

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 18-1353V
Filed: February 25, 2026

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CHERYL DEWIT, *
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Petitioner, *
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v. *
SECRETARY OF HEALTH *
AND HUMAN SERVICES, *
*
Respondent. *
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Howard Mishkind, Esq., Mishkind Kulwicki Law Co., L.P.A., Cleveland, OH, for petitioner.
Catherine Stolar, Esq., U.S. Department of Justice, Washington, DC, for respondent.

DECISION¹

Roth, Special Master:

On September 5, 2018, Cheryl Dewit (“Ms. Dewit” or “petitioner”) filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. §300aa-10, et seq.² (the “Vaccine Act” or “Program”). The petition alleges that Ms. Dewit received an influenza (“flu”) vaccination on October 7, 2015, and thereafter developed transverse myelitis. Petition at 1, ECF No. 1.

An entitlement hearing was held on December 8 and 9, 2022. Additional filings, including expert reports, were required following the hearing. After careful review and analysis of all evidence submitted in this matter, I find that petitioner has failed to provide preponderant evidence that the flu vaccine caused her injury. Accordingly, she is not entitled to compensation.

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, the undersigned finds that the identified material fits within this definition, such material will be redacted from public access.

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

I. Procedural History

The petition and accompanying medical records were filed on September 5, 2018 and assigned to the undersigned the following day. ECF Nos. 1, 4. Respondent filed his Rule 4(c) Report on September 16, 2019. ECF No. 19. Petitioner then filed additional medical records and expert reports over the course of the following year. ECF Nos. 22-23, 25, 27, 30, 32, 35.

Respondent filed responsive expert reports on January 19, 2021. Respondent's Exhibits ("Resp. Ex.") A-D, ECF No. 39. Petitioner filed a supplemental expert report on March 27, 2021. Petitioner's Exhibit ("Pet. Ex.") 62, ECF No. 41. Respondent filed responsive expert reports on May 13, 2021 and on November 23, 2021. Resp. Ex. E-H, ECF Nos. 42, 48.

The parties filed pre-hearing submissions, and a two-day entitlement hearing was held on December 8 and 9, 2022. ECF Nos. 53, 55, 60-61, 63, 65-66.

Following the hearing, additional expert reports, medical literature, and medical records were filed. ECF Nos. 78, 82, 84, 86. The parties filed simultaneous post-hearing briefs on October 6, 2023. ECF Nos. 91-92.

This matter is now ripe for decision.

II. Factual Background

A. Medical History Prior to the Flu Vaccine

Petitioner's past medical history includes severe allergies, asthma, insomnia with sleep apnea, hypothyroidism, vertigo, 18 years of smoking (quit in 1999), obesity, chronic sinusitis, GERD, headaches, and a family history of stroke and multiple sclerosis. Pet. Ex. 4 at 1, 3-4, 7-8; Pet. Ex. 6 at 1, 3, 4; Pet. Ex. 7 at 1, 9; Pet. Ex. 9 at 1; Pet. Ex. 12 at 563; *see generally* Pet. Ex. 5; Pet. Ex. 16. Petitioner reported that she "always" had body aches. Pet. Ex. 82 at 47. She was under the care of an endocrinologist since at least 2007. *See generally* Pet. Ex. 3.

Petitioner received massage therapy from Mr. Jones at Burt Jones Massotherapy³/Burt's Remedies on various occasions since 2010 for back, neck, and shoulder pain. Pet. Ex. 70 at 3-4.

In June of 2013 she presented to her medical doctor and reported "left arm numbness down her entire arm into her thumb which started 2 weeks ago-keeps her awake at night" and weakness in her left arm when elevated. Left arm pain was 7-8/10. No trauma was reported. Examination was normal but for mild bicep tenderness. She was diagnosed with carpal tunnel syndrome. Pet. Ex. 7 at 7.

Petitioner was diagnosed with pre-diabetes in January 2015. Pet. Ex. 9 at 9-11. At that time, she started a weight loss program which included Qsymia (Phentermine-topiramate ER) with

³ Massotherapy refers to the treatment of disease by massage. Massotherapy, DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1095 (33rd ed. 2020) [hereinafter *Dorland's*].

protein-sparing modified fast (“PSMF”). She had lost 35 pounds by April of 2015. *Id.* at 15.

Petitioner received all routine vaccines including her yearly flu vaccines through her employment without event. *See generally* Pet. Ex. 2. Petitioner received the subject influenza vaccine on October 7, 2015 in her left arm while at work. *Id.* at 7.

B. Medical History After the Flu Vaccine

Three weeks after receipt of her flu vaccine, on October 26, 2015, petitioner presented to her endocrinologist for follow up of hypothyroidism and weight management. Pet. Ex. 9 at 30. The record documents that she was doing well and was involved in aerobics and weight training three times a week. She was taking phentermine-topiramate daily. *Id.* She reported weight loss while doing PSMF and taking Qsymia and felt great, but when she reintroduced carbs, she gained 17 pound back and felt sluggish and less energetic. She wanted to restart PSMF. *Id.* at 31. Examination was normal but for some bloating and heartburn. *Id.* A weight loss plan including a restart of PSMF for three months with uric acid and vitamin K checked monthly and weight loss medications was discussed. *Id.* at 32. Her HbA1c had normalized before she put the weight on again. *Id.*

Addressed below in detail are two visits with Mr. Jones at Burt’s Remedies for massotherapy on November 4, 2015 and on November 13, 2015. Pet. Ex. 8; Pet. Ex. 65; Pet. Ex. 66.

Petitioner returned to the endocrinologist on December 7, 2015. She was taking Phentermine daily and had lost 3 pounds. Pet. Ex. 9 at 37-38. Examination was normal. *Id.* at 38. The record includes that she was overweight, goes to the gym, was given another prescription for Phentermine, and was prediabetic. *Id.* at 39. The importance of diet was discussed, and she was to follow up in a month. *Id.*

Ten days later, on December 17, 2015 or 71 days following the subject flu vaccination, petitioner presented to the Painesville Quick Care Center with “numbness and pain in the [left] neck all the way down to [left] hand. Started this morning” which included her left arm, neck, and hand. Pet. Ex. 10 at 2. She reported “constant and sharp” pain that was “uncomfortable” for “several hours.” She stated she “woke up with pain in [left] neck, shoulder area and tingling in [left] arm”. The Review of Symptoms included, “claims joint pain and claims muscle pain” but “denies motor weakness.” *Id.* There was no left scapula winging, atrophy, ecchymosis, erythema, swelling, or scars. *Id.* at 2-3. There was tenderness over the trapezius when pressed but no weakness or tingling of the left lower extremity. *Id.* at 3. The assessment was “[p]ain in left shoulder. Intractable pain – not applicable.” X-rays of the cervical spine were ordered, and she was prescribed muscle relaxers. *Id.*

The following day, December 18, 2015, petitioner presented to the emergency room (“ER”) with left-sided weakness. Pet. Ex. 11 at 4. She was unable to move her left arm or leg and said the pain started in her left shoulder and arm. *Id.* at 10. She rated the pain as 6/10, at worst an 8. She reported onset 12 hours prior to arrival. *Id.* The record also included left shoulder pain two weeks ago. She reported being able to move all extremities last night but awoke this morning at 7am

unable to move her left arm or leg. Sensation was intact and she had good pulses. *Id.* On examination, there was weakness of the left arm and leg, but right arm strength was intact and right leg strength was a 2 out of 4. *Id.* at 12. The ER physician noted left scapular area pain for about 2 months treated with massage therapy with no effective change. *Id.* at 19. “The left shoulder pain progressed yesterday with tingling to the left arm and then subsequently gradually getting weaker.” *Id.* She went to urgent care, had x-rays, and was prescribed Naprosyn for pain. She has since progressed to complete weakness on the left with decreasing ability to control her right lower extremity. Her right arm was normal. She had no other issues. *Id.* The physician assistant documented a 53-year-old with chronic pain in her neck that became acutely worse yesterday. *Id.* at 23. She awoke this morning with weakness in her left arm and leg. She said the last time she had normal strength in her left arm and leg was when she went to bed the night before. *Id.* CT of brain and cervical spine and labs were normal. *Id.* at 22, 62. An MRI of the cervical spine was read as showing mild cervical spondylolysis with mild disc bulges at C3-4 and C6-7 mildly flattening the ventral thecal sac. *Id.* at 23, 56, 60. However, neurologist Dr. Kendis reviewed the film with a colleague and noted an “abnormality along the cervical spine on the T2 weighted images that is likely to cause [petitioner’s] symptoms.” *Id.* at 23. Petitioner scored a 12 on the NIH stroke scale with 4 points for left arm and leg weakness and 2 points for right arm and leg weakness. Light touch sensation was fully intact. It was noted that if her symptoms were due to stroke, it was too late for “TPA” with symptoms present for over 6 hours prior to arrival. *Id.* at 23-24. Patellar reflexes were diminished bilaterally. A lumbar puncture was not recommended. *Id.* at 24. She had no complaints of pain on the right but had increasing difficulty holding her right arm up. Her right side was notably stronger that morning. *Id.* at 15. She was transferred to University Hospital for “Acute neurological change without identifiable cause.” *Id.* at 16.

Petitioner was seen by neurology upon arrival at University Hospital. Pet. Ex. 12 at 562-572. Her history included a 53-year-old with a past medical history of hypothyroidism and prediabetes who presented with worsening left shoulder pain radiating to her left hand for two days and progressive left-greater than right-sided weakness since this morning. “Since about three months ago, patient had neck pain shooting to her whole left hand and left hip, for which she had neck massage without significant improvement (last massage about 2-3 weeks ago).” *Id.* at 562. At 7am on December 17, 2015, she had shooting pain from her neck radiating to her whole left arm which acutely worsened and felt like “someone sitting on her left shoulder & excruciating pain” with numbness. *Id.* She reported needing more time to urinate. She reported cold-like symptoms last week with some sneezing and a cough; her husband had the same. She reported two episodes of transient vertigo, blurred vision and double vision for 2 weeks about 2 months ago and 1.5 years ago. She reported chronic sinus headache for years and 50-pound weight loss in the past year after a diagnosis of pre-diabetes. *Id.* She received a flu shot in September 2015. She had no fever, chills, recent change in vision or hearing, facial numbness, drooping, choking, speech difficulties, skin rash, chest pain, abdominal pain, nausea, vomiting, diarrhea, urinary or bowel incontinence, saddle anesthesia, history of neck trauma, or recent travel. *Id.* She went to Tri-Point for left-sided weakness and while there developed new weakness of the right arm and leg and urinary retention; a Foley was inserted. Her blood cell counts were normal, but the differential showed mild leucopenia with predominant neutrophil.⁴ Urinalysis was negative. MRI of the

⁴ Neutrophils are a type of white blood cell with a primary function of killing and digesting bacterial microorganisms. Acute bacterial infection and trauma will trigger neutrophil production. Leucopenia (or leukopenia) refers to decreased white blood cell count. MOSBY’S MANUAL OF DIAGNOSTIC AND LABORATORY TESTS 466-72 (Elsevier 6th ed. 2018).

cervical spine with and without contrast showed T2 intramedullary hyperintense lesion in the left anterior-lateral spinal cord from C2-C4 (however radiology reported mild spondylitic changes only). Head CT was unremarkable. She was transferred for further evaluation and treatment. *Id.* She had a family history of multiple sclerosis and stroke. *Id.* at 563. Her past medical history included hypothyroidism and pre-diabetes. The “Last Known Well – Date/Time” was listed as December 17 at 7:30am. *Id.* Review of symptoms included dry cough, pain, weakness, dizziness, and headache. *Id.* at 564. Neurological examination revealed no distress, alert, interactive, and cooperative. *Id.* at 566. Motor examination showed normal muscle bulk in the upper and lower extremities, but there was reduced muscle tone diffusely with no fasciculations, tremor, or other abnormal movements. *Id.* at 567. Reflexes were abnormal. *Id.* Sensation was intact to light touch, vibration, and joint position sense. *Id.* at 568. There was reduced sensation to temperature in her arms and legs and pinprick on the left and right arms below the shoulder, but intact on the anterior chest T1-T4 level. Gait and coordination were not done due to weakness. *Id.* The assessment included chest x-ray showing possible right lower lobe nodule, MRI of the cervical spine showing C2-4 hyperintense lesion with no enhancement, quadriparesis left greater than right, decreased sensation to palpation and temperature below C5, possibly spared T1-T4, decreased reflex over left side, upgoing toes, loose rectal tone, and “C2-C4 Myelopathy.”⁵ *Id.* at 569.

The neurology resident’s note included differentials of longitudinally extensive TM suspected to be idiopathic given the lesion involving 3 vertebra and recent cold-like symptoms, however usually acute demyelination would have some enhancement; TM secondary to multifocal neurologic disease with NMO possible since she had eye symptoms in the past; TM secondary to inflammatory process/systemic autoimmune disease also possible; spinal infarction less likely given progressive course and location of the lesion (anterior only on the left); infection less likely because WBC and ESR were normal; metabolic less likely due to acute course and sparing light touch/vibration/proprioception. Pet. Ex. 12 at 570.

Methylprednisolone was started on December 19, 2015, for “C2-C4 myelopathy compatible with longitudinal [TM] concerning for NMO vs [MS] vs other inflammatory/autoimmune etiology.” Pet. Ex. 12 at 597. Some improvement was noted overnight and on December 19, 2015, petitioner was able to raise her right arm more. *Id.* at 599. LP was negative with normal WBC, protein, and glucose, and no oligoclonal bands. *Id.* at 681.

Neurological evaluation on December 21, 2015 revealed improvement on the right side but still plegic on the left. She reported neck pain mainly on the left with similar pain for the past few months, but she sometimes sleeps on the couch with her head tilted in an uncomfortable position according to her husband. She also uses a phone for hours with head tilted. She had increased neck pain and some burning with movement. Pet. Ex. 12 at 618. She received three doses of IV methylprednisolone then prednisone taper by mouth with continued improvement. *Id.* at 686. She was pending placement to rehabilitation. *Id.*

However, on December 28, 2015, she developed profound weakness with nausea and vomiting and required oxygen via nasal cannular. Pet. Ex. 12 at 703. Initial improvement was noted with IV steroids, then no further significant improvement. *Id.* at 705. MRI of the thoracic

⁵ Myelopathy is 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* at 1203.

spine on December 19 revealed herniated disc at T8-T9. *Id.* at 708. She now reported bowel/bladder incontinence and intermittent double vision. *Id.* She continued to deteriorate, became hypotensive, bradycardic, and hypothermic and was transferred to the NSU for monitoring and initiation of BiPAP. *Id.* at 711. Broad spectrum antibiotics were started. *Id.* at 719. There was diffuse congestion on chest x-ray. She developed a fever with marked drowsiness and poor effort or ability to move her left side. Lung sounds were coarse bilaterally. *Id.* IVIG was prescribed. *Id.* at 729. The differential included high cervical cord lesion causing autonomic instability vs. medication effect TTE. *Id.* at 734. Head CT on December 29, 2015 was normal. *Id.* at 208-09.

On December 30, 2015, petitioner was noted as having resolving hypercapnia⁶ of unknown etiology, possibly from spinal cord lesion, anxiety, or medications that potentially suppress respiration. Pet. Ex. 12 at 735. The record included MRI enhancement consistent with TM, respiratory failure, and receiving IVIG. *Id.* at 750. Heparin was slightly increased. *Id.* She had significant improvement by December 31, 2015 and was tolerating IVIG. *Id.* at 752.

The neurology record included a diagnosis of C2-C4 lesion from TM vs. alternative process. Pet. Ex. 12 at 758. MRI of the cervical spine with gadolinium was performed on December 31, 2015 due to clinical history of “High cervical transverse myelitis versus dural AV fistula with a cord infarct.” *Id.* at 225. It was compared with the December 18, 2015 cervical MRI and December 19, 2015 brain MRI. *Id.* It was documented “that the study was not completed with dural AV fistula protocol.” *Id.* at 226. The findings included a “redemonstration of abnormal hyperintense T2-weighted signal throughout the cervical cord from the inferior aspect of the C2 to the superior aspect of the C7 levels. On the prior comparison MRI, abnormal T2 hyperintense signal extended from [C2 to C4] levels. It also now extends to the central and bilateral aspect of the spinal cord and appears more well-defined in appearance when compared with the prior MRI of the cervical spine dated 12/18/2015. These findings present progressive cytotoxic edema within the spinal cord. Following contrast administration there is new patchy curvilinear enhancement of the cervical cord from C2 to C6 levels, primarily involving the gray matter as well as the leptomeninges. Constellation of these findings suggests acute to subacute infarct. Alternatively, transverse myelitis may demonstrate similar appearance although it is felt less likely given the interval development of postcontrast enhancement of the spinal cord and leptomeninges. Potentially, dural arteriovenous fistula with venous infarction may represent an etiology. However, evaluation of the dural arteriovenous fistula is limited on this examination secondary to incorrect protocol.” *Id.* at 226-27. There were multilevel discogenic degenerative changes of the cervical spine unchanged from December 18, 2015. *Id.* at 228. The differential included demyelinating – first episode of MS or NMO – inflammatory/post-infectious, malignant/paraneoplastic, or spinal cord infarct/AVM. There was a lot of spinal cord swelling and tenuous but improving respiratory status. *Id.* at 760. LP was not to be repeated now. It was “[c]urious that first LP so bland.” Continue IVIG with “ultimate diagnosis not clear”. *Id.*

MRA of the brain with contrast on December 31, 2015 showed abnormal FLAIR hyperintense signal along the bifrontal cortex within the vertex, which may represent exudative subarachnoid fluid of unclear etiology, with no evidence of subarachnoid hemorrhage or postcontrast enhancement. The possible etiologies included meningitis or high concentration oxygen therapy. Pet. Ex. 12 at 942-43.

