

In the United States Court of Federal Claims

No. 15-1137
(Filed: 17 June 2020*)

PITEY MORGAN,

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Petitioner,

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v.

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Vaccine Act; off-table case; influenza vaccine; longitudinally-extensive transverse myelitis (“LETM”); neuromyelitis optica spectrum disorder (“NMOSD”).

SECRETARY OF HEALTH AND HUMAN SERVICES,

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Respondent.

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Sylvia Chin-Caplan, of Law Office of Sylvia Chin-Caplan, with whom was *Timothy J. Mason*, both of Boston, MA for petitioner.

Zoe Wade, Trial Attorney, Torts Branch, Civil Division, U.S. Department of Justice, with whom were *Joseph H. Hunt*, Assistant Attorney General, *C. Salvatore D’Alessio*, Acting Director, *Catharine E. Reeves*, Deputy Director, *Heather L. Pearlman*, Assistant Director, all of Washington, DC, for respondent.

OPINION AND ORDER

Petitioner Pitey Morgan (“petitioner”) moved for review of Chief Special Master Corcoran’s decision that petitioner is not entitled to compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. §§ 300aa-10–300aa-34 (“Vaccine Act”). Petitioner claims he suffered longitudinally extensive transverse myelitis (“LETM”) caused by the influenza (“flu”) vaccine he received on 16 October 2012. The Special Master denied compensation and found petitioner did not “offer[] preponderant evidence to support the alleged diagnosis of LETM, whereas the record evidence preponderates in favor of an alternative diagnosis: Neuromyelitis Optica Spectrum Disorder (“NMOSD”).” *Morgan v. Sec’y of Health & Human Servs.*, No. 15-1137V, 2019 WL 7498665, at *1 (Fed. Cl. Spec. Mstr. Dec. 4, 2019). Petitioner contends this decision was arbitrary and capricious because it ignored factual evidence in the record, particularly portions of the expert reports and testimony, as well as medical literature. For the following reasons, the Court **DENIES** petitioner’s motion and **SUSTAINS**

* This opinion was initially filed under seal pursuant to Vaccine Rule 18(b) of the Rules of the Court of Federal Claims. The Court provided the parties 14 days to submit proposed redactions, if any, before the opinion was released for publication. Neither party proposed redactions. This opinion is now reissued for publication in its original form.

the decision of the Chief Special Master. Additionally, the Court **GRANTS** petitioner's motion for leave to exceed the page limit.¹

I. Background

A brief recitation of the facts provides necessary context.²

A. Petitioner's Medical History and the Vaccination

Petitioner, who was 54 at the time of his October 2012 flu vaccination, suffered preexisting conditions, including: "lower back pain, lower extremity radiculopathy, multi-level degenerative disc disease, lumbar spondylosis, and prostatitis." *Id.* at *1, *3. Beginning in August 2009, petitioner was under the care of Physician Assistant Deborah Stayman ("PA Stayman") and Dr. Anthony Wilson, M.D. of Orthopaedic Associates of Muskegon for lower back pain. *Id.* Petitioner "complained of pain radiating to his left thigh," and PA Stayman noted petitioner "exhibited decreased reflexes in his left achilles tendon." *Id.* A Magnetic Resonance Imaging ("MRI") study conducted on 1 September 2009 showed "mild foraminal narrowing at the L3–L4 and L4–L5 levels . . . with moderate foraminal narrowing bilaterally at L5–S1 level. No significant spinal canal narrowing. There are disc bulges involving the lower two lumbar levels." *Id.* (quoting Pet'r's Ex. 2, at 1; Pet'r's Ex. 4, at 32–33). Petitioner was referred to physical therapy for his pain but complained the physical therapy "was not assisting." *Morgan*, 2019 WL 7498665, at *2. An electromyography test ("EMG") and nerve conduction study, both conducted on 24 November 2009, returned normal results. *Id.* Throughout 2009 and 2010, petitioner received spinal nerve injections. *Id.*

In January 2011, petitioner began to visit Shoreline Family Medicine, complaining of "muscle stiffness, decreased range of motion, weakness, and radiating lower back pain." *Id.* At that time, "he was diagnosed with chronic lower back pain and degenerative disc disease." *Id.* Thereafter, he was seen monthly and "consistently complained of persistent pain, stiffness, weakness, and radiating lower back pain, though not every symptom was present at every visit." *Id.* In May 2011, he began to complain of dizziness and neck pain. *Morgan*, 2019 WL 7498665, at *2. On 30 June 2011, petitioner underwent another MRI, which showed "[s]pondylosis causing some mild to moderate spinal canal stenosis at C5-6 and C6-7. No frank herniated disc is appreciated." *Id.* (quoting Pet'r's Ex. 5, at 92).

On 11 March 2012, petitioner underwent an additional MRI "for continued lower back pain and lower extremity radiculopathy."³ *Id.* The MRI "showed '[m]ulti-level degenerative

¹ Petitioner also filed a motion for leave to exceed the page limit contemporaneously with filing his motion for review. See Pet'r's Mot. for Leave of Court to Exceed the Page Limit, ECF No. 66. Respondent indicated during oral argument he does not oppose the motion. See Tr. at 5:8–10, ECF No. 73.

² As the basic facts in this case have not changed significantly since the Special Master's 4 December 2019 decision in this case, the Court's recitation of the background facts herein draws from that decision.

³ Radiculopathy is a "[d]isorder of the spinal nerve roots." *Radiculopathy*, Stedmans Medical Dictionary (Westlaw, last updated Nov. 2014). "Radiculopathy describes a range of symptoms produced by the pinching of a nerve root in the spinal column. The pinched nerve can occur at different areas along the spine." *Radiculopathy*, John's Hopkins Medicine, <https://www.hopkinsmedicine.org/health/conditions-and-diseases/radiculopathy> (last visited May 21, 2020).

disc disease and lumbar spondylosis with slight interval progression and worsening in the appearance of degenerative change at the L4-5 level.” *Id.* (quoting Pet’r’s Ex. 8, at 193).

On 6 August 2012, petitioner was diagnosed with prostatitis⁴ after being seen for his “trouble urinating and related concerns.” *Id.* (citing Pet’r’s Ex. 5, at 145). A month later, he was also diagnosed with “lower back pain and bilateral sciatica” during a follow-up visit when he “complained of stiffness and lower back pain in addition to citing the urological symptoms of frequency and oliguria.” *Id.* On 24 September 2012, petitioner was seen for “toe and thigh numbness with an onset of three weeks prior, as well as difficulty initiating urination and waking up during the night to urinate.” *Morgan*, 2019 WL 7498665, at *2. Petitioner “underwent an ultrasound of his prostate,” which returned negative results. *Id.*

On 9 October 2012, petitioner was seen again at Shoreline Family Medicine for “lower back and pelvic pain, weakness, poor balance, fatigue, and sleep disturbances.” *Id.* Petitioner underwent a CT scan on 12 October 2012, but “the results were unremarkable.” *Id.*

On 16 October 2012, petitioner received the flu vaccination at issue in this case. *Id.* at 3. The next day, he was seen for a urologic consultation with Dr. Arthur Golin, M.D. *Id.* Petitioner explained to the physician his urinary symptoms began a year prior but had worsened over the previous two and a half months. *Morgan*, 2019 WL 7498665, at *3. He also noted “increasing pain and some weakness in the right lower extremity . . . numbness, right lateral thigh.” *Id.* Dr. Golin found an “enlarged, benign-appearing [prostate] gland’ and reduced tone of the anal sphincter” during a physical examination and determined petitioner suffered “urinary retention—but with a possible neurologic component.” *Id.* (quoting Pet’r’s Ex. 22, at 7). On 22 October 2012, petitioner was seen for persistent symptoms at Shoreline Family Medicine and “was prescribed medication for his prostatitis and instructed to return in one week for a follow-up appointment.” *Id.* The next day, petitioner “was seen by Scott Greenwald, M.D. at Michigan Pain Consultants for his lower back pain.” *Id.* He complained of lower back pain with pain “radiating down both of his legs,” as well as “new numbness in bilateral calves.” *Id.* Petitioner “was treated with a lumbar epidural steroid injection.” *Morgan*, 2019 WL 7498665, at *3.

On 24 October 2012, petitioner went to the emergency room complaining of urinary retention and “again reported that he had been experiencing urinary incontinence issues for about a year.” *Id.* A catheter was placed. *Id.* “Later that same day, however, [petitioner] gradually lost the strength in his legs until he was too weak to ambulate.” *Id.*

Petitioner was taken by ambulance to the emergency room of a different hospital, where he was seen “for leg weakness and urinary retention.” *Id.* Petitioner underwent an MRI, which showed “[m]ild lumbar disc degeneration, which does not appear significantly changed as compared to 3/11/2012 . . . conus medullaris appears somewhat indistinct with a suggestion of some increased T2-weighted signal intensity, of uncertain significance given the limitations of the low field strength magnet.” *Id.*

⁴ Prostatitis is “[i]nflammation of the prostate.” *Prostatitis*, Stedmans Medical Dictionary (Westlaw, last updated Nov. 2014).

Petitioner was thereafter transferred to a different hospital. *Morgan*, 2019 WL 7498665, at *3. He reported “that he had first noticed mild weakness in his lower extremities in August 2012 (two months before the vaccination in question)” and “patchy numbness and tingling he experienced progressively increased over the previous two months and coincided with his worsening urologic symptoms.” *Id.* Dr. Christopher Marquart, M.D., the treating physician, noted, after physical examination, petitioner had “crude sensory level at about T12-L1 level” and “‘patchy decreased pinprick and light touch over the anterior thighs bilaterally, top of the right foot and bottom both the left heel and patchy over the area of the cath[eter],’ decreased strength, and absent reflexes in his lower extremities.” *Id.* at *4 (quoting Pet’r’s Ex. 9, at 898). Dr. Marquart “observe[d] evidence of nerve root clumping and enhancing in the conus—leading him to question whether [petitioner] had experienced transverse myelitis (“TM”) or some other acute, neuro-inflammatory process.” *Id.* Petitioner “was admitted to the intensive care unit for observation, ‘to make certain he does not have any type of ascending paralysis with the recent flu vaccination.’” *Id.* (quoting Pet’r’s Ex. 9, at 899).

On 25 October 2012, petitioner underwent another MRI, which showed “edema within the cord from T8 to the inferior tip of the cord . . . but no significant contrast enhancement.” *Id.* An infectious disease consultant, Dr. Roni Devlin, M.D. saw petitioner the same day and “indicated that the MRI was suggestive of myelitis of indeterminate etiology—though he did later implicate the flu vaccine, noting that ‘[c]ase reports of myelitis following vaccination have certainly been reported, but rarely.’” *Id.* (quoting Pet’r’s Ex. 9, at 714).

