In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 16-1494V

(to be published)

EBONIE WEAVER *parent of T. M. a minor*,	*	Filed: September 23, 2022
Petitioner,	*	
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Edward Kraus, Kraus Law Group, LLC, Chicago, IL, for Petitioner.

Megan R Murphy, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION DENYING ENTITLEMENT[1]

On November 14, 2016, Ebonie Weaver, on behalf of her minor daughter, T.M., filed a Petition under the National Vaccine Injury Compensation Program (the "Vaccine Program"),[2] alleging that as a result of receiving several vaccines on December 10, 2013, T.M. experienced a seizure disorder and a significant worsening of her preexisting developmental delays. Petition

[1] This Decision will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet**. As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Ruling's inclusion of certain kinds of confidential information. Specifically, 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 entire Decision will be available to the public in its current form. *Id.*

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended at 42 U.S.C. §§ 300aa-10–34 (2012)) (hereinafter "Vaccine Act" or "the Act"). All subsequent references to sections of the Vaccine Act shall be to the pertinent subparagraph of 42 U.S.C. § 300aa.

(ECF No. 1) ("Pet.") at 1–2, 40. An entitlement hearing in the matter was held on February 9–10, 2022.

Having reviewed the record, all expert reports, and testimony provided at trial, I hereby deny an entitlement award. Petitioner has offered sufficient reliable evidence for me to conclude that simply by producing a fever attributable to the oft-experience vaccination "malaise," the vaccines T.M. received could be, and were, likely responsible for her *initial* febrile seizure in December 2013. Febrile seizures can lead to a more all-encompassing seizure disorder—but the record *in this case* does not support the conclusion that T.M.'s subsequent seizures were likely caused by the first, given an absence of evidence that her brain was likely damaged by the first febrile seizure. Petitioner otherwise has not established that T.M.'s preexisting developmental problems were aggravated by the vaccine-caused febrile seizure.

I. Factual Background

Pre-Vaccination History

T.M. was born on March 29, 2013, with Apgar scores of 9/9. Ex. 6 at 32–34. She received a vaccine at this time and was released home two days later. Ex. 3 at 3; Ex. 6 at 4. T.M. had her five-day well-child visit on April 3, 2013, at the Aunt Martha's Health Center ("AMHC"), and was deemed normal. Ex. 4 at 11.

On July 15, 2013, T.M. received her four-month vaccinations at AMHC—the DTaP, Hib, a second dose of hepatitis B, inactivated poliovirus vaccine, and pneumococcal vaccines. Ex. 3 at 3; Ex. 4 at 8. The physician performing the associated exam observed at this time (as memorialized under the record subheading "Concerns") that T.M.'s eyes were different sizes. Ex. 4 at 8. T.M. was also at this exam assessed for developmental delays. Based on an "Ages and Stages Questionnaire" (ASQ), Petitioner reported to the examining treater that T.M. could not push up on her elbows, her movements were not symmetrical, and she did not roll and reach for objects. *Id.* It was also noted, however, that it might be premature to conclude anything about T.M.'s developmental status, since T.M. was not quite four months old. *Id.*

T.M. had another well-child visit on December 6, 2013, at which time Ms. Weaver requested administration of six-month vaccinations. Ex. 8 at 5. However, the previously-received round of vaccines could not be verified, so Petitioner was told to return another time. *Id.* At this appointment Petitioner put down on the intake form that there was a smoker in the home. *Id.* at 1. It was also noted that T.M. was not meeting certain developmental milestones; thus, she did not sit alone, roll from front to back, pass a toy from hand to hand, imitate vowel sounds, make constant sounds, or say "mama" or "dada." *Id.*

Upon returning to AMHC a few days later (December 10, 2013), T.M. received additional vaccines—her second DTaP dose, second Hib, third hepatitis B, second IPV, first rotavirus, and second pneumococcal dose. Ex. 3 at 3. Ms. Weaver now reported that T.M. was "babbling—little[;] smiles at mom[;] mom said she was rolling up but now she doesn't do much[;] doesn't sit up." Ex. 8 at 4. She further stated that T.M. "was doing everything normal until 4 months now she is not doing much." *Id.* Based upon this reported history, T.M. was referred to a developmental clinic. *Id.* The record thus establishes the existence of developmental concerns before, or at least at the time of, the vaccinations at issue in this case.

First Post-Vaccination Febrile Seizure

The next day—December 11, 2013—T.M. was taken by ambulance to the emergency room ("ER") at Franciscan Health in Chicago Heights, Illinois, for treatment of a 25-minute seizure that had occurred prior to arrival, and which thereafter manifested intermittently for 10 additional minutes. Ex. 25 at 5–11. Ms. Weaver reported that she had heard T.M. grunt in her crib that morning, and had observed her shaking with clenched hands and pulsating limbs. Ex. 16 at 2. Upon admittance, the ER physician took specific note of the fact that T.M. had received several vaccines the day before, and was febrile upon arrival, although Petitioner was uncertain if she had experienced a fever earlier in the day. Ex. 25 at 8. T.M.'s father reported that her tongue was swollen, and she was drooling more than usual. *Id.*

On exam, T.M. was minimally responsive, and displayed myoclonus-like twitching in all extremities. Ex. 25 at 9. She had to be intubated due to periods of apnea, and initial treater impressions were that T.M. was experiencing seizure, unspecified fever, and respiratory distress. *Id.* at 11. Testing was conducted, including a flu panel, brain CT and chest x ray, but all yielded negative results, although T.M.'s bloodwork did show some abnormalities, such as a high white blood cell count ("WBC") of 18.7 × 10^9/L.[3] *Id.* at 10, 26, 30. T.M. was given antibiotics and transferred to Advocate Children's Hospital in Oak Lawn, Illinois, for treatment in its pediatric intensive care unit ("PICU"). *Id.* at 68.