⁶ Hypercapnia is excess of carbon dioxide in the blood. Hypercapnia, *Dorland's* at 876.

On January 2, 2016, petitioner was on day 5 of IVIG for TM vs. alternate process. The plan was to repeat lumbar puncture once the cervical spine lesion had more time for the swelling to reduce. CT of chest, abdomen, and pelvis were to be done that day to assess for malignancy, possibly myelitis as a malignant or paraneoplastic phenomenon. Pet. Ex. 12 at 786. CTs revealed nonspecific pulmonary nodules within the bilateral lungs which may be infectious, inflammatory, or neoplastic; bilateral effusion with associated consolidation representing pneumonia or compressive atelectasis and tiny nonspecific hypodensities within the thyroid. *Id.* at 794. There was no acute abdominopelvic pathology. *Id.*

Petitioner received a final dose of IVIG on January 3, 2016 and was noted to be doing “extremely well.” Pet. Ex. 12 at 790. She was alert, cooperative, oriented, and had a strong voice. There was no dysarthria, diplopia, or facial asymmetry. She had diminished strength of her extremities on the left side and reduced strength on the right. She could wiggle her toes on the right, had sensation throughout, brisk reflexes at her knees, and muted plantar response on the right. *Id.* The assessment remained TM vs alternative process. *Id.* at 794.

Pulmonary was consulted on January 3, 2016 due to chest CT findings on the left side and concern for worsening TM vs. cord infarct vs. malignancy. Pet. Ex. 12 at 803. Bilateral effusions were present with lung base atelectasis. *Id.* at 942. PET scan to better delineate pulmonary nodules was limited due to motion. There was diffuse thyroid activity, bilateral pleural effusions, and bilateral lung-based atelectasis. Thoracentesis to decrease fluid overload was indicated and performed on January 4, 2016. *Id.* at 293-295, 803, 810, 819-20, 942. Repeat LP was done on January 6, 2016 and showed lymphocytes but was negative for B-cell lymphoma. *Id.* at 849, 900-01.

Petitioner developed decreasing platelets (thrombocytopenia) and a GI bleed. Hematology and gastroenterology were consulted with 2 units of blood transfused. Pet. Ex. 12 at 858. She was noted to be stable neurologically with mounting medical issues. *Id.* at 888. Enteroscopy on January 8 showed some gastritis but was otherwise normal with no active bleeding. *Id.* at 906.

MRI of the brain with and without contrast on January 10, 2016 when compared to December 31, 2015 showed abnormal signal and contrast enhancement in the spinal cord especially to the left side. Pet. Ex. 12 at 369, 942. Findings on the brain were compatible with acute to early subacute infarction. *Id.* at 369-71. Due to concern for infarct, the stroke team was consulted. *Id.* at 938. The spinal lesions were now thought to be infarcts rather than TM or other process. *Id.*

A neurology note on January 11, 2016 included new incidental discovery of left cerebellar strokes of unclear etiology with prior imaging unrevealing. It was unclear whether recent issues with GI bleeding and thrombocytopenia were related to new strokes. “TTE with bubble study” was recommended. Pet. Ex. 12 at 945.

On January 18, 2016, following a successful coil embolization of a ruptured left P-comm aneurysm which measured 9.2 mm tall x 6.6 mm wide with a 3mm neck in the left posterior communicating artery that originated from the medial neck of the aneurysm, a cerebral angiogram

was performed for left sided weakness, spinal cord edema/myelomalacia, cerebral infarcts, and to rule out dural arteriovenous fistula. Pet. Ex. 12 at 435. The results revealed unremarkable intracranial and cervical vasculature with no evidence of aneurysm, arteriovenous malformation, or stenosis. *Id.* at 438.

Petitioner was discharged to a rehabilitation facility on January 22, 2016 following bowel and bladder incontinence, right paracentral herniated disc at T8/T9, hypotension, bradycardia, concern for sepsis, hypercapnic respiratory failure requiring BiPAP, IVIG for TM, abnormal signal on brain MRI, prominent C2-C6 lesion on cervical spine MRI, large bilateral pleural effusions, multiple small pulmonary nodules requiring thoracentesis, thrombocytopenia, black tarry stool, repeat brain and C-spine MRIs revealed small cerebellar embolic infarcts without contrast-enhancing lesion, chest pain on January 11, 2016 with shortness of breath, transfer to MICU, likely bradycardia and hypotension episodes due to the presence of a high cervical cord lesion, with susceptibility to autonomic dysregulation causing low HR and BP when vagal nerve was stimulated especially while eating. Angiogram on January 18 had no vascular changes, UTI requiring antibiotics, and discharged on January 22, 2016 to acute rehabilitation on prednisone taper. Pet. Ex. 12 at 626-27. Her final discharge summary problem list included “cerebrovascular accident (CVA) involving cerebellum”, melena, transverse myelitis, stroke, urinary tract infection associated with catheterization, and autonomic dysfunction. *Id.* at 1, 1780.

At Euclid, petitioner’s problem list included impaired mobility and locomotion; impaired activities of daily living; neurogenic bowel dysfunction; dysautonomia; cerebellar infarcts; TM; left hemiplegia; neurogenic bladder dysfunction; and orthostatic hypotension. Pet. Ex. 13 at 1. She made slow progress and was discharged almost two months later on March 21, 2016 to a skilled nursing facility at Heather Hill. *Id.* at 117; Pet. Ex. 14. The admitting diagnosis was transverse myelitis and cerebellar infarcts. Pet. Ex. 14 at 3. She continued to complain of left sided weakness. *Id.*

Petitioner received home care for about a month after discharge, then attended outpatient therapies. She reported to her therapist that she believed her flu shot caused her illness because she developed back and neck pain that became severe a few days after the shot. Pet. Ex. 19 at 164-65.

On May 16, 2016, petitioner presented to the hematologist for follow up of anemia. Pet. Ex. 15 at 1. The impression included recent TM and cerebellar stroke, on Xarelto with heme positive stool and chronic anemia. *Id.* at 3. Colonoscopy was recommended after 6 months and neurology review. *Id.*

Petitioner had a follow up with neurology on May 19, 2016 for TM with left-sided hemiparesis. Pet. Ex. 17 at 4. She had a home health aide, home PT 3 times weekly, and took Xarelto after strokes, discontinued that day. *Id.* at 5. The impression was a 53-year-old with an episode of TM after a flu shot without any other reported etiology found in extensive workup. She was to continue intensive therapy, take gabapentin and tizanidine as needed at night for neck pain, and to follow up in 6 weeks. *Id.* She had in-home care from May 16, 2016 through June 20, 2016. *See generally* Pet. Ex. 19.

Petitioner returned to the neurologist on June 29, 2016 for follow up of cervicalgia and TM. Gabapentin and tizanidine had helped. She was receiving outpatient therapy for her neck, had incontinence at night and shallow breathing. She had increased movement of her left arm and leg. She was starting pelvic rehabilitation next week for incontinence. Pet. Ex. 17 at 4. She was being treated for obstructive sleep apnea with a CPAP. *See generally* Pet. Ex. 20.

Repeat cervical MRI on August 4, 2016 revealed that the previous edema seen on the January 1, 2016 MRI within the cervical cord had largely converted to myelomalacia⁷ change extending from the C2-3 through C5-6 level. Prior enhancement had resolved. Pet. Ex. 22 at 92.

Petitioner had ongoing sequela with left sided weakness as well as other health issues requiring constant treatment. Pet. Ex. 17 at 11.

In January of 2017, petitioner presented to Dr. Serels reporting a history of neck and low back pain 3 days after a flu vaccine in October of 2015. Pet. Ex. 28 at 177. Trigger point injections were started in March of 2017 for left upper extremity weakness “following a prolonged course of transverse myelitis that began in October 2015”. *Id.* at 162.

Petitioner was hospitalized on December 29, 2017 with pneumonia, acute lower urinary tract infection, and dehydration. Pet. Ex. 26 at 3-7. In January of 2018, she received a left myoelectric brace for her left arm weakness to restore function in the left arm. *See generally* Pet. Ex. 25.

The most recent medical records show petitioner’s continued difficulty with ongoing sequela. Pet. Ex. 32 at 5; *see also* Pet. Ex. 39; Pet. Ex. 82. She lost her job, was distressed about losing independence, and is dependent on others for activities of daily living. *See* Pet. Ex. 36. She believes she has undiagnosed GBS from her flu vaccine. *Id.*

C. Affidavit and Testimony of Petitioner

Petitioner submitted an affidavit with her petition and testified at the hearing. Pet. Ex. 1.

Petitioner affirmed receipt of a flu vaccine on October 7, 2015 and “[o]n or about December 17, 2015,” started to have numbness and pain in the left side of her neck that also involved her left arm. She then developed an inability to move her left arm and leg. Pet. Ex. 1 at 1.

According to petitioner, based on the medical records and her physician’s opinion, she believes she developed TM “as a direct result of the flu vaccination administered on October 7, 2015.” She continues to suffer pain and disability, left sided weakness, and neurogenic bowel/bladder. Pet. Ex. 1 at 1-2.

Petitioner testified to being unable to work due to her injuries from the vaccine. Tr. 9-10.

Petitioner stated that she was being treated for hyperthyroidism and was pre-diabetic which she addressed as soon as she learned of it. Tr. 10-12.

⁷ Myelomalacia refers to morbid softening of the spinal cord. Myelomalacia, *Dorland’s* at 1202.

Petitioner previously received flu vaccines in October of 2011, August of 2012, August of 2013, and September of 2014 without complication. Tr. 12.

She did not recall complaining to her family doctor of numbness in her left arm prior to the flu vaccine, only that she had tingling in her left hand thought to be carpal tunnel. A splint and some exercises took care of it. Tr. 12-13. She attributed her “migraines” to work stress. Tr. 13-14. She never had issues with blood pressure or cholesterol. Tr. 14. She periodically saw Burt Jones for back massages of “the trigger points” from sitting all day or for neck pain from sleeping incorrectly, maybe once every three to five months. Tr. 14-15.

Petitioner stated that her arm was sore after she received the flu vaccine on October 7, 2015, then “[w]ithin the two to three weeks”, she had “unbearable” pain in her neck, back and shoulder that she never felt before, like “two elephants” standing on each shoulder pressing down. Tr. 16-17. She went to Burt Jones on November 4, 2015 thinking it was stress or that she slept wrong but never gave any thought to the flu vaccine. Tr. 17, 19. Her pain was so bad her husband had to help her out of bed that day, took her to Mr. Jones using his appointment, and stayed while she was treated. Tr. 17-18. She saw Mr. Jones again on November 13, 2015. She did not see any doctors in between. Tr. 19. She stated that Mr. Jones’ first treatment helped but by evening the pain returned. She was fortunate to be able to see him again within two weeks. Tr. 19. She then stated the treatment did not help. Tr. 19-20.

Petitioner stated there was a lot going on in her life between October 7 and December 17, 2015. Her daughter was part of a competitive dance team with plans to perform at Disney World and she was “one of the dance moms” active with fundraising and coordinating the trip. Her father had mini-strokes, and his wife had Alzheimer’s so she was helping him take care of her. Tr. 20-21. During this timeframe, she had good days and bad days, but didn’t see a doctor with everything going on in her life. She just took Motrin. Tr. 21-22.

Petitioner described the events of December 17, 2015 and December 18, 2015. She went to work but by midmorning had terrible pain, like something pressing down on her neck and pulling her head and neck apart. She went to urgent care, was prescribed muscle relaxers, and given a script to go to the hospital for x-rays of her spine. Tr. 23. She went for the x-rays, filled the script for muscle relaxers, and went home. Tr. 24. Petitioner stated she never experienced the kind of pain she had on December 17, 2015 before which included pain in her left neck and shoulder area with tingling of the left arm. Tr. 23. She still did not feel well the next morning, called out sick from work, and went back to bed. When she got up to go to the bathroom, her legs felt like they were knocked out from under her and she “fell flat on [her] face”. Tr. 24. She still had use of her right hand at the time and called her husband to come home. She was still on the floor when he got home. She couldn’t move, couldn’t roll over or “do anything.” Tr. 25. He put her on the bed and called 911. Tr. 25.

Petitioner stated that she provided her medical history at the hospital on December 18, 2015 of left leg and arm weakness for “two days”. Tr. 25. She then stated she did not recall what she reported at the hospital because it was a “whirlwind” of questions. Tr. 28-29; Pet. Ex. 11 at 10.

Petitioner recalled being transferred to University Hospital by ambulance. By that time, she couldn't move and was being asked a lot of questions including if she had received a flu vaccine. Tr. 30-31. No one ever said anything about a stroke. Tr. 31. She heard them say transverse myelitis, which she never heard of. Tr. 31.

Petitioner stated that she was hospitalized for 5 weeks with tests being done. She was asked every day if she had a flu vaccine. Tr. 32. She had a blood transfusion, pneumonia, and her lungs drained. Tr. 32. Transverse myelitis was her discharge diagnosis, and she was admitted to Euclid for transverse myelitis. Tr. 33. She received 8 weeks of grueling therapy at Euclid and was then transferred to Heather Hill for therapy and blood pressure monitoring. Tr. 33-34. She had home health after being discharged home. Tr. 34.

Petitioner stated after discharge she was followed by Dr. Kendis, the neurologist she saw while in the hospital until 2021 when Dr. Kendis left the practice. Tr. 35. Dr. Kendis told her she had transverse myelitis. Tr. 35. She asked Dr. Kendis why all the doctors kept asking if she had a flu vaccine and "in her roundabout way", Dr. Kendis told her she thought the flu vaccine caused her paralysis. Tr. 35-36. Dr. Kendis never mentioned a spinal cord infarct. Tr. 36, 38.

Petitioner was then treated by Dr. Sunshine who agreed she had transverse myelitis. Tr. 39-40.

Petitioner recalled being told she had "two little minor strokes" and internal bleeding while she was in the hospital. Tr. 41-42. Seven years later, she still receives therapy, uses a wheelchair, has pain and weakness, and needs assistance with activities of daily living. Tr. 40-41.

D. Affidavit and Testimony of Petitioner's Husband

Mr. Dewit is petitioner's husband. Mr. Dewit affirmed that in April of 2016, he called Mr. Jones' office, asked for petitioner's records for two visits in 2015, received "2 invoices for the visits" and without looking at them, and faxed them to counsel's office on a fax machine at a friend's garage. Pet. Ex. 67 at 1; Pet. Ex. 8. He affirmed that the record "clearly in error was filed with the court". Pet. Ex. 67 at 1.

At hearing, Mr. Dewit stated that he was asked by someone at petitioner's counsel's office for receipts for petitioner's visits with Mr. Jones in November 2015. He either called or went to the office, but Mr. Jones and his wife were not there. Another woman was in the office, and he told her the attorney needed records for those dates. Mrs. Jones called him later and said she would have someone get the information for him. Tr. 49-50. He faxed the records to the attorney from a buddy's towing company. Tr. 50. Mr. Dewit stated he had nothing to do with any other records from Mr. Jones. Tr. 50-51.

Mr. Dewit stated that he had been seeing Mr. Jones for around twenty years due to his work as foreman and heavy machine operator. He has since retired. Tr. 45-47. He stated that petitioner may have seen Mr. Jones once or twice a year before 2015. Tr. 48.

Mr. Dewit recalled petitioner receiving a flu vaccine in mid-to-late October and her arm

being sore. But then she got worse. Tr. 49. He had an appointment with Mr. Jones scheduled for November 4, 2015, but petitioner started “complaining of back, shoulder stuff” couldn’t sleep and was having a hard day so he gave his appointment to her because Mr. Jones had no other appointments available. Tr. 47. He stayed in the room during petitioner’s treatment but “I really did not think too much of it.” Tr. 48. Petitioner scheduled an appointment for November 13, 2015 because she was still having problems sleeping and with her with back. “It seemed like it was better for a while...but...still there.” Tr. 48.

Petitioner stated that when petitioner was in the hospital, they ran all kinds of tests and after everything was eliminated said she had transverse myelitis. Tr. 51-52. He recalled a conversation with Dr. Kendis after petitioner was out of the hospital about transverse myelitis and Dr. Kendis said on rare occasions transverse myelitis is triggered by flu vaccine. Tr. 52-53.

Mr. Dewit stated that petitioner will never walk again but she works really hard at therapy and maybe with time will get better but will never be the same. Tr. 53-54; *see also*, Pet. Ex. 67 at 2.

E. Affidavit of Hallie Kendis, M.D.

Dr. Hallie Kendis, a neurologist, treated petitioner.⁸ She submitted an affidavit. Pet. Ex. 21. According to her affidavit, petitioner established care with her in May of 2016, seven months after the subject vaccination.⁹ Pet. Ex. 21 at 1.

Dr. Kendis was familiar with medical literature that discussed the association between transverse myelitis and influenza vaccination. Pet. Ex. 21 at 1. No other etiology was found for petitioner’s TM after extensive work up. Dr. Kendis concluded that, “given the association for this clinical syndrome in which an immune-mediated process causes neural injury to the spinal cord, [petitioner’s] history and symptoms are entirely consistent with a post-influenza vaccination neurological complication.” While impossible to establish a definite causal relationship, given the “mechanisms by which vaccines may induce TM, it is reasonable to conclude that in her case, it is probable that her TM is vaccine related.” *Id.* at 1-2.

Dr. Kendis did not affirm that she reviewed all petitioner’s medical records, MRIs, and test results and did not address other diagnoses included in the differentials.

F. Evidence from Burt Jones

There were several filings from Burt’s Remedies over the course of these proceedings. Pet.

⁸ Dr. Kendis was sent a letter by counsel seeking her assistance in proving petitioner’s case and enclosing the Vaccine Injury Table and a proposed affidavit for her to sign. Dr. Kendis informed counsel that she could not sign the affidavit. Counsel sent another letter asking for the opportunity to discuss the content of the proposed affidavit, assuring Dr. Kendis she would not have to testify. Counsel also wrote he was certain she would “want to do the right thing for [her] patient and help as much as [she] can.” Dr. Kendis then signed an affidavit filed as Pet. Ex. 21. Pet. Ex. 22 at 68, 82-86.