On 27 October 2012, while still at the same hospital, petitioner was seen by Dr. Larry Wahl, D.O. at Mercy Health “who noted that [petitioner] had experienced ‘increasing urinary retention and some difficulty with strength in his lower extremities, climbing stairs as much as 5-1/2 weeks ago that gradually increased’ and ‘seemed to reach a critical level 1 day after having an epidural steroid injection on Tuesday [October 23, 2012].’” *Morgan*, 2019 WL 7498665, at *4 (quoting Pet’r’s Ex. 9, at 715). Dr. Wahl’s differential diagnosis included TM, but the physician “express[ed] some skepticism towards TM as explanatory given the extensive nature of [petitioner’s] spinal cord edema.” *Id.* While in the hospital, petitioner “gradually recovered the ability to stand, bear weight, and walk short distances with the assistance of a walker, but he continued to experience numbness and tingling in his lower extremities.” *Id.*

On 29 October 2012, petitioner was discharged from the hospital to outpatient rehabilitation. *Id.* During a follow-up visit on 15 November 2012, Dr. Marquart “reiterated his belief that Petitioner’s myelitis was ‘probably a reaction to his flu vaccine for lack of a better explanation.’” *Id.* (quoting Pet’r’s Ex. 10, at 1). Another MRI conducted on 21 November 2012 “showed marked improvement in the appearance of the spinal cord with only ‘very mild patchy cord edema.’” *Id.* (quoting Pet’r’s Ex. 5, at 79).

On 13 December 2012, petitioner was seen by Dr. Douglas Gelb, M.D. at the University of Michigan Neurology Clinic. *Morgan*, 2019 WL 7498665, at *4. Petitioner noted during this visit, “since his hospital discharge on October 29, 2012, he had not noticed much improvement in his ability to ambulate and felt as though his neurologic symptoms were worsening.” *Id.* At this time, he “complained of persistent loss of sensation in his lower extremities, bladder, and [bowels], burning pains, the development of a lump on his neck, worsening vision, sudden arm

jerks, and cramping or spasms in his fingers.” *Id.* During a physical examination, petitioner “exhibited mild spasticity in both lower extremities, reduced sensation from the waist down, and absent reflexes in his ankles.” *Id.* at *5. Dr. Gelb “proposed that [petitioner] was experiencing either an isolated episode of TM or the first instance of a recurrent, central nervous system (“CNS”) demyelinating disease, such as Multiple Sclerosis (“MS”) or Neuromyelitis Optica (“NMO”).” *Id.* Dr. Gelb “acknowledged that [petitioner’s] pre-vaccination symptoms ‘raise[d] some concern that he might have had an ongoing disease process in his nervous system that “flared up” on Oct. 24,’ but noted that those earlier symptoms were non-specific, or could be explained by [petitioner’s] degenerative disc disease and enlarged prostate.” *Id.* (quoting Pet’r’s Ex. 14, at 10–11). Dr. Gelb determined petitioner’s neurologic disease was not progressing, and “attribut[ed] his change in vision to . . . one of the medications [petitioner] was taking.” *Morgan*, 2019 WL 7498665, at *5 (citing Pet’r’s Ex. 14, at 10–11). Dr. Gelb subsequently directed petitioner to “taper off” the medication “and suggested a follow-up MRI as well as a serum NMO antibodies test.” *Id.*

On 20 December 2012, petitioner had a follow-up visit with Dr. Wahl. *Id.* Petitioner “described continuing improvement of his neurologic symptoms, and he demonstrated almost full strength throughout his lower extremities during his physical evaluation.” *Id.* Dr. Wahl “ordered laboratory testing—including an NMO serum antibodies test and a brain MRI.” *Id.* Between this visit and 14 February 2013, petitioner experienced some improvement in his neurologic symptoms. *Id.* Both his NMO serum antibodies test and a 7 January 2013 brain MRI returned negative results, although the NMO serum antibodies test results summary noted, “seronegativity does not necessarily preclude a diagnosis of [NMO].” *Morgan*, 2019 WL 7498665, at *5 (quoting Pet’r’s Ex. 5, at 56).

On 29 April 2013, petitioner returned to Dr. Wahl, where “he exhibited decreased strength in both legs.” *Id.* Another MRI was performed on 20 May 2013, which showed, “[i]nterval change in the appearance of the thoracic spinal cord which demonstrates diffuse but mild expansion and increased intermedullary signal centrally between the T6 level and the conus,” which appearance was “suggestive of [TM].” *Id.* (quoting Pet’r’s Ex. 5, at 70).

Petitioner’s condition continued to deteriorate. *Id.* “[B]y June 17, 2013, he was unable to stand independently and exhibited increasingly diminished strength in his bilateral lower extremities.” *Id.* On 23 July 2013, petitioner, “concern[ed] that he was experiencing a relapse of his symptoms,” was seen by a neurologist, Dr. Ivan Landon, M.D. *Id.* “Dr. Landon concluded that [petitioner] had suffered at least one, maybe two, relapses and that he was likely suffering from a polyphasic TM.” *Morgan*, 2019 WL 7498665, at *5.

On 31 July 2013, petitioner “was admitted to . . . inpatient rehabilitation” for nine days, “during which time he was treated with high dose steroids, [intravenous immunoglobulin (“IVIG”)], and intensive physical therapy.” *Id.* at *6. Petitioner “saw some improvement with these treatments.” *Id.* Over the next year, petitioner was continually seen by Dr. Landon, who “noted that [petitioner’s] condition appeared to be deteriorating, as he continued to experience recurrent symptoms relapses.” *Id.* By 17 June 2014, petitioner became “restricted to a wheelchair and complained of symptoms in his upper extremities.” *Id.* Dr. Landon observed

petitioner “seemed to respond best to IVIG coupled with steroids, but [petitioner’s] insurance company was no longer covering the cost of the IVIG treatment.” *Id.*

On 15 August 2014, petitioner returned to the University of Michigan Neurology Clinic, where he “reported that he had developed numbness in his trunk that ascended from his waist to his mid-back, numbness in the tips of his fingers, and blurry spots of vision within the past few months.” *Morgan*, 2019 WL 7498665, at *6. Dr. Gelb was uncertain “whether [petitioner’s] clinical deterioration was due to [a] new episode of spinal cord inflammation, or simply some systemic illness exacerbating his deficits from his initial episode (although new episodes of inflammation seem more likely, given the severity and persistence of the new deficits, and given the higher sensory level).” *Id.* (quoting Pet’r’s Ex. 14, at 95).

Dr. Gelb therefore referred petitioner to a MS clinic and ordered “repeat MRIs of [petitioner’s] cervical and thoracic spine and brain as well as a repeat serum NMO antibodies test.” *Id.* The NMO antibodies test returned negative results, but the MRI of his thoracic MRI showed:

[V]olume retraction/myelomalacia, seen caudal to T8 level and extending down to the conus, is non masslike abnormal enhancement predominantly involving central and posterior portions of the spinal cord, which is more conspicuous at T12 and T10-T11 levels . . . The spinal cord volume loss likely represent[s] myelomalacia as the sequela of previous inflammatory process. Areas of T2 signal change and abnormal enhancement could represent reactivation of inflammatory process, this possibility should be correlated with deficits on physical exam and paraclinical test/parameters.

Id. (quoting Pet’r’s Ex. 14, at 135, 137). Petitioner’s brain MRI also showed “nonspecific small areas of nonenhancing T2 signal prolongation in predominantly left supratentorial white matter, these findings may represent sequela from previous inflammatory, infectious or small vessel white matter ischemic process.” *Id.* (quoting Pet’r’s Ex. 14, at 137).

On 26 November 2014, petitioner was seen by Dr. Robert Pace, M.D. at the University of Michigan MS Clinic. *Id.* Dr. Pace reviewed the August 2014 MRI scans and noted:

[S]everal nonspecific T2/FLAIR hyperintensities seen in the brain. These are not in a pattern that is strongly suggestive of demyelination such as would be seen with [MS]. However, there is a T2 hyperintensity in the fourth ventricle surrounding the cerebral aqueduct. This is of unclear significance, but can be seen in [NMO] spectrum.

Morgan, 2019 WL 7498665, at *6 (quoting Pet’r’s Ex. 14, at 110). Dr. Pace also observed “patchy enhancement of the lower thoracic spine/conus medullaris that appears to involve some of the cauda equina.” *Id.*

Based on his review of the August 2014 MRIs and laboratory testing, Dr. Pace formally diagnosed petitioner with “longitudinal myelitis due to [NMO], sero-negative.” *Id.* at *7

(quoting Pet'r's Ex. 14, at 110). Dr. Pace "advised [petitioner] to begin immune modulation therapy" given the "high likelihood that [petitioner's] condition would cause 'recurrent and potentially devastating episodes of myelitis if untreated.'" *Id.* (quoting Pet'r's Ex. 14, at 110). Although petitioner "had experienced significant improvement with IVIG treatment in the past," Dr. Pace "opined that the most effective treatment for patients with NMO is Rituximab." *Id.*

Petitioner did not pursue either recommended treatment, but "he pursued physical therapy from July to September 2015." *Id.* On 18 August 2015, petitioner returned to Dr. Pace, who listed his diagnoses as "relapsing-remitting MS, Devic's disease, and flaccid paralysis of the lower extremities."⁵ *Morgan*, 2019 WL 7498665, at *7. Petitioner "reported persistent paralysis in his lower extremities and numbness from the midthoracic region down," but noted he felt "he was cognitively doing better than before." *Id.* Dr. Pace again ordered "MRIs and hepatitis serologies." *Id.* The MRIs "showed that the nonspecific signal hyperintensities located in the periventricular area of the brain were stable since January." *Id.* There were no reported abnormalities of the cervical spine, "and the previously documented areas of abnormal signal in the thoracic region of the spinal cord had resolved." *Id.*

On 20 April 2016, petitioner again returned to Dr. Pace, and reported he "was seeing improvement with physical therapy." *Id.* Although he was still wheelchair-bound, "he had not developed any new or worsening symptoms." *Morgan*, 2019 WL 7498665, at *7. "Dr. Pace again opined that [petitioner's] diagnosis was 'most likely seronegative [NMO].'" *Id.* (quoting Pet'r's Ex. 53, at 42).

Petitioner had a follow-up appointment with Dr. Pace in April 2017 with similar reports that he continued to improve with physical therapy and did not experience new or worsening symptoms. *Id.* "A physical exam revealed that [petitioner] was able to activate his hip flexors and extensors, . . . actions he was incapable of performing the year prior." *Id.* Dr. Pace also "noted that the changes in [petitioner's] spine were stable and that there was no evidence of new or enhancing lesions." *Id.* Dr. Pace included the following diagnoses after the appointment: "NMO, acute TM, paralytic syndrome, and spinal stenosis of the cervical region." *Id.*

B. The Petition and Hearing Before the Special Master

Petitioner filed his vaccine petition against the Secretary of Health and Human Services ("respondent") on 7 October 2015. *See* Pet., ECF No. 1. Petitioner requested compensation for the transverse myelitis he allegedly developed after receipt of a flu vaccination on 16 October 2012. *See id.* at 1.