Upon admittance, T.M.'s parents informed treaters that they had found her shaking and grunting around 2:00 a.m., leading them to take her to the ER, where she was given Tylenol for her fever (101.1 degrees), which in turn had helped to end the shaking. Ex. 7 at 59–60; 84. They also reported her receipt of six-month vaccines the day prior, as well as her referral to early intervention ("EI") due to suspected developmental delay. *Id.* at 84; Ex. 8 at 4. An EEG[4] was

[3] While this medical record does not specify what a normal range would be, a typical white blood cell count for children will generally range between 5 to 10 per 10^9/L. Leukemia & Lymphoma Society, *Understanding Blood Counts*, https://www.lls.org/treatment/lab-and-imaging-tests/understanding-blood-counts (last visited Sept. 23, 2022).

[4] An electroencephalogram, or EEG, is a "diagnostic test that measures the currents emanating from nerve cells in the brain. The fluctuations in current are shown in waves, which correlate with different neurologic conditions." *See*

performed with negative/normal results, and T.M. was extubated with no further seizure activity while at the PICU, and remained afebrile. Ex. 7 at 68, 83, 88. T.M. tested negative for a staph infection but was nevertheless treated with an antibiotic. *Id.* at 83, 93. One treater opined that T.M. had experienced a complex febrile seizure. *Id.* at 83. A flu vaccine was ordered, although it is unclear from the records whether it was received. *Id.* at 85. T.M. was discharged on December 12, 2013, with a diagnosis of febrile seizure. *Id.* at 88.

Four days later, T.M. returned to AMHC for a follow-up. Ex. 4 at 1. By this time, Ms. Weaver had prepared and submitted a VAERS report[5] implicating the vaccines received earlier that month in T.M.'s febrile seizure. *Id.* at 7. T.M. was now doing well, but was referred to a neurologist for an outpatient EEG study. *Id.* Two days later, on December 18, 2013, T.M. returned to the ER with a history of three episodes of vomiting, a small wet stool, and no fever the previous day. Ex. 7 at 5–16, 37. T.M.'s diagnosis was for vomiting, and she was discharged with a prescription for Zofran. *Id.* at 7. No seizures were reported at this time.

Treatment in 2014 and Monitoring of Developmental Problems

T.M. had a follow-up at AMHC on January 14, 2014. Ex. 4 at 5. The treating physician noted that T.M. had been intubated after a febrile seizure, specifically identifying as causal "DTaP induced versus febrile seizure." *Id.* Several months passed, however, before T.M.'s second seizure manifested.

On March 18, 2014, T.M. was taken back to the ER for evaluation of a second febrile seizure and upper respiratory infection symptoms. Ex. 7 at 221–22. The consulting physician was informed that T.M. "was not acting herself," and at daycare she had been "[t]ired and w[]oozy," having a temperature of 99.1 degrees. *Id.* at 241. T.M. was also reported to have displayed full body, tonic-clonic movements lasting less than ten minutes, with a post-ictal state lasting one to two hours. *Id.* Prior to this event T.M. had displayed a runny nose, congestion and cough for a week, with one episode of diarrhea. *Id.* at 242. One physician deemed these symptoms to constitute the likely etiology of T.M.'s subsequent fever. *Id.* at 244. She was also noted to have low truncal tone, but with good head control, and no focal neurological deficits were observed during physical exam. *Id.* at 241. Bloodwork resulted in normal findings. *Id.* at 240.

Caredio v. Sec'y of Health & Hum. Servs., No. 17-0079V, 2021 WL 4100294, at *2 (Fed. Cl. Spec. Mstr. July 30, 2021), *mot. for review den'd*, __ Fed. Cl. __, 2021 WL 6058835 (2021).

[5] The Vaccine Adverse Event Reporting System ("VAERS") is a national warning system designed to detect safety problems in U.S.-licensed vaccines. *See About VAERS*, VAERS, https://vaers hhs.gov/about html (last visited Sept. 23, 2022). It is managed by both the CDC and the FDA. VAERS monitors and analyzes reports of vaccine related injuries and side effects from both healthcare professionals and individuals. *See generally Carda v. Sec'y of Health & Hum. Servs.*, No. 14-191V, 2017 WL 6887368, at *6 (Fed. Cl. Spec. Mstr. Nov. 16, 2017).

T.M. remained hospitalized overnight in order to monitor her condition after the observed seizure activity, although treaters noted that at the time of her admission that she was "back to baseline" already. Ex. 7 at 239, 240–45. That night, T.M. had a fever that resolved with Tylenol, and displayed no further seizures. *Id.* at 240. The attending physician also discussed T.M.'s developmental delays with Petitioner and her grandmother, referring her to EI. *Id.* at 239–40. T.M. was discharged with febrile seizure and told to alternate between Motrin and Tylenol. *Id.* at 241.

The following month, T.M. had a well-child visit at AMHC on April 1, 2014. Ex. 4 at 4. Her diagnoses at that time included developmental delay and febrile seizure, she was again referred to EI plus neurology. *Id.* Her developmental problems continued to be of concern going forward—although the record does not at this time reveal an increased tempo in reported issues or a heightening of concern beyond what had previously been reported or discussed with prior treaters. *See, e.g.*, Ex. 3 at 85–88 (April 8, 2014, pediatric well-check visit).

On May 1, 2014, T.M. was brought to the ER by ambulance after a purported third seizure (reported by a daycare educator). Ex. 1 at 1. T.M. had begun to "shake, foam at the mