In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 20-1924V

YE XIA, *as administrator for* ESTATE OF S.L.,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: August 29, 2025
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Ronald C. Homer, Conway Homer P.C., Boston, MA, for Petitioner.

Voris E. Johnson, Jr., U.S. Department of Justice, Washington, D.C., for Respondent.

RULING ON ENTITLEMENT[1]

On December 21, 2020, Ye Xia, on behalf of her minor child, S.L., filed this action seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petition (ECF No. 1) ("Pet.") at 1. Petitioner alleges that the influenza ("flu") vaccine administered to S.L. on February 1, 2020, caused S.L. to experience autoimmune encephalitis, and ultimately led to her tragic death. Pet. at 1–2.

A trial was held in this matter on October 7, 2024. Now, based upon my review of the record and consideration of the hearing testimony, including expert input, I find Petitioner entitled to compensation.

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public in its present form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 State. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references will be to § 300aa of the Act (but will omit that statutory prefix).

I. Factual Background

Vaccination and Emergency Hospitalization

S.L., born on December 7, 2008, was eleven years old at the time of the vaccination at issue, and had no prior medical history bearing on this claim. Ex. 4; *see generally* Ex. 3. On February 1, 2020, S.L. presented to Nationwide Children's Hospital with a two-day history of congestion, cough, fever, headache, sore throat and swollen glands, along with a faint rash around her mouth. Ex. 2 at 30–31. A physical exam revealed that S.L. was running a high fever (104.2 degrees), and her left ear's tympanic membrane appeared inflamed. *Id.* at 30. Treaters assessed S.L. with left otitis media,[3] "likely adeno"[4] and prescribed oral antibiotics.

It was during this treatment visit that S.L. also received a flu vaccine. Ex. 2 at 32. Thus, the record reveals that S.L. was unquestionably experiencing some kind of severe infection *at the time of vaccination*, and which had already begun. And there is no record evidence of any noticeable vaccine reaction in the immediate days after.

Three days later, on February 4, 2020, S.L.'s father called her primary care provider's ("PCP") office regarding her ongoing cough and other persistent symptoms (including sore throat and headache) over the last five days. Ex. 3 at 20. He also reported S.L.'s recent exposure to an individual with flu-like symptoms. *Id.* The next day (February 5, 2020), at approximately 1:00 p.m., S.L.'s mother called the PCP again, reporting that S.L. was experiencing a "URI and influenza like illness," as well as congestion, cough, vomiting, diarrhea, headache, fatigue, shortness of breath, wheezing, chills, shaking of the "hands and face," and fever. *Id.* at 18–19. It was recommended that S.L. undergo further evaluation at Nationwide Children's Hospital urgent care. *Id.*

To that end, S.L. was taken to the emergency room ("ER") at Nationwide Children's Hospital via emergency medical services. Her parents reported that S.L. had a preceding six- to seven-day history of fevers, and that she was taking amoxicillin for an acute ear infection. Ex. 2 at 8539. They noted further that S.L. recently developed several episodes of vomiting the prior evening and had a temperature of 103.3 degrees that morning. *Id.* As the day progressed, S.L. became "combative," and began to exhibit intermittent left facial twitching, left arm shaking, and right eye deviation. *Id.* at 8539, 8544.

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[3] "Otitis Media" is defined as "inflammation of the middle ear; subtypes are distinguished by length of time from onset (*acute* versus *chronic*) and by type of discharge (*serous* versus *suppurative*)." *Otitis Media*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=95455&searchterm=otitis+media (last visited Aug. 29, 2025).

[4] Adenoviruses can cause a wide range of signs and symptoms, including common cold or flu-like symptoms, fever, sore throat, acute bronchitis, pneumonia, pink eye, and acute gastroenteritis. Less common symptoms are bladder inflammation or infection, and neurologic disease. *About Adenovirus*, Center for Disease Control and Prevention, https://www.cdc.gov/adenovirus/about/index.html (last visited Aug. 29, 2025).

Assessment of S.L. while Hospitalized

Upon arrival, S.L.'s temperature was 101.1 degrees, she was tachycardic and hypotensive with a mean arterial pressure[5] of 50–55, and she exhibited seizure-like activity with an altered mental state. Ex. 2 at 8546–44. S.L.'s treating providers initiated treatment for presumed encephalopathy, focal seizure, and septic shock,[6] and initially proposed a wide differential diagnosis, including in it "sepsis, meningitis, encephalitis (Herpes Simplex Virus ("HSV") vs other viral vs autoimmune), status epilepticus, toxic ingestion, space occupying lesion in the brain, influenza, UTI" and "pneumonia, bacteremia, intercranial hemorrhage, mass effect, cerebral edema." *Id.* at 8539, 8543.

S.L. received two doses of Ativan to control her seizures, but no success was achieved until an alternative medication was administered. Ex. 2 at 8543. She was eventually placed on antibiotics (Vancomycin and Ceftriaxone), an antiviral (Acyclovir), and was given two liters of fluid bolus, but demonstrated little to no improvement in her hypotension. *Id.* Thereafter, S.L. underwent a head CT and was then intubated due to a concern for respiratory failure. *Id.* at 8545. Despite some transient improvement in her blood pressure, S.L.'s systolic blood pressure dropped, leading treaters to start a vasoconstrictor. *Id.* at 8545. She underwent a lumbar puncture and ultimately was admitted to the pediatric intensive care unit, where she remained during her hospitalization. *Id.* at 8541.

Neurologist Whitney Woodhull, M.D., evaluated S.L. shortly after her arrival and noted that although S.L. was not alert, she still exhibited some ability to move her extremities in response to painful stimuli. Ex. 2 at 8522. After a review of S.L's lab testing and imaging results, Dr. Woodhull deemed a bacterial infectious explanation unlikely, favoring instead HSV encephalitis as a proper diagnosis, based upon the evidence of high cerebral spinal fluid ("CSF") of 57 mg/dL (reference range 14–45 mg/dL) with a red blood cell count ("RBC") of 570/mm3. *Id.* at 8520, 8535–36. Dr. Woodhull ordered a brain MRI and long-term electroencephalographic monitoring ("LTM"), as well as developed a seizure management plan that included benzodiazepines and anti-epileptic drugs ("AED") should S.L.'s seizure activity continue. *Id.*

[5] "Mean Arterial Pressure" is defined as "the average pressure within an artery over a complete cycle of one heartbeat." *Mean Arterial Pressure*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=99948&searchterm=mean+arterial+pressure (last visited Aug. 29, 2025).

[6] "Septic Shock" is defined as "shock associated with overwhelming infection, usually infection with gram-negative bacteria, although it may be produced by other bacteria, viruses, fungi, or protozoa. It is thought to result from the action of endotoxins or other products of the infectious agent on the vascular system, causing large volumes of blood to be sequestered in capillaries and veins; activation of the complement and kinin systems and the release of histamine, cytokines, prostaglandins, and other mediators may be involved." *Septic Shock*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=105946&searchterm=septic+shock (last visited Aug. 29, 2025).

The next day (February 6, 2020), neurologist Lisa Pabst, M.D., provided a detailed consult of S.L. Ex. 2 at 8465–71. Despite the AED trials of Keppra and Vimpat, Dr. Pabst expressed concern for S.L's ongoing status epilepticus with rhythmic twitching in the face, left and right hands and legs. *Id.* She was given phenobarbital which stopped the activity. *Id.* In reviewing S.L.'s lab work, imaging studies, and LTM, she noted that S.L's LTM was significant "for a high burden of periodic discharges with right frontal area being the most prominent/evolving region" and the brain MRI revealed mild parenchymal volume loss, with no evidence of demyelination or focal abnormalities. *Id.* at 1796, 8440, 8470.

S.L. was then placed into a drug-induced coma to suppress her seizure activity. Ex. 2 at 8333. Dr. Pabst's "immediate goal [was] to put [S.L.] into burst suppression with pentobarbital given the duration and refractory nature of seizures while continuing to evaluate for underlying etiology"—the main areas needed for further investigation included infectious, autoimmune, and genetic conditions. *Id.* at 8470. At this time, the differential diagnosis was most concerning for infectious and para-infectious etiologies. But Dr. Pabst also considered HSV encephalitis, ADEM, autoimmune encephalitis, fulminant onset of epilepsy in the setting of febrile illness ("FIRES"),[7] toxicological process, vascular process, and underlying metabolic or mitochondrial epilepsy. *Id.* at 8417, 8470–71. S.L.'s etiology work-up began with an infectious disease consult, an ophthalmology consult, comprehensive genetic epilepsy panel, autoimmune encephalopathy panel, as well as a pelvic ultrasound to evaluate for ovarian teratoma. *Id.* at 8417.