1. Expert Reports

Petitioner filed his first expert report authored by Dr. Carlo Tornatore, M.D. on 27 October 2016. *See* Pet'r's Ex. 24, ECF No. 18-1. Dr. Tornatore is Professor and Chairman of the Department of Neurology at the Georgetown University Medical Center. *Id.* at 7. He is also

⁵ The Special Master's decision noted that "NMO is sometimes referred to as Devic's disease." *Morgan*, 2019 WL 7498665, at *7 n.12.

the Chairman, Neurologist-in-Chief, and Executive Director of the Multiple Sclerosis Patient Centered Specialty Home at Medstar Georgetown University Hospital. *Id.* Upon reviewing petitioner's medical history, Dr. Tornatore asserted:

I agree with the treating physicians that [petitioner] suffered a profound demyelinating event within a week of receiving an influenza vaccination. This event was characterized by symptoms of lower extremity weakness and bladder dysfunction that were subsequently diagnosed as longitudinally extensive transverse myelitis. Notably[,] the spinal cord was edematous at multiple levels in October 2012, consistent with an acute event.

Id. at 2. Dr. Tornatore explained the physiology of his preferred diagnosis:

Transverse Myelitis (TM) is a rare clinical syndrome in which an immune-mediated process causes neural injury to the spinal cord, resulting in varying degrees of weakness, sensory alterations[,] and autonomic dysfunction. The term *myelitis* refers to inflammation of the spinal cord; *transverse* describes the position of the inflammation, across the width of the spinal cord. In TM, inflammation damages or destroys myelin, the fatty insulating substance that covers nerve cell fibers, causing scars that interrupt communications between the nerves and the rest of the body.

Id. at 2–3. Dr. Tornatore stated “[t]he immunopathogenesis of TM is not fully understood,” but “[i]t is thought that a variety of immune stimuli, through such processes as molecular mimicry or superantigen-mediated immune activation, may trigger the immune system to injure the nervous system.” *Id.* at 3. Based on this background, Dr. Tornatore cited to various medical journal articles, which state “TM has been reported following vaccinations,” including the influenza vaccination. *Id.* Quoting another medical journal article, Dr. Tornatore highlighted that “it is widely reported in neurology texts that [acute TM] is a post-vaccination event.” *Id.* at 3–4. Dr. Tornatore further emphasized that “the Johns Hopkins Transverse Myelitis Center’s model diagnostic approach for evaluating patients with acute myelopathies[] includes determining whether there is a history of recent vaccination or systemic illness.” *Id.* at 4. A neuroimmunology textbook, in addition to a study of MRIs following flu vaccines, also show an association between the flu vaccine and acute TM.⁶ *Id.* at 5–6. Based on petitioner’s medical history and the various cited medical journal articles, Dr. Tornatore concluded:

[I]t is my opinion, to a reasonable degree of medical probability, that the influenza vaccine [petitioner] received on 10/16/2012 resulted in transverse myelitis, within a week of vaccination. . . . [T]here was no evidence in [his] medical record for any alternate cause for his condition and the temporal relationship between the vaccination and the onset of [his] symptoms was in an appropriate time frame . . .

⁶ The authors of the Bakshi et al. article Dr. Tornatore cites in his report concluded, however, “association of TM following the influenza vaccination does not *prove* cause and effect, however, because no other known causes of [acute TM] were identified, [and] a postvaccination syndrome was diagnosed by exclusion. Pet’r’s Ex. 24, at 6.

Lastly, [his] treating physician also speculated that the vaccination could have been the etiology of the transverse myelitis.

Id. at 6–7.

On 23 June 2017, respondent filed an expert report authored by Dr. Subramaniam Sriram. *See* Resp’t’s Ex. A, ECF No. 33-1. Dr. Sriram is “a Professor of Neurology and Microbiology, Immunology and head of the Multiple Sclerosis (MS) Clinic at Vanderbilt Medical Center.” *Id.* at 1. He cares for “over 1,000 patients with MS and allied neuro-inflammatory disorders including NMO,” in addition to performing “research on the causes and treatment of MS.” *Id.* After detailing petitioner’s medical history, Dr. Sriram opined “the final diagnosis in [petitioner] is not transverse myelitis, which is attributed to monophasic disease; rather[,] his diagnosis is relapsing longitudinal myelitis, which suggests that the condition that [petitioner] had was a recurring relapsing disease of the spinal cord.” *Id.* at 5.

Dr. Sriram “agree[d] with the final assessment of Dr. Pace from the University of Michigan that the most likely diagnosis is longitudinal extensive myelitis seronegative Neuromyelitis Optica.” *Id.* Dr. Sriram laid out the diagnostic criteria for Seronegative NMO as follows:

1. At least 2 core clinical characteristics occurring as a result of one or more clinical attacks and meeting all of the following requirements:
 - a. At least 1 core clinical characteristic must be optic neuritis, acute myelitis with LETM, or area post-rema syndrome.
 - b. Dissemination in space (2 or more different core clinical characteristics).
 - c. Fulfillment of additional MRI requirements, as applicable.
2. Negative tests for AQP4-IgG using best available detection method, or testing unavailable
3. Exclusion of alternative diagnoses.

Id. at 6. Dr. Sriram determined petitioner “most likely satisfies the criteria for seronegative neuromyelitis optica,” satisfying subsection 1(a), but expressed uncertainty “as to the definitiveness of the diagnoses . . . because [he has] not been able to look at the MRI scans to ensure that he has had dissemination in space (Item 1b above).” *Id.* Dr. Sriram suggested “anti-Myelin Oligodendrocyte Protein (MOG) antibody mediated longitudinal transverse extensive myelitis” as an alternative diagnosis, but “[a]t present, the serological tests for the MOG antibody are not available, and hence will be diagnosis of exclusion.” *Id.* Dr. Sriram further emphasized “[n]one of the neurologists [who treated petitioner] suggested that the vaccination played a role in his disease,” and petitioner’s neurologic symptoms predated his vaccination. *Id.*

Dr. Sriram also opined, contrary to Dr. Tornatore’s report, that “[t]he current literature does not support a causal connection between influenza vaccine and a relapsing myelitis of any cause.” *Id.* Dr. Sriram distinguished two of the case reports Dr. Tornatore cited because neither of the patients studied in each report suffered “isolated cases of Transverse Myelitis.” *Id.* at 6–7. Dr. Sriram distinguished the other case study cited for the association between flu vaccination

and TM because “the clinical findings [in that study] are very suggestive of Neuromyelitis Spectrum disorder and not an isolated TM,” also noting that report “preceded the advent of our current understanding of NMO.” *Id.* at 7. Dr. Sriram further responded to Dr. Tornatore’s report, stating he did “not provide a causal connection between TM and the influenza vaccine, and he provides no supporting evidence to show that the influenza vaccine can cause TM.” *Id.* Regarding causation, Dr. Sriram contended, “[t]he prevailing opinion on the neurological condition of [petitioner] is that he has Neuromyelitis Optica Spectrum disorder (NMOSD), a condition caused by an auto- antibody response to Aquaporin IV, a protein in brain cells. There is no evidence that there is any cross reactivity between Influenza vaccine and Aquaporin IV protein.” *Id.* Dr. Sriram therefore concluded: “It is my opinion that [petitioner] most likely had NMOSD. In addition, [petitioner’s] receipt of the influenza vaccine on 10/16/2012 did not cause or contribute to the development or the subsequent course of the disease. I hold these opinions to a reasonable degree of medical probability.” *Id.*

Petitioner filed a responsive expert report also authored by Dr. Tornatore on 5 October 2017. *See* Pet’r’s Ex. 47, ECF No. 36-1. In this report, Dr. Tornatore focused his analysis on “whether [petitioner’s] disc disease could have been the etiology of his myelitis.” *Id.* at 1. He answered this question in the negative because the disc disease petitioner suffered was in the lumbar region, lower on the spine than the level at which he suffered myelitis. *Id.* at 1–2. According to the 1 September 2009 MRI, petitioner had “mild foraminal narrowing at the L3-L4 and L4-L5 levels with moderate foraminal narrowing bilaterally at the L5-S1 level,” and there were “disc bulges involving the lower two lumbar levels.” *Id.* at 2. The 25 October 2012 MRI, on the other hand, identified “evidence of edema within the cord from T8 to the inferior tip of the cord,” with the spinal cord ending at the L1 level. *Id.* Therefore, “from an anatomical standpoint, the myelitis cannot be attributed to lumbar disc disease.” *Id.* Dr. Tornatore also opined petitioner’s “symptoms of weakness, bladder changes[,] and sensory symptoms are due to the myelitis and not lumbar disc disease” based on the discharge summary from petitioner’s 25 October 2012 hospital admission because “[t]he abrupt change in neurologic symptoms, in the absence of lumbar disc disease that encroached upon the spinal canal, clearly speaks to the myelitis as the etiology of the acute symptoms and subsequent disability.” *Id.* at 3.

On 17 October 2017, the Chief Special Master ordered respondent to file a supplemental expert report “addressing a) the distinction made by Petitioner’s Expert regarding Petitioner’s preexisting pain, b) the recently filed MRIs and c) stating if there is a meaningful difference between the injuries claimed by the experts for the purposes of causation.” Respondent filed this supplemental expert report, also authored by Dr. Sriram, on 22 December 2017, responding to the Chief Special Master’s inquiries. Resp’t’s Ex. F at 1, ECF No. 38-1. Given petitioner’s “long history of low back pain,” Dr. Sriram agreed with Dr. Tornatore’s assessment that “the back pain was from degenerative lumbo-sacral disc disease and does not have any bearing on the diagnosis of inflammatory thoracic myelopathy.” *Id.* Dr. Sriram summarized the timeline from when petitioner was hospitalized 24 October 2012, his continued neurologic deterioration throughout 2013, and the 30 August 2014 MRI, which “showed abnormal enhancement predominantly involving the central and posterior portions of the spinal cord which is more conspicuous at thoracic 12 and thoracic 10 and thoracic 11 levels.” *Id.* at 2. Based on this summary, Dr. Sriram stated:

The sum of these observations suggests that [petitioner's] symptoms of spinal cord dysfunction:

- Preceded the receipt of the flu vaccination on 10/16/2012;
- Was relapsing remitting in nature;
- At least one clinical relapse and two radiological relapses were observed; and
- Was not the consequence of his back pain or the local steroid therapy.

Id. Therefore, Dr. Sriram asserted:

Any causal connection between [petitioner's] receipt of the flu vaccine and the development of neurological deficits consistent with transverse myelitis is unlikely since the development of gait instability and bladder complaints preceded the receipt of the vaccine. Furthermore, the underlying diagnosis of [petitioner's] condition is relapsing myelitis, and the clinical relapse and the radiological relapse were seen 6-9 months after his initial presentation. Therefore, it is my opinion that there is no causal connection between the vaccine and the neurological problems that followed, given [petitioner's] chronic course. I agree with the physicians who evaluated [petitioner] at the University of Michigan that he has neuromyelitis optica spectrum disorder.

