson argued that Petitioner's variable presentations upon examination were consistent with a stuttering course seen within 12 hours in patients with a longer period from onset to nadir. Petitioner's treaters do not note this factor in Petitioner's records and do not base her diagnosis on the timing of her symptom onset or nadir of symptoms.

Dr. Jamieson also discussed exclusionary criteria for TM and argued that Petitioner had a "clear arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery" that rules out TM. Resp't's Ex. F at 1. Petitioner's pinprick sensory deficit was inconsistent, and Dr. Andersson disagreed with this assertion outright.

Particularly in a case like this where the experts don't agree, the opinions of Petitioner's treating physicians, even in their equivocations, are instructive, because they are "in the best position" to determine Petitioner's injury. *See Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326; *Cucuras*, 993 F.2d at 1528 (noting contemporaneous medical records, "in general, warrant consideration as trustworthy evidence"). Petitioner's treaters remained unable to make a definitive diagnosis during the course of her hospitalization. They maintained the differential diagnoses of paraparesis, SCI versus thoracis myelopathy/myelitis. A medical record dated October 3, 2016, recorded neurologist Dr. Jones' opinion that Petitioner likely had SCI. *See* Pet'r's Ex. 5 at 42. Petitioner's neurology hospital service records at the time of discharge provide the most definitive assertation by a treater, namely that she had probable SCI. *See id.* at 51. There is no mention of TM in this record, but myelitis does reappear later in the record as a differential.

Petitioner has the burden of establishing each of the elements of her claim by a preponderance of the evidence. There is evidence in the record that TM was considered by Petitioner's treaters as a possible diagnosis. This is seen in the multiprong treatment approach from her doctors, the continued testing and follow-ups, the precautionary vaccination prohibition, and the equivocation in the medical record. However, the neurology records from Petitioner's treatment at the most crucial points during her hospitalization and throughout the trajectory of her condition noted probable SCI. I am not a physician, and it is not possible nor required that I accurately diagnose Petitioner when her treaters could not, and the medical experts disagree. I must determine if, based on the evidence presented, Petitioner's has established it more likely than not that she suffers from TM. The evidence does not identify a definitive diagnosis, and the "probable SCI" note is the most decisive opinion from a treater. Therefore, Petitioner has failed to establish TM as the more likely than not condition she suffers from.

### B. *Althen* Prong One – Medical Theory

Dr. Andersson asserted that the "etiology of vaccination-induced TM is the same as" in GBS cases. Pet't's Ex. 10 at 10. He argued further that this theory is applicable to many other myelin-related disorders, including MOG-Antibody disorder and neuromyelitis optica. Dr.

Andersson acknowledged that the published literature to support his theory is rare as applied to TM, but he explained that molecular mimicry is commonly accepted as a mechanism for vaccine-caused neurological disease and TM specifically. In support of this contention, Dr. Andersson relied on general references from sources that suggest a link without further analysis. For example, Dr. Andersson referred to the NINDS TM Fact Sheet for evidence of a causal link between flu vaccination and TM. The authors did note that TM can result from "a post-vaccine autoimmune phenomenon, in which the body's immune system mistakenly attacks the body's own tissue while responding to the infection or, less commonly, a vaccine." Pet'r's Ex. 14 at 2. However, this nonspecific reference to molecular mimicry does not articulate a sound and reliable theory that connects the flu vaccine to TM. Dr. Andersson admitted that he is limited by existing knowledge and without specifics of shared homology for cross-reactivity or pathological antibodies for evidence specific to flu and TM. He further admitted that a direct causal relationship "has still not been definitively established." Pet'r's Ex. 10 at 10. In fact, none of Petitioner's submissions endeavor to explain how the flu vaccine causes TM via the mechanism of molecular mimicry. The papers that do deliberate a causal process for vaccine-induced disease focus on the polio, rabies, and hepatitis B vaccines. Dr. Forsthuber argued that these vaccines are not similar to the flu vaccine, and Dr. Anderson did not explain why papers discussing these vaccines are instructive or identify relevant comparable qualities to the flu vaccine. I must stress that conclusive medical literature is not a requirement in order to establish causation in the Program. However, to the extent it is filed and relied upon, the authors' opinions, suggestions, and omissions may be considered. *See Knudsen*, 35 F.3d at 549; *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano*, 440 F.3d at 1325–26).