The aforementioned consultations shed little light on possible etiologies for S.L.'s illness, however. An ophthalmology consultation confirmed no significant ophthalmologic abnormalities, and the pelvic ultrasound was negative for concern for ovarian mass. Ex. 2 at 8354, 1805–06. On February 7, 2020, Angelique Boutzoukas, M.D., an infectious disease specialist, provided a consultation and opined that HSV was the "most worrisome and consistent" given S.L.'s age group. Although S.L.'s recent labs for HSV were negative, Dr. Boutzoukas recommended follow-up testing to confirm the results were "not a result of early testing," as well as other possible, if atypical, infectious causes. Ex. 2 at 8390. Dr. Boutzoukas further recommended discontinuing antibiotic medications should repeat CSF testing fail to identify the presence of a bacterial infection, and proposed that steroid treatment be avoided until HSV encephalitis was officially ruled out. *Id.*

Subsequent Treatment

Despite continued efforts, treaters made little progress in identifying the cause of S.L.'s condition, and her seizures did not respond to their efforts. A nasopharynx respiratory viral panel,

[7] "Febrile Infection-Related Epilepsy Syndrome" is understood as "a catastrophic epileptic syndrome that strikes previously healthy children aged 3–15 years and has an unknown pathogenesis and few treatments. These children experience a nonspecific febrile illness that is followed by prolonged refractory status epilepticus." National Library of Medicine, https://pubmed.ncbi.nlm.nih.gov/28898171/ (last visited Aug. 29, 2025).

collected on February 7, 2020, resulted in a positive finding for adenovirus. Ex. 2 at 1811. Dr. Woodhull, however, doubted the significance of this finding, observing that it was not consistent with S.L.'s ongoing seizure activity, and adding that "[r]epeat [lumbar puncture][8] with further elevated protein and cell count points toward [an] autoimmune process." *Id.* at 8354. S.L. was subsequently administered a five-day course of high dose Solu-Medrol (a steroid treatment) beginning on February 9, 2020. *Id.* at 7101. She also underwent five sessions of plasmapheresis treatment between February 12 to February 20, 2020. *Id.* Later, after developing a pleural effusion,[9] S.L. required chest tube placement, and was treated with heparin to address a deep vein thrombosis. *Id.* at 8065, 8158. She received IVIG treatments, and for two weeks, beginning on February 21, 2020, S.L. received Anakinra (an immunosuppressive medication). However, these treatments also failed at arresting S.L.'s ongoing seizure activity. *Id.* at 8152.

During a neurology consult on March 2, 2020, S.L.'s treating providers noted minimal improvement after steroids, plasmapheresis, IVIG, and Anakinra, but recommended Cytoxan, a stronger immunosuppressant, based on their continued belief that the etiology for her illness was likely autoimmune-driven. Ex. 2 at 7321, 7516. Her clinical presentation was complicated by concerns for a potential CPT1 deficiency[10] and Drug Reaction with Eosinophilia and Systemic Symptoms,[11] leading to an abrupt discontinuation of certain medications. The working diagnosis remained an autoimmune mediated process "despite the lack of positive antibodies (other than anti GAD in the blood at a level that is not clinically significant)."

On March 4, 2020, neurologist Hera Kamdar, M.D., noted that "[c]linically, at the top of the differential diagnosis continues to remain a primary autoimmune etiology/inflammatory

[8] The results yielded a CSF with a total cell count of 43, with RBS of 1, white blood cell ("WBC") of 42 and protein of 106. Ex. 2 at 1815, 8354.

[9] "Pleural Effusion" is "the presence of fluid in the pleural space; types include chylothorax, hemothorax, hydrothorax, and pyothorax (empyema)." *Pleural Effusion*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=72555&searchterm=pleural+effusion (last visited Aug. 29, 2025).

[10] "Carnitine Palmitoyltransferase I Deficiency" is defined as "a rare autosomal recessive disorder caused by mutations in the *CPT1A* gene (locus: 11q13), which encodes carnitine palmitoyltransferase Ia. It is characterized by severe episodes of hypoketotic hypoglycemia, hepatomegaly, and encephalopathy, usually occurring after fasting or illness; onset is in infancy or early childhood." *Carnitine Palmitoyltransferase I Deficiency*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63335 (last visited Aug. 29, 2025).

[11] "Drug Reaction with Eosinophilia and Systemic Symptoms Syndrome, or "DRESS," is "a rare, potentially life-threating adverse drug reaction with cutaneous manifestations and internal organ involvement" and is caused by a "severe hypersensitivity to a medication and its reactive drug metabolites." *Nguyen v. Sec'y of Health & Hum. Servs.*, No. 17-2051V, 2022 WL 4392971, at *3 (Fed. Cl. Spec. Mstr. Aug. 29, 2022) (citing Z. Husain et al., *DRESS Syndrome: Part 1. Clinical Perspectives*, 68 J. Am. Acad. Dermatology 693e.1 (2013).

epilepsy based upon [S.L.'s] clinical course and biochemical features." *Id.* at 7224. Additionally, S.L.'s brain MRI,[12] dated March 3, 2020, explained her present differential diagnosis as:

> Structural etiology (not supported by imaging), primary genetic cause (less likely as she was previously healthy and developmentally normal, panel with multiple VUS, but KCTN1 mutation is a consideration), infections (she qualifies for a diagnosis of [FIRES] however infection and known pathogenic antibodies have not been identified), primary metabolic etiology (CPT1 deficiency was thought to be a possible etiology of her abnormal acylcarnitine profile however genetic testing is negative), or possible mitochondrial pathology (testing is being sent).

Id. Because the results of the genetic testing returned negative, S.L.'s treating providers proceeded with plans to initiate a ketogenic diet. *Id.* at 7216.

The following day, on March 5, 2020, a coordination of care conference was held with S.L.'s medical care team and her family, at which time future care elements were discussed (such as beginning Cytoxan once a month, beginning a ketogenic diet, completing genetic and mitochondrial tests, as well as needing a long-term ventilator, tracheostomy, and percutaneous endoscopic gastrostomy ("PEG") tube). Ex. 2 at 7186. S.L. underwent another round of IVIG treatment from March 12 to March 13, 2020, and was subsequently scheduled to undergo a muscle biopsy during her tracheotomy and PEG placement. *Id.* at 6786, 6610, 6826. However, a brain MRI performed prior to S.L.'s procedure revealed:

> 1. Further decreased cerebral volume with increased ex-vaco enlargement of the supratentorial ventricles. Unchanged cerebellar volume loss; 2. Persistent parenchymal abnormalities of the bilateral basal ganglia and thalami, which show decreased volume; 3. Multiple nonenhancing, scattered foci of T2 prolongation throughout the cerebral cortex, increased in number and conspicuity, especially in the occipital lobes. Persistent increased T2 signal of the hippocampi; 4. Normal appearing pituitary gland; 5 Unchanged marked pansinus disease and fluid-filled bilateral mastoid air cells and middle ear cavities.

Id. at 6434. S.L. underwent the contemplated treatment-oriented procedures on March 19, 2020, with minimal complications.

By March 20, 2020, S.L. was completely weaned from the pentobarbital, and ketosis was briefly obtained thereafter, leading to *some* improvement in seizure control on EEG. Ex. 2 at 6205. However, due to an upward trend in lab results for amylase, lipase, transaminase, and bilirubin, S.L.'s treating providers were now concerned for pancreatic inflammation, hepatotoxicity, and

[12] Results of which revealed "similar bilateral symmetric basal ganglia T2 hypersensitivity with patch enhancement" and "increase FLAIR signal in bilateral hippocampus which is not surprising given her frequency of seizures." Ex. 2 at 7222.

hepatic failure, causing the need to alter her medication and diet. *Id.* at 4172. These changes led to a loss of ketosis, resulting in increased seizure activity. *Id.*

S.L. received another round of IVIG treatment on April 8, 2020. Ex. 2 at 5499. That same day, however, S.L. was documented to have cytomegalovirus[13] ("CMV") viremia in her blood in relatively high quantities, deemed to be a delayed reactivation as a result of her extensive immunosuppressive medications. *Id.* at 5523. A subsequent lumbar puncture, performed on April 9, 2020, revealed a total cell count of 0, RBC of 0, WB of 0, glucose of 62, and protein of 29. Ex. 2 at 2273–74. In addition, repeat meningitis and encephalitis panels remained negative, and a repeat oligoclonal band profile was negative. *Id.* Because S.L. had been diagnosed with a CMV infection, her treating providers' ability to establish ketosis or administer further immunosuppressive therapies was greatly impacted. *Id.* at 5426.