Id.

2. Expert Testimony

The Chief Special Master held a one-day entitlement hearing on 23 January 2019. *See* Order, ECF No. 41. Both parties' experts testified during the hearing.

i. Testimony of Dr. Tornatore

Dr. Tornatore began his testimony recognizing the complexities of petitioner's medical record due to his preexisting symptoms but stated there was "no question" petitioner had LETM. Hr'g Tr. at 9:7–15, ECF No. 57. He asserted that the dramatic change between the slow tempo of petitioner's symptoms before the vaccination and petitioner's condition a week after the vaccination suggested to him the vaccine caused petitioner's LETM. *See id.* at 10:16–11:25.

In contrasting petitioner's post-vaccination condition from his pre-vaccination condition, Dr. Tornatore emphasized the progression of petitioner's symptoms. *See id.* at 11:5–8. Dr. Tornatore opined petitioner's long-standing bladder issues and lower back pain were likely attributable to petitioner's history of prostatitis, bilateral sciatica, and degenerative disc disease. *See id.* at 12:18–23, 26:20–24. He also acknowledged petitioner's neurological exams were "checked off as negative" until the week after the vaccination. *Id.* at 15:25–16:1; *see also id.* at 24:4. Dr. Tornatore further emphasized the importance of the tempo of change in petitioner's symptoms by explaining that petitioner's spinal inflammation was so profound it could not have predated the vaccine because petitioner would have experienced more symptoms than his bladder issues and lower back pain with that degree of inflammation. *See id.* at 29:11–30:2. Dr.

Tornatore similarly urged that the slow progression of petitioner's pre-vaccination symptoms is inconsistent with the "profound inflammatory event" characteristic of either LETM or NMOSD. Hr'g Tr. at 30:3–18.

Dr. Tornatore cited an article authored by Dr. Kerr, petitioner's Exhibit 26, which states "acute transverse myelitis ["ATM"] exists on a continuum of neuroinflammatory disorders," including NMOSD, all of which are "related to an autoimmune response." *Id.* at 35:18–36:15. Dr. Kerr writes that "it is widely reported in neurology text that ATM is a post-vaccination event," and it is important for a physician treating these neuroinflammatory disorders to "determine if there's a recent history of vaccination or systemic illness." *Id.* at 37:7–8, 37:21–38:4. Relying on the Kerr article, Dr. Tornatore invoked the theory of molecular mimicry to explain how the flu vaccination caused petitioner's LETM. *See id.* at 38:18–39:1. According to this theory, bacteria "triggers an immune system and then the immune system attacks the heart and the brain, as well as the joints." *Id.* at 39:20–23.

Dr. Tornatore contended it did not matter whether petitioner had LETM or NMO for purposes of determining whether the vaccination caused the injury. *See id.* at 46:6–18. The significance of diagnosis, Dr. Tornatore maintained, was merely for finding the proper treatment. *See id.* at 46:19–47:9. He further explained the uncertainties with treating and diagnosing patients with neuroinflammatory conditions, but test negative for the Aquaporin-4 antibodies that are typical of NMOSD. Hr'g Tr. at 47:10–49:1. Moreover, Dr. Tornatore suggested petitioner, as a male in his fifties, does not fit the typical demographic for NMOSD. *See id.* at 49:2–16.

Dr. Tornatore opined the "temporal relationship . . . between the antigenic exposure and the onset" of petitioner's spinal inflammation was suggestive of vaccine-induced TM because it made sense "from an immune standpoint" that petitioner's spinal cord inflammation would begin "roughly seven or eight days following the vaccination." *Id.* at 50:5–14.

Lastly, Dr. Tornatore noted TM is not always monophasic, meaning "it can relapse, whether it's from NMO or other things that can cause transverse myelitis," and petitioner's relapse did not affect his assessment of petitioner's condition. *Id.* at 50:18–51:9.

On cross examination, Dr. Tornatore acknowledged petitioner's pre-vaccination symptoms could be symptoms of demyelinating disease, but conditioned that they could also be attributed to other causes. *See id.* at 54:18–55:22, 56:19–57:8. Despite this, Dr. Tornatore reiterated his opinion that "the greater probability is that these were preexisting symptoms that were not referable to the spinal cord" due to the difference in the tempo of petitioner's pre-vaccination symptoms versus his post-vaccination symptoms. *Id.* at 57:20–22, 58:9–16, 59:2–6.

ii. Testimony of Dr. Sriram

Dr. Sriram opined petitioner had a relapsing form of Aquaporin-4 negative NMOSD, also called serologically negative or seronegative NMOSD. *Id.* at 82:18–83:6. He began his testimony by providing an overview of the different types of demyelinating disorders of the central nervous system. *Id.* at 84:5–89:10. Dr. Sriram explained "there is not a typical course" of NMOSD, but it is considered a chronic condition because "[a]bout 60 to 70 percent of patients

will relapse.” *Id.* at 90:2, 90:9–10. He also indicated a physician would reconsider his initial diagnosis of TM if the patient relapsed because TM is monophasic. *Id.* at 90:20–91:4.

Addressing the testing for NMOSD, Dr. Sriram explained there is a blood test used to detect Aquaporin-4 autoantibodies, the autoantibodies which tend to be elevated in NMOSD patients. *Id.* at 91:15–18. Dr. Sriram indicated, though, the test is “not very sensitive,” and estimated “20 to 25 percent [of patients] are seronegative. . . . for a number of reasons.” *Id.* at 91:19–23. Therefore, notwithstanding petitioner’s negative Aquaporin-4 test, it was likely his physicians “missed the window of opportunity where [they were] more likely to have the test come [back] positive.” *Id.* at 92:7–9. Dr. Sriram opined petitioner’s pre-vaccination symptoms “were indicative of NMOSD.” *Id.* at 92:17–19. Reviewing the progression of petitioner’s pre-vaccination symptoms, Dr. Sriram asserted “[t]hey represent an ongoing process that began in the spinal cord sometime in August [2012].” *Id.* at 96:6–7.

Dr. Sriram contended there was no “scientifically reliable evidence to show that flu vaccine can cause TM . . . [o]r NMOSD” or that show the “flu vaccine can worsen an individual’s clinical course if he has TM . . . [o]r NMOSD.” *Id.* at 97:16–20, 98:14–19. Discussing the Kerr article Dr. Tornatore relied on to assert the flu vaccine can cause TM, Dr. Sriram warned “that extreme caution should be exercised in drawing a causal connection” because it was a case report and “case reports must be viewed with caution as it is entirely possible that the two events occurred in close proximity by chance alone.” *Id.* at 99:8–17.

Dr. Sriram also addressed the timeline of the progression of petitioner’s symptoms, contending that an onset of six to eight weeks would be a reasonable progression for petitioner’s demyelinating condition. *Id.* at 100:20–101:24.

Lastly, Dr. Sriram stated he did not believe petitioner’s vaccination caused or worsened his condition. *Id.* at 105:2–4.

On cross examination, Dr. Sriram agreed that in patients who test negative for Aquaporin-4, the diagnostic criteria for NMSOD are more stringent. *Id.* at 107:10–17. According to the seronegative NMOSD diagnostic criteria, individuals must exhibit two or more different core clinical characteristics. *Id.* at 107:18–20. Dr. Sriram agreed petitioner had myelitis, but he did not have optic neuritis or any other core clinical characteristics of seronegative NMOSD. *Id.* at 108:1–4, 9–20. Further, although Dr. Sriram agreed petitioner did not have symptoms associated with the area postrema of the brain, “he had a lesion there.” *Id.* at 108:5–8. Dr. Sriram therefore maintained petitioner has seronegative NMOSD based on petitioner’s myelitis and an extending spinal lesion. *Id.* at 108:21–109:9. While Dr. Sriram acknowledged petitioner’s spinal lesion having extended is not considered dissemination in space for purposes of satisfying a second requisite core clinical characteristic of seronegative NMOSD, he asserted the lesion “[l]iterally . . . disseminated from one region of the spinal cord to an additional region of the spinal cord.” *Id.* at 109:3–15. Admittedly using the diagnostic criteria as “guidelines,” and conceding that petitioner did not have symptoms related to the area postrema of the brain, Dr. Sriram asserted it is “an unusual place to develop a T2 lesion” and “[i]t’s not uncommon to have silent lesions.” *Id.* at 110:10, 111:21–112:20. Dr. Sriram recognized it is uncommon for NMOSD to progress over a course of months or years but

contended petitioner's symptoms progressed over a six-week period, which he considered a reasonable timeframe for NMOSD onset. *Id.* at 127:12–129:20.

Finally, Dr. Sriram asserted there is no human evidence, nor is there epidemiological evidence, that the flu vaccine can cause demyelinating disorders. *Id.* at 138:12–139:12.

C. The Special Master's Decision Denying Compensation

On 4 December 2019, Chief Special Master Corcoran issued his decision denying petitioner's claim and denying compensation because petitioner did not “offer[] preponderant evidence to support the alleged diagnosis of LETM, whereas the record evidence preponderates in favor of an alternative diagnosis: [NMOSD],” and petitioner did not “establish[] a reliable theory explaining how the flu vaccine could have caused his NMOSD.” *Morgan*, 2019 WL 7498665, at *1.

The Chief Special Master began by defining TM and NMOSD because “[s]uch distinctions are critical for purposes of evaluating causation in this case.” *Id.* at *16. The Chief Special Master first “noted that acute demyelinating neurologic conditions like TM are understood to occur rapidly, proceed in a monophasic manner, and often resolve without recurrence.” *Id.* The Chief Special Master contrasted this with “chronic demyelinating conditions affecting the [central nervous system (“CNS”)], like MS, [which] can initially present as if they were TM but will invariably recur.” *Id.* He thus differentiated NMOSD, which “is understood to be a relapsing and chronic CNS disease, like MS,” from “monophasic conditions like TM, even though both involve CNS demyelination.” *Id.* Therefore, “[w]hile a chronic CNS demyelinating disease may *begin* with an occurrence that appears discrete, like TM, the later overall course of disease will establish that the patient did not *only* experience a one-time event.” *Id.* This distinction was important to the Chief Special Master's decision because although “petitioners have on many occasions successfully established that acute forms of CNS demyelinating conditions . . . were likely vaccine-caused[.]. . . claimants have less consistently succeeded in establishing that a vaccine . . . could cause a person to develop a chronic demyelinating condition, like MS or NMOSD.” *Morgan*, 2019 WL 7498665, at *16.