Dr. Forsthuber's report conceded that, although not perfect, molecular mimicry could be a viable mechanism for neurological disease under very rare and specific circumstances. He defined molecular mimicry generally as a pathogenic cross-reaction between microorganism antigens and host antigens that induce immune cells and antibodies and cause autoimmune disease. However, he asserted that other processes, such as bystander activation, may also be necessary for the type of sustained immune response that could dysfunction and ultimately lead to autoimmune disease. I find this reasoning persuasive. Furthermore, as my colleagues and I have said in previous decisions, to the extent that molecular mimicry is offered as a theory, it must be supported by a sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548.

There must also be some degree of selectivity. *See W.C. v. Sec'y of Health & Hum. Servs.,* 704 F.3d 1352, 1360 (2013) (finding that a petitioner cannot prevail by simply invoking a biological term, or by showing that the mechanism is a valid theory to explain how *other* triggers may have induced *other* diseases and determining that a petitioner must produce additional evidence that the mechanism can cause that vaccine to cause a specific disease); *Caves v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 135 (2011), *aff'd*, 463 F. App'x. 932 (2012); *McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451, 2019 WL 4072113, *50 (Fed. Cl. Spec. Mstr. July 15, 2019). Petitioner does not have to provide a specific mechanism, but it must be detailed enough to apply to the administered vaccine and alleged injury in this case. Otherwise, any vaccination, by nature of its purpose to illicit an immune response, could be asserted as the cause of any autoimmune disease that later developed in an individual, and *Althen* prong one "would be rendered meaningless." *See Caves*, 100 Fed. Cl. at 135; *see also McKown*, 2019 WL 4072113, *50 ("[M]erely chanting the words 'molecular mimicry' in a Vaccine Act case does not render a

causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or the vaccine in question."). Petitioner is not limited to any one type of evidence in support of a molecular mimicry mechanism. A non-exhaustive list of potential evidence includes homology evidence, epidemiology studies, pathogenic antibodies, relevant animal models, or disease etiology specific to the vaccine's live virus counterpart. *See Broekelschen*, 618 F.3d 1339; *Dougherty v. Sec'y of Health & Hum. Servs.*, 141 Fed. Cl. 223 (2018); *Brayboy v. Sec'y of Health & Hum. Servs.,* No. 15-183, 2021 WL 4453146, (Fed. Cl. Spec. Mstr. August 30, 2021). I reiterate, Petitioner is under no obligation to provide any one or more of those types of evidence. This list serves only to illustrate that even in cases of rare and understudied phenomena, Petitioner's must provide preponderant evidence of causation in support of any identified mechanism. Petitioner did not provide such evidence here, and therefore, does not meet her burden pursuant to *Althen* prong one.

### C. *Althen* Prong Two – Actual Causation

It is difficult to evaluate whether a Petitioner has provided preponderant evidence of a causal relationship between her vaccination and injury when preponderant evidence of the injury has not been provided. That difficulty is magnified when Petitioner has not provided preponderant evidence that the injury alleged can be caused by the vaccine, because the mechanism itself is unclear. Dr. Andersson noted the absence of any other known cause for Petitioner's condition and her symptom onset within two days of vaccination as evidence of causation. He declared "to a reasonable medical probability" that Petitioner received a flu vaccine on September 28, 2016, and became symptomatic at approximately 9:00 pm the next day. Pet'r's Ex. 10 at 12. He then declared that TM was the cause of Petitioner's symptoms. At no point during the section in Dr. Andersson's report that purported to discuss actual causation is the molecular mimicry theory applied to Petitioner. Dr. Andersson correctly titled the section: "A Logical Sequence of Cause and Effect," but he neglected the directive that the sequence must show the vaccination was the reason for the injury. *See id.*