⁹ Dr. Kendis was the neurologist at Tri-Point when petitioner presented on December 18, 2015 and read the MRI on that date with a colleague. Pet. Ex. 11 at 23.

Ex. 8; Pet. Ex. 65; Pet. Ex. 66; Pet. Ex. 70.

The first record filed includes visits on 11-13-16 and 1-4-16 (the page was cut off and could also be read as 11-4-16). Both records include checkmarks next to hot/cold packs and soft tissue mobilization under “office services”; back pain and restriction of motion under “diagnosis”; and cervical, thoracic, and shoulder under “strains/sprains”. Pet. Ex. 8. These were the records Mr. Dewit faxed from his friend’s business. Tr. 50. The timestamp on the fax is April 14, 2016 at 7:27am. Mr. Jones affirmed that these records were erroneous because at least one visit date (11-13-16) had not yet happened at the time of the fax. *See* Pet. Ex. 8; Pet. Ex. 67.

A second set of records filed contains additional pages, but most important to this case, visits on 11-4-2015 and 11-13-2015 but not the visits on 11-13-16, 1-4-16, or 11-4-16 contained in Exhibit 8. Apart from the date and a seemingly different handwriting, both visits contain the same checkmarks as contained in the Exhibit 8. *See* Pet. Ex. 65. Along with the filing of this record, Mr. Dewit’s affidavit (discussed above) and an affidavit of Mr. Jones (discussed next) were filed. Pet. Ex. 66; Pet. Ex. 67.

Mr. Jones affirmed that he is a licensed medical massotherapist. Petitioner was his patient “beginning on November 4, 2015.” Mr. Dewit, a patient prior to this date, asked that his wife be seen on November 4 at his scheduled appointment to address “some nonspecific symptoms involving her left shoulder, arm and neck. [He] provided soft tissue mobilization to the areas involved.” Pet. Ex. 66 at 1. He provided the same treatment to petitioner on November 13, 2015. He did not see her again until February of 2017. *Id.*

Mr. Jones affirmed that upon review of his records and calendars, his office was closed on January 4, 2016, and he did not see petitioner on November 13, 2016 and therefore Exhibit 8 is incorrect. Pet. Ex. 66 at 2. He provided no explanation for the existence of the records filed as Exhibit 8. *Id.*

Mr. Jones then wrote a letter dated October 12, 2022, in response to a request from petitioner’s counsel, that he learned that Mr. Dewit requested statements for services rendered while he and his wife were out of the office and records were generated in error. “Bottom line, [petitioner] was seen for her acute onset of symptoms for the very first time on November 4, 2015.” Pet. Ex. 70 at 1. He then discussed his review of Dr. Lindsey’s expert report (provided by counsel) which addressed Mr. Jones’ records and, upon “further [search of] my archives” was able to locate the original notes from the visits in question and attached them to his letter. He wrote that these notes were “made at the time of [his] original examinations and contain to the best of [his] memory the sum and substance of the dates that [petitioner] first appeared” and that petitioner’s symptoms at those visits “were profound and some of the most difficult therapy sessions” he had ever performed on a patient. *Id.*

Also included with this filing were handwritten visit notes for various visits beginning in 2010. The note for 11-4-15 starts with “see next page . . . extensive notes”, with the remainder of the page left blank. Pet. Ex. 70 at 4. That “next page”, also dated 11-4-15, contains details of petitioner’s symptoms and the treatment provided. *Id.* at 5. A note for 11-13-15 includes severe headaches with back, neck, and shoulder pain which feels like an elephant sitting on her shoulders.

“I will make an assessment (extensive) along w/ treatment that I did.” *Id.* at 6-7. The next seven pages are typed therapy notes only for the 11-4-15 and 11-13-15 visits with elaborate details about petitioner’s presentation. There is no indication when this document was created. *Id.* at 8-14.

Contrary to his affidavit in which Mr. Jones affirmed treating petitioner for nonspecific symptoms involving her left shoulder, arm, and neck, the typed therapy notes document treatment for “severe bilateral pain in her neck, shoulders, and thoracic back area” with “terrible burning sensations” and “severe tight, spastic muscle pain with muscle spasms in her shoulders and thoracic back area” and that she developed a “considerable gelotic mass at the base of the Occiput (skull) that is causing inflammatory responses in the areas mentioned above.” Pet. Ex. 70 at 8; Pet. Ex. 66 at 1. Mr. Jones then detailed his treatment. Pet. Ex. 70 at 8-14.

The various versions of Mr. Jones’ records are concerning. No explanation was provided for the existence of Exhibit 8 which documented visits in 2016 that apparently never occurred. Exhibit 65 provided little clarification and Mr. Jones affirmed treating petitioner for “nonspecific symptoms”, making it difficult to reconcile the later appearing letter with therapy notes describing “the most profound symptoms he had ever treated” some handwritten some typed. Further, Mr. Jones affirmed “first” seeing petitioner on November 4, 2015, yet he later produced records showing that he treated her dating back to 2010. Finally, Mr. Jones’ therapy records detailing extreme debilitating symptoms conflict with petitioner’s medical visits with her medical provider in which she reported no complaints and was going to the gym during the same time period. *See* Pet. Ex. 9 at 37-39. Therefore, I do not find these records reliable and, although considered, are given little weight.

III. Expert Opinions

A. Petitioner’s Expert, Dr. Michael Levy¹⁰

Dr. Levy was recognized as an expert in neurology and neuroimmunology. Tr. 66. Although he has no specialized training in reading MRI films, he stated that in practice he reviews films and does not always agree with the radiologists’ interpretation or defer to them.¹¹ Tr. 101-05.

Dr. Levy detailed petitioner’s medical history as follows: flu vaccine on October 7, 2015; normal neurological exam on October 19, 2015; normal neurological examination on October 25,

¹⁰ Dr. Levy is a neurologist with eleven years of clinical and research expertise in rare neuroimmunological disorders. Pet. Ex. 42 at 1. He graduated with his M.D. and his PhD from Baylor College of Medicine and completed his residency at Johns Hopkins Neurology. *Id.* Dr. Levy was previously on the faculty at Johns Hopkins University and was the Director of the Neuromyelitis Optica Clinic at Johns Hopkins Hospital. *Id.* He currently leads the Neuroimmunology research unit at Massachusetts General Hospital and Harvard Medical School where he manages over 425 patients, eighty of whom have transverse myelitis. *Id.* He has published more than 125 peer-reviewed research articles and holds three patents covering tolerization therapy, TM diagnostics, and stem cell regeneration approaches. *Id.* Additionally, Dr. Levy is the Chief Editor of Multiple Sclerosis and Related Disorders and sits on the editorial board for the Journal of Neurological Diseases and on the scientific advisory boards for Alexion, MedImmune, Chugai, Shire, and Quest Diagnostics. *Id.*

¹¹ Respondent’s counsel objected to whether Dr. Levy was qualified to offer an opinion on the radiological images in this case. I agreed that Dr. Levy could offer his opinion based on his experience in reading films but noted that he was not a specialist who is trained to read films. His testimony on this topic will be weighed accordingly. Tr. 102-05.

2015; onset of left-sided neck and arm pain 45 days after flu vaccine on November 21, 2015; normal neurological examination on December 7, 2015; acute onset of sensory changes on December 17, 2015 to the left side of her neck, shoulder, and arm with no weakness prompting a visit to Painesville Quick Care Center; ER visit on December 18, 2015 for left arm and leg weakness and ongoing sensory changes in those areas. Normal brain MRI, stroke ruled out, and transfer to University Hospital where an MRI confirmed longitudinally extensive T2 hyperintense lesion from C2-4, spinal fluid normal other than elevated myelin basic protein (“MBP”). She received 3 days of high dose IV corticosteroids followed by oral steroids on December 19, 2015; episode of bradycardia, hypothermia, and CO₂ retention prompting transfer to ICU attributed to TM, and IVIG started on December 28, 2015; evolving cervical lesion with new patchy post contrast enhancement including meningeal enhancement on December 31, 2015; pleural effusions on January 3, 2016; thoracentesis for fluid in the lungs on January 4, 2016; MRI of the brain now showing cerebellar infarcts with patent vessels on January 10, 2016; stroke workup negative on January 11, 2016; cerebral angiogram negative on January 18, 2016; Aquaporin 4 antibody testing negative and discharged to rehabilitation on January 22, 2016. Pet. Ex. 42 at 2; Tr. 88. The discharge diagnosis was transverse myelitis. Tr. 86-87.

Dr. Levy explained that the central spinal cord is made up of grey matter consisting of neuronal cells that process information and generate electrical signals from the brain to the limbs and organs, and sensory signals from the body back to the brain. The peripheral cord is made up of white matter consisting of myelinated axons that carry electrical signals. Pet. Ex. 42 at 3. The spinal cord is protected by the spinal vertebral bones – 7 cervical, 12 thoracic, and 5 lumbar. *Id.*

Dr. Levy distinguished myelitis, an inflammation of the spinal cord, from myelopathy, which broadly refers to disease of the spinal cord. “Inflammatory myelopathy” is interchangeable with “myelitis”. Tr. 74. Vascular myelopathy causes spinal cord dysfunction but there is no inflammation. Tr. 74.

Dr. Levy explained that TM is an immunological attack on the spinal cord causing dysfunction of the nerves at the level of the lesion and potentially anything below. Pet. Ex. 42 at 3; Tr. 71-72. TM has no known infectious organism but is thought to be an unwarranted and aberrant autoimmune attack. Pet. Ex. 42 at 3. The 2002 Working Group criteria for diagnosing idiopathic TM serves as a starting point to rule in and out different diseases including TM. Tr. 78. Advancements in medicine such as in MRI technology and blood testing have been made since the criteria was published in 2002 but it is still the best currently available. He agreed TM is a diagnosis of exclusion. Tr. 78-80.

Dr. Levy explained that in the acute stage of TM, the lesion on the spinal cord contains immune cells, edema, swelling of the axons, tissue, and debris. In the chronic stage scar formation occurs from the healed tissue causing disruption of the signals traveling up and down the spinal cord. Pet. Ex. 42 at 3. It is like cutting a wire with the outgoing motor and autonomic/sensory signals to any muscle or organ below the lesion being compromised. *Id.* at 3-4.

Dr. Levy continued that TM can be part of broader chronic diseases such as MS, neuromyelitis optica (“NMO”), and MOG antibody disease. Pet. Ex. 42 at 4. However, TM is monophasic or a single immunological attack of the spinal cord with no known trigger. It causes

damage to the spinal cord but has no biomarker to identify it or distinguish it from the other diseases. *Id.*; Pet. Ex. 44 at 2; Tr. 72.

i. Prong I - Molecular Mimicry as the causal mechanism of TM from the flu vaccination.

Dr. Levy's theory in this case relies on molecular mimicry. He explained that the immune attack in TM is thought to be "most likely" T cell-mediated and sometimes B cell-facilitated. Pet. Ex. 85 at 2. Animal studies and clinical studies in humans can track and explain how T cells may fail to regulate when faced with an environmental stimulus, causing the onset of immune-mediated disease. *Id.* at 2-3. He opined that the flu vaccine caused immune dysregulation through either B or T cell immune-mediated pathophysiology. He added that the IOM includes the function of T cells under immune-mediated mechanisms likely to contribute to adverse events post-vaccination. *Id.*; Pet. Ex. 77.¹²

Dr. Levy described T cells as "highly promiscuous" and necessary in activating both primary and secondary immune response, with memory T and B cells activated as well. T cells also play a "pivotal" role in the signaling between the innate and adaptive immune systems. The IOM also discusses T cell contribution to establishment and maintenance of immune responses, clearance of pathogens, and maintenance of self-tolerance. Pet. Ex. 85 at 2.

He explained that the spinal cord is an "immune privileged area" which does not grant free access but "when the immune system breaches into the spinal cord, it may discover proteins not typically found in the periphery. If any of these proteins resemble a protein from a recently eradicated foreign organism, the immune system will re-activate and attack the spinal cord in a mistaken case of molecular mimicry." Pet. Ex. 42 at 3-4; Tr. 81-82. Typically, when presented with a virus or bacteria, the innate immune system attacks the perceived invasion by "non-specific" means, which causes swelling and infiltration of immune cells to attack what is in front of them. Pet. Ex. 48 at 1-2; Tr. 82-83. Then the adaptive immune system provides the "specific approach" to the problem, which requires 2-6 weeks to carry out. "Specialized adaptive immune cells engulf the foreign substance, break down the antigens, and sort them into self and non-self. The non-self antigens are chosen as the target of a specific, neutralizing immune response." Pet. Ex. 48 at 2.

According to Dr. Levy, the immune system has evolved to recognize that some invaders can mutate to avoid specific immune response. The influenza virus has several proteins that mutate or change every year and can evade immune detection. Through "antigen spreading", the immunological response that expands against a specific antigen more broadly covers the mutated forms so that infection with one strain of flu can provide some protection against other strains. But "antigen spreading" also carries the risk of cross over reactivity against self-proteins, creating the basis for molecular mimicry, which is the medical theory that covers all auto-reactive immunological attacks on the nervous system. "The theory posits that exposure to an environmental foreign (non-self) antigen sets in motion an immunological reaction to protect the host against it. In the process of protecting against a specific non-self antigen, the immune system produces an immune reaction that crosses the boundary between self and non-self." Pet. Ex. 48 at

¹² Institute of Medicine of the National Academies, *Influenza Vaccine*, in *Adverse Effects of Vaccines: Evidence and Causality*, 309-10 (2012), filed as "Pet. Ex. 77".

2; Tr. 82-83.

According to Dr. Levy, the immunological reaction to foreign proteins that triggers neurological consequences by molecular mimicry are not the result of the invasion of the foreign substance in the central nervous system (“CNS”) but rather the cross-reaction induced by the foreign substances to target a self-protein in the nervous system. Pet. Ex. 48 at 2; Tr. 165.

Dr. Levy explained that the concept of molecular mimicry is based on experimental autoimmune encephalomyelitis (“EAE”) models, used to study TM, MS, and NMO for decades and applied to other neurological diseases. Pet. Ex. 42 at 4; Pet. Ex. 44 at 2-3; Pet. Ex. 48 at 2-4; Tr. 84, 164. He submitted that over 80 years of animal studies support molecular mimicry as the basic underlying mechanism for TM. Pet. Ex. 48 at 2, 3; Tr. 83-84, 162.

Additionally, Dr. Levy submitted “among qualified treating physicians” various inflammatory demyelinating diseases such as NMO, TM, acute disseminated encephalomyelitis (“ADEM”), MS, and Guillain-Barre Syndrome (“GBS”) are believed to be caused by vaccines with the most likely mechanism of causation being molecular mimicry. Pet. Ex. 44 at 1-2. Vaccines “are known triggers of TM” via molecular mimicry because vaccines provide a large dose of foreign proteins and an adjuvant to activate the immune system, and while safe, some including flu vaccine can trigger TM. Pet. Ex. 42 at 4; Pet. Ex. 83 at 2, 3. He added that TM is very rare and live attenuated vaccines such as varicella zoster, tetanus, and live influenza are more likely to trigger TM. Pet. Ex. 62 at 3.

Dr. Levy stated that the best evidence of molecular mimicry is *Campylobacter jejuni* (“*C. jejuni*”) infection and GBS where human nerves contain protein complexes that are similar to ganglioside antigens on the outer coat of the *C. jejuni* bacterium, and these antibodies created against the outer coat of *C. jejuni* have been found in GBS patients within 2-3 weeks of the initial infection. Animal studies have shown that these antibodies bind to peripheral nerves and trigger an immunological response similar to GBS. Pet. Ex. 48 at 2; Tr. 83.

Dr. Levy summarized that sufficient evidence exists in the medical literature to support molecular mimicry as the mechanism by which TM can be triggered by foreign antigens, such as infection or vaccine including flu vaccine although it does not have a known antigenic target. Pet. Ex. 44 at 2; Pet. Ex. 48 at 3-4. Dr. Levy submitted that in *Day v. Secretary of HHS*,¹³ which involved the Gardasil vaccine and NMO, the special master found the literature cited to be persuasive on his causation theory. *See generally* Pet. Ex. 44.

ii. Prong III – Any time of onset is a reasonable time from flu vaccine to development of TM

Regarding a medically reasonable timeframe for TM, Dr. Levy provided that the majority of spinal cord attacks post-vaccination occur within 30 days, but studies have shown up to 90 days. Pet. Ex. 42 at 4. The attack can be systemic with rash and fever or insidious with minor neurological symptoms to full blown TM over several weeks. *Id.*; Tr. 116. He proposed that the time course for EAE and TM are similar, within 2-3 weeks from antigen exposure to first

¹³ The *Day* decision was filed into the record as an exhibit but not any of the literature discussed therein.

neurological symptom which coincides with the time required for the adaptive immune system to process foreign antigens and activate a response to them. Pet. Ex. 48 at 2-3; Tr. 96-98.

Dr. Levy referred to *Schonberger*, which studied the 1976 flu vaccine and the onset of GBS, as the basis for later studies to determine a risk-interval primarily within the 5-week period after vaccination but lasting approximately 9-10 weeks in total. Pet. Ex. 83 at 2. “Prior to the 10th week after vaccination, all relative risks were significantly greater than 1.” *Id.*; Pet. Ex. 84.¹⁴ He referred to *Baxter*, a 2016 study which used VAERS data and included 67 cases of TM occurring within 9 months of immunization and showed 38 of the 67 cases following flu vaccine. Dr. Levy agreed that if the timing was confined to 5-28 days post-vaccination, then there would have been only 7 cases. Pet. Ex. 62 at 3; Tr. 170; Resp. Ex. A Tab 6.¹⁵

At one point, Dr. Levy stated that petitioner reported onset two weeks prior to her presentation to the hospital and reached nadir on December 18, 2015 when she presented nearly paralyzed. Tr. 93, 98, 115, 125, 176. He also placed onset as November 4, 2015 based on Mr. Jones’ billing records,¹⁶ stating that she then had myelopathy exacerbation on December 18, 2015, extending the progression to nadir beyond the 21-day window imposed by the criteria of the 2002 TM Working Group “if this criteria were deemed absolute.” Pet. Ex. 62 at 1; Tr. 115-16, 133-34, 177. Admitting that this would not meet 2002 Working Group criteria for idiopathic TM, he claimed that having worked with Dr. Kerr, the senior author of the 2002 TM Working Group manuscript and having treated over 500 patients with TM and vascular myelopathies, his experience is that the 21-day cut off from onset to nadir excludes some true acute TM patients. Pet. Ex. 62 at 2; Tr. 60, 111-13. He referred to *Barreras* as showing that 27% of the 247 cases of inflammatory TM studied evolved to nadir in more than 21 days. Pet. Ex. 62 at 2; Pet. Ex. 64;¹⁷ Tr. 75-77, 134-36, 188.

Dr. Levy also placed onset on November 21, 2015 but could not recall where this date came from. Pet. Ex. 62 at 1; Tr. 177. He referenced the December 18, 2015 hospital record which included left shoulder pain for two months treated with massage therapy stating that this could have been the start of her symptoms because her left arm symptom was different than what she complained of historically and could correlate with a TM lesion. Tr. 91-92. He also stated that a vaccine given on October 7 could cause TM that starts to develop around November 4 with symptoms staying the same for another six weeks until there was a rapid, abrupt onset of more severe symptoms. Tr. 118-19.

After the hearing, Dr. Levy was asked to address an onset of TM 8-10 weeks after

¹⁴ Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. Epidemiology 105 (1979), filed as “Pet. Ex. 84”.

¹⁵ Roger Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis*, 63 Clinical Infectious Diseases 1456 (2016), filed as “Resp. Ex. A Tab 6”.

¹⁶ When it was noted that petitioner presented to medical doctors in the time between the vaccine and her presentation on December 17 without any complaints and that Mr. Jones’ records were the only records to note left arm and neck pain around November 4, Dr. Levy argued that he was not relying on Mr. Jones’ records “any more than you do.” Tr. 124. He submitted that the best evidence in his opinion that the symptoms started before the hospitalization was the intake record on December 18 that the symptoms started two weeks prior. Tr. 124-25.

¹⁷ Paula Barreras et al., *Clinical biomarkers differentiate myelitis from vascular and other causes of myelopathy*, 90 Neurology e12 (2018), filed as “Pet. Ex. 64” and “Resp. Ex. G”.

vaccination. He responded that 8-10 weeks was medically appropriate and supported by sound and immunologically reliable evidence. The flu vaccine has hemagglutinin (“HA”) and neuraminidase (“NA”) as surface proteins, utilized to designate influenza virus subtypes such as H1N1. Serum antibody levels to these proteins are measured in humans and correlate with protection from the wild-type virus. Human studies have shown peak HA response to flu vaccine around 4-6 weeks after vaccination, with peak titers as early as 2 weeks and up to 16 weeks after administration, but he conceded the longer intervals of 8-16 weeks occur with adjuvanted vaccines. Pet. Ex. 83 at 1; Pet. Ex. 76.¹⁸

He added that flu vaccines induce a peak antibody response similar to that of natural infection with seroprotective levels of antibody present for at least 90 days. Pet. Ex. 83 at 1; Pet. Ex. 85 at 3. He explained that “seroprotective antibodies” are a clinical marker of immune response after vaccinations that researchers use to study the efficacy of vaccine immune responses in humans. The antibodies circulate and can be measured in the blood as a marker of an ongoing and active immune response. Pet. Ex. 85 at 1. Some studies indicate that circulating levels of antibodies can remain high for many months to years. Pet. Ex. 83 at 1-2.

Dr. Levy relied on *Mealy*, an NMO study, to show a similar time interval for increased risk of relapse in those with identified autoimmune disease and vaccination. *Mealy* included increased risk up to 90 days with the highest risk during the first 30 days. Pet. Ex. 83 at 2; Pet. Ex. 45;¹⁹ Tr. 119, 180-88. When questioned about this study, Dr. Levy responded that “The 30-day time point was chosen as the primary analysis based on a large study that suggested that there was no increased association of any CNS demyelination beyond 30 days for time of vaccine administration” and beyond 30 days “is not as well accepted.” Tr. 180-81. But sound epidemiological studies confirm that flu vaccines can produce an increased safety risk in a time interval that corresponds with the biological and clinical data from animal and human studies to support the onset of TM between 8-10 weeks after receipt of vaccination as appropriate although rare. Pet. Ex. 83 at 2; Tr. 99-100, 121, 125-26.

Further, Dr. Levy asserted that not all patients will reach peak antibody response in that 2-6-week period and that antibody levels do not rapidly decline and can remain active for at least 90 days. Pet. Ex. 85 at 2-3; Pet. Ex. 83 at 3. He proposed that petitioner’s immune response to the flu vaccine could have peaked after 2 weeks but remained at a sufficient seropositive level for at least 3 months; alternatively, it is possible that her immune system did not peak until 8-16 weeks post vaccine, although this is more likely for adjuvanted vaccines but does not eliminate the explanation for petitioner individually. Pet. Ex. 83 at 3; Tr. 162-63. He argued that petitioner developed TM within two months (or 8 weeks) of the flu vaccine with no alternative etiology for the development of her neurological disabilities but for the flu vaccination. Pet. Ex. 42 at 5. He later argued that petitioner’s flu vaccine elicited a sufficient seropositive immune antibody response at some point prior to 10 weeks post-vaccination, but her peak antibody response did not turn off immediately, and ultimately, she had a change in health culminating in hospitalization and TM diagnosis on December 17. Pet. Ex. 83 at 3. He conceded that 10 weeks was on the longer side of the risk

¹⁸ Institute of Medicine of the National Academies, *Adverse Effects of Vaccines: Evidence and Causality* (2012), filed as “Pet. Ex. 76”.

¹⁹ Maureen A. Mealy et al., *Vaccines and the association with relapses in patients with neuromyelitis optica spectrum disorder*, 23 *Multiple Sclerosis and Related Disorders* 78 (2018), filed as “Pet. Ex. 45”.

interval but “realistically [was] not too far removed to be an immunologically appropriate etiological factor.” There is not much data beyond 10-12 weeks other than compelling factual and logical sense that in some individuals a longer time is medically appropriate based on biologic understanding of the human immune response to a vaccination and what is understood about the pathophysiology of the disease from biologic and clinical studies in animals and humans. *Id.* at 2, 3. Since petitioner’s peak immune response time cannot be determined with certainty, the medical facts and her onset of neurological symptoms correlate with biological understanding and timing of antibody responses to flu vaccines. *Id.* at 3. Therefore, the flu vaccine was the most likely immune trigger here, and 8-10 weeks post-vaccination is medically appropriate for the onset of idiopathic TM. *Id.* at 3-4.

Dr. Levy opined that the timeline in this case is consistent with a link between the vaccine and petitioner’s later acute myelopathic decline. Pet. Ex. 62 at 2. He stated that onset anytime between October 7 and December 18, 2015 was “consistent with [his] theory that the vaccine started this transverse myelitis process, that the first symptom of that transverse myelitis process was left arm pain, and that it evolved into this point of no return event which manifested as paralysis.” Tr. 179.

iii. Prong II – A logical sequence of cause and effect exists that the flu vaccination caused petitioner’s TM

Dr. Levy opined that TM was the correct diagnosis. The December 18, 2015 MRI showed a non-enhancing C4 lesion, and the MRI 13 days later showed an enhanced, developing lesion, suggesting some sort of quickly evolving process that is consistent with TM more than vascular neuropathy. Initial CSF testing was normal but 19 days later showed the presence of inflammatory cells. Pet. Ex. 42 at 5; Pet. Ex. 62 at 2; Tr. 95, 98-99, 106-08, 123, 142-45. He agreed repeat assessments were not done within 7 days as required by the 2002 TM Working Group, technically making the diagnosis “possible ATM” since the criteria for TM was not met. Nevertheless, he asserted that the swelling and enhancement on repeat MRI was more severe and inflamed than would be expected from vascular myelopathy, and the CSF findings mirrored the MRI findings, both evolving. Even though the testing did not fit the 7-day window proposed, “the workup confirms the presence of inflammation close enough to the acute decline in function that fits with the clinical diagnosis of transverse myelitis.” Pet. Ex. 62 at 2; Tr. 189-91. Further, the contrast enhancement on “initial presentation acutely” was more consistent with TM than a vascular myelopathy. Tr. 77, 94; Pet. Ex. 64.²⁰ The remainder of her examination during her December 2015 hospitalization was negative for autoimmune disease. Pet. Ex. 42 at 5.

Dr. Levy argued that petitioner’s subsequent complications including embolic strokes in the cerebellum were “directly” linked to her TM, hypercoagulability associated with hospitalization and immobility that leads to weakness and autonomic dysfunction where venous circulation is compromised. Pet. Ex. 42 at 5; Pet. Ex. 62 at 3; Tr. 155-58. The other complications including lung nodules, pleural effusion, sepsis, and thrombocytopenia are all inflammatory or infectious in nature and contributed to her myelopathy. Pet. Ex. 62 at 3. He added, although petitioner had no pre-existing vascular disease, the embolic strokes indicated that “her blood

²⁰ Barreras et al., *supra* note 17.

vessels aren't great". Tr. 156; Pet. Ex. 42 at 5. She also had no prior neurological history until she had left-sided neck and shoulder pain around 8 weeks after the vaccination. Pet. Ex. 42 at 4.

Dr. Levy disagreed that petitioner suffered a spinal cord infarct. He described two types of vascular myelopathies, one that is hyperacute progressing to nadir within minutes to hours, the other chronic progressing to nadir over several weeks to months with interval declines in neurological function. Petitioner did not fit the clinical picture of either because the lesion was predominantly within the grey matter of the spinal cord and onset was not hyperacute nor did "the chronology of this case" fit the profile of the second. Pet. Ex. 62 at 2; Tr. 70-71, 152. Additionally, an angiogram to rule out arteriovenous malformation or other causes of vascular myelopathy did not reveal any vascular abnormalities. Pet. Ex. 62 at 2; Tr. 89-90, 153-54. Further, the immune-mediated treatments petitioner received would not have improved an arteriovenous malformation. Tr. 90-91. Finally, the risk factors for vascular myelopathies generally involve men of advanced age, hyperlipidemia, hypertension, and diabetes, and petitioner fit none of those risk factors. Pet. Ex. 62 at 3. He later conceded that petitioner "had maybe hypertension." Tr. 160.

Dr. Levy concluded that he has spent his life caring for people like petitioner and conducting research in this area. He recognizes that inflammatory TM and vascular myelopathies are hard to distinguish. Pet. Ex. 62 at 4; Tr. 58, 75. He also agreed that *Barreras* concluded that a TM diagnosis is wrong 54% of the time with a different diagnosis fitting better. Tr. 196-97; Pet. Ex. 64.²¹ He agreed that vascular myelopathy was possible here but was less supported by the medical records. He believed petitioner more likely suffered from TM because: 1. The vaccine was a preceding inflammatory risk and there were no preceding vascular risk factors; 2. The inflammatory process brewed over weeks culminating in an acute exacerbation; 3. The second MRI and spinal fluid testing were consistent with myelitis and not vascular myelopathy; 4. The clinical context was inflammatory including sepsis with other complications; and 5. The vascular myelopathy workup was negative. Also, petitioner responded to corticosteroids which is more consistent with TM than with vascular myelopathy. Therefore, the flu vaccine was the cause of petitioner's TM, the complications she suffered while hospitalized, and her resulting disabilities. Pet. Ex. 62 at 4; Tr. 70-71, 148, 150-51, 169, 197-98.

B. Respondent's Expert, Dr. J. William Lindsey²²

Dr. Lindsey summarized petitioner's medical history and objective testing. Resp. Ex. A at 2-3. He submitted that petitioner suffered a rapid onset of severe weakness affecting both sides of her body, left more than right, with sensation to pain and temperature affected but other modalities preserved 2.5 months post flu vaccine, placing onset of new left neck pain and severe left shoulder

²¹ *Id.*

²² Dr. Lindsey received his M.D. from Harvard Medical School. Resp. Ex. A at 1. His residency program was in neurology at Stanford University Medical Center, where he was also a fellow in neuroimmunology and studied under Dr. Lawrence Steinman. *Id.* After his fellowship training, he was on faculty in the Department of Neurology at the University of Texas Health Science Center at Houston, the position he currently holds. *Id.* He is also currently a professor of Neurology and the Director of the Division of Multiple Sclerosis and Neuroimmunology. *Id.* Throughout his career, he has cared for patients, participated in clinical trials, and conducted research in the field of neuroimmunology. *Id.* Roughly half of his patients suffer from transverse myelitis, neuromyelitis optica, neurosarcoidosis, Behcets, autoimmune encephalitis, and other related diseases. *Id.* When he is not seeing patients, he spends his time conducting research and participating in clinical trials. *Id.*

pain on December 17, 2015. *Id.* at 3; Tr. 300-01, 303, 308, 323-24. Initial CSF testing was normal. Repeat CSF testing two weeks later showed mild inflammation. Cervical spine MRI initially showed small non-enhancing lesions but repeat MRI two weeks later demonstrated an increased lesion that was enhanced. Resp. Ex. A at 3-4; Tr. 301-02. Petitioner was initially treated with high-dose IV steroids then switched to oral steroid taper, but “presumably because the response to the steroids was unsatisfactory”, IVIg was implemented. Tr. 307. According to Dr. Lindsey, petitioner’s neurologic condition did not change much with either the steroids or IVIg suggesting a non-inflammatory cause of her condition. Tr. 307-08. If petitioner was his patient, he would have diagnosed a vascular myelopathy but agreed that it was appropriate to treat her for TM just in case. Tr. 351-52. Subsequent imaging showed the enhancement stopped and the area became atrophic. Resp. Ex. A at 4.

Dr. Lindsey disagreed that the repeat MRI and CSF imaging suggested myelitis, arguing instead that the swelling and enhancement seen on the repeat MRI was consistent with evolving spinal cord infarct. Additionally, the number of white blood cells in the second CSF testing was only minimally increased above the upper limit of normal. *Barreras* showed that the spinal fluid results for inflammatory myelitis and ischemic myelopathy overlap, with no definitive distinction between the two. Resp. Ex. E at 1; Pet. Ex. 64, Table 2.²³

Dr. Lindsey stated that petitioner’s presentation of prodromal symptoms on December 17, 2015 with a rapid progression to nadir on December 18, 2015 fit the clinical picture of a hyperacute stroke. An angiogram was not done until January 18, 2016 to rule out dural arteriovenous fistula, therefore the angiogram results were not informative of whether she had a spinal vascular occlusion a month earlier. Resp. Ex. E at 1; Pet. Ex. 12 at 438.

Dr. Lindsey acknowledged the many complicating medical issues that arose during petitioner’s hospitalization but disagreed they were informative of the etiology of her myelopathy. If anything, the cerebellar strokes were more suggestive of her susceptibility to hypercoagulation which could have contributed to a vascular myelopathy. Resp. Ex. E at 1-2; Resp. Ex. A at 4-5.

Dr. Lindsey opined that petitioner had severe myelopathy (spinal cord dysfunction) characterized by rapid onset of disability with limited recovery. Resp. Ex. A at 4. He described TM as focal inflammation of the spinal cord and is a term often imprecisely used for any spinal cord problem with acute or subacute onset and no obvious etiology. Citing a recent Mayo Clinic study, Dr. Lindsey pointed out that only 18% of the 226 cases diagnosed as TM met the diagnostic criteria and 70% had a different diagnosis after further investigation. *Id.*; Resp. Ex. A Tab 1;²⁴ Tr. 304-05. In this case, petitioner did not meet the 2002 TM Working Group Criteria. Resp. Ex. A at 4, 7; Resp. Ex. A Tab 2.²⁵ She did not have inflammation within the spinal cord evidenced by no increased protein or white blood cells in her CSF, no elevation of the IgG index, and no contrast enhancement on the December 18, 2015 MRI. MRI and CSF testing were not repeated within 7 days as suggested. Resp. Ex. A at 4.

²³ *Barreras et al., supra* note 17.

²⁴ Nicholas L. Zalewski et al., *Evaluation of idiopathic transverse myelitis revealing specific myelopathy diagnoses*, 90 *Neurology* e96 (2018), filed as “Resp. Ex. A Tab 1” and “Resp. Ex. C Tab 4”.

²⁵ Transverse Myelitis Consortium Working Group, *Proposed diagnostic criteria and nosology of acute transverse myelitis*, 59 *Neurology* 499 (2002), filed as “Resp. Ex. A Tab 2”.

In Dr. Lindsey's opinion, petitioner more likely suffered from spinal cord infarct based on the clinical and objective findings which were consistent with infarct. Resp. Ex. A at 4, 6; Tr. 326. She presented with rapid progression of deficits, pain at onset, sensory loss selective for pain and temperature, bladder dysfunction, and areflexia. Resp. Ex. A at 4; Tr. 301-02. The MRI results on December 18, 2015 were consistent with small, limited lesions of the cord that greatly increased by the time of the December 31, 2015 MRI, which showed new contrast enhancement and which the radiologist read as consistent with cytotoxic edema from acute to subacute cord infarct. Resp. Ex. A at 4. Diffusion weighted imaging of the cord was not done but petitioner still met the diagnostic criteria for probable spontaneous spinal cord infarction, lacking only the specific MRI criteria. *Id.* The primary clinical findings that argue against TM are the relatively abrupt onset, the lack of inflammation seen on the December 18 MRI, and normal CSF. Tr. 302, 305-06.