With this background, the Chief Special Master distinguished cases in which compensation was awarded where the petitioner showed a flu vaccination caused NMOSD because the theories offered in those cases were not the same as the medical theory of causation posited in this case. *Id.* For example, in two of the cited cases, “the theories offered in both cases associating the flu vaccine with NMOSD relied on the concept that the components of the flu vaccine first caused direct injury to the endothelial cells in the body, thereby producing a breach in the blood brain barrier, and resulting in further injury via a subsequent antibody attack on the myelin sheath.” *Id.* at *17 (citing *Calise v. Sec'y of Health & Human Servs.*, No. 08-85V, 2011 WL 1230155, at *12–21 (Fed. Cl. Spec. Mstr. Mar. 14, 2011); *Davis v. Sec'y of Health & Human Servs.*, No. 07-451V, 2010 WL 1444056, at *8–9 (Fed. Cl. Spec. Mstr. Mar. 16, 2010), *aff'd*, 94 Fed. Cl. 53). In this case, however, “[p]etitioner simply proposes that molecular mimicry between antigens in the vaccine and self-structures of the CNS caused harm, with less explanation as to how the process occurred.” *Id.*

The Chief Special Master next considered whether petitioner’s pre-vaccination symptoms were related to his neurologic injury. *Id.* Although some of petitioner’s post-vaccination treating physicians considered his pre-vaccination symptoms related to his post-vaccination symptoms, the Chief Special Master determined “the record preponderates *against* the conclusion that Petitioner’s injury, however characterized, predated his receipt of the flu vaccine.” *Id.* (emphasis added). The Chief Special Master based this determination on Dr. Tornatore’s testimony “that there was a difference between the tempo of Petitioner’s long-standing pre-vaccination symptoms and those he experienced thereafter.” *Id.* The Chief Special Master additionally noted, “Dr. Sriram seems to have conceded the low likelihood that an individual with his preferred diagnosis of NMOSD would experience a slow and progressive series of symptoms over the relevant time period at issue.” *Morgan*, 2019 WL 7498665, at *17 (citing Hr’g Tr. at 127).

Next, the Chief Special Master determined, based on his “[c]onsideration of the record as a whole” that the record preponderates in favor of a “seronegative NMOSD diagnosis.” *Id.* at *18. The Chief Special Master cited treating physicians’ views, “especially those from physicians who saw Petitioner later in time” for persuasive evidence that petitioner suffered NMOSD. *Id.* Additionally, the Chief Special Master reasoned “the record upon which treaters based the NMOSD diagnosis preponderantly supports Respondent’s position.” *Id.* For example, “the relapsing and remitting nature of [petitioner’s] disease process, plus the existence of a lesion in the area of the brain most commonly associated with NMOSD” made Dr. Pace’s diagnosis of NMOSD persuasive. *Id.* Considering the diagnostic criteria for seronegative NMOSD, the Chief Special Master indicated despite “the difficulty in establishing those criteria, . . . there was still evidence to fit each criterion—Petitioner initially exhibited acute myelitis with LETM, and demonstrated brain lesions in the area postrema region of the brain.” *Id.* Further, responding to petitioner’s argument that evidence of area postrema syndrome was not supported by the record, the Chief Special Master cited “evidence of dissemination in space, because later MRI reports from September 2015 show a brain lesion in the periventricular region of the brain . . . , as well as a lesion extending to T6, where it had previously extended only to T8 before resolving.” *Morgan*, 2019 WL 7498665, at *18. Moreover, “while Petitioner did not experience some of the *symptoms* that would be associated with an area postrema lesion . . . , his treating physicians nevertheless noted that the mere *existence* of an area postrema lesion supported a diagnosis of NMOSD by itself.”⁷ *Id.*

The Chief Special Master determined “the overall record does *not* preponderate in favor of the TM diagnosis proposed by Petitioner.” *Id.* While at first petitioner’s treating physicians thought petitioner “experienced a one-time, monophasic event,” which would support a TM diagnosis, “over time, Petitioner began experiencing a progressive course of symptoms that suggested a relapse, and certainly resulted in more severe symptoms that impacted his ambulation.” *Id.* Thus, “[t]he overall progressive course of Petitioner’s symptoms from October 2012 to 2016 . . . suggests Petitioner’s initial symptoms were part of something chronic that took

⁷ As discussed *infra*, the record also shows the international consensus diagnostic criteria for seronegative NMOSD merely provides guidelines for treating physicians. See Hr’g Tr. at 110:8–14. Petitioner’s treating physicians, especially the later treating physicians, could interpret the criteria in light of their expertise treating NMO-spectrum diseases.

time to unfold,” which the Chief Special Master deemed more suggestive of NMOSD than TM. *Id.*

Applying the three-pronged test for causation the Federal Circuit articulated in *Althen v. Secretary of Health & Human Services*, 418 F.3d 1274, 1278 (Fed. Cir. 2005), the Chief Special Master decided “[t]his case largely turns on Petitioner’s inability to satisfy the first and second *Althen* prongs.” *Id.* at *19. These prongs require a petitioner to demonstrate: “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury.” *Althen*, 418 F.3d at 1278. Concerning the first prong, the Chief Special Master noted “that Petitioner’s causation theory includes elements that are routinely deemed valid in the Vaccine Program[;] . . . molecular mimicry has repeatedly been embraced in Program cases as a reliable scientific mechanism for explaining the pathophysiology of certain immune-mediated conditions, including many demyelinating disorders.” *Morgan*, 2019 WL 7498665, at *19. Notwithstanding the general recognition of molecular mimicry as a reliable medical theory in vaccine cases, “[f]or molecular mimicry to have utility herein as a reliable mechanism, there should be some evidence that the relevant autoantibodies that are known to drive, or are at least associated with, the resulting demyelinating disease are likely produced as a result of the flu vaccine.” *Id.* The Chief Special Master indicated, “[p]etitioner, however, offered little such evidence.” *Id.* at *20. Petitioner’s medical literature “indicat[ed] that NMOSD initially manifesting as LETM could be caused by a variety of infectious agents,” but not the influenza vaccine, and petitioner’s filings did not “establish how an initial reaction to vaccination might be sufficient to create the kind of chronic, CNS-oriented inflammatory process that would ultimately morph into NMOSD.” *Id.* The Chief Special Master similarly found petitioner failed to satisfy the second prong because “[t]he record does not support the conclusion that the progression of [petitioner’s] symptoms over a period of four or more years could reasonably be attributed to a vaccination received at the outset of that timeframe.” *Id.* Moreover, the Chief Special Master determined it was not “evident from the record that the vaccine, even if it had played some role in his initial presentation, continued to drive a pathologic process over such a lengthy period of time.” *Id.*

Importantly, the Chief Special Master acknowledged “the overall record in this case makes it difficult to establish *with certainty* Petitioner’s correct diagnosis (a task that I am not even called upon to perform, since diagnosing an illness falls well beyond the purview of the special masters in resolving Vaccine Act claims).” *Morgan*, 2019 WL 7498665, at *19.

D. Petitioner’s Motion for Review

On 3 January 2020, petitioner filed his motion for review. *See* Pet’r’s Mot. for Review, ECF No. 65. Petitioner argues “[t]he Chief Special Master abused his discretion, and erred as a matter of law, in ruling that the Petitioner’s injury was [NMOSD].” *Id.* at 13. Petitioner points to the diagnostic criteria for seronegative NMOSD as set out in the article entitled, “International consensus diagnostic criteria for neuromyelitis optica spectrum disorders,” written by Dr. Dean M. Wingerchuk, MD, FRCP(C) et al. (“Wingerchuk”), respondent’s Exhibit E. *Id.* at 14. Wingerchuk provides, in relevant part, a patient who tests seronegative must exhibit:

1. At least 2 core clinical characteristics occurring as a result of one or more clinical attacks and meeting all of the following requirements:
 - a. At least 1 core clinical characteristic must be optic neuritis, acute myelitis with LETM, or area postrema syndrome
 - b. Dissemination in space (2 or more different core clinical characteristics)
 - c. Fulfillment of additional MRI requirements, as applicable
2. Negative tests for AQP4-IgG using best available detection method, or testing unavailable
3. Exclusion of alternative diagnoses

Resp't's Ex. E, at 3, ECF No. 33-5. Wingerchuk also lists the following core clinical characteristics:

1. Optic neuritis
2. Acute myelitis
3. Area postrema syndrome: episode of otherwise unexplained hiccups or nausea and vomiting
4. Acute brainstem syndrome
5. Symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions . . .
6. Symptomatic cerebral syndrome with NMOSD-typical brain lesions

Id. Quoting expert testimony from the hearing, petitioner contends “the testimony of both experts demonstrated that the Petitioner did not meet the diagnostic criteria for seronegative NMOSD” due to what petitioner characterizes as “major concessions during cross examination” of respondent’s expert, Dr. Sriram. Pet’r’s Mot. for Review at 17, 18. While both experts agreed petitioner had acute myelitis, satisfying one of the above diagnostic criteria, petitioner emphasizes Dr. Sriram’s testimony that petitioner’s “thoracic spinal cord lesion (i.e., his myelitis), and the extension thereof over time, did not constitute dissemination in space.” *Id.* at 19 (emphasis and internal quotation marks omitted). Petitioner contends the diagnostic requirement that a patient exhibit “2 or more different core clinical characteristics” means the dissemination in space must “affect[] different neuroanatomic regions”—“[t]hus, by definition, an extensive spinal cord lesion, on its own, is not evidence of dissemination in space.” *Id.* (emphasis omitted). Similarly, “despite Dr. Sriram’s contention that the Petitioner had a brain lesion ‘at the floor of the fourth ventricle,’ he conceded that the Petitioner lacked an ‘[area] postrema syndrome, which is nausea, vomiting[,], or hiccups, which he did not have[.]’” *Id.* at 20 (quoting Hr’g Tr. at 83) (emphasis omitted). Petitioner therefore argues, “[d]espite this testimony and the undisputed literature, the Chief Special Master, citing to Wingerchuk, found that there was ‘still evidence to fit each criterion,’” a finding which petitioner claims had “no basis in the record.” *Id.* at 21 (emphasis omitted).

Petitioner further asserts the Chief Special Master’s reasoning that petitioner “had evidence of a ‘brain lesion in the periventricular region of the brain’ based upon a September 2015 brain MRI” was contrary to the record because area postrema syndrome “requires associated dorsal medulla/area postrema lesions,” which is a smaller region of the CNS than the

periventricular region.⁸ *Id.* at 24 (emphasis omitted). Petitioner maintains this finding was accordingly contrary to portions of the record, such as Dr. Pace’s notes reviewing petitioner’s brain MRIs. *Id.* at 25–26. Petitioner points to Dr. Tornatore’s testimony to show the Chief Special Master’s “discussion that TM is a strictly ‘monophasic condition’ whereas MS and NMOSD are ‘relapsing and chronic CNS’ diseases” was likewise unsupported by the record. *Id.* at 26. Furthermore, petitioner argues “[t]he Chief Special Master’s overreliance on Dr. Pace’s diagnosis is misplaced” because Dr. Pace “did not always adhere” to his diagnosis that petitioner suffered “longitudinal myelitis due to [NMO], seronegative.” *Id.* at 28 (emphasis omitted).