In this case, Petitioner presented an association between her vaccine and her condition, specifically that the former directly preceded the latter. However, a chronological relationship and lack of alternative cause, without more, are insufficient to meet the preponderant standard. Furthermore, as discussed below, the temporal relationship between Petitioner's vaccination and symptom onset is not consistent with the general understanding of molecular mimicry as asserted. Recall challenge notwithstanding, Dr. Andersson did not analyze Petitioner's clinical presentation in the context of his theory to distinguish an idiopathic TM or SCI from what happened in Petitioner's case. He argued that the specifics of the immune pathogenesis of TM are unknown, including "how exactly and by what antigen exactly." Pet'r's Ex. 31 at 8. However, "its existence as a disease and its association with vaccination stands undiminished by this ignorance, and so too the theory [he] offered to explain all that is known, with what is currently known." *Id.* at 9. This acknowledgment that there is no mechanism or subsequent application to Petitioner's presentation best illustrates the lack of preponderant evidence here to satisfy *Althen* prong two.

**D. *Althen* Prong Three – Temporal Relationship**

There is also strong disagreement between the parties' experts, Drs. Andersson and Jamieson, regarding both the onset and progression of Petitioner's symptoms. The temporal relationship between vaccination and symptom onset is a key supporting factor for the viability of a molecular mimicry mechanism in this case. Dr. Andersson argued that Petitioner's symptoms began two days post vaccination, while Dr. Jamieson more precisely placed Petitioner's initial feeling of fatigue "in the back of her legs," at 1.5 days post vaccination. Pet'r's Ex. 4 at 1; *see* Resp't's Ex. F at 3. The medical records do not specify what time of day Petitioner received her flu shot; and Petitioner's expert ultimately incorporated 1.5 days into his supplemental report, asserting that this shortened time is of no consequence.

However, Dr. Andersson argued that "the etiology of vaccination-induced TM is the same as for vaccination-induced [GBS]." Pet'r's Ex. 10 at 10. The appropriate timeframe for symptom onset of flu vaccine-caused GBS, as articulated on the Vaccine Injury Table, is between three and 42 days. 42 C.F.R. § 100.3(a)(XIV)(D). Indeed, the initial immune system processes that occur as a prerequisite to molecular mimicry and are described by Dr. Forsthuber, including T and B cell activation and the production of IgG antibodies, occur during this time. Dr. Andersson addressed this discrepancy by conceding that a 1.5-day time frame is shorter than the case studies he presented. However, he relied on case studies with two-day (ADEM) and four-day (TM) onset periods and reiterated that the difference of a few days is inconsequential. It is unclear whether he is contending that Petitioner's case is an example of "the activation of a memory response," or "a primary immune response and this rapid onset of [TM]." Pet'r's Ex. 10 at 17. The latter argument is inconsistent with his supplemental report. Dr. Andersson stated that "[a] probable and logical theory was offered in my report by invoking a memory response for the rapid time course and molecular mimicry as mechanism for the host attack." Pet'r's Ex. 31 at 8. He never provided a mechanism based on a primary immune response that is consistent with such a short onset. In fact, Dr. Andersson asserted that the short onset of Petitioner's symptoms "demand . . . a memory response mechanism." *Id.* In the case of the former, Dr. Forsthuber noted the peculiarity of Petitioner experiencing a pathological memory response that caused a neurological disorder after 15 prior exposures to the flu vaccine without incident. Dr. Andersson's response to this concern was that "[t]he demand for a probable theory [is] unreasonable." *Id.* He argued that this "is a skepticism that could be applied to every case of vaccination associated [TM] with a prior similar vaccination, and every case of vaccination associated [GBS] with a similar prior vaccination too." *Id.* Petitioner did not provide any information on the number of GBS-flu claims involving a Petitioner previously vaccinated over a decade without incident. Petitioner did not acknowledge that Table cases, which require a three-day onset, do not rely on a biological mechanism including a memory response. Petitioners are not required to provide conclusive evidence to establish any element of their claim. The temporal relationship need only be a medically acceptable inference, "given the medical understanding of the disorder's etiology." *de Bazan*, 539 F.3d at 1352. Dr. Andersson's reliance on memory response to account for the shortened time frame is ill-reasoned and not persuasive.