Dr. Lindsey noted that there is no evidence that connects flu vaccine to spinal cord infarct. Even if TM is the accepted diagnosis, there is no convincing evidence connecting vaccination to myelitis. Resp. Ex. A at 6; Tr. 357.

Dr. Lindsey added that his research found only one review article from 1983 which included two case studies from 1953 of post-vaccine myelopathy in children thought to be of vascular etiology. Resp. Ex. A at 5. A PubMed search using the search terms "influenza vaccine" and "myelitis" resulted in 13 case reports, 6 of which were related to the 2009 H1N1 vaccine, but no larger studies. The other seven cases involved the multi-valent seasonal vaccine with a latency of 7 days or fewer between vaccination and onset of symptoms. Petitioner's onset here was about 70 days after vaccination. *Id.* Dr. Lindsey noted with the number of flu vaccines administered each year, this is a small number of cases, and it is telling that the literature has not progressed beyond case reports, which suggests that the vaccine causation is questionable, as is the temporal relationship. *Id.*

Dr. Lindsey submitted that *Baxter* used the Vaccine Safety Datalink database and identified 7 cases of acute TM out of 19 million flu vaccines administered, with no increase when compared to the control population. Resp. Ex. A at 5-6; Resp. Ex. A Tab 6;²⁶ Tr. 334. *Nordin* studied TM and GBS in over 76,000 vaccinated pregnant women compared to 148,000 unvaccinated pregnant women and found no increase of TM or GBS. Resp. Ex. A at 6; Resp. Ex. A Tab 7.²⁷ Comprehensive review of the literature showed no concern for influenza and TM. Resp. Ex. A at 6; Resp. Ex. A Tab 8;²⁸ Tr. 328.

Dr. Lindsey asserted that Dr. Levy relied on his own study of NMO published in 2018 involving vaccines in those who had already been diagnosed with NMO who suffered relapses after vaccination, not new onset. Resp. Ex. A at 6; Pet. Ex. 45.²⁹ The study included 90 NMO patients, 211 vaccinations (61% being flu vaccine), and 340 relapses in the group. They compared

²⁶ Baxter et al., *supra* note 15.

²⁷ James D. Nordin et al., *Maternal Safety of Trivalent Inactivated Influenza Vaccine in Pregnant Women*, 121 *Obstetrics & Gynecology* 519 (2013), filed as "Resp. Ex. A Tab 7".

²⁸ Agency for Healthcare Research and Quality, *Safety of Vaccines Used for Routine Immunization in the United States*, AHRQ Publication No. 14-E002-EF (2014), filed as "Resp. Ex. A Tab 8".

²⁹ Mealy et al., *supra* note 19.

the observed number of relapses at various intervals following vaccination (30, 60, and 90 days) with randomly selected time points. There were 17 relapses within 90 days of vaccination but no increase in relapses following vaccination. They found that the patients untreated for NMO were more likely to relapse after vaccination. He further noted that the applicability of the results from NMO to isolated TM is unclear. *Id.* At hearing, Dr. Lindsey agreed that TM and NMO both involve inflammation of the spinal cord, though NMO has a known, non-autoimmune target. Tr. 337-39.

Dr. Lindsey disagreed that TM was the correct diagnosis or that vaccines are a known trigger for TM. He added that a 70-day onset is atypical for a trigger of autoimmune disease. *Agmon Levin* is “merely a collection of previous case reports published between 1971 and 2007, which varied widely in quality and detail”, with only two of the 37 cases involving influenza vaccine. This only demonstrates how rare myelitis is and says nothing about causality. Resp. Ex. A at 7; Pet. Ex. 49;³⁰ Tr. 339.

Dr. Lindsey further disagreed that there is significant molecular mimicry between flu vaccine and spinal cord antigens. If there were, there would be an increase of TM following flu vaccine but there is not. Dr. Lindsey acknowledged that *C. jejuni* infection has been found to have an antigen capable of triggering GBS but argued that is irrelevant here in discussing a vaccine not infection and because GBS is different disease. Similarly, animal models of EAE and Dr. Levy’s own work with NMO are irrelevant to whether the flu vaccine in this case caused myelopathy. Resp. Ex. A at 7.

In addressing whether 8-10 weeks is a medically appropriate time frame for a post-vaccine autoimmune syndrome, Dr. Lindsey pointed out that petitioner’s vaccination was on October 7, 2015, and she presented to urgent care on December 17, 2015, 71 days or slightly over 10 weeks after vaccination. Resp. Ex. I at 1. He stated that a very long prodromal period with little progression until a sudden, severe onset of symptoms does not “seem terribly plausible.” Tr. 309-10, 313, 319-20.

Dr. Lindsey argued that a risk interval—the time period for immune-mediated problems to arise following a potential trigger—of 4-6 weeks for the onset of TM after influenza vaccine has been demonstrated in two vigorous studies. *Nordin* studied TM and GBS in relation to flu vaccine and considered only events occurring within 6 weeks from vaccination to be vaccine-related and *Baxter* used an interval of 5-28 days after vaccination. Resp. Ex. I at 2; Resp. Ex. E at 2; Resp. Ex. A Tab 6;³¹ Resp. Ex. A Tab 7;³² Tr. 308-13, 319-23. He explained that the interval of 43 days to 9 months was used in *Baxter* as a comparison interval, and it is unlikely that cases in the comparison interval were causally related to vaccination. Resp. Ex. E at 2; Resp. Ex. A Tab 6.

Dr. Lindsey also addressed *Schonberger*, which provided the relative risk of GBS in a vaccinated group compared to an unvaccinated group with 95% confidence intervals and concluded that between weeks 1 and 5 post-vaccination, the risk of GBS is significantly increased, but in weeks 10 through 17, the risk is not significantly elevated. The usefulness of *Schonberger*

³⁰ N Agmon-Levin et al., *Transverse myelitis and vaccines: a multi-analysis*, 18 *Lupus* 1198 (2009), filed as “Pet. Ex. 49”.

³¹ Baxter et al., *supra* note 15.

³² Nordin et al., *supra* note 27.

in this case is limited because it was done in 1979 and studied a different version of the flu vaccine and a different autoimmune disease. Resp. Ex. I at 1; Pet. Ex. 84 at 9.³³ The time course of experimental animal studies of induced autoimmunity varies with antigen, adjuvant, and the species and strain of animal. *Id.* The same can be said for autoimmunity with different vaccines depending on the rapidity and durability of the induced immune response and the antigens contained in the vaccine. *Id.* at 1-2. Therefore, findings related to GBS cannot necessarily be accurately ascribed to TM. *Id.* at 2.

Dr. Lindsey responded to Dr. Levy's time course of seroprotective levels of antibodies in circulation following vaccination and the exhibits filed in support thereof, noting that this issue has been well studied and is not informative for post-vaccine autoimmunity. He agreed antibodies may be elevated for months after exposure, but the "generally accepted period for vaccine-induced autoimmunity is much shorter" likely because autoimmunity requires both autoimmune T cells and autoantibodies with effector T cell responses being short lived. Resp. Ex. I at 2; Pet. Ex. 86 at 16.³⁴

Dr. Lindsey concluded that 71 days from vaccination to myelopathy is a longer than expected timeframe for autoimmune-mediated disease; Dr. Levy acknowledged that the causal role of a vaccine is rare and "10 weeks is on the longer end of the medically appropriate vaccine safety risk interval." Resp. Ex. A at 7; Resp. Ex. H at 1; Resp. Ex. I at 2; Pet. Ex. 83 at 3; Tr. 324. Therefore, in Dr. Lindsey's opinion, petitioner's illness was more likely than not unrelated to her flu vaccine. Resp. Ex. I at 2.

Dr. Lindsey believes petitioner had a spinal cord infarct based on clinical, radiological, and laboratory findings, unlikely to be caused by the flu vaccine. Resp. Ex. A at 7; Tr. 357-58. An abrupt onset of petitioner's left-sided weakness is consistent with stroke. Resp. Ex. H at 1. Her presentation and objective testing were more consistent with vascular myelopathy than with TM. The initial MRI and CSF findings with the evolution over time as seen on repeat testing were consistent with vascular myelopathy. The negative angiogram findings were not informative of etiology because it was done over a month later. Finally, there is no evidence of causal relationship between the flu vaccine and petitioner's myelopathy. Resp. Ex. E at 2.

C. Respondent's Expert, Dr. William B. Zucconi³⁵

As a board-certified neuroradiologist, Dr. Zucconi focused his review on petitioner's neuroimaging. Resp. Ex. C; Tr. 203.

³³ Schonberger et al., *supra* note 14.

³⁴ Claire-Anne Siegrist, *Vaccine Immunology*, in *General Aspects of Vaccination*, filed as "Pet. Ex. 86".

³⁵ Dr. Zucconi obtained his D.O. from New York College of Osteopathic Medicine in 2001. Resp. Ex. D. He is board-certified in radiology with a Certificate of Added Qualification in Neuroradiology. Resp. Ex. C. At the time of hearing, he served as the Section Chief of Neuroradiology at the Yale School of Medicine, Department of Radiology and Biomedical Imaging, and as the Program Director of the Neuroradiology Fellowship program. *Id.* While he has published peer-reviewed literature and lectured on the topic of autoimmune disease of the central nervous system, he primarily works as a clinician. *Id.* In the last five years, Dr. Zucconi has been directly involved in the care of approximately 30-40 transverse myelitis patients, but he has reviewed many more cases through consultations, interdepartmental conferences, teaching sessions, lectures, and research activities at Yale. *Id.*

Dr. Zucconi noted a history of left arm numbness down her entire arm to her thumb in 2013. Resp. Ex. C at 2; Pet. Ex. 7 at 7. She received the subject flu vaccine on October 7, 2015 in her left arm. She presented to urgent care on December 17, 2015 for “Pain in left neck shoulder area and tingling in L arm.” She denied weakness. Resp. Ex. C at 2; Pet. Ex. 10 at 2. This was preceded by left neck and hip pain over the past three months. Resp. Ex. C at 2; Pet. Ex. 12 at 562. On December 18, 2015, she awoke with left arm and leg weakness, fell when attempting to walk, presented to urgent care and was sent for CTs of head and neck and cervical spine MRI. Resp. Ex. C at 2; Pet. Ex. 11 at 56, 60, 62. She had decreased reflexes bilaterally and decreased strength on the right as well. Resp. Ex. C at 2; Pet. Ex. 11 at 22. She was transferred to another facility where extensive work up and treatment was started for presumed spinal cord inflammation with intravenous corticosteroids followed by intravenous immunoglobulin therapy. Thereafter she had a complicated course with pulmonary effusions, cerebellar infarcts, possible HIT, respiratory failure, bacteremia, complicated UTI, melena requiring blood transfusion, and autonomic dysfunction. She was discharged to a rehabilitation facility then skilled nursing. Resp. Ex. C at 2; Pet. Ex. 12 at 1150.

Dr. Zucconi discussed the imaging studies in detail. *See generally* Resp. Ex. C. The cervical spine radiographs done on December 17, 2015 showed disc narrowing at C5-6 and C6-7, mild facet arthropathy and spurring with foraminal narrowing on the right at C3-4 and C4-5, and mild right-sided arthritis with foraminal encroachment at C6-7. Resp. Ex. C at 2-3; Pet. Ex. 54. There was evidence of foraminal narrowing at C3-4 on the left side due to facet encroachment and mild-moderate foraminal stenosis at C5-6 and C6-7. Resp. Ex. C at 3. No other abnormalities were noted. *Id.*

CT of the brain on December 18, 2015 revealed no relevant abnormalities with the exception of a small hypoattenuating focus within the inferior aspect of the anterior limb of the left internal capsule, open perivascular space, or possibly sequela of remote small lacunar infarct. It was read as normal. Resp. Ex. C at 3; Pet. Ex. 54. CT of the cervical spine on December 18, 2015 included arthritic findings with neural foraminal stenosis at C3-4, C4-5, C5-6, and C6-7. There were no additional significant abnormalities found. The original interpretation did not include the foregoing and read only a small disc protrusion at C3-4. *Id.*

Cervical MRI on December 18, 2015 confirmed the findings of the CT of mild to moderate spinal stenosis at the C4-5 disc level with mild cord deformity and mild spinal stenosis at C5-6. Resp. Ex. C at 3; Pet. Ex. 54. There was a subtle abnormal signal hyperintensity on T2 weighted sequences within the cord from the lower C2 through C5 levels. Resp. Ex. C at 4; Pet. Ex. 54; Tr. 210-11. Dr. Zucconi noted that the subtle spinal cord edema seen between C2 and C5 levels was characteristic of early spinal cord infarction. Resp. Ex. F at 1; Resp. Ex. C at 4. The abnormal signal at C3 occupied the central, anterior left aspect of the spinal cord corresponding to grey matter and its left anterior horn. Resp. Ex. C at 4; Pet. Ex. 54. Slightly inferiorly, there was bilateral central cord involvement also confined to the central grey matter which “is a feature supporting anterior spinal artery distribution SCI”. *Id.*; Resp. Ex. F at 1; Resp. Ex. C at 12; Tr. 211-12. This type of distribution is “highly suggestive of an ischemic stroke” as noted by *Barreras*. The relatively mild MRI signal changes would be expected at this point in time. Resp. Ex. F at 1; Resp. Ex. G.³⁶ It would be very unusual for an acute, severe transverse myelitis to present with such

³⁶ *Barreras et al., supra* note 17.

subtle MRI findings. Resp. Ex. F at 1. Summarily, the December 18 imaging showed no enhancement, no additional cord lesions identified, no lesions within the portion of the cerebellum or brain stem, and no significant abnormalities identified within the vertebrae or paraspinal soft tissue. Resp. Ex. C at 4.

Dr. Zucconi asserted based on petitioner's clinical history and the pattern of cord disease his primary consideration at that time would have been acute spinal cord infarct due to acute onset, anterior cord and specific central grey/anterior horn involvement, and absence of initial enhancement. He acknowledged that the initial radiology report did not mention the subtle cord signal abnormality. Resp. Ex. C at 5; Pet. Ex. 11 at 56; Tr. 214. Dr. Zucconi added that the spondylotic changes seen on the imaging may be partly responsible for petitioner's prior complaints of left neck and left arm pain into the thumb in 2013 and not related to the myelopathy that ensued. Resp. Ex. C at 5; Tr. 238.

A brain MRI on December 19, 2015 showed no evidence of prior cortical infarct or definitive lacunar infarct within the cerebellar hemispheres or brain stem. Resp. Ex. C at 5-6; Pet. Ex. 55. There was no abnormal enhancement of the brain or meninges. Resp. Ex. C at 6. Incidental findings were nonspecific but commonly seen in the setting of microvascular ischemia. *Id.*; Tr. 219. He also cautioned against using a brain MRI to draw conclusions about the spinal cord. Tr. 216-19, 280-81.

Dr. Zucconi submitted that the MRI of the thoracic spine performed on December 20, 2015 showed prominent right central disc extrusion at T8-9 which deformed the right ventral cord surface. There was no myelopathic signal abnormality associated. The spinal cord was otherwise unremarkable with mild upper facet arthropathy and foraminal narrowing at the T3-T5 levels. There was no abnormal enhancement. Resp. Ex. C at 6; Pet. Ex. 55; Tr. 219-20.

A brain MRI performed on December 31, 2015 revealed no significant interval findings and the abnormal FLAIR referenced in the initial interpretation was highly likely artifactual since it was not reproduced on other imaging. Resp. Ex. C at 6; Pet. Ex. 56; Tr. 220. The cervical MRI on December 31, 2015 however revealed much more conspicuous cord pathology spanning the C2-6 vertebral segments with a greater degree of T2 signal hyperintensity and volume of cord involvement. The T2 signal abnormality was predominantly central and anterior, and the abnormal enhancement now well visualized conformed to the morphology of the central grey matter. Resp. Ex. C at 6-9; Pet. Ex. 56; Tr. 220-21. This MRI showed the evolution of the cord abnormality and enhancement for a spinal cord infarct as expected. Resp. Ex. F at 1; Resp. Ex. C at 6; Tr. 220. Gadolinium enhancement is seen in 39-43% of spinal cord infarcts. Resp. Ex. F at 1; Resp. Ex. C Tab 4.³⁷

Dr. Zucconi continued that the January 5, 2016 cervical MRI showed that the pattern of enhancement was better delineated and increased, but stable in distribution, characteristic of evolving cytotoxic edema in SCI. Resp. Ex. C at 9; Pet. Ex. 57; Tr. 226-28.

Dr. Zucconi added that the original interpretation of abnormal signal in the central portion of the cord was unchanged but now included "consider acute demyelinating process, vascular

³⁷ Zalewski et al., *supra* note 24.

malformation with venous infarction or transverse myelitis”, which departed from the prior interpretation of acute-subacute spinal cord infarct. Resp. Ex. C at 9; Pet. Ex. 12 at 319. He agreed with the impression but noted that he would have probably ordered the differential in a different hierarchy, favoring SCI. Tr. 228.

Dr. Zucconi noted that the MRI of the brain on January 10, 2016 showed relevant interval changes within the left cerebellar hemisphere compatible with small, acute embolic infarcts. Resp. Ex. C at 9-10; Pet. Ex. 58. A cerebral and cervical angiogram performed on January 18, 2016 to exclude shunting vascular malformation as the cause of the SCI was normal. Resp. Ex. C at 10; Pet. Ex. 59; Tr. 229-30. Dr. Zucconi added that an embolus and arterial dissection several weeks after infarct would likely not be visualized. Resp. Ex. C at 10; Tr. 277-78. He explained that preexisting atherosclerotic disease risk factors are not necessary to sustain a SCI. Resp. Ex. F at 2. Further, neither a “dissection protocol” MRI (which utilizes special sequences to evaluate the blood vessels) or a CTA (computed tomography angiogram) of the neck arteries (which are sensitive to the presence of blood within the wall of an artery which can form clots and embolize or occlude vessels that arise in the area of or distal to the dissection) was performed here. “It is well known that an arterial dissection, or blood collecting within the wall of an artery, can lead to spinal cord and brain infarctions and can be missed on catheter angiography, especially when delayed”. *Id.*; Resp. Ex. F Tab 1;³⁸ Resp. Ex. F Tab 2;³⁹ Resp. Ex. F Tab 3.⁴⁰ Dr. Zucconi acknowledged that petitioner’s catheter angiogram was normal but performed over a month after the initial MRI. Resp. Ex. F at 2.