Attending infectious disease specialist Rouba Sayegh, M.D., completed a thorough review of S.L.'s condition. Ex. 2 at 4992–5000. Dr. Sayegh noted that S.L. was intermittently febrile, with worsening seizures throughout the week, and her lab work further demonstrated ongoing liver abnormalities as a result of drug induced hepatotoxicity. *Id.* Due to these abnormalities and the progression of her MRI brain imaging, liver and brain biopsies were scheduled. *Id.* at 5007.

On April 16 and 17, 2020, Dr. Pabst reviewed S.L.'s overall course, and now characterized her condition as New Onset Refractory Status Epilepticus ("NORSE") of unknown etiology, but with the *highest* suspicion for autoimmune encephalitis. Ex. 2 at 5046 (emphasis added). Epilepsy and mitochondria gene panels produced unremarkable results that did not aid in identifying an etiologic explanation for S.L.'s NORSE, and the same was true for the lab testing and lumbar puncture results. *Id.* at 4969, 5056. Dr. Pabst further noted that the management of S.L.'s epilepsy had been significantly impacted by the need to avoid treatments that would themselves results in debilitating side-effect diseases or other biologic deficiencies. Ex. 2 at 5037. She recommended that S.L.'s treating providers continue to administer Ketamine for seizure suppression, but with close monitoring of her pancreas and liver function; pursue IVIG treatment on a four-week schedule; adjust S.L.'s diet; and obtain a brain tissue biopsy in the hopes of aiding in S.L.'s overall diagnostic workup. *Id.* at 4968, 5037.

On April 20, 2020, S.L. underwent a right anterior craniotomy to obtain a brain tissue sample, but the biopsy results were deemed clinically unremarkable. Ex. 2 at 4640. Additionally,

[13] "Cytomegalovirus" is defined as "a genus of ubiquitous viruses of the subfamily Betaherpesvirinae (family Herpesviridae) that infect humans and nonhuman primates, with the production of unique large cells bearing intranuclear inclusions. It includes the species human herpesvirus 5, the agent of cytomegalic inclusion disease." *Cytomegalovirus*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=12438 (last visited Aug. 29, 2025).

the results of a CSF neurotransmitter test[14] and pterin testing were also diagnostically unhelpful. Thus, the etiology of S.L.'s condition remained non-specific. *Id.* at 4670.

Seven days later, on April 27, 2020, S.L.'s medical care team met with her parents to discuss her condition and treatment in light of her prognosis, as she remained critically ill, intubated, mechanically ventilated, had persistent seizure activity refractory to treatment, and her brain MRI demonstrated tissue damage. Ex. 2 at 4084. Moreover, and despite extensive efforts, S.L.'s "testing [remained] unrevealing as to an etiology." *Id.* at 3980. Based on their discussion, S.L.'s family made the difficult decision to transition her to comfort care and remove her from life support. Ex. 2 at 4293.

On May 4, 2020, and following eighty-eight days of hospitalization, S.L. was officially removed from life support and passed away shortly after 10:00 p.m. that evening. Ex. 2 at 3966–67. Her death certificate, dated May 11, 2020, listed the cause of death as "Super Refractory Status Epilepticus, suspected autoimmune encephalitis." Ex. 4 at 1.

II. Witness Testimony

A. *Petitioner's Expert – Dr. AHM Mahbubul Huq*

Dr. Huq, a pediatric neurologist and clinical geneticist, prepared two written reports and testified on behalf of Petitioner. Report, dated Jan. 26, 2023, filed as Ex. 6 (ECF No. 29-1) ("First Huq Rep."); Report, dated Dec. 10, 2023, filed as Ex. 149 (ECF No. 56-1) ("Supp. Huq Rep."); Tr. at 5–100. Dr. Huq opined that S.L.'s receipt of the flu vaccine on February 10, 2020, more likely than not triggered her super refractory status epilepsy and autoimmune encephalitis and/or NORSE/FIRES, leading to her untimely death. First Huq Rep. at 36.

Dr. Huq received his Bachelor of Medicine and Bachelor of Surgery at Dhaka University/Dhaka Medical College in Bangladesh. *Curriculum Vitae*, filed as Ex. 7 (ECF No. 29-2) ("Huq CV") at 1; Tr. at 5. Thereafter, he received his Ph.D. at Tokushima University in Japan and completed residency training in Pediatrics and Neurology at the Children's Hospital of Michigan, Wayne State University, before completing clinical and post-doctoral fellowships in Medical Genetics and Pediatric Neurology. *Id.* Dr. Huq is a Professor of Pediatrics at Central Michigan University and Professor of Neurology at Wayne State University. Huq CV at 2; Tr. at 6; First Huq Rep. at 1. He is board certified in Neurology with a special qualification in Child Neurology by the American Board of Psychiatry and Neurology, as well as by the Royal College of Physicians and Surgeons of Canada. Huq CV at 4; Tr. at 5. Dr. Huq has treated approximately thousands of individuals with epilepsy—seeing 15 to 20 patients with epilepsy per week. First Huq

[14] CSF Neurotransmitter test "are integral to the diagnosis and management of multiple inborn metabolic errors, some of which require prompt identification and intervention to improve outcome." *Clinical Use of CSF Neurotransmitters*, National Library of Medicine, https://pubmed.ncbi.nlm.nih.gov/26194033/ (last visited Aug. 29, 2025).

Rep. at 1. He has also published several peer reviewed articles and abstracts on the genetics of epilepsy. *Id.*; Huq CV at 21–33

Dr. Huq began his testimony with an overview of S.L.'s medical history prior to her receipt of the flu vaccine. Tr. at 12. He noted that S.L. was in general good health—actively participating in sports, such as swimming, and excelling academically. *Id.* He then briefly turned to the events of February 1, 2020, when S.L. presented to Nationwide Children's Hospital with a one to two-day history of fever, headaches, and sore throat. *Id.* He noted the initial impression was otitis media and possible viral infection, of which her flu test was negative and thus she received the flu vaccine at this time. *Id.* at 13. Dr. Huq also acknowledged that S.L. presented with a "high fever of 104.2" during this visit (which thus could not be attributed to the vaccine she received after this temperature reading had been taken). Tr. at 13.

Over the course of the next several days, S.L. continued to experience symptoms (i.e., fever, headache, and sore throat), became gradually less responsive, and began exhibiting behavioral and personality changes. Tr. at 13. In addition to multiple episodes of vomiting, S.L. also started having focal seizures[15]—characterized mostly by twitching of the left side of her face, lips, arm, and leg, as well as a right-sided gaze deviation. *Id.* S.L. was subsequently brought to the emergency department at Nationwide Children's Hospital on February 5, 2020, and treaters now expressed concerned for encephalitis, meningitis, or some infectious process. *Id.* at 14. S.L. was transferred to the intensive care unit and underwent a series of extensive testing.

Dr. Huq then spent some time discussing the relevance of S.L.'s various diagnostic results. The findings of the initial EEG, he explained, demonstrated "periodic discharges," which is suggestive of epilepsy. Tr. at 13; Ex. 2 at 2424–25. Similarly, her lumbar punctures from February 5th and 7th revealed inflammatory changes (i.e., elevated RBC, WBC, and protein), although later CSF testing returned negative results for meningitis and encephalitis infection. Tr. at 16. Although records indicate that S.L. "intermittently" tested positive for adenovirus throughout the course of her hospitalization, her treaters were not convinced it adequately explained S.L.'s condition, in Dr. Huq's opinion. *Id.* at 17.