Decisions from other special masters where petitions alleging vaccine-induced TM in the Program have been dismissed for similar onset being found too close in time to vaccination to be medically reasonable. *True v Sec'y of Health & Hum. Servs.,* No. 21-1100V, 2025 WL 1343027

at *30 (Fed. Cl. Spec. Mstr. April 1, 2025); *See, e.g., Martinez ex rel. W.M. v. Sec'y of Health & Hum. Servs.*, No. 16-738V, 2022 WL 4884923, at *27 (Fed Cl. Spec. Mstr. Sept. 9, 2022) ("Because TM is reasonably understood to be mediated by an autoimmune reaction involving antibodies or other immune cells associated with the adaptive, lagging immune response in reaction to antigenic exposures . . . , a relatively short onset timeframe is simply not medically acceptable." (emphasis omitted)); *Palattao v. Sec'y of Health & Hum. Servs.*, No. 13-591V, 2019 WL 989380, *35 (Fed. Cl. Spec. Mstr. Feb. 4, 2019) (citing dismissals of cases with 24-hour onset for vaccine-related TM and finding a 30- to 36-hour onset to not be medically acceptable); *Brancheau v. Sec'y of Health & Hum. Servs.*, No. 21-1209V 2024 WL 1619606, at *23-26 (Fed. Cl. Spec. Mstr. Mar. 21, 2024) (finding a one-day onset of TM following flu vaccination not appropriate given the theory of molecular mimicry).

Specifically, in *Helen Forrest v. Sec'y of Health & Hum. Servs.*, the petitioner's expert proposed that the flu vaccine can cause TM via molecular mimicry. No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 28, 2019). To explain the 36-hour onset, the expert proposed that the petitioner had a recall response due to previous flu vaccinations. *Id.* at *4. The special master found that "[e]ven if molecular mimicry could be accepted to explain how the flu vaccine can cause [TM] abstractly, . . . a preponderance of the evidence shows that molecular mimicry is not likely to happen within 36 hours, even for a recall response." *Id.* at *6; *see also Mosley v. Sec'y of Health & Hum. Servs.,* No. 08-724V, 2015 WL 2354316, at *19 (Fed. Cl. Spec. Mstr. Apr. 27, 2015) (denying compensation where "onset of TM one day after tetanus vaccination [was] too soon to support vaccine causation"); *Jagoe v. Sec'y of Health & Hum. Servs.*, No. 08-678V, 2012 WL13036265, at *28 (Fed. Cl. Spec. Mstr. Aug. 3, 2012) (finding a 24-hour onset not medically appropriate for a vaccine-induced TM injury); *Crosby v. Sec'y of Health & Hum, Servs.*, No. 08-799V, 2012 WL 13036266, at *38–39 (Fed. Cl. Spec. Mstr. June 20, 2012) (same).

Petitioner has not provided preponderant evidence of an appropriate temporal relationship for the onset of TM symptoms following flu vaccination generally, or in Petitioner's specific case. Accordingly, Petitioner has failed to satisfy *Althen* prong three.

## VI. Conclusion

After a careful review of the record, Petitioner has failed to prove by preponderant evidence that she suffered from TM as a result of her September 28, 2016 flu vaccination. Further, Petitioner has failed to provide preponderant evidence that her condition was caused by vaccination. Accordingly, I **DENY** Petitioner's claim and **DISMISS** her petition.[29]

**IT IS SO ORDERED.**

s/Herbrina D.S Young
Herbrina D.S. Young
Special Master

[29] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.