MRI of the brain on August 4, 2016 showed that the cerebellar infarcts had resolved, with no acute or interval findings. The study was reported as normal. Resp. Ex. C at 10; Pet. Ex. 60; Tr. 231. The cervical MRI on that date showed the expected interval atrophy of the spinal cord most notably anteriorly by concavity of the spinal cord surface at the C3-4 level and abnormal T2 signal confined to the anterior aspect of the spinal cord through the C5 level, suggesting SCI. There was no interval cord lesion identified. Resp. Ex. C at 10; Tr. 232-33.

Finally, the MRIs of the brain and cervical spine on July 15, 2019 were unchanged. Resp. Ex. C at 11; Pet. Ex. 61.

Based on the clinical information and imaging, the differential diagnosis for petitioner’s spinal cord disease is essentially limited to spinal cord infarct or TM, but in Dr. Zucconi’s opinion, more likely SCI. Resp. Ex. C at 11; Tr. 206, 223, 225, 258, 261. However, he agreed that if he were viewing petitioner’s records and MRIs on December 31, having no other information, the differential would contain both TM and SCI. Tr. 222-23. Dr. Zucconi defined TM as involving both right and left halves of the spinal cord, requiring laboratory evidence of inflammation and exclusion of other diagnoses. TM inflammation causes edema with enhancement of the spinal cord and blood vessels within the cord. Imaging patterns include involvement of the entire cross-

³⁸ Hidefumi Suzuki et al., *Cervical Cord Infarction Caused by Dissection of the Intracranial Segment of the Vertebral Artery*, 57 *Internal Medicine* 3321 (2018), filed as “Resp. Ex. F Tab 1”.

³⁹ Marcel Arnold et al., *Vertebral Artery Dissection: Presenting Findings and Predictors of Outcome*, 37 *Stroke* 2499 (2006), filed as “Resp. Ex. F Tab 2”.

⁴⁰ James M. Provenzale, *MRI and MRA for evaluation of dissection of craniocerebral arteries: lessons from the medical literature*, 16 *Emergency Radiology* 185 (2009), filed as “Resp. Ex. F Tab 3”.

sectional diameter of the cord and affect grey and white matter on both T2 and contrast enhanced sequences involving anterior, posterior, central, peripheral regions, and may be asymmetric. Resp. Ex. C at 11. Spinal cord disease is typically conspicuous at the time of diagnosis and shows contrast enhancement. *Id.* at 12; Resp. Ex. C Tab 1;⁴¹ Tr. 206-07. Cord enhancement with TM is characterized as eccentric, peripheral and nodular, as well as diffuse and heterogeneous. Resp. Ex. C at 12; Resp. Ex. C Tab 5;⁴² Tr. 222.

Dr. Zucconi explained that the spinal cord tissue insult in SCI is primarily an infarction where the spinal cord is deprived of blood and/or oxygen, causing rapid dysfunction and death to neurons of the spine. The edema that follows is due to cytotoxic edema or cell death and preferentially affects the grey matter of the cord which is more susceptible to infarction than white matter. Despite the rapid onset of symptoms in SCI, MRI manifestations are delayed on routine MRI because cellular injury and death need time to evolve to the point of detection on imaging. Resp. Ex. C at 12; Resp. Ex. F at 1. “Diffusion weighted MRI” is challenging to obtain, but has the ability to document cytotoxic edema usually within hours because of its sensitivity to restricted cellular and interstitial water diffusion in the tissue before swelling manifests. However, diffusion weighted MRI was not done. “Because the grey matter is more sensitive to blood and oxygen deprivation, the pattern of injury on MRI will show preferential disease in this distribution.” *Id.*; Resp. Ex. C Tab 5.⁴³ The grey matter within the central cord is usually described as “butterfly shaped” due to its symmetric, mirrored anterior and posterior horns on the right and left side of the cord. Resp. Ex. C at 12. The anterior aspect of the cord is most commonly affected in the area of the anterior spinal artery which lies in front of the cord at the midline and is primarily supplied by the vertebral arteries. *Id.*; Tr. 278-79.

Dr. Zucconi concluded that petitioner’s imaging was compatible with SCI, specifically the preferential signal abnormalities affecting the grey matter that were subtle (or even read as normal) initially but evolved over a period of days to reflect cytotoxic edema and cell death. Resp. Ex. C at 12. Regions of the cord in petitioner’s case showed pathologic signal that conformed to the confines of the central grey matter, which is specific for cord infarction and incompatible with the imaging appearance of TM. *Id.* at 12-13; Resp. Ex. C Tab 1;⁴⁴ Resp. Ex. C Tab 2;⁴⁵ Tr. 206.

Dr. Zucconi then pointed to the features of on petitioner’s imaging compatible with SCI including the “anteromedial Spot” sign, (left sided) and anterior central grey matter involvement on the initial MRI on December 18, 2015. The December 31, 2015 MRI showed characteristic delayed increase in volume and intensity of T2 signal abnormality. Resp. Ex. C at 13; Resp. Ex. C Tab 1.⁴⁶ He agreed with the initial interpretation that included “[p]rogressive cytotoxic edema of the entire cervical spinal cord with new patchy postcontrast enhancement of the cord primarily involving the grey matter and leptomeninges when compared to the prior MRI of the cervical spine

⁴¹ Nicholas L. Zalewski et al., *Characteristics of Spontaneous Spinal Cord Infarction and Proposed Diagnostic Criteria*, 76 JAMA Neurology 56 (2018), filed as “Resp. Ex. A Tab 3” and “Resp. Ex. C Tab 1”.

⁴² Christine Goh et al., *MRI in Transverse Myelitis*, 40 J. Magnetic Resonance Imaging 1267 (2014), filed as “Resp. Ex. C Tab 5”.

⁴³ *Id.*

⁴⁴ Zalewski et al., *supra* note 41.

⁴⁵ M.I. Vargas et al., *Spinal Cord Ischemia: Practical Imaging Tips, Pearls, and Pitfalls*, 36 American J. Neuroradiology 825 (2015), filed as “Resp. Ex. C Tab 2”.

⁴⁶ Zalewski et al., *supra* note 41.

dated 12/18/2015. The constellation of findings suggest acute to subacute infarct.” Resp. Ex. C at 13; Pet. Ex. 12 at 228. He added that at the time it was felt that TM “may demonstrate similar appearance although is felt less likely given the interval development of postcontrast enhancement of the spinal cord and leptomeninges.” Resp. Ex. C at 13. Further, the imaging features over time remained consistent with acute SCI. Resp. Ex. F at 1; Resp. Ex. C at 10.

Dr. Zucconi opined that the embolic infarcts petitioner suffered in her cerebellum seen on the January 10, 2016 brain MRI “strongly supports” a diagnosis of SCI because the region of the affected spinal cord and cerebellum, particularly the left side where the spinal cord pathology was most pronounced, share a common blood supply in the vertebral arteries. Resp. Ex. C at 14; Resp. Ex. C Tab 6;⁴⁷ Tr. 228-29. A disease process affecting vertebral arteries is most likely the cause of both infarct events. Resp. Ex. C at 14; Resp. Ex. F at 2; Resp. Ex. C Tab 7;⁴⁸ Resp. Ex. C Tab 8.⁴⁹ It is common that no potential cause for petitioner’s SCI and cerebral infarct was identified. Resp. Ex. C at 14. He offered fibrocartilaginous embolism as a potential etiology of SCI here, given that petitioner had disc bulging and small protrusions at C4-5 and C5-6. Resp. Ex. F at 2; Resp. Ex. C Tab 4.⁵⁰

From a clinical standpoint, Dr. Zucconi concluded that petitioner’s acute presentation was consistent with SCI with corresponding neurological features and absence of evidence of neuroinflammation as noted by Dr. Lindsey. Resp. Ex. C at 14; *see* Resp. Ex. A. He submitted that, if onset were determined to be on November 4 and there was some sort of “stuttering onset” until December 18, he would expect much more apparent signal changes on the initial MRI as opposed to the subtle T2 signal that was actually seen. Tr. 235-36.

Based on the literature, the imaging here is “highly suggestive of SCI.” Resp. Ex. C at 14. The most compelling findings were the features of grey matter injury specific to SCI, the evolution of enhancement and edema, and the subsequent infarct in the same arterial distribution on brain MRI three weeks later. *Id.* Additionally, the evolution and spatial distribution of the abnormal spinal cord MRI findings are highly suggestive of acute spinal cord infarction and would be unusual for transverse myelitis. Resp. Ex. F at 2. It is also notable that 14-16% of patients diagnosed with TM are ultimately diagnosed with SCI upon further evaluation. Resp. Ex. C at 14; Resp. Ex. C Tab 1.⁵¹

IV. Legal Standards

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a “Table” injury—i.e., an injury listed on the Vaccine Injury Table

⁴⁷ Ethan A. Prince & Sun Ho Ahn, *Basic Vascular Neuroanatomy of the Brain and Spine: What the General Interventional Radiologist Needs to Know*, 30 *Seminars in Interventional Radiology* 234 (2013), filed as “Resp. Ex. C Tab 6”.

⁴⁸ Ken Johkura et al., *Combination of infarctions in the posterior inferior cerebellar artery and anterior spinal artery territories*, 207 *J. Neurological Sciences* 1 (2003), filed as “Resp. Ex. C Tab 7”.

⁴⁹ S. Ben Sassi et al., *Man-in-the-Barrel Syndrome with Combination of Infarctions in the Anterior Spinal Artery and Posterior Inferior Cerebellar Artery Territories*, 27 *Cerebrovascular Diseases* 201 (2009) filed as “Resp. Ex. C Tab 8”.

⁵⁰ Zalewski et al., *supra* note 24.

⁵¹ Zalewski et al., *supra* note 41.

that occurred within the provided time period. § 11(c)(1)(C)(i). “In such a case, causation is presumed.” *Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); see § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an “off-Table” injury, which requires that the petitioner “prove by a preponderance of the evidence that the vaccine at issue caused the injury.” *Capizzano*, 440 F.3d at 1320; see § 11(c)(1)(C)(ii). Initially, a petitioner must provide evidence that he or she suffered, or continues to suffer, from a definitive injury. *Broekelschen v. Sec’y of Health & Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a “substantial factor” and a “but for” cause of the injury is sufficient for recovery. See *Pafford v. Sec’y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec’y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999).⁵²

To prove causation for an “off-Table” injury, petitioners must satisfy the three-pronged test established in *Althen v. Sec’y of Health & Human Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioners show by preponderant evidence that a vaccination petitioner received caused his or her injury “by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.” *Id.* at 1278. Together, these prongs must show “that the vaccine was ‘not only a but-for cause of the injury but also a substantial factor in bringing about the injury.’” *Stone v. Sec’y of Health & Human Servs.*, 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with “no hard and fast per se scientific or medical rules.” *Knudsen v. Sec’y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify “specific biological mechanisms” to establish causation, nor are they required to present “epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities.” *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). “[C]lose calls regarding causation are resolved in favor of injured claimants.” *Althen*, 418 F.3d at 1280.

Each of the *Althen* prongs requires a different showing. The first *Althen* prong requires petitioner to provide a sound and reliable medical theory demonstrating that the vaccines received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted); *Knudsen*, 35 F.3d at 548. This theory need only be “legally probable, not medically or scientifically certain.” *Pafford*, 451 F.3d at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, “petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature.” *LaLonde v. Sec’y of Health & Human Servs.*, 746 F.3d 1334, 1341 (Fed. Cir. 2014).

The second *Althen* prong requires proof of a “logical sequence of cause and effect.” *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). In other words, even if the vaccinations can cause the injury, petitioner must show “that it did so in [this] particular case.”

⁵² The Vaccine Act also requires petitioners to show by preponderant evidence the vaccinee suffered from the “residual effects or complications” of the alleged vaccine-related injury for more than six months, died from the alleged vaccine-related injury, or required inpatient hospitalization and surgical intervention as a result of the alleged vaccine-related injury. § 11(c)(1)(D). It is undisputed that this requirement is satisfied in this case.

Hodges v. Sec’y of Health & Human Servs., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). A sound and reliable “medical or scientific explanation must support this logical sequence of cause and effect,” *id.* at 961 (citation omitted), and “treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury,” *Paluck v. Sec’y of Health & Human Servs.*, 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu ex rel. Andreu v. Sec’y of Health & Human Servs.*, 569 F.3d 1367, 1375 (Fed. Cir. 2009)). Petitioner is not, however, required “to eliminate alternative causes as part of establishing [their] prima facie case.” *Doe v. Sec’y of Health & Human Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); see *Walther v. Sec’y of Health & Human Servs.*, 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a “petitioner does not bear the burden of eliminating alternative independent potential causes”).

To satisfy the third *Althen* prong, 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 & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, “a petitioner’s failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause.” *Id.* However, “cases in which onset is too soon” also fail this prong; “in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked.” *Id.*; see also *Locane v. Sec’y of Health & Human Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) (“[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.”).

A. Factual Issues

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

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining facts such as the onset of a petitioner’s symptoms. *Vallenzuela v. Sec’y of Health & Human Servs.*, No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also *Eng v. Sec’y of Health & Human Servs.*, No. 90-175V, 1994 WL 67704, at *3 (Fed.

Cl. Spec. Mstr. Feb 18, 1994) (explaining that § 13(b)(2) “must be construed so as to give effect to § 13(b)(1) which directs the special master or court to consider the medical record...but does not require the special master or court to be bound by them”); *see also Burns v. Sec’y of Health & Human Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (holding that it is within the special master's discretion to determine whether to afford greater weight to medical records or to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is rational).

There are situations in which compelling oral testimony may be more persuasive than written records. *See Campbell ex rel. Campbell v. Sec’y of Health & Human Servs.*, 69 Fed. Cl. 775, 779 (2006). When witness testimony contradicts medical records, such testimony must be consistent, clear, cogent, and compelling to be persuasive. *See Sanchez v. Sec’y of Health & Human Servs.*, No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (vacated on other grounds, *Sanchez by & through Sanchez v. Sec’y of Health & Human Servs.*, No. 2019-1753, 2020 WL 1685554 (Fed. Cir. Apr. 7, 2020), review denied, *Sanchez by & through Sanchez v. Sec’y of Health & Human Servs.*, 152 Fed. Cl. 782 (2021)) (quoting *Blutstein v. Sec’y of Health & Human Servs.*, No. 90-2808V, 1998 WL 408611, at *85 (Fed. Cl. Spec. Mstr. June 30, 1998)); *see, e.g., Stevenson ex rel. Stevenson v. Sec’y of Health & Human Servs.*, No. 90-2127V, 1994 WL 808592, at *7 (Fed. Cl. Spec. Mstr. June 27, 1994) (crediting the testimony of a fact witness whose “memory was sound” and “recollections were consistent with the other factual evidence”). Special masters may also consider other types of evidence, such as unsworn statements, on the grounds that the Vaccine Program was designed to have “flexible and informal standards of admissibility of evidence.” 42 U.S.C. § 300aa-12(d)(2)(B); *see also Munn v. Sec’y of Health & Human Servs.*, 970 F.2d 863, 873 (Fed. Cir. 1992).

On the whole, a special master’s fact findings are to be upheld when the special master’s evaluation is evidence-based and not wholly implausible. *See Colon v. Sec’y of Health & Human Servs.*, 156 Fed. Cl. 534 (2021).

B. Evaluating Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec’y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court’s opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. “In short, the requirement that an expert’s testimony pertain to ‘scientific knowledge’ establishes a standard of evidentiary reliability.” *Id.* at 590 (citation omitted). Thus, for Vaccine Act claims, a “special master is entitled to require some indicia of reliability to support the assertion of the expert witness.” *Moberly ex rel. Moberly v. Sec’y of Health & Human Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010). The *Daubert* factors are used in the weighing of the reliability of scientific evidence proffered. *Davis v. Sec’y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen*, 618 F.3d at 1347 (citing

Lampe, 219 F.3d at 1362). And nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the ipse dixit of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder ex rel. Snyder v. Sec’y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

C. Consideration of Medical Literature

Finally, although this decision discusses some but not all of the literature in detail, the undersigned reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec’y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision.”); *Simanski v. Sec’y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) (“[A] Special Master is ‘not required to discuss every piece of evidence or testimony in her decision.’” (citation omitted)), *aff’d*, 601 F. App’x 982 (Fed. Cir. 2015).

V. Discussion

Because petitioner does not allege an injury listed on the Vaccine Injury Table, her claim is “off-Table.” As noted above, for petitioner to prevail on an “off-Table” claim, she must submit a sound and reliable theory that her alleged injury of transverse myelitis was caused by the flu vaccine she received on October 7, 2015. *Capizzano*, 440 F.3d at 1320.

Although all three *Althen* Prongs are at issue, the primary disputes in this case are the onset of petitioner’s injury and whether that injury was TM or a spinal cord infarct.

A. Petitioner failed to demonstrate a medically reasonable timeframe under *Althen* Prong III that supports an inference of causation.

There were several different onset dates proposed. Petitioner affirmed that “[o]n or about December 17, 2015,” she started to have numbness and pain in the left side of her neck that also involved her left arm. Pet. Ex. 1 at 1. But she testified that “[w]ithin the two to three weeks” post-vaccine, she had “unbearable” pain in her neck, back, and shoulder that she never felt before, like “two elephants” standing on each shoulder pressing down, prompting a visit to Mr. Jones on November 4, 2015 using her husband’s appointment that day. Tr. 16-17, 19, 47. She then described terrible pain that she had never experienced before on the morning of December 17, 2015 prompting her to present to urgent care. Tr. 23-24. The following morning, December 18, 2015, she fell and couldn’t move. Tr. 24-25.

Dr. Levy’s opinion on onset was difficult to follow because every time he was asked, he changed the onset of petitioner’s symptoms. He initially opined that petitioner suffered an onset of left-sided neck and arm pain on November 21, 2015, 45 days after the flu vaccine, with an acute onset of sensory changes to the left side of her neck and shoulder on December 17, 2015 and progression from onset to peak within 4 hours-21 days, meeting the 2002 Working Group criteria for idiopathic TM. Pet. Ex. 42 at 2, 4; Tr. 88, 117, 177. In a subsequent report, he again stated that

onset was November 21, 2015 but testified that he could not recall where this date came from. He then claimed that onset was November 4, 2015 based on Mr. Jones' records. Pet. Ex. 62 at 1; Tr. 177. He also opined that petitioner's myelopathy exacerbated on December 18, 2015, extending the progression to nadir beyond the 21-day window imposed by the criteria of the 2002 TM Working Group, "if this criteria were deemed absolute." Pet. Ex. 62 at 1; Tr. 115-16, 133-34. He argued that in his experience, the 21-day cut off from onset to nadir excludes some true acute TM patients. Pet. Ex. 62 at 2; Tr. 60, 111-13. He cited *Barreras* as support, noting 27% of the 247 cases of inflammatory TM studied evolved to nadir in more than 21 days. Pet. Ex. 62 at 2; Pet. Ex. 64;⁵³ Tr. 75-77, 134-36, 188.

At hearing, Dr. Levy pointed to the December 18, 2015 hospital record which documented left shoulder pain for two months treated with massage therapy stating that this supported that some of her neurological symptoms could have started two months prior to the abrupt, acute onset of symptoms on December 17. Tr. 91-92. He then stated that her symptoms started two weeks prior to her presentation around December 4, 2015 with nadir on December 18, 2015 when she presented to the hospital and was nearly completely paralyzed. Tr. 98, 115, 125, 176. Therefore, "[s]omething in the between there is probably accurate." Tr. 93. He argued that the timeline in this case was consistent with a link between the vaccine and petitioner's later acute myelopathic decline. Pet. Ex. 62 at 2. He ultimately stated that so long as the date of onset is determined to be between October 7 when the vaccine was given and the "event in December", "then it's consistent with [his] theory that the vaccine started this transverse myelitis process". Tr. 179.

Dr. Levy agreed that the medically appropriate timeframe for the onset of TM in the majority of spinal cord attacks post-vaccination is within 30 days but added that studies have shown up to 90 days for a vaccine attack on the spinal cord. Pet. Ex. 42 at 4. He explained that the onset of TM is expected within 2-3 weeks after antigen exposure, which coincides with the time required for the adaptive immune system to process foreign antigens and activate a response to them. Pet. Ex. 48 at 2-3; Tr. 96-98. Using GBS as a proxy and relying on *Schonberger*, he argued that the risk-interval post-vaccination is primarily within 5 weeks but can last 9-10 weeks in total. Pet. Ex. 83 at 2; Pet. Ex. 84.⁵⁴ Additionally, *Baxter* included 67 cases of TM occurring within 9 months of immunization, with 38 of the 67 cases following flu vaccine. However, he agreed that if the timing was confined to 5-28 days post-vaccination, then there would have been only 7 cases. Pet. Ex. 62 at 3; Resp. Ex. A Tab 6;⁵⁵ Tr. 170.

Following the hearing, Dr. Levy opined that 8-10 weeks between vaccine and onset was medically appropriate. Serum antibody levels to surface proteins on the flu vaccine peak around 4-6 weeks following vaccination but could peak as early as 2 weeks and up to 16 weeks after administration; Dr. Levy conceded that the longer interval of 8-16 weeks occurs in adjuvanted vaccines. Pet. Ex. 83 at 1; Pet. Ex. 76.⁵⁶ Further, seroprotective levels of antibodies are present for at least 90 days, with some studies indicating that circulating levels of antibodies can remain high for many months to years. Pet. Ex. 83 at 1-3; Pet. Ex. 85 at 2-3.

⁵³ *Barreras et al., supra* note 17.

⁵⁴ *Schonberger et al., supra* note 14.

⁵⁵ *Baxter et al., supra* note 15.

⁵⁶ Institute of Medicine of the National Academies, *supra* note 18.

He argued that *Mealy* determined an increased risk of relapse in NMO patients who had already been diagnosed with NMO of up to 90 days post-vaccine with the highest risk during the first 30 days. Pet. Ex. 83 at 2; Pet. Ex. 45;⁵⁷ Tr. 119, 180-88. He stated that the 30-day risk interval was chosen in the study because beyond 30 days “is not as well accepted.” Tr. 180-81. Dr. Levy submitted the biological and clinical data from animal and human studies supports the onset of TM between 8-10 weeks after receipt of vaccination as appropriate although rare. Pet. Ex. 83 at 2; Tr. 99-100, 121, 125-26.

Dr. Levy proposed it was reasonable that petitioner’s immune response to the flu vaccine peaked after 2 weeks but remained at a sufficient seropositive level for at least 3 months, or, alternatively, that her immune response did not peak until 8-16 weeks post vaccine, even though it was not an adjuvanted vaccine. Pet. Ex. 83 at 3; Tr. 162-63. He opined that a vaccine given on October 7 could cause TM that starts to develop around November 4 with symptoms staying the same for another six weeks until there is a rapid, abrupt onset of more severe symptoms. Tr. 118-19. He added that based on literature and his clinical experience, a flu vaccine 10 weeks prior to onset is “realistically not too far removed to be an immunologically appropriate etiological factor”. He conceded there is not much reliable data beyond 10-12 weeks, other than compelling factual and logical sense that in some individuals a longer time is medically appropriate. Pet. Ex. 85 at 2, 3. However, it is his opinion that the timeline in this case is consistent with a link between the vaccine and petitioner’s later acute myelopathic decline. Pet. Ex. 62 at 2.

Dr. Levy did not offer an opinion on a medically reasonable timeframe for onset of SCI following flu vaccine.

Dr. Lindsey opined that onset in this case was on December 17, 2015—71 days or roughly 10 weeks post-vaccination—when petitioner presented with new left neck pain and severe left shoulder pain. Resp. Ex. A at 3; Resp. Ex. I at 1; Tr. 300-01, 303, 308, 323-24. He disagreed that the records supported a “stuttering onset” at some point in November of 2015 which rapidly worsened on December 17, 2015, stating that a very long prodromal period with little progression until a sudden, severe onset of symptoms does not “seem terribly plausible” in his opinion. Tr. 309-10, 313, 319-20; Resp. Ex. H at 1.

He added that a 70-day onset is atypical for a trigger of autoimmune disease. Resp. Ex. A at 7. Rather, *Nordin* and *Baxter* demonstrated the risk interval for onset of TM after flu vaccine to be 4-6 weeks. Resp. Ex. I at 2; Resp. Ex. E at 2; Resp. Ex. A Tab 6;⁵⁸ Resp. Ex. A Tab 7;⁵⁹ Tr. 308-13, 319-23. He pointed out Dr. Levy’s acknowledgment that “10 weeks is on the longer end of the medically appropriate vaccine safety risk interval.” Resp. Ex. I at 2; Pet. Ex. 83 at 3; Tr. 324.

Dr. Lindsey addressed Dr. Levy’s opinion regarding seroprotective levels of antibodies following vaccination. He agreed antibodies may be elevated for months after vaccination, but the “generally accepted period for vaccine-induced autoimmunity is much shorter” likely because

⁵⁷ Mealy et al., *supra* note 19.

⁵⁸ Baxter et al., *supra* note 15.

⁵⁹ Nordin et al., *supra* note 27.

autoimmunity requires both autoimmune T cells and autoantibodies, and effector T cell responses are short lived. Resp. Ex. I at 2; Pet. Ex. 86 at 16.

Dr. Lindsey concluded that petitioner had a rapid onset of myelopathy 71 days from vaccination, longer than the expected timeframe for autoimmune-mediated disease. Resp. Ex. A at 7; Resp. Ex. H at 1.

The totality of the evidence filed in this case supports an abrupt onset of pain and symptoms on December 17, 2015 which petitioner admitted she had never experienced before. Tr. 23-25. Petitioner presented to her endocrinologist on October 26, 2015, and reported doing well and exercising three times per week. The examination that day was normal. Pet. Ex. 9 at 30-32. She presented again to the endocrinologist on December 7, 2015, and reported exercising and going to the gym. The examination at this visit was also normal. *Id.* at 37-39.

Petitioner presented to urgent care on December 17, 2015, and reported symptoms of numbness and pain that “[s]tarted this morning” upon waking. Pet. Ex. 10 at 2. When she presented again the following day, December 18, 2015, she reported onset of pain in the left shoulder and arm and inability to move her left arm or leg which started 12 hours prior. Pet. Ex. 11 at 4, 10. She reported being able to move all extremities the night before but unable to move when she woke up that morning. *Id.* at 10. She also reported left shoulder pain two months and two weeks prior. *Id.* at 19, 23.

Petitioner’s medical records on December 17 and 18, 2015 are consistent with her initial affirmation that “[o]n or about December 17, 2015,” she started to have numbness and pain in the left side of her neck that also involved her left arm. Pet. Ex. 1 at 1. In addition, at hearing, she stated that she had “terrible pain that she had never experienced before” on the morning of December 17, 2015. Tr. 23-25.

The assertion that petitioner’s symptoms began at some point in November of 2015 occurred after the filing of the petition, medical records, and petitioner’s affidavit and with the appearance of records from Mr. Jones for petitioner’s visits on November 4 and November 13, 2015. For the reasons detailed above, the various records from Mr. Jones that became more elaborate with each filing raise questions of credibility. Besides being internally inconsistent, Mr. Jones’ records are inconsistent with the endocrinologist records, which documented normal examinations, no complaints, and petitioner’s reports of regular exercise following her visits with Mr. Jones. *See* Pet. Ex. 9 at 30-32, 37-39. It is difficult to believe that anyone would report being well with no complaints, exercising, and going to the gym regularly if they were in unbearable, excruciating pain as described in Mr. Jones’ subsequently filed records. It is further difficult to reconcile petitioner’s report to her medical providers, her affirmations, and the petition that the onset of her severe pain was on December 17, 2015, if she had been suffering from excruciating and debilitating pain since November 4, 2015. Finally, while petitioner testified to unbearable pain “[w]ithin the two to three weeks” of her vaccination, prompting the visit to Mr. Jones, this onset was not mentioned in her initial affidavit. Tr. 16-17, 19; Pet. Ex. 1; *see, e.g., R.K. v. Sec’y of Health & Human Servs.*, No. 03-0632V, 2015 WL 10936124, at *76 (Fed. Cl. Spec. Mstr. Sept. 28, 2015) (holding that more remote histories of illness do not have sufficient indicia of reliability to be credited over conflicting contemporaneous medical records and earlier reported histories), *mot. for*

rev. denied, 125 Fed Cl. 57 (2016), *aff'd*, 671 F. App'x 792 (Fed. Cir. 2016); *see also Vergara v. Sec'y of Health & Human Servs.*, No. 08-882V, 2014 WL 2795491, *4 (Fed. Cl. Spec. Mstr. May 15, 2014) (“Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recounted in later medical histories, affidavits, or trial testimony”). The allegation of symptoms beginning in November of 2015 is inconsistent with the contemporaneous medical records and with petitioner’s own statements elsewhere in the record.

Even if I were to accept Mr. Jones’ records as credible and, more broadly, that onset was in November of 2015, petitioner failed to demonstrate how onset of symptoms could begin in November, stay relatively stable for well over six weeks, then abruptly and severely worsen to the point of paralysis over 71 days post-vaccination. Dr. Levy stated that this could occur based on his experience but cited no literature to support it. Tr. 118-19; *Snyder*, 88 Fed. Cl. at 742-43; *Perreira ex rel. Perreira v. Sec'y of Health & Human Servs.*, 33 F.3d 1375, 1377 n. 6 (Fed.Cir.1994) (“An expert opinion is no better than the soundness of the reasons supporting it.” (citing *Daubert*, 509 U.S. at 579)).

The evidence herein supports onset on December 17, 2015, 71 days or 10 weeks post-vaccination. Whether this time interval is medically appropriate for TM or SCI following flu vaccination is therefore at issue. On this point, respondent’s experts were more persuasive. Both Dr. Lindsey and Dr. Levy agreed that the generally accepted timeframe for immune-mediated disease is around 4-6 weeks post-exposure. Pet. Ex. 83 at 2; Tr. 119, 180-88, 308-13, 319-23; Resp. Ex. I at 2; Resp. Ex. E at 2. Petitioner’s onset here is more than double that. Dr. Levy conceded that longer timeframes for onset typically occur in adjuvanted vaccines. Pet. Ex. 83 at 1, 3. The subject vaccine in this case did not contain an adjuvant.

Dr. Levy’s argument regarding seroprotective antibodies was not persuasive. He explained, and Dr. Lindsey agreed, that antibodies circulate for 90 days, if not more. Dr. Levy, however, failed to explain how circulating seroprotective antibodies can trigger a T-cell or B-cell mediated autoimmune process 10 weeks after exposure. Pet. Ex. 85 at 1-3. Dr. Lindsey pointed out that autoimmune disease requires both autoimmune T cells and autoantibodies, and “[e]ffector T-cell responses are short-lived” with most effector T-cells dying within a few days. Resp. Ex. I at 2; Pet. Ex. 86 at 16.

Dr. Levy claimed it made “logical sense” that a longer onset may occur in some people. Pet. Ex. 85 at 2, 3. Recognizing that there are always those who may fall outside of what is generally accepted as the norm based on individual differences and that this an important consideration when dealing with adverse reactions to vaccines, that alone is not sufficient to satisfy petitioner’s burden in proving by preponderant evidence that a 10-week onset is medically reasonable. This would be a different case if the onset of a spinal cord injury was 43 days, rather than 3-42 days post-vaccination as reflected in the Vaccine Injury Table as a medically accepted timeframe for GBS after flu vaccine. Indeed, special masters including myself have found entitlement, albeit rarely, in TM, ADEM, and GBS cases up to 65 days after vaccination. *See, e.g., Songero v. Sec'y of Health & Human Servs.*, No. 18-300V, 2025 WL 3013090, at *19 (Fed. Cl. Spec. Mstr. Oct. 3, 2025) (onset of TM 63 days after vaccine was reasonable “on rare occasion”); *Brown v. Sec'y of Health & Human Servs.*, No. 09-426V, 2011 WL 5029865, at *43-44 (Fed. Cl. Spec. Mstr. Sept. 30, 2011) (onset of ADEM 60 days after flu vaccine based

on *Schonberger and Langmuir*); *Doe v. Sec'y of Health & Human Servs.*, 2010 WL 4205677, at *25 (Fed. Cl. Spec. Mstr. Oct. 20, 2010), *vacated sub nom. on other grounds Doe 93 v. Sec'y of Health & Human Servs.*, 98 Fed. Cl. 553 (2011) (onset of TM 65 days after flu vaccine); *Spayde v. Sec'y of Health and Human Servs.*, No. 16-1499V, 2021 WL 686682, at *19 (Fed. Cl. Spec. Mstr. Jan. 27, 2021) (60 days is “reasonable and appropriate” in a flu/GBS case); *Cooper v. Sec'y of Health & Human Servs.*, No. 18-1885V, 2024 WL 1522331, at *20 (Fed. Cl. Spec. Mstr. Mar. 12, 2024) (60-day onset acceptable period for causation in a Prevnar/GBS case).

Here, petitioner’s onset was 71 days post-vaccination, which is far outside what is generally accepted in the scientific community for immune-mediated diseases and outside the outer limit of what has been found reasonable in rare vaccine cases. *See, e.g., Ray v. Sec'y of Health & Human Servs.*, No. 20-321V, 2025 WL 3900631, at *22 (Fed. Cl. Spec. Mstr. Dec. 12, 2025) (finding 74 days or more than 10 weeks post-vaccination too long for the petitioner's GBS/CIDP to be causally associated with the subject vaccine); *Randolph v. Sec'y of Health & Human Servs.*, No. 18-1231V, 2020 WL 542735, at *8 (Fed. Cl. Spec. Mstr. Jan. 2, 2020) (denial due to onset of GBS 76 days post-vaccination, “well outside the 3-42 day window set by the Table for a flu-GBS claim”). Further, petitioner offered no evidence to support a medically reasonable timeframe between a flu vaccine and SCI.

As such, petitioner has failed to satisfy *Althen* Prong III.

B. The evidence best supports SCI as the diagnosis. But regardless of diagnosis, the onset of symptoms was too remote in time to demonstrate a logical sequence of cause and effect under *Althen* Prong II.

Having concluded that the onset of petitioner’s illness was December 17, 2015, 71 days or 10 weeks after her receipt of the subject flu vaccination on October 7, 2015, whether she suffered from TM or SCI is irrelevant since there is no support for either resulting from a flu vaccine 10 weeks after vaccination. Nevertheless, petitioner’s diagnosis will be briefly addressed as it is the second fatal flaw to this case.

As a threshold matter, petitioner must establish that she actually suffered the injury alleged in the petition. *See Broekelschen v. HHS*, 618 F.3d 1339,1346 (Fed. Cir. 2010). The Federal Circuit has made clear that, “the statute places the burden on petitioner to make a showing of at least one defined and recognized injury.” *Lombardi v. HHS*, 656 F.3d 1343,1353 (Fed. Cir. 2011) (affirming a special master’s decision to dismiss a petition when the petitioner could not establish that she had any of the three diagnoses alleged). “The function of a special master is not to ‘diagnose’ vaccine-related injuries, but instead to determine based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of the evidence that a vaccine caused [petitioner’s] injury.” *Lombardi*, 656 F.3d at 1352-53 (internal citation omitted). Thus, where “the existence and nature of the injury itself is in dispute, it is the special master’s duty to *first determine* which injury is best supported” by the evidence before applying the *Althen* test to determine causation. *Id.* at 1352 (citing *Broekelschen*, 618 F.3d at 1345) (emphasis added).

The Federal Circuit has recognized that the opinions of treating providers are generally owed a level of deference. *See, e.g., Capizzano*, 440 F.3d at 1326. This recognition is “especially

apt” in determining diagnosis, since “no person is better qualified to opine on [a petitioner’s] condition . . . than the physicians that treated [them] at that time.” *Smith v. Sec’y of Health & Human Servs.*, No. 15-1194V, 2018 WL 6242453, at *1, *5 (Fed. Cl. Spec. Mstr. Oct. 31, 2018). Medical records and statements of a treating physician, however, do not per se bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion judgment test result, report, or summary shall not be binding on the special master or court”); *Snyder*, 88 Fed. Cl. at 746 n.67 (“there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”). The views of treating physicians should be weighed against other, contrary evidence also present in the record. *Hibbard v. Sec’y of Health & Human Servs.*, 100 Fed. Cl. 742,749 (2011), *aff’d*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec’y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den’d*, 100 Fed. Cl. 344, 356 (2011), *aff’d without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

Dr. Levy opined that TM was the correct diagnosis. Pet. Ex. 42 at 5; Tr. 98, 106, 123. Despite being contrary to his opinion on onset, Dr. Levy argued that repeat MRI on December 31, 2015, 13 days after petitioner’s presentation, showed an enhanced, developing lesion, suggestive of some sort of quickly evolving process more consistent with TM as opposed to vascular neuropathy. Additionally, repeat CSF at that time showed the presence of inflammatory cells, suggestive of an inflammatory cause of symptoms. Pet. Ex. 62 at 2; Tr. 95, 98-99, 106-08, 142-45. He added that the inflammatory cells on repeat CSF testing were beyond what would be expected in a vascular myelopathy, and the swelling and enhancement seen on the repeat MRI was more severe and inflamed than would be expected for vascular myelopathy. Pet. Ex. 62 at 2; Tr. 189-91.

Dr. Levy also argued that petitioner’s subsequent complications including embolic strokes in her cerebellum were “directly” linked to her TM due to hypercoagulability associated with hospitalization and immobility that leads to weakness and autonomic dysfunction where venous circulation is compromised. Pet. Ex. 42 at 5; Pet. Ex. 62 at 3; Tr. 155-58. Other complications including lung nodules, pleural effusion, sepsis, and thrombocytopenia were all inflammatory or infectious in nature and contributed to petitioner’s myelopathy. Pet. Ex. 62 at 3.

Dr. Levy concluded that SCI was less likely because petitioner did not have pre-existing vascular disease prior to symptom onset and the angiogram, performed to rule out arteriovenous malformation or other causes of vascular myelopathy, was normal. Pet. Ex. 42 at 5; Pet. Ex. 62 at 2; Tr. 89-90, 153-54. However, Dr. Levy conceded the embolic strokes indicated that “her blood vessels aren’t great”. Tr. 156. He also later conceded that petitioner “had maybe hypertension”, which is a risk factor for vascular myelopathy. Tr. 160.

Dr. Levy ultimately agreed that inflammatory TM and vascular myelopathies are hard to distinguish. Pet. Ex. 62 at 4; Tr. 75. He also recognized that *Barreras* noted that a TM diagnosis is wrong 54% of the time with a different diagnosis a better fit. Tr. 196-97; Pet. Ex. 64.⁶⁰ Dr. Levy agreed that vascular myelopathy was possible, but TM was more likely because she had no preceding vascular risk factors but did have an inflammatory challenge in the form of a vaccine,

⁶⁰ *Barreras et al.*, *supra* note 17.

repeat MRI and CSF were both consistent with myelitis and not vascular myelopathy, the complications during hospitalization were inflammatory in nature, and the vascular myelopathy workup was negative. He also noted that petitioner responded to corticosteroids which is more consistent with TM than with vascular myelopathy. Pet. Ex. 62 at 4; Tr. 70-71, 148, 150-51, 169, 197-98.

On the other hand, respondent argued that petitioner's course was more consistent with a spinal cord infarct than with TM.

Dr. Lindsey argued that petitioner's presentation did not meet the 2002 TM Working Group Criteria. Resp. Ex. A at 4, 7; Resp. Ex. A Tab 2.⁶¹ He opined that petitioner's presentation of prodromal symptoms on December 17, 2015 with a rapid progression to nadir on December 18, 2015 fits the clinical picture of a hyperacute stroke. Resp. Ex. E at 1. Further, petitioner's initial CSF was normal and repeat testing two weeks later showed only mild inflammation. Additionally, repeat cervical MRI showed an increased enhanced lesion, consistent with evolving spinal cord infarct. Resp. Ex. A at 3-4; Resp. Ex. E at 1; Tr. 301-02. Petitioner was treated with steroids and IVIg, neither of which resulted in significant neurologic improvement, suggesting a non-inflammatory cause of her condition. Tr. 307-08. He disagreed that the angiogram performed one month after onset of symptoms was informative of the etiology of petitioner's symptoms. Resp. Ex. E at 1. He also disagreed that the complications petitioner experienced during hospitalization were informative of etiology. If anything, he submitted the cerebellar strokes were more suggestive of her susceptibility to hypercoagulation which could have contributed to a vascular myelopathy. *Id.* at 1-2; Resp. Ex. A at 4-5.

Dr. Lindsey added that *Barreras* showed that the spinal fluid results for inflammatory myelitis and ischemic myelopathy overlap, thus there was no definitive distinction between the two. Resp. Ex. E at 1; Pet. Ex. 64, Table 2.⁶² Though he would have diagnosed a vascular myelopathy if petitioner had been his patient, he agreed it was appropriate to treat her for TM because there was a chance that this was indeed TM. Tr. 351-52.

In Dr. Lindsey's opinion, petitioner more likely suffered from spinal cord infarct based on the clinical and objective findings all consistent with infarct. Resp. Ex. A at 4, 6; Tr. 326. She presented with rapid progression of deficits, pain at onset, sensory loss selective for pain and temperature, bladder dysfunction, and areflexia. Resp. Ex. A at 4; Resp. Ex. H at 1; Tr. 301-02. Between the initial MRI and CSF testing and subsequent testing 13 days later, there was an evolution consistent with vascular myelopathy. Resp. Ex. E at 2. The primary clinical findings that argue against TM are the relatively abrupt onset, the lack of inflammation seen on the December 18, 2015 MRI, and normal CSF. Tr. 302, 305-06.

Similarly, Dr. Zucconi opined that SCI was a more likely diagnosis. He detailed all of the imaging in this case, including the cervical MRI on December 18, 2015 which showed subtle spinal cord edema between C2 and C5 levels, suggestive of early spinal cord infarction. Resp. Ex. F at 1; Resp. Ex. C at 4. The early left anterior and central grey matter involvement seen is expected in

⁶¹ Transverse Myelitis Consortium Working Group, *supra* note 25.

⁶² *Barreras et al.*, *supra* note 17.

SCI. Resp. Ex. F at 1; Resp. Ex. C at 5, 12; Resp. Ex. G;⁶³ Tr. 211-14. In contrast, acute, severe transverse myelitis would likely not correspond with such subtle MRI findings. Resp. Ex. F at 1; Tr. 235-36.

Dr. Zucconi pointed out that the subsequent cervical MRI on December 31, 2015 showed the evolution of the cord abnormality and enhancement for a spinal cord infarct as expected. Resp. Ex. F at 1; Resp. Ex. C at 6; Tr. 220. It revealed a much more conspicuous cord pathology and T2 signal hyperintensity. The hyperintensity was predominantly central and anterior, and it conformed to the morphology of the central grey matter. Resp. Ex. C at 6-9; Pet. Ex. 56; Tr. 220-21. The evolution and spatial distribution of the abnormal spinal cord MRI findings are highly suggestive of acute spinal cord infarction and would be unusual for transverse myelitis. Resp. Ex. F at 2.

Further, Dr. Zucconi argued that the pattern of enhancement on the January 5, 2016 cervical MRI was better delineated and increased, but stable in distribution, which is characteristic of evolving cytotoxic edema in SCI. Resp. Ex. C at 9; Pet. Ex. 57; Tr. 226-28. The brain MRI on January 10, 2016 showed acute embolic infarcts in the cerebellar hemisphere, which shares a common blood supply with the spinal cord. Resp. Ex. C at 9-10; Pet. Ex. 58; Tr. 228-29. He argued that a disease process affecting vertebral arteries is most likely the cause of both the initial infarct and the later embolic infarcts. Resp. Ex. C at 14; Resp. Ex. F at 2; Resp. Ex. C Tab 7;⁶⁴ Resp. Ex. C Tab 8.⁶⁵

Dr. Zucconi concluded that the imaging here was compatible with SCI, specifically the preferential signal abnormalities affecting the grey matter that evolved, initially seen as subtle but over a period of days reflected cytotoxic edema and cell death. Resp. Ex. C at 12. The signal changes confined to the central grey matter is specific for cord infarction and incompatible with TM. *Id.* at 10, 12-13; Resp. Ex. C Tab 1;⁶⁶ Resp. Ex. C Tab 2;⁶⁷ Tr. 206.

Dr. Zucconi opined that based on the clinical information and imaging, the differential diagnosis for petitioner's spinal cord disease is essentially limited to spinal cord infarct or TM, but more likely SCI. Resp. Ex. C at 11; Tr. 206, 223, 225, 258, 261. However, he agreed that if he were viewing petitioner's records and MRIs on December 31, having no other information, he would have included both TM and SCI in the differential. Tr. 222-23.

Ultimately, all three experts agreed that significant overlap exists between what is seen in TM and SCI, making them difficult to distinguish. Pet. Ex. 62 at 4; Pet. Ex. 64;⁶⁸ Resp. Ex. C at 11; Resp. Ex. E at 1; Tr. 70-71, 75, 148, 150-51, 169, 196-98, 206, 222-23, 225, 258, 261, 351-52. They also agreed that TM is often misdiagnosed, with SCI frequently being the correct diagnosis upon further investigation. Tr. 196-97, 304-05; Pet. Ex. 64; Resp. Ex. A at 4; Resp. Ex. A Tab 1;⁶⁹ Resp. Ex. C at 14; Resp. Ex. C Tab 1.⁷⁰

⁶³ *Id.*

⁶⁴ Johkura et al., *supra* note 48.

⁶⁵ Sassi et al., *supra* note 49.

⁶⁶ Zalewski et al., *supra* note 41.

⁶⁷ Vargas et al., *supra* note 45.

⁶⁸ Barreras et al., *supra* note 17.

⁶⁹ Zalewski et al., *supra* note 24.

⁷⁰ Zalewski et al., *supra* note 41.

Throughout the medical records and petitioner's extensive treatment, she received several differential diagnoses, including both TM and spinal cord infarct. Though TM was initially listed high in the differential, the progression of petitioner's condition during her hospitalization, including the evolution of CSF and MRI findings, indicated the most likely diagnosis was SCI. Pet. Ex. 12 at 226-28 (radiologist noting that, when compared with the December 18 MRI of the cervical spine, the December 31, 2015 findings present progressive cytotoxic edema of the entire spinal cord with new patchy curvilinear enhancement from C2 to C6, primarily involving the gray matter as well as the leptomeninges. "Constellation of these findings suggests acute to subacute infarct. Alternatively, transverse myelitis may demonstrate similar appearance although it is felt less likely given the interval development of postcontrast enhancement of the spinal cord and leptomeninges. Potentially, dural arteriovenous fistula with venous infarction may represent an etiology. However, evaluation of the dural arteriovenous fistula is limited on this examination secondary to incorrect protocol."); Pet. Ex. 12 at 369-71, 938 (January 10, 2016 MRI findings compatible with acute to early subacute infarction).

Respondent's experts pointed out compelling evidence in the medical records to support SCI as the diagnosis. They explained that an abrupt onset of symptoms is consistent with SCI and not with TM. Resp. Ex. E at 1; Tr. 302, 305-06. As detailed above, onset of symptoms petitioner had never experienced before was on December 17, and petitioner was nearly completely paralyzed by the morning of December 18. Pet. Ex. 10 at 2-3; Pet. Ex. 11 at 4, 10, 19, 23; Pet. Ex. 12 562.

Dr. Zucconi was particularly persuasive in discussing the imaging as it relates to the issue of diagnosis, given that neuroradiology is his area of expertise. Resp. Ex. C. He submitted that grey matter involvement is specific to SCI and incompatible with TM. Resp. Ex. C at 5-9, 10, 12-13; Resp. Ex. C Tab 1;⁷¹ Resp. Ex. C Tab 2;⁷² Pet. Ex. 56; Resp. Ex. F at 1; Resp. Ex. G;⁷³ Tr. 206, 211-14, 220-21. Furthermore, the evolution of MRI findings—from subtle on the initial MRI on December 18 to clear hyperintensity on the repeat MRIs on December 31 and January 5—reflects cytotoxic edema and cell death that occurs in SCI. Resp. Ex. C at 4, 6-9, 12, 14; Resp. Ex. F at 1. The subtle findings on imaging and normal CSF on December 18 were inconsistent with TM in the presence of such severe symptoms. Pet. Ex. 12 at 150-51; Resp. Ex. A at 4; Resp. Ex. E at 2.

The experts also explained that an inflammatory process like TM would improve with immunotherapies, unlike SCI. Here, the records show that petitioner's condition improved only slightly with corticosteroids which prompted the initiation of IVIg, but any progress was not sustained. Pet. Ex. 12 at 599, 686, 703-05; Tr. 307-08.

Additionally, petitioner suffered cerebellar strokes, which Dr. Zucconi noted is consistent with SCI because the cerebellar hemisphere shares a common blood supply with the spinal cord, so it is most likely that a single underlying process affecting the vertebral arteries was responsible for both the initial stroke and the later strokes during hospitalization. The fact that petitioner

⁷¹ *Id.*

⁷² Vargas et al., *supra* note 45.

⁷³ Barreras et al., *supra* note 17.

suffered later cerebellar strokes also underscores her susceptibility to hypercoagulation. Resp. Ex. A at 4-5; Resp. Ex. E at 1-2; Resp. Ex. C at 9-10, 14; Pet. Ex. 58; Tr. 228-29; Resp. Ex. F at 2; Resp. Ex. C Tab 7;⁷⁴ Resp. Ex. C Tab 8.⁷⁵ Dr. Levy conceded that petitioner's blood vessels were not in the best condition, as evidenced by the cerebellar strokes she suffered, and that she had hypertension, which is a risk factor for SCI. Pet. Ex. 42 at 5; Pet. Ex. 62 at 3; Tr. 156, 160.

Based on the evidence filed, I find that SCI is the best supported diagnosis for petitioner's condition. In addition to failing to provide a medically reasonable timeframe for onset, petitioner also failed to support by preponderant evidence the diagnosis alleged. As such, petitioner has not demonstrated a logical sequence of cause and effect between the flu vaccination and her injury under *Althen* Prong II.

C. Petitioner failed to provide preponderant evidence of a sound and reliable theory that a flu vaccine could cause SCI under *Althen* Prong I.

The first *Althen* prong requires petitioner to provide a sound and reliable medical theory demonstrating that the vaccines received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted); *Knudsen*, 35 F.3d at 548. This theory need only be "legally probable, not medically or scientifically certain." *Pafford*, 451 F.3d at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1341 (Fed. Cir. 2014).

Petitioner's causation theory hinges on a finding that she had TM. Dr. Levy's theory that flu vaccine can cause TM is based on molecular mimicry. Dr. Levy admitted that there is no known cause or mimic for TM, but molecular mimicry would best describe the immunological process by which a vaccine or infection could cause TM. Pet. Ex. 42 at 4; Pet. Ex. 44 at 2; Pet. Ex. 48 at 1-4; Pet. Ex. 85 at 2-3. Dr. Levy provided no evidence that a flu vaccine could cause SCI. Dr. Lindsey submitted that there is no convincing evidence connecting vaccines to myelitis. Resp. Ex. A at 5-7.

As detailed at length above, preponderant evidence supports SCI as the diagnosis—not TM. "Because causation is relative to the injury, a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case, although the explanation need only be 'legally probable, not medically or scientifically certain.'" *Broekelschen*, 618 F.3d at 1345 (quoting *Knudsen*, 35 F.3d at 548-49). Thus, my finding that petitioner did not suffer from TM renders her causation theory that a flu vaccine can cause TM completely irrelevant. Because she provided no evidence that the subject vaccine could cause SCI, petitioner failed to provide a sound and reliable theory under *Althen* Prong I.

VI. Conclusion

Petitioner has my sincere sympathies for all that she has and continues to endure. However, my decision must be based on thorough consideration of all the evidence and application of the

⁷⁴ Johkura et al., *supra* note 48.

⁷⁵ Sassi et al., *supra* note 49.

law. Petitioner in this matter failed to provide preponderant evidence to demonstrate that the flu vaccine she received on October 7, 2015 played any role in the spinal cord infarct she suffered on December 17, 2015. Thus, she is not entitled to compensation.

In the absence of a timely filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accordance with this decision.⁷⁶

IT IS SO ORDERED.

s/ Mindy Michaels Roth

Mindy Michaels Roth
Special Master

⁷⁶ Pursuant to Vaccine Rule 11 (a), if a motion for review is not filed within 30 days after the filing of the special master's decision, the clerk will enter judgment immediately.