Finally, petitioner asserts “[b]y ignoring expert agreement that the diagnostic criteria for seronegative NMOSD had not been met, and relying on his own broader criteria, for which there was no basis in the record, the Chief Special Master substituted his own judgment for that of the medical community.” *Id.* at 29. Petitioner claims the Chief Special Master’s decision constituted legal error warranting reversal. *Id.*

On 3 February 2020, respondent filed its response to petitioner’s motion for review. *See* Resp’t’s Resp. to Pet’r’s Mot. for Review, ECF No. 69. Respondent argues “[p]etitioner makes only one objection that the Chief Special Master erred in concluding that petitioner’s injury was NMOSD, an argument that amounts to nothing more than a request that this Court impermissibly reweigh the evidence regarding the nature of his condition.” *Id.* at 12. Respondent asserts, “[c]onsistent with the Chief Special Master’s duty to determine which injury petitioner suffered from, but ‘not through the lens of the laboratorian,’ he admitted that not all of the evidence on diagnosis was one-sided.” *Id.* at 14 (quoting *Morgan*, 2019 WL 7498665, at *12). Therefore, “it is unsurprising that petitioner can point to evidence in the record that cuts against the Chief Special Master’s ultimate determination that petitioner suffered from NMOSD, but those arguments fall far short of demonstrating that the Chief Special Master’s conclusion was based on evidence that is ‘wholly implausible.’” *Id.* Pointing to Dr. Sriram’s testimony and medical records from petitioner’s visits with Dr. Pace, respondent maintains “even though petitioner disagrees with the Chief Special Master’s weighing of the evidence, the record provides—at minimum—a basis for his determination that petitioner suffered from NMOSD that is not ‘wholly implausible’ and must be affirmed.” *Id.* at 16. Additionally, respondent argues “[e]ven if petitioner did not have NMOSD, he has not established by preponderant evidence that he had TM, much less that his TM was caused by an influenza vaccine.” *Id.* at 17. Respondent suggests that “petitioner appears to assume that the nature of his condition is an either/or proposition: if it is not NMOSD, it must be TM. But the record does not support this assumption.” *Id.* Lastly, respondent contends “regardless of the diagnosis for petitioner’s alleged injury, his claim would have failed because he could not have satisfied either *Althen* prong one or prong two based on the evidence provided.” *Id.* at 19.

The Court held oral argument on 20 May 2020. Order, ECF No. 71. Petitioner’s motion for review is now ripe for decision.

⁸ Petitioner explains the periventricular region “refers to the entire region near or around any of the cerebral ventricles, rather than the fourth ventricle specifically. . . . [T]he fourth ventricle is the only ventricle that houses the area postrema.” Pet’r’s Mot. for Review at 24, n.10.

II. Discussion

A. Legal Standards

1. The Court's Standard of Review of a Special Master's Decision

The Vaccine Act provides this Court jurisdiction to review a Special Master's decision upon timely motion of either party. *See* 42 U.S.C. § 300aa-12(e)(1)–(2). In reviewing the record of the proceedings before the Special Master, the Court may: (1) “uphold the findings of fact and conclusions of law of the special master and sustain the special master's decision;” (2) “set aside any findings of fact or conclusion of law of the special master found to be arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law and issue its own findings of fact and conclusions of law;” or (3) “remand the petition to the special master for further action in accordance with the court's direction.” *Id.* § 300aa-12(e)(2). “Fact findings are reviewed . . . under the arbitrary and capricious standard; legal questions under the ‘not in accordance with law’ standard; and discretionary rulings under the abuse of discretion standard.” *Saunders v. Sec'y of Dept. of Health & Human Servs.*, 25 F.3d 1031, 1033 (Fed. Cir. 1994) (quoting *Munn v. Sec'y of Dept. of Health & Human Servs.*, 970 F.2d 863, 870 n.10 (Fed. Cir. 1992)).

It is not the Court's role “to reweigh the factual evidence, or to assess whether the special master correctly evaluated the evidence.” *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1360 (Fed. Cir. 2000) (quoting *Munn*, 970 F.2d at 871). The Court also does “not examine the probative value of the evidence or the credibility of the witnesses. These are all matters within the purview of the fact finder.” *Id.* (quoting *Munn*, 970 F.2d at 871). “Reversal is appropriate only when the special master's decision is arbitrary, capricious, an abuse of discretion, or not in accordance with the law.” *Snyder ex rel. Snyder v. Sec'y of Dept. of Health & Human Servs.*, 88 Fed. Cl. 706, 718 (2009). The arbitrary and capricious standard “is a highly deferential standard of review.” “[i]f the special master has considered the relevant evidence of record, drawn plausible inferences and articulated a rational basis for the decision, reversible error will be extremely difficult to demonstrate.” *Hines ex rel. Sevier v. Sec'y of Dept. of Health & Human Servs.*, 940 F.2d 1518, 1528 (Fed. Cir. 1991).

2. The Standard of Causation in Vaccine Cases

“A petitioner seeking compensation under the Vaccine Act must prove by a preponderance of the evidence that the injury or death at issue was caused by a vaccine.” *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1341 (Fed. Cir. 2010) (citing 42 U.S.C. §§ 300aa-11(c)(1), -13(a)(1)). “A petitioner can show causation under the Vaccine Act in one of two ways:” (1) “by showing that she sustained an injury in association with a vaccine listed in the Vaccine Injury Table,” in which case “causation is presumed;” or (2) “if the complained-of injury is not listed in the Vaccine Injury Table . . . the petitioner may seek compensation by proving causation in fact.” *Id.* at 1341–42 (internal citations omitted). Vaccine cases employ a burden shifting standard: “[o]nce the petitioner has demonstrated causation, she is entitled to compensation unless the government can show by a preponderance of the evidence

that the injury is due to factors unrelated to the vaccine.” *Id.* at 1342 (citing *Doe v. Sec’y of Health & Human Servs.*, 601 F.3d 1349, 1351 (Fed. Cir. 2010); 42 U.S.C. § 300aa-13(a)(1)(B)).

“When a petitioner has suffered an off-Table injury, as is the case here, [the Federal Circuit] has established the following test for showing causation in fact under the Vaccine Act:”

[The petitioner’s] burden is to show by preponderant evidence that the vaccination brought about 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.

Broekelschen, 618 F.3d at 1345 (quoting *Althen*, 418 F.3d at 1278). Under the first prong, “[a] petitioner must provide a ‘reputable medical or scientific explanation’ for its theory.” *Boatmon v. Sec’y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Moberly ex rel. Moberly v. Sec’y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010)). “While it does not require medical or scientific certainty, it must still be ‘sound and reliable.’” *Id.* (quoting *Knudsen ex rel. Knudsen v. Sec’y of Dept. of Health & Human Servs.*, 35 F.3d 543, 548–49 (Fed. Cir. 1994)). Petitioners “need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act.” *Andreu ex rel. Andreu v. Sec’y of Dept. of Health & Human Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009). Where such evidence is introduced, however, it must not be viewed “through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. For satisfying the second prong, “medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether ‘a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.’” *Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, 1326 (Fed. Cir. 2006) (quoting *Althen*, 418 F.3d at 1280). Lastly, “the proximate temporal relationship prong 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).

B. Analysis

Petitioner argues the Chief Special Master erred in determining petitioner’s injury was NMOSD because in doing so, he “relied upon broader diagnostic criteria, for which there was no basis in the record.” Pet’r’s Mot. for Review at 13. Petitioner identifies three findings in the Chief Special Master’s decision he claims were not in accordance with law because they were not “based ‘on the record as a whole:’” (1) that petitioner had dissemination in space due to the extension of his thoracic spinal lesion; (2) that petitioner had dissemination in space because he had a lesion in the periventricular region of his brain; and (3) petitioner had an area postrema lesion due to the lesion at the floor of the fourth ventricle. *Id.* at 12, 13, 24 (quoting 42 U.S.C. § 300aa-12(e), § 300aa-13(a)(1)).

Respondent responds, highlighting the Chief Special Master’s acknowledgment that “[p]etitioner has raised reasonable objections’ to the NMOSD diagnosis, ‘such that I could not

find that the NMOSD diagnosis is supported by even 75 percent of the record. However, the evidence still *preponderates* in favor of the NMOSD diagnosis (a determination that merely means more than 50 percent of the record favors that determination).” Resp’t’s Resp. to Pet’r’s Mot. for Review at 14 (quoting *Morgan*, 2019 WL 7498665, at *19). Respondent therefore argues “it is unsurprising that petitioner can point to evidence in the record that cuts against the Chief Special Master’s ultimate determination that petitioner suffered from NMOSD, but those arguments fall far short of demonstrating that the Chief Special Master’s conclusion was based on evidence that is ‘wholly implausible.’” *Id.*

The Chief Special Master stated, “[p]etitioner is correct in pointing out the criteria that apply in the context of a seronegative [NMOSD] patient, as well as the difficulty in establishing those criteria, but there was still evidence to fit each criterion—Petitioner initially exhibited acute myelitis with LETM, and demonstrated brain lesions in the area postrema region of the brain.” *Morgan*, 2019 WL 7498665, at *18 (citing Pet’r’s Ex. 5, at 56; Pet’r’s Ex. 14, at 110, 135; Pet’r’s Ex. 21, at 1; Pet’r’s Ex. 53, at 21; Resp’t’s Ex. E at 3). As previously discussed, the diagnostic criteria for seronegative NMOSD, as outlined by Wingerchuk, require an individual experience two or more different core clinical characteristics, in addition to MRI imagery showing NMOSD lesions. *See* Resp’t’s Ex. E, at 3. Wingerchuk provides “[a]t least 1 core clinical characteristic must be optic neuritis, acute myelitis with LETM, or area postrema syndrome.” *Id.* Both experts agreed petitioner had acute myelitis, fulfilling this requirement. *See* Hr’g Tr. at 68:11–12 (Dr. Tornatore); 108:1–2 (Dr. Sriram).

1. Whether the Chief Special Master’s Finding that Petitioner had Dissemination in Space due to the Extension of his Thoracic Lesion was Supported by the Record

The Wingerchuk diagnostic criteria require “[d]issemination in space (2 or more different core clinical characteristics).” Resp’t’s Ex. E, at 3. Besides acute myelitis, the other core clinical characteristics a seronegative NMOSD patient might exhibit include: (1) “Optic neuritis;” (2) “Area postrema syndrome: episode of otherwise unexplained hiccups or nausea and vomiting;” (3) “Acute brainstem syndrome;” (4) “Symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions;” and (5) “Symptomatic cerebral syndrome with NMOSD-typical brain lesions.”⁹ *Id.* The Chief Special Master noted, however, there was, “as Dr. Sriram proposed, evidence of dissemination in space, because later MRI reports from September 2015 show . . . a lesion extending to T6, where it had previously extended only to T8 before resolving.” *Morgan*, 2019 WL 7498665, at *18 (citing Pet’r’s Ex. 5, at 70, 80; Pet’r’s Ex. 14, at 110, 137; Pet’r’s Ex. 21, at 1). Dr. Sriram testified on cross-examination during the hearing that he would diagnose petitioner with seronegative NMOSD due to petitioner’s myelitis and lesions. Hr’g Tr. at 108:21–109:9. Dr. Sriram explained, describing the visible progression of petitioner’s disease: “He had an old lesion that stopped at T8. His new lesion in the relapse involved up to T6. So there was an extension of the old – or a new lesion, T6 down.” *Id.* at 109:4–7. While Dr. Sriram acknowledged that

⁹ Dr. Sriram testified that petitioner did not have any symptoms representative of acute brain syndrome, symptomatic narcolepsy or acute diencephalic clinical syndrome, or symptomatic cerebral syndrome with NMOSD-typical brain lesions. Hr’g Tr. at 108:9–19.

progression is “not typically” considered dissemination in space, the lesion “disseminated from one region of the spinal cord to an additional region of the spinal cord.” *Id.* at 109:12–15.

Petitioner quotes portions of Wingerchuk to argue Dr. Sriram’s and the Chief Special Master’s determinations that petitioner’s spinal lesion growth satisfied the dissemination in space requirement was contrary to evidence in the record. Pet’r’s Mot. for Review at 19–20. For example, petitioner clarifies Wingerchuk’s requirement that a seronegative NMOSD patient exhibit dissemination in space means “dissemination in space, affecting different neuroanatomic regions;” in other words, “an extensive spinal cord lesion, on its own, is not evidence of dissemination in space.” *Id.* at 19 (quoting Resp’t’s Ex. E, at 3) (emphasis omitted). Further, petitioner quotes from Dr. Brian G. Weinshenker’s article titled “Neuromyelitis Spectrum Disorders,” respondent’s Exhibit G (“Weinshenker”)¹⁰: “[r]ecurrent isolated episodes of . . . myelitis do not qualify [as NMOSD] [for seronegative patients].” *Id.* (quoting Resp’t’s Ex. G, at 4, ECF No. 47-1). Weinshenker continues, however: “NMOSD *cannot be excluded* in this situation” Resp’t’s Ex. G, at 4 (emphasis added).

To the extent petitioner contends the Chief Special Master disregarded the medical literature in finding petitioner had dissemination in space, “[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.” *Moriarty ex rel. Moriarty v. Sec’y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (citing *Hazlehurst v. Sec’y of Health & Human Servs.*, 604 F.3d 1343, 1352 (Fed. Cir. 2010)). In fact, the Chief Special Master wrote, “[w]hile I have reviewed all of the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner’s case.” *Morgan*, 2019 WL 7498665, at *16.

Moreover, there was sufficient evidence in the record to support the Chief Special Master’s determination that petitioner satisfied the dissemination in space requirement for a seronegative NMOSD diagnosis. Dr. Sriram testified petitioner’s “myelitis also involved . . . an extension of an old lesion,” which although is “not typically” considered dissemination in space, “[l]iterally . . . disseminated from one region of the spinal cord to an additional region of the spinal cord.” Hr’g Tr. at 109:3–4, 10–15. Further, despite acknowledging he was “probably a little more liberal with the interpretation of the criteria,” Dr. Sriram explained the diagnostic criteria “are usually guidelines to physicians” and “we are not necessarily . . . boxed into this alone.” *Id.* at 109:23–24, 110:9–10, 13–14. While petitioner contends there was no basis in the record for the Chief Special Master’s findings, Dr. Sriram’s testimony provides a basis in the record. It is not the Court’s role “to reweigh the factual evidence, or to assess whether the special master correctly evaluated the evidence. And of course we do not examine the probative value of the evidence or the credibility of the witnesses. These are all matters within the purview

¹⁰ Dr. Dean M. Wingerchuk, M.D., is also an author of the Weinshenker article, respondent’s Exhibit G. See Resp’t’s Ex. G, at 1. Both Wingerchuk and Weinshenker outline the diagnostic criteria for NMOSD. Compare Resp’t’s Ex. E, at 3, with Resp’t’s Ex. G, at 4. Throughout the Chief Special Master’s decision and subsequent briefing, respondent’s Exhibit E is referred to as “Wingerchuk” and respondent’s Exhibit G as “Weinshenker.” See, e.g., *Morgan*, 2019 WL 7498665, at *8; Pet’r’s Mot. for Review at 14–15.

of the fact finder.” *Munn v. Sec’y of Dept. of Health & Human Servs.*, 970 F.2d 863, 871 (Fed. Cir. 1992).

With Dr. Sriram’s testimony, based on treating physician’s records, the Court cannot say the Chief Special Master’s finding that petitioner had dissemination in space was “wholly implausible.” See *Lampe*, 219 F.3d at 1363 (“Since the special master’s conclusion was based on evidence in the record that was not wholly implausible, we are compelled to uphold that finding as not being arbitrary and capricious.”). The Chief Special Master reasonably relied on expert testimony and evidence of dissemination in space to support his finding that petitioner suffered from seronegative NMOSD. See *Broekelschen*, 618 F.3d at 1348 (quoting *Hines*, 940 F.2d at 1528) (“[R]eversible error is “extremely difficult to demonstrate” if the special master “has considered the relevant evidence of record, drawn plausible inferences and articulated a rational basis for the decision.””). The Chief Special Master’s finding that evidence supported dissemination in space was accordingly not “arbitrary, capricious, an abuse of discretion, or otherwise not in accordance with law” because it was supported by the record. 42 U.S.C. § 300aa-12(e)(2)(B).

2. Whether the Chief Special Master’s Finding that Petitioner had Dissemination in Space Because of a Periventricular Brain Lesion was Based in the Record

Petitioner similarly challenges the Chief Special Master’s finding that there was, “as Dr. Sriram proposed, evidence of dissemination in space, because later MRI reports from September 2015 show a brain lesion in the periventricular region of the brain.” *Morgan*, 2019 WL 7498665, at *18 (citing Pet’r’s Ex. 14, at 110; Ex. 21, at 1). Petitioner quotes the Wingerchuk criteria, which state “area postrema syndrome ‘requires associated dorsal medulla/area postrema lesions,’” and explains “[t]he periventricular region, by contrast, expands beyond the dorsal medulla and area postrema.” Pet’r’s Mot. for Review at 24. Petitioner therefore argues “the Chief Special Master appears to have conflated the ‘periventricular region of the brain’ with the more specific MRI requirements for area postrema syndrome.” *Id.* (quoting Resp’t’s Ex. E, at 3). Additionally, petitioner claims although the Chief Special Master suggested Dr. Sriram proposed a periventricular lesion constitutes evidence of dissemination in space, there is no evidence in the record of Dr. Sriram “rel[ying] upon a ‘periventricular lesion’ in his opinion.” *Id.* Petitioner quotes from Dr. Pace’s notes where he states the MRIs show “nonspecific . . . hyperintensities seen in the brain,” which he specifies are “not in a pattern that is strongly suggestive of demyelination such as would be seen with multiple sclerosis.” *Id.* at 25 (quoting Pet’r’s Ex. 14, at 110).

Petitioner cites Dr. Pace’s notes to support his contention; however, reviewing the MRI images of hyperintensities in petitioner’s brain, Dr. Pace noted they “can be seen in neuromyelitis optica spectrum, as this is the location of high concentration Aquaporin 4 channels.” Pet’r’s Ex. 14, at 110. Additionally, the Chief Special Master cites petitioner’s 19 September 2015 MRI report. See *Morgan*, 2019 WL 7498665, at *18; Pet’r’s Ex. 21. That report notes, “[t]here are scattered foci of hyperintense FLAIR and T2-weighted signal identified within the *bilateral periventricular* and subcortical white matter.” Pet’r’s Ex. 21, at 1 (emphasis added). Contrary to petitioner’s contentions, this report suggests petitioner had lesions in the

periventricular region of the brain. Moreover, Dr. Sriram testified that “very few diseases . . . give you a postrema lesion in the floor of the fourth ventricle.” Hr’g Tr. at 110:15–17.

To the extent petitioner disagrees with the terminology the Chief Special Master used, this does not rise to the level of arbitrary or capricious that would justify setting aside factual findings. Given Dr. Pace’s notes suggesting petitioner’s brain lesion could be seen with NMOSD, the radiology report of petitioner’s September 2015 MRI suggesting petitioner had a bilateral periventricular lesion, and Dr. Sriram’s testimony, the Court cannot say the Chief Special Master’s finding was “wholly implausible.” *Cedillo v. Sec’y of Health & Human Servs.*, 617 F.3d 1328, 1338 (Fed. Cir. 2010) (quoting *Lampe*, 219 F.3d at 1363). Petitioner’s arguments amount to a request that the Court reweigh the evidence—a task the Chief Special Master completed. Since the Chief Special Master’s finding that petitioner had a periventricular lesion suggestive of NMOSD was based in the record, the Court must uphold it as not arbitrary or capricious. *Id.* (quoting *Lampe*, 219 F.3d at 1363) (“We ‘do not sit to reweigh evidence. [If] the Special Master’s conclusion [is] based on evidence in the record that [is] not wholly implausible, we are compelled to uphold that finding as not arbitrary or capricious.’”).

3. Whether the Chief Special Master’s Finding that Petitioner’s Area Postrema Lesion Supported a NMOSD Diagnosis was Based in the Record

Petitioner also challenges the Chief Special Master’s finding that petitioner’s “brain lesions in the area postrema region of the brain” supported a seronegative NMOSD diagnosis by satisfying area postrema syndrome as the second core clinical characteristic. *See* Pet’r’s Mot. for Review at 21; *Morgan*, 2019 WL 7498665, at *18. Petitioner points to Wingerchuk’s criteria, which explain that area postrema syndrome, a core clinical characteristic of seronegative NMOSD, “requires both a presentation of symptoms (i.e., ‘intractable hiccups or nausea and vomiting’) and MRI findings ‘meant to enhance diagnostic specificity [that] must also be present.’” Pet’r’s Mot. for Review at 20 (quoting Resp’t’s Ex. E, at 3) (emphasis and internal footnotes omitted). The Chief Special Master acknowledged petitioner’s argument that “evidence of area postrema syndrome [was] not strongly supported by the record.” *Morgan*, 2019 WL 7498665, at *18 (emphasis omitted). The Chief Special Master also noted, however, “while Petitioner did not experience some of the *symptoms* that would be associated with an area postrema lesion . . . , his treating physicians nevertheless noted that the mere *existence* of an area postrema lesion supported a diagnosis of NMOSD by itself.” *Id.* (citing Pet’r’s Ex. 14, at 110).

During oral argument, petitioner argued the exhibit the Chief Special Master cited for the proposition that petitioner’s “treating physicians note[d] that the mere *existence* of . . . [an] area of postrema lesion supported a diagnosis of NMO by itself” did not in fact support that assertion. Tr. at 21:23–22:10, ECF No. 73. Respondent conceded the Chief Special Master likely conflated treating physician statements with Dr. Sriram’s testimony during the hearing. *See id.* at 26:24–28:2. While the exhibit the Chief Special Master cited—Dr. Pace’s notes from petitioner’s 26 November 2014 visit—does not state the mere existence of an area postrema lesion meant petitioner had NMOSD, Dr. Pace noted in his review of petitioner’s 31 August 2014 MRI:

There are several nonspecific T2/FLAIR hyperintensities seen in the brain.¹¹ These are not in a pattern that is strongly suggestive of demyelination such as would be seen with multiple sclerosis. However, there is T2 hyperintensity in the fourth ventricle surrounding the cerebral aqueduct. This is of unclear significance, *but can be seen in neuromyelitis optica spectrum*, as this is the location of high concentration of Aquaporin 4 channels.

Pet’r’s Ex. 14, at 110 (emphasis added). Dr. Sriram testified on cross-examination during the hearing “there are very few diseases that give you a postrema lesion in the floor of the fourth ventricle.” Hr’g Tr. at 110:15–17. Dr. Sriram continued: “[I]rrespective if the patient did not have hiccups and did not have vomiting and nausea, this is something that clinicians pay attention to.” *Id.* at 110:19–21. He also testified the lesion in the floor of the fourth ventricle was “very persuasive for an NMO spectrum disorder.” *Id.* at 111:12–13. Further, he testified that although petitioner did not exhibit the clinical systems of area postrema syndrome (hiccups or nausea and vomiting), “[i]t’s not uncommon to have silent lesions.” *Id.* at 112:20.

As previously discussed, to the extent petitioner claims the Chief Special Master disregarded the Wingerchuk criteria, the Court “presume[s] that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.” *Moriarty ex rel. Moriarty*, 844 F.3d at 1328 (citing *Hazlehurst*, 604 F.3d at 1352). Additionally, although citation to one treating physician’s records does not support the proposition that petitioner’s area postrema lesion standing alone supports a NMOSD diagnosis, there is ample evidence in the record to support the Chief Special Master’s finding that petitioner’s area postrema lesion supported a seronegative NMOSD diagnosis. Both Dr. Pace’s notes from reviewing petitioner’s MRIs and Dr. Sriram’s testimony lend further support for the Chief Special Master’s finding. Petitioner’s argument asks the Court to reweigh which pieces of evidence support discrete findings, but that is not the Court’s role in reviewing a special master’s decision. “We ‘do not sit to reweigh the evidence. [If] the Special Master’s conclusion [is] based on evidence in the record that [is] not wholly implausible, we are compelled to uphold that finding as not being arbitrary or capricious.’” *Cedillo v. Sec’y of Health & Human Servs.*, 617 F.3d 1328, 1338 (Fed. Cir. 2010) (quoting *Lampe*, 219 F.3d at 1363). The Chief Special Master’s finding, based on the evidence, is not “wholly implausible.” *Id.* The Court therefore finds the Chief Special Master’s finding was not arbitrary or capricious and was supported by the record. *Lampe*, 219 F.3d at 1360 (“The arbitrary and capricious standard of review is difficult for an appellant to satisfy with respect to any issue, but particularly with respect to an issue that turns on the weighing of evidence by the trier of fact.”).

4. Petitioner’s Burden to Prove the Flu Vaccine Caused LETM

¹¹ Upon further review by the Court, it seems the confusion may be that Dr. Pace notes T2/FLAIR “hyperintensities” rather than “lesions.” Hyperintensities on T2-weighted MRI images of the brain depict white matter lesions, as “[w]hite matter *lesions* are considered present if *hyperintense* on T2 weighted . . . images.” See Stéphanie Debette & H.S. Markus, *The clinical importance of white matter hyperintensities on brain magnetic resonance imaging: systematic review and meta-analysis*, *The BMJ* (July 26, 2010), <https://www.bmj.com/content/341/bmj.c3666>.

Respondent argued in its response to petitioner’s motion for review “[m]erely attempting to cast doubt about the Chief Special Master’s conclusion regarding the diagnosis does not address” the requirements that “petitioner . . . prove by preponderant evidence which injury he suffered, and that the injury was caused by a vaccine.” Resp’t’s Resp. to Pet’r’s Mot. for Review at 17. Respondent thus asserted “petitioner appears to assume that the nature of his condition is an either/or proposition: if it is not NMOSD, it must be TM. But the record does not support this assumption.” *Id.* During oral argument, respondent expounded on these arguments, contending it was not sufficient for petitioner to allege molecular mimicry, a medical theory which has been accepted in Vaccine Program cases before, establishes the link of causation between the vaccine and injury. *See* Tr. at 60:14–61:3. Respondent explained, “irrespective of which diagnosis the [Chief] Special Master ultimately landed on, one of the things that he did note was that for molecular mimicry to have utility in this case, there should be some evidence of the relevant antibodies that are known to drive or at least are associated with the resulting demyelinating disease are likely produced as a result of the flu vaccine.” *Id.* at 61:4–11. Dr. Tornatore’s testimony, in contrast, was “exceptionally vague,” respondent contends, and “[t]here was really nothing sufficient to tether this theory to the flu vaccine that the Petitioner received and this theory does not explain the presence of the lesion that was in the Petitioner’s brain.” *Id.* at 62:1–5. Respondent therefore argued, even if “the Court were to rule that NMOSD is not the correct diagnosis, that does not mean that LETM is the correct diagnosis.” *Id.* at 62:6–8.

In response, petitioner argued “[w]hat the [Chief] Special Master is looking for here is, in fact, direct contradiction to his own finding from just three years ago,” citing *Johnson v. Secretary of Health & Human Services*, No. 14-113V, 2017 WL 772534 (Fed. Cl. Spec. Mstr. Jan. 6, 2017). *Id.* at 63:22–25. Petitioner pointed to footnote 25 of that case, where the Chief Special Master wrote: “Petitioner did not show exactly which antigen would be involved in the proposed cross-reactivity process, nor did she offer any studies showing molecular mimicry could happen between [immune thrombocytopenic purpura (“ITP”)] and [human papillomavirus (“HPV”)]. But to require Petitioner to have done so amounts to heightening the burden of proof beyond what a claimant need offer.” *Johnson*, 2017 WL 772534, at *19 n.25. Petitioner therefore asserted, “[a] petitioner does not have to show a precise, exact biological mechanism of injury. To do so inappropriately heightens the petitioner’s burden to the level of scientific certainty, a level that’s not required under preponderant evidence.” Tr. at 64:14–18.

The Chief Special Master recognized “molecular mimicry has repeatedly been embraced in Program cases as a reliable scientific mechanism for explaining the pathophysiology of certain immune-mediated conditions, including many demyelinating disorders.” *Morgan*, 2019 WL 7498665, at *19. As applied to petitioner’s case, however, the Chief Special Master found petitioner offered little evidence to show “the relevant autoantibodies that are known to drive, or at least associated with, the resulting demyelinating disease are likely produced as a result of the flu vaccine,” which the Chief Special Master noted was “reasonable to require” of a petitioner “when evaluating the success of the claimant’s [*Althen*] prong one showing.” *Id.* (citing *W.C. v. Sec’y of Health & Human Servs.*, 704 F.3d 1352, 1361 (Fed. Cir. 2013)). The Chief Special Master observed, “[a]t best, there are some references in the literature indicating that NMOSD initially manifesting as LETM could be caused by a variety of infectious agents (i.e., the herpes virus, dengue fever, tuberculosis, etc.). But this list does not also include the influenza wild virus. Nor did petitioner’s filings establish how an initial reaction to a vaccination might be

sufficient to create the kind of chronic, CNS-oriented inflammatory process that would ultimately morph into NMOSD.” *Id.* at *20. The Chief Special Master therefore found petitioner failed to demonstrate with “reliable and persuasive evidence . . . that the flu vaccine could cause a chronic form of CNS demyelinating disease such as NMOSD, that would unfold over a lengthy period of time.” *Id.*

The *Johnson* case is inapposite to the instant case. In *Johnson*, the parties largely agreed the petitioner suffered from ITP; they merely disagreed whether the HPV vaccine was a contributing factor to the petitioner’s condition. *Johnson*, 2017 WL 772534, at *15. Here, the parties dispute what petitioner’s injury was, and this is the crux of petitioner’s motion for review. Notably, however, petitioner does not argue in his motion for review the flu vaccine caused LETM, nor does he challenge the Chief Special Master’s holding that petitioner failed to meet his burden to prove the flu vaccine caused his injury. Respondent therefore asserted during oral argument “even if [the Court] determine[s] that it’s not NMOSD, . . . it’s not necessarily the case that that finding would translate to a finding that the Petitioner has met his burden to demonstrate a legally cognizable injury.” Tr. at 13:25–14:4. Likewise, respondent argued “ruling out NMOSD does not automatically rule in LETM as the Petitioner’s ultimate diagnosis. And, again, it is the Petitioner that bears the burden of demonstrating that and they did not challenge or raise an appeal” of the Chief Special Master’s “finding that petitioner failed to meet its burden of showing a cognizable injury.” *Id.* at 28:11–12, 19–23. Contrary to petitioner’s argument, the Chief Special Master did not raise the burden of causation in this case; petitioner simply failed to meet it. As the Chief Special Master stated, the medical literature petitioner offered did not connect the flu vaccine or influenza wild virus to a demyelinating disorder that eventually manifests as NMOSD. *Morgan*, 2019 WL 7498665, at *20. The Chief Special Master was not requiring petitioner to “show exactly which antigen would be involved in the proposed-cross reactivity process.” *Johnson*, 2017 WL 772534, at *19 n.25. The Chief Special Master was looking for evidence “involving the flu vaccine . . . and its association with NMOSD, or proof that immune system stimulation can at least *initiate* a chronic process.” *Morgan*, 2019 WL 7498665, at *19. Therefore, even if the Court found the Chief Special Master’s finding that petitioner suffered from NMOSD was arbitrary and capricious, that does not mean petitioner met his burden of proof to show the flu vaccine caused LETM.

III. Conclusion

For the foregoing reasons, the Court **SUSTAINS** the Chief Special Master’s decision because it was not arbitrary, capricious, or not otherwise in accordance with law. The Court therefore **DENIES** petitioner’s motion for review. The Clerk of Court is directed to enter judgment for respondent.

IT IS SO ORDERED.

s/ Ryan T. Holte
 RYAN T. HOLTE
 Judge