Additionally, multiple imaging studies (i.e., brain MRIs, Pet CT scans) were conducted. S.L. underwent brain MRIs on February 6th and 18th, March 17th, and April 13th—the results of which revealed persistent parenchymal volume loss and abnormalities, T2 flare changes in the basal ganglia, hippocampus, and defined cortical areas, as well as scattered foci optic to prolongation throughout the cerebral cortex. Tr. at 18. Based on these findings, radiologists opined that autoimmune encephalitis was a likely explanation for S.L.'s symptoms. Tr. at 19.

[15] Dr. Huq noted that S.L. was "in a constant like status epilepsy arising from the right temporal lobe" despite noticeable seizure-like activity externally or on clinical presentation. Tr. at 20. Moreover, the records indicate that S.L. did not respond to either the anti-seizure treatment or the aggressive immune therapies. *Id.* at 25.

Despite a negative autoimmune encephalopathy panel, S.L.'s brain biopsy (conducted on April 26, 2020) showed "astrocyte proliferation gliosis, which is a [type] of scarring in the brain, as well as infiltration of T lymphocytes"—a finding strongly suggestive for autoimmune encephalitis and/or FIRES, Dr. Huq opined. Tr. at 23, 24; Ex. 2 at 4329–93. Dr. Huq further stated, and relying on some independent literature, that approximately fifteen percent of patients with FIRES or NORSE have some demonstrated T-cell lymphocyte infiltration, suggesting that autoimmune encephalitis "could be the cause" of FIRES. Tr. at 24.

Based on his review of the medical records, Dr. Huq opined that the most appropriate diagnosis for S.L.'s neurologic condition was super-refractory status epilepsy due to autoimmune encephalitis. Tr. at 28. At the same time, however, Dr. Huq allowed that FIRES was also "an appropriate label" for her diagnosis (noting that nowadays it is common to give "multilevel diagnos[es]"), along with NORSE. *Id*. at 35. FIRES, he explained, "is a subcategory or subgroup of NORSE in which there is a fever between 24 hours [ ] to 2 weeks prior to the onset of the symptoms." *Id.* at 33.

Dr. Huq then discussed the diagnostic criteria for autoimmune encephalitis in pediatric patients—explaining that the clinical presentation is often different from the adult presentation. Tr. at 29. The proposed diagnostic criteria require: (1) evidence of acute or subacute symptoms; (2) at least two clinical features of neurologic dysfunction; (3) paraclinical evidence of neuroinflammation; (4) autoimmune encephalitis serology; and (5) exclusion of other etiologies. T. Cellucci et al., *Clinical Approach to the Diagnosis of Autoimmune Encephalitis in the Pediatric Patient*, 7 Neurol Neuroimmunol. Neuroinflamm. 1, 7 (2020), filed as Ex. 171 (ECF No. 65-1) ("Cellucci").

S.L.'s clinical presentation was relatively acute, Dr. Huq felt. Tr. at 30. She exhibited more than two of the clinical features of neurologic dysfunction required by Cellucci, including EEG slowing and epileptiform activity as well as altered mental status; seizures not explained by previous seizures; myorhythmia (a movement disorder); and some psychiatric symptoms (i.e., her behavioral and personality changes). *Id.* at 31. Dr. Huq also mentioned that S.L. demonstrated paraclinical evidence of neuroinflammation via CSF inflammatory changes (i.e., T2 flare changes) and brain MRI and brain biopsy features which showed infiltration of T lymphocytes. *Id.* Although there was no autoimmune encephalitis serology identified, there was still reasonable exclusion of alternative causes and etiologies, in Dr. Huq's opinion. *Id.* at 32.

As for the cause of S.L.'s condition, Dr. Huq deemed her receipt of the flu vaccine, in combination with the possible viral infection, as working synergistically to result in autoimmune encephalitis with a clinical presentation as FIRES. Both infections and vaccinations, Dr. Huq opined, can "trigger inflammation and secretion of proinflammatory cytokines and can thus amplify the effects of cytokines on endothelium and blood brain barriers." Tr. at 38, 39; First Huq Rep. at 16. While vaccinations are intended to provoke an immune response, they can also produce other, adverse symptoms, such as fever, headache, or sore throat. *Id*. at 40; First Huq Rep. at 28

(referencing C. Herve et al., *The How's and What's of Vaccine Reactogenicity*, 4 Vaccines 1 (2019), filed as Ex. 58 (ECF No. 34-3) ("Herve"). Relying on Figure Three of Herve, Dr. Huq pointed out the various factors that can impact a vaccine's "reactogenicity," or the body's immediate response to vaccination—some specific to mechanistic or formulaic factors (injection route; needle form or length; adjuvant type; dose); some intrinsic to an individual (i.e., age or gender); and some purely associated with an individuals' experience during and after vaccination. Herve at 5; Tr. at 41.

Dr. Huq opined that S.L.'s high fever when she was vaccinated was "definitely a factor that probably affected her [reactogenicity]," as she was already in a higher state of inflammation, and thus likely resulting in more severe, faster inflammatory and adaptive immune responses. Tr. at 42, 43. Inflammation can be a trigger to autoimmunity. *Id*. at 54. Activation of the innate immune cells produce inflammation, and in turn activate the adaptive immune cells (i.e., T lymphocytes, B lymphocytes, or other antigen-presenting cells), leading to adaptive immune responses. *Id.* at 55. Moreover, autoimmunity in and of itself will "of course" trigger additional inflammation, and thus "the relationship [between] inflammation and autoimmunity is very intertwined." *Id.*

Dr. Huq then discussed specifically the role of inflammation in the pathogenesis of seizure disorders, referencing a chart from one item of literature. Tr. at 51–54; *see also* A. Vezzani et al., *The Role of Inflammation in Epilepsy*, 1 Nat. Rev. Neurology 1, 23 (2011), filed as Ex. 131 (ECF No. 41-6) ("Vezzani"). Vezzani is a review article discussing other medical/scientific studies that evaluate the extent to which "inflammatory mediators" throughout the body (both in the brain and periphery) might contribute to "the origin of individual seizures and the epileptogenic process." Vezzani at 1.

Although Vezzani lists infection as a trigger for inflammation (and thus a potential factor in epileptogenesis), Dr. Huq maintained that vaccinations can be viewed as "analogous in this regard," since both can produce systemic inflammation leading to the secretion of proinflammatory cytokines. Tr. at 51; First Huq Rep. at 32–33; Vezzani at 7–8. Vaccines trigger the production of cytokines (analogously with infections) which in turn activate immune cells, such as neutrophils or macrophages. Tr. at 52; Vezzani at 23 Figure 1. These immune cells will then release "inflammatory mediators" through the blood brain barrier, resulting in a cascade of inflammatory events and a wide array of physiopathological outcomes. Vezzani at 23 Figure 1.

While acknowledging the rarity of autoimmune encephalitis, FIRES, and NORSE, Dr. Huq testified that the "the only logical" explanation for why individuals develop these conditions is a result of "a perfect storm"—specifically, the body's condition at the time of vaccination, plus an individual's immunogenetic makeup and microbiome profile. Tr. at 58. Autoimmune disease processes are clearly, Dr. Huq contended, influenced by both genetic and environmental factors, further increasing a likelihood for encephalitis given the right combination of factors. *Id.* at 57. Genetics often affect autoimmunity in how susceptible or vulnerable an individual is, whereas environmental factors, such as a flu vaccine or an infection, will be the trigger. *Id.*

Accordingly, although S.L. may have possessed a viral infection prior to vaccination, in Dr. Huq's view, that infection only made her "more vulnerable and susceptible" to a disease process that was in this case likely assisted along due to vaccination. In fact, Dr. Huq gave more weight to S.L.'s receipt of the flu vaccine as a trigger for her autoimmune encephalitis than any preexisting infection, noting that "even if [he] assum[ed] that [S.L.] had Adenovirus … the literature of the adenovirus relation with autoimmune encephalitis or FIRES or NORES" is limited. *Id.* at 59–60.

Dr. Huq proposed molecular mimicry as the mechanism by which the flu vaccine likely triggered S.L.'s autoimmune encephalitis (with a clinical presentation of FIRES). Tr. at 61. The flu vaccine has been consistently demonstrated to cause a variety of autoimmune diseases through molecular mimicry, such as Guillain Barré syndrome ("GBS") or narcolepsy. *Id.* at 62; M. Fujimori et al., *A Study of the Association between Seasonal Influenza Vaccines and the increased Risk of Guillain-Barré syndrome using Vaccine Adverse Event Reporting System*, 76 Pharmazie 437 (2021), filed as Ex. 44 (ECF No. 32-9) ("Fujimori") (finding an association between the seasonal influenza vaccines and the incidence of GBS); J. Stowe et al., *Risk of Narcolepsy after AS03 adjuvanted Pandemic A/H1N1 2009 Influenza Vaccine in Adults: A Case-Coverage Study in England*, 39 Sleep 1051 (2016), filed as Ex. 117 (ECF No. 40-2) (finding a significantly increased risk in adult narcolepsy following receipt of the Pandemrix vaccination).[16] FIRES has also been documented after vaccination (although nothing was filed herein specific to the flu vaccine). Tr. at 65–66; K. Bauman et al., *New-Onset Refractory Status Epilepticus after Pfizer-BioNTech COVID-19 Vaccination*, 98 Neurology (2022), filed as Ex. 150 (ECF No. 58-1); R. Kilic et al., *New Onset Refractory Status Epilepticus after BNT162b2 nCoV-19*, 3 J. Clinical Images Med. Case Reps. 1, 2 (2022), filed as Ex. 160 (ECF No. 58-11) (attributing the development of NORSE to receipt of the BNT162b2 nCoV-19 because the patient did not have a pre-vaccination history of epilepsy).

As for timing, Dr. Huq opined that S.L.'s onset of her refractory status epilepsy and autoimmune encephalitis occurred approximately four days post-vaccination and six days post-infection. Tr. at 68. He further stated that "the initial component" sparking her overall condition was likely innate immunity and activation of inflammation—thus, the timing of onset was consistent, at least as seen in cases involving the flu vaccine and GBS. *Id.*; *see also* Fujimori at 441. Additionally, because S.L. was already in a higher inflammatory state due to her high fever and possible viral infection, more rapidly-progressive inflammation was likely, as well as faster activation of the innate and adaptive immune responses. Tr. at 68. Her prior exposure to the flu vaccine in the past also might have increased the speed of response in this case. *Id.* at 69.

[16] I have repeatedly rejected the contention that the flu vaccine (at least in the form administered in the United States) can cause narcolepsy. *See D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 766475 (Fed. Cl. Spec. Mstr. Nov. 28, 2016), *mot. for review den'd, decision aff'd*, 132 Fed. Cl. 421 (2017), *aff'd*, 726 F.App'x 809 (Fed. Cir. 2018).

On cross-examination, Dr. Huq admitted that despite ample testing, no foreign antigen had been identified that would explain the etiology of S.L.'s encephalitis. Tr. at 74. Instead, he relied on the criteria set forth in Cellucci and another article to support his opinion that S.L. developed autoimmune encephalitis. *Id.*; Cellucci at 7, Table 4; F. Graus et al., *Recommended Diagnostic Criteria for Paraneoplastic Neurological Syndromes*, 75 J. Neurology Neurosurgery Psychiatry 1135 (2004), filed as Ex. 48 (ECF No. 33-3) ("Graus"). Antibody serology is not available for most antigens possibly associated with autoimmune encephalitis, and the relied-upon criteria are otherwise generally accepted by the medical community at large in making a diagnosis of autoimmune encephalitis. Tr. at 75.

Dr. Huq also tried to bulwark his proposed mechanism despite the absence of cited medical literature supporting the conclusion that the flu vaccine can result in the production of proinflammatory cytokines that cross the blood-brain barrier into the central nervous system at levels clinically sufficient to cause encephalitis. Tr. at 83, 84. In so doing, Dr. Huq referenced a single article as "substantial evidence" that peripheral inflammation can induce experimental autoimmune encephalomyelitis. *Id.* at 84; S. Blackmore et al., *Influenza Infection Triggers Disease in a Genetic Model of Experimental Autoimmune Encephalomyelitis*, Proc. Natl. Acad. Sci. U. S. A 114: E6107 (2017), filed as Ex. 24 (ECF No. 30-9) (in context of animal model used to simulate multiple sclerosis, peripheral influenza infection shown to be able to cause activation of immune cells in brain).

B. *Respondent's Experts*

1. Dr. Michael Sweeney

Dr. Sweeney, a pediatric neurologist, prepared one written report and testified on behalf of Respondent. Report, dated July 27, 2023, filed as Ex. A (ECF No. 47-1) ("Sweeney Rep."); *See generally* Tr. at 104–49. Dr. Sweeney opined that S.L.'s encephalitis was most likely triggered by an abnormal immune response to an adenovirus infection. Sweeney Rep. at 9.

Dr. Sweeney received his undergraduate degree from Purdue University and his medical degree from the Medical College of Wisconsin. *Curriculum Vitae*, filed as Ex. B (ECF No. 47-16) ("Sweeney CV") at 1; Tr. at 105. Thereafter, he completed residency training in Pediatrics/Child Neurology at the University of Cincinnati and Cincinnati Children's Hospital Medical Center before completing a fellowship in Autoimmune Neurology at the University of Utah. *Id.*; Sweeney Rep. at 1. Dr. Sweeney is an Associate Professor in the Department of Neurology at Norton Children's Hospital at the University of Louisville. *Id.* He is board certified by the American Academy of Neurology with special qualifications in Child Neurology. Tr. at 107; Sweeney Rep. at 1. He specializes in treating children and adults with various autoimmune or inflammatory conditions that affect the nervous system, such as multiple sclerosis, neuromyelitis optica spectrum disorder, anti-MOG related demyelinating disease, and encephalitis. Sweeney Rep. at 1. Dr.

Sweeney has also published several peer-reviewed articles on the topic of neurology. *See* Sweeney CV at 5–6.

Dr. Sweeney began his testimony by first describing encephalitis. He characterized it as an inflammatory condition of the brain, in which patients often present with an acute or subacute onset of progressive neurologic symptoms. Tr. at 109. There are, however, different classifications of encephalitis. Historically, encephalitis has been broken down into two broad categories: infectious (i.e., attributable to an active infection directly impacting the brain) and autoimmune (i.e., driven by an autoantibody, whether detected or not). *Id.* at 109, 110.

Dr. Sweeney disputed Dr. Huq's proposed diagnosis of autoimmune limbic encephalitis. Tr. at 111. Referencing the criteria set forth by Graus, Dr. Sweeney agreed that S.L. met the first and third criteria, as she exhibited a subacute onset of working memory deficit seizures or psychiatric symptoms, and had demonstrated changes on her EEG and CSF pleocytosis present on her second lumbar puncture. *Id.* at 112. But S.L. did not meet the second criteria, because she did not display any temporal lobe abnormalities on her first MRIs. *Id.* at 113. He further critiqued Dr. Huq's reliance on another article purporting to illustrate the difference in neuroimaging studies among autoimmune encephalitis patients, noting that none of the imaging discussed therein was "reminiscent of what [is] described in S.L.'s MRIs." *Id.* at 114; B. Kelley et al., *Autoimmune Encephalitis: Pathophysiology and Imaging Review of an Overlooked Diagnosis*, 38 AJNR Am. J. Neuroradiology 1070 (2017), filed as Ex. 159 (ECF No. 81-1).

Application of the Cellucci criteria, Dr. Sweeney maintained, also did not support the conclusion that S.L. had experienced an antibody-positive kind of autoimmune encephalitis. Tr. at 115. Dr. Sweeney acknowledged that S.L. "met enough" of the clinical and paraclinical criteria to at least put that diagnosis into reasonable contention, but in his view the absence of an identified antibody and the inability to exclude alternative etiologic classifications (like FIRES), instead placed S.L. merely in the category for "possible" autoimmune encephalitis. *Id.* at 116. In general, Dr. Sweeney acknowledged an overlap in clinical signs and symptoms between FIRES and autoimmune encephalitis. *Id.* at 117. But S.L.'s overall clinical picture—including the evidence of her febrile illness preceding the onset of her symptoms, the fact that she went into status epilepticus and became refractory so quickly, her imaging changes over the course of her illness, as well as S.L.'s unfortunate outcome—was more consistent with NORSE and FIRES, according to Dr. Sweney. Tr. at 117–18. (Although Dr. Sweeney further acknowledged that he likely would not have done anything differently at S.L.'s bedside, given the extensive overlap in autoimmune encephalitis and FIRES/NORSE. *Id.*at 119).

As to causation, Dr. Sweeney opined that S.L.'s condition was most likely due to her adenovirus infection, which in turn likely caused her to develop an extremely robust inflammatory response, leading to FIRES. Tr. at 123. Although the record lacked evidence of treater views that an adenovirus infection was causal (and perhaps endorsed the view that this infection was even inconsequential), she clearly had a viral infection before vaccination, and there was *some* treater

acknowledgement that such infection could have triggered her symptoms that lead to her condition. *Id*. at 124–25; Ex. 2 at 7186 (referencing family care conference held on March 4, 2020, in which treaters acknowledged "that even adeno virus 'could have started the fire' in [S.L.'s] brain."). S.L. had a documented febrile illness prior to the onset of her symptoms, and the viral infection responsible for it likely led to a "predisposition" to eliciting a rather robust and dysregulated response, causing her to develop FIRES. Tr. at 126.

2. Dr. Hayley Gans

Dr. Gans, a pediatric infectious disease specialist, prepared one written report and testified on behalf of Respondent. Report, dated July 24, 2023, filed as Ex. C (ECF No. 50-3) ("Gans Rep."); *See generally* Tr. at 149–88. In Dr. Gans' opinion, S.L.'s seizure disorder was the likely result of a predisposing condition in conjunction with a preceding febrile infection. Gans Rep. at 16.

Dr. Gans is a Clinical Professor in the Department of Pediatrics and Division of Pediatric Infectious Disease at Stanford University. Curriculum Vitae, filed as Ex. D (ECF No. 47-18) ("Gans CV") at 1; Tr. at 150. She received her medical degree from SUNY at Syracuse and completed a residency and fellowship at Stanford University. *Id.* In her clinical capacity, Dr. Gans has cared for hundreds of infants and children with infections and has been involved with disease prevention through mitigation and immunizations. Gans Rep. at 1. She currently conducts immunology research in the field of infectious diseases and studies vaccine responses in several populations including normal hosts, HIV-infected children, premature children, children who have received organ transplants and children with autoimmune diseases. *Id.* Dr. Gans serves on several regulatory boards overseeing the safety of vaccines and is involved with case adjudication for vaccine studies. She is board certified in both Pediatrics and Pediatric Infectious Diseases. Gans CV at 2.

Dr. Gans first discussed what she considered the most appropriate diagnosis for S.L.'s condition. Tr. at 155. She agreed that S.L. clearly had a refractory seizure disorder which followed an acute febrile illness. *Id.* However, unlike Dr. Huq, Dr. Gans opined that FIRES was not a "catch-all term" for autoimmune encephalitis, but rather has its own pathophysiology. *Id.* at 156. To bulwark this assertion, Dr. Gans provided a brief explanation about the distinction between autoimmune conditions and *autoinflammatory* conditions—stating that the latter "are a group of disorders that actually reflect dysregulation in the innate immune system," whereas autoimmunity conditions involve self-attack driven by an overactive *adaptive* immune system. *Id.* at 157; *see also* Gans Rep. at 14; N. Gaspard et al., *New-Onset Refractory Status Epilepticus (NORSE) and Febrile Infection-Related Epilepsy Syndrome (FIRES): State of the Art and Perspectives*, 4 Epilepsia 1956 (2018), filed as Ex. C-5 (ECF No. 51-5) ("Gaspard") (suggesting a role for a postinfectious cytokine-mediated mechanism).

The "exact mechanism for FIRES," Dr. Gans emphasized, likely involves the pathologic

function of proinflammatory cytokines. Tr. at 159. In individuals with FIRES, "these proinflammatory cytokines that are produced from [the] innate immune system are dysregulated," and are thus able to incite an inflammatory process within the central nervous system. *Id.* at 160. But in her view, an adenovirus infection was more likely to cause a cytokine response capable of triggering an inflammatory response, as in S.L.'s case, than a flu vaccine. *Id.* Adenovirus, Dr. Gans contended, is "one of the most inflammatory viruses that there are"—whereas a wild flu infection has reduced immunogenic capacity, and a flu vaccine even less so. *Id.* at 161. She thus criticized applying studies that involve cytokine responses to a wild flu infection to the context of receipt of a flu vaccine, which she deemed "a fairly weak vaccine compared to other vaccines," and thus "there [is] really no comparison of the immunity between the natural disease and vaccines for influenza." *Id.* at 163.

Dr. Gans then responded to Dr. Huq's downplaying of S.L.'s positive adenovirus testing results, and his reliance on medical literature suggesting that adenovirus can be detected by PCR[17] testing (but was not here). Tr. at 163. She noted that for all viruses, some individuals can be infected but present asymptomatically, whereas others will develop a severe disease, and thus a spectrum exists for what clinical symptoms can be detected at any given time. *Id.* at 165. The fact that adenovirus was only detected in S.L.'s respiratory tract was significant to Dr. Gans, given that the medical records indicated S.L. was not suffering from an acute viral encephalitis, and therefore it would be unlikely that this pathogen would have shown up within her central nervous system. *Id.* And because S.L. presented as an immunocompetent individual, her body naturally responded to the virus, meaning it likely presented with symptoms consistent with a respiratory illness, as here. *Id.*

Dr. Huq otherwise did not persuasively link the flu vaccine to FIRES, in the view of Dr. Gans. She denied the existence of medical literature supporting the proposition that proteins in the flu vaccine can cross-react with neural tissues leading to FIRES. Tr. at 168. Medical science presently understands FIRES to be propagated by an aberrant innate immune response, and thus it would not also be mediated by an adaptive immune response involving autoantibodies. *Id.* In addition, because the flu vaccine is an inactivated, immunogenically weak form of vaccine, the likelihood that it could produce an acute inflammatory response resulting in substantial neurological injury via molecular mimicry was unlikely. *Id.* at 169. The flu vaccine in fact is given annually to millions of individuals and is under very strict safety surveillance, resulting in studies that have demonstrated no scientifically significant pathophysiologic support for adverse neurologic outcomes. Tr. at 169–70.

[17] "Polymerase Chain Reaction" is a "laboratory technique for rapidly producing (amplifying) millions to billions of copies of a specific segment of DNA, which can then be studied in greater detail. PCR involves using short synthetic DNA fragments called primers to select a segment of the genome to be amplified, and then multiple rounds of DNA synthesis to amplify that segment." *Polymerase Chain Reaction (PCR)*, National Human Genome Research Institute, https://www.genome.gov/genetics-glossary/Polymerase-Chain-Reaction-PCR (last visited Aug. 29, 2025).

Overall, because of S.L.'s febrile infection, Dr. Gans opined that it was unnecessary to even invoke the flu vaccine as causal in S.L.'s case. The clinical evidence of S.L.'s febrile infection established that she was already "clearly" in an inflamed state, and thus a preexisting infection was the most likely cause for S.L.'s condition. *Id.* at 170–71. On cross-examination, however, Dr. Gans agreed that S.L.'s presenting fever was evidence of an existing "inflammatory state." *Id.* at 177. And while she denied that the flu vaccine would necessarily "augment" that state (by increasing the amount of inflammation), she acknowledged that administration of a vaccine at that time may not have been the treatment practice choice she would have made. *Id.* at 179, 181.

III. Procedural History

This case was initiated in December 2020, and (after the process for evaluating the sufficiency of document filings in the case was completed) it was assigned to my own docket in February 2022. *See* Docket Entry, dated Feb. 18, 2022 (ECF No. 23). Respondent filed his Rule 4(c) Report contesting Petitioner's right to compensation on July 15, 2022. *See* Report, dated July 15, 2022 (ECF No. 26). Thereafter, the parties engaged in a round of expert reports, with the final report from Dr. Huq filed on December 15, 2023. The parties submitted pre-hearing submissions, and a one-day entitlement hearing occurred on October 7, 2024. When the filing of post-trial briefs by both sides was concluded, the matter became ripe for resolution. *See* Petitioner's Post-Hearing Submission, dated January 24, 2025 (ECF No. 89) ("Br."); Respondent's Post-Hearing Brief, dated January 24, 2025 (ECF No. 88) ("Opp.").

IV. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[18] Although there is a Table claim for encephalitis after receipt of the flu vaccine, Petitioner has only plead a causation-in-fact claim.

[18] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. App'x. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. V. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(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 proximate temporal relationship between vaccination and injury."

Each *Althen* prong requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Distinguishing between "preponderant evidence" and "medical certainty" is important because special masters must take care not to impose an evidentiary burden that is too high. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed.Cir.1991) ("The standard of proof required by the [Vaccine] Act is simple preponderance of evidence; not scientific

certainty.... [I]t is not plaintiff's burden to disprove every possible ground of causation suggested by defendant nor must the findings of the court meet the standards of the laboratorian.") (citations and internal quotation marks omitted).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Cerrone v. Sec'y of Health & Hum. Servs.*, No. 2024-1281, 2025 WL 2110246 (Fed. Cir. July 29, 2025); *Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Demore v. Sec'y of Health & Hum. Servs.*, No. 20-1265V, 2024 WL 4542934 (Fed. Cl. Spec. Mstr. Sept. 26, 2024), *aff'd*, No. 20-1265V, 2025 WL 868902, at *4 (Fed. Cl. Mar. 20, 2025) (rejecting the argument that a petitioner's burden is to prove that a causation theory is *plausible* and instead requiring petitioner to prove the theory by a preponderance of the evidence) (emphasis added). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("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'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the

opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *De Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is20edicaly acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. Den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11I(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95

Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras*, 993 F.2d at 1525 ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and

compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

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

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

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88

Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particul'r expert's credibility, is part'of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[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") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. FIRES Best Categorizes S.L.'s Clinical Presentation—But This Does Not Rule Out an Autoimmune/Vaccine-Associated Pathogenesis

Petitioner via Dr. Huq has proposed that while S.L.'s clinical *presentation* could be consistent *both* with FIRES and NORSE, in either case her illness was ultimately attributable to an autoimmune form of encephalitis. Br. at 54. In effect, to Dr. Huq the FIRES/NORSE characterizations of S.L.'s clinical features are far less significant than whether S.L.'s injury had some autoimmune derivation (which in turn opens the door to a finding that the flu vaccine's impact on the immune system could have caused it). Petitioner ultimately contends that S.L. likely meets the definitions for "probable antibody-negative pediatric autoimmune encephalitis" as defined by Cellucci. Br. at 56; Tr. at 30–33.

Respondent, by contrast, favors FIRES almost as an outright diagnosis, adding that FIRES is best understood as *autoinflammatory,* and thus can be propagated without autoantibodies. Opp. at 18–19. For Respondent's experts, FIRES is not simply a clinical catch-all description of S.L.'s presentation, but subsumes within it notions about etiology as well. But Dr. Sweeney allowed that it was reasonable for treaters to consider an autoimmune *cause* for S.L.'s seizure activity, although he contended that the medical evidence only supported a "possible" autoimmune encephalitis, under the applicable diagnostic criteria set forth in articles like Cellucci or Graus. Opp. at 20.

Even though the parties appear to disagree as to the proper diagnosis for S.L.'s illness, there is considerable overlap in their respective positions. Literature establishes that FIRES is a sub-set of NORSE—but that *both* terms are clinical descriptors rather than diagnoses. *See, e.g.*, L. Hirsch et al., *Proposed Consensus Definitions for New-Onset Refractory Status Epilepticus (NORSE), Febrile Infection-Related Epilepsy Syndrome (FIRES), and Related Conditions*, 59 Epilepsia 4:739 (2018), filed as Ex. C Tab 6 (ECF No. 51-6) ("Hirsch"), at 740. More significantly for present purposes, FIRES *could* have an autoimmune etiology—even though it may be more likely propagated by the effects of uncontrolled cytokines than damage from cross-reactive autoantibodies. Hirsch at 741–42; Gaspard at 748.

Based on the record and the party's arguments, I deem FIRES the most reasonable clinical descriptive explanation for S.L.'s presentation and course. But I do not also find that her FIRES *could not* be autoimmune in nature, or vaccine-caused (at least partially), even if its pathology depends more on inflammation, the instigation of which is not necessarily dependent on an autoimmune reaction. Indeed, FIRES could occur without an initial autoimmune spark—although this does not mean it never would begin in this manner. And here, there is indisputable evidence that some unidentified *prior* infectious process was substantially responsible for S.L.'s injury. This leaves for resolution the question of whether it has been shown the flu vaccine could *also* have been a substantial factor in her disease process.

II. Petitioner's Claim Has Been Preponderantly Established

This case presents a very close call (albeit more on the resolution of the first than second *Althen* prong). But its extreme facts, and tragic outcome, justify a finding in Petitioner's favor. Indeed, this matter epitomizes the kind of case where such an outcome is consistent with Program goals. *Roberts v. Sec'y of Health & Hum. Servs.*, No. 09-427V, 2013 WL 5314698, at *10 (Fed. Cl. Spec. Mstr. Aug. 29, 2013) (noting petitioners are afforded the benefit of close calls in the Vaccine Program). Although the sad circumstances *alone* do not justify a finding of entitlement, the facts of this case make it exceedingly difficult to conclude that the vaccine likely did *not* play some role in S.L.'s disease course—enough to find causation is met, despite many reasoned doubts about the overall strength of Petitioner's showing. I address below the *Althen* prongs in order of their significance to my decision.

A. *Prong Two*

There is sufficient evidence in this record to find that the flu vaccine likely was enough of a factor in S.L.'s disease course to deem it to have contributed to her FIRES and subsequent death. In so finding, I give significant weight to contemporaneous treater views. Although no possibly-causal autoantibody was ever identified (and certainly the flu vaccine was not *itself* identified as likely causal), S.L.'s treaters repeatedly endorsed an autoimmune explanation for S.L.'s course, even as they evaluated alternative explanations. *See, e.g.*, Ex. 2 at 2106, 8235, 8386–8390, 8410. And Petitioner has shown that the diagnostic criteria for an autoimmune form of encephalitis are more likely than not met (even if I find—which I do—that the flu vaccine has not been shown to be *primarily* causal of S.L.'s FIRES). Br. at 56–60.

In addition, the alternative explanation favored by Respondent's experts (who obviously were evaluating the record after-the-fact) of an adenovirus infection is intriguing but far less-well supported by the evidence. The fact of the infection was established by different aspects of the record, but contemporaneous treaters never placed a great deal of faith in its explanatory value. Ex. 2 at 8354 (indicating that "the [infectious disease] team is comfortable starting steroids and that the adeno+ nasal swab *does not explain* her refractory status epilepticus") (emphasis added), 8361 (documenting exchange between treaters and S.L.'s mother that "the respiratory virus is very unlikely to have caused this abrupt onset of status, especially at [S.L.'s] age"). Thus, treater thinking on S.L.'s condition was inconsistent with this alternative explanation for S.L.'s course—and therefore this is not a matter where probative evidence undermines the conclusion that the vaccine was in some part causal of injury.

Importantly, this is not a case in which the flu vaccine *by itself* has been shown to have caused the relevant injury. FIRES by definition involves a fever—but S.L. was unquestionably suffering from a preexisting infectious process that made her ill enough (and febrile at the time of vaccination—not after) to require emergency treatment. *See* Ex. 2 at 30–32. The flu vaccine clearly did not cause that initial fever. And there is little in the way of evidence that receipt of the flu

vaccine sparked any immediate reaction after administration (although as discussed below the timeframe for onset post-vaccination was medically acceptable nonetheless). But the Program has in prior cases recognized the capacity of vaccines to synergistically encourage disease processes in some limited and rare circumstances. *See, e.g.*, *Lehrman v. Sec'y of Health & Hum. Servs.*, No. 13-901V, 2018 WL 1788477, at *16–19 (Fed. Cl. Spec. Mstr. Mar. 19, 2018) (finding a 24-hour onset post-vaccination was medically acceptable because petitioner's expert persuasively explained how a preceding upper respiratory infection acted synergistically with the flu vaccine, leading to a rapid onset of GBS). Here, the medical record allows for the conclusion that this likely occurred here.

B. *Prong One*

The evidence in favor of the conclusion that the flu vaccine "can cause," at least in part, a FIRES presentation is mixed, and far from robust. But I can find in Petitioner's favor on this *Althen* prong even if other factors were relevant to the cause of the injury, so long as the vaccine also likely was a "substantial factor" in the illness. *Shyface*, 165 F.3d at 1350, 1352–53. The circumstances of this case—receipt of the flu vaccine in the midst of a preexisting infection that prompted a fever—allow for that conclusion.

Here, Petitioner has offered enough linked contentions to knit together a theory sufficient to cross the preponderant line—albeit *barely*. Through the testimony of Dr. Huq and literature filed in the matter, Petitioner has shown that FIRES could have an autoimmune origin. Firs Huq Rep. at 21; *see also* A. Yeshokumar & C. Pardo, *Autoimmune Epilepsies*, 24 Seminars Pediatric Neurology 161, 166 (2017), filed as Ex. 148 (ECF No. 43-3); M. Basso et al., *Connections between Febrile Infection-Related Epilepsy Syndrome and Autoimmune Encephalitis. A Case Report of a Child with New Anti-neuronal Antibodies*, 10 Frontier Pediatrics 1 (2022), filed as Ex. 20 (ECF No. 30-5).

I credit the contentions of Dr. Gans that FIRES may *eventually* be determined to be primarily, if not exclusively, driven by inflammation attributable to an aberrant innate response (in which out-of-control cytokine production promotes excessive inflammation). And when/if medical science reaches that conclusion, it may be far *less* likely that the flu vaccine could meaningfully contribute to such a process (especially since, as Dr. Gans credibly opined, the version of flu vaccine commonly received—an inactivated preparation including several different viral strains, with no adjuvant or live reactive components—is not known to be especially immunogenic). But at the moment, application of the FIRES classification to a patient's clinical presentation does not rule out an autoimmune element or cause. And as many other Program cases have recognized, the flu vaccine has been understood to be capable of triggering an autoimmune form of epilepsy. *Ginn v. Sec'y of Health & Hum. Servs.*, No. 16-1466V, 2021 WL 1558342 (Fed. Cl. Spec. Mstr. Mar. 26, 2021) (finding that five vaccines, including the flu vaccine, triggered a febrile seizure in four-year-old that contributed/led to the development of epilepsy).

Especially important herein is the fact, discussed above, that S.L. received the flu vaccine at the time she presented with a febrile illness of a likely infectious origin. Dr. Huq was unable to establish the infection had nothing to do with S.L.'s injury, and did not try. First Huq Rep. at 16, 33, 35. But Petitioner offered reliable evidence suggesting that medical science recognizes the possible dangers of receipt of a vaccine when an individual is already sick, as was the case with S.L. Br. at 85 (citing Advisory Committee on Immunization Practices (ACIP), General Best Practice Guidelines for Immunization). Respondent's experts for their part *acknowledged* the risk of vaccination for such an ill patient, and that they themselves admitted that they might have opted not to administer a vaccine at time S.L. was first taken for urgent care. Tr. at 141–42, 43, 181.

Although Respondent's experts did vouch for the *general* safety of the flu vaccine, that kind of consideration does not bear on the outcome of a vaccine injury case.[19] And Respondent could not rebut the possibility of a synergistic reaction between S.L.'s existing infectious illness and vaccine receipt. Dr. Gans offered some items of (now-old) literature about the risks posed by administration of *other* vaccines to ill recipients, but she did not also show the same was true in this context. *See, e.g.*, M. Harris, *The Safety of Measles Vaccine in Severe Illness*, SA Medical Journal (1979), filed as Ex. C Tab 25 (ECF No. 53-6); N. Halsey, M.D., et al., *Response to Measles Vaccine in Haitian Infants 6 to 12 Months Old*, 313 N. Eng. J. Med. 544, 546, 548 (1985), filed as Ex. C Tab 24 (ECF No. 53-5) (involving the measles vaccine). And literature supporting the conclusion that a wild flu infection poses greater risks than vaccination does not mean that the combination of a vaccine *and* illness is not potentially problematic. Gans Rep. at 13.

I thus find on the basis of this record that it has been preponderantly shown that *in the specific, limited context of an existing febrile illness,* receipt of the flu vaccine could interact sufficiently[20] with an existing inflammatory state to result in FIRES, by causing additional stress from an immune response that would prove enough to cause the kind of seizure injury S.L. so tragically experienced. In so determining, I do *not* find that the flu vaccine has been shown *alone* to be capable of resulting in a FIRES-like presentation. I certainly did not find Dr. Huq more persuasive overall than Respondent's experts, and his contentions did not rise to a level of preponderance *absent* the existing febrile infection. If this theory (as embraced by Dr. Huq, and with the numerous items of literature filed by Petitioner) were applied to a case in which a child simply developed refractory epilepsy after receipt of the flu vaccine, I would find causation *had not* been preponderantly established (absent evidence the vaccination likely promoted a reaction, like a fever). I also deem the testimony provided by Respondent's experts about the likely

[19] Indeed, one of the Vaccine Program's unacknowledged foundations is the concept that vaccines are safe for the majority of recipients—and that vaccine-associated injuries are quite rare—allowing the Program to be a tribunal in which otherwise-uncommon injuries may be litigated, thereby serving the public goal of encouraging vaccination for the majority, who are not otherwise at risk.

[20] Although molecular mimicry was trotted out by Dr. Huq to explain how vaccination could encourage disease process herein, I do not find that mechanism was all that well substantiated for a seizure disorder presenting as FIRES—but since litigants need never prove a mechanism to prevail, this omission in his opinion does not weigh significantly against this prong one finding.

pathogenesis of FIRES (which might not be autoimmune at all) to have scientific merit, although the matter remains incompletely evaluated. I am, in the end, *far from certain* that S.L.'s ultimate illness and death had anything to do with her receipt of a flu vaccine. But the evidentiary standard utilized in the Program is mere preponderance, not certainty. And so long as the scales tip, even slightly, in a claimant's favor, petitioners meet their burden, no matter how much reliable evidence has been offered against the contention.

C. *Prong Three*

The medical record clearly demonstrates that S.L. first displayed the kinds of neurologic symptoms likely reflective of her ultimate FIRES presentation within three days of vaccination—but also about a week after her febrile illness had begun. Ex. 2 at 8539, 8544. As noted, I am able to conclude in this case the flu vaccine likely played some substantial role in S.L.'s course, although I cannot say for sure what that role was. Consistent with FIRES (and as elucidated by Dr. Gans), the vaccine could have promoted some cytokine production, within a preexisting context that was already primed to overreact to stimulation of proinflammatory cytokines. First Huq Rep. at 17.

The evidence in this case thus supports the medical acceptability of timing. No matter how interaction between vaccination and S.L.'s preexisting infectious febrile illness occurred, Petitioner's onset happened within a reasonable post-vaccination timeframe. Three days was long enough either for antibodies produced in reaction to the vaccine to begin to show symptoms, or (and more likely, in light of what science seems to be viewing as the pathogenesis of FIRES) for the upregulation of proinflammatory cytokines that the flu vaccine is known to at least transiently cause to peak[21]—and in an already demonstrably excessively-inflammatory environment.

CONCLUSION

The facts of this case are devastating—involving the death of a wholly innocent child. Of course, severity of injury alone is not an occasion for a special master to "put his thumb on the scale" and decide the case in the claimant's favor, no matter the Program's emphasis on generosity and fairness to the petitioner. The Program does not establish an "insurance policy" against post-vaccination adverse events.

But the Program's policy goals have some bearing in how the evidence is weighed in close cases. This matter is the epitome of such a close case. Petitioner's showing *would not have been enough to carry the day* had S.L. not also been suffering from a concurrent severe illness. And Respondent offered not just good faith objections, but numerous items of persuasive literature to support his defense. As more is learned about FIRES and its interaction with factors like

[21] *See Crawford v. Sec'y of Heath & Hum. Servs.*, No. 18-198V, 2023 WL 5815719, at *18 (Fed. Cl. Spec. Mstr. July 27, 2023) (relying on literature indicating that pro-inflammatory cytokines post-flu vaccination remain elevated for a period of two to three days).

vaccination, I cannot say that the outcome of a factually-similar case would be the same. But here, Petitioner has met the burden of proof.

A damages order will follow issuance of this Ruling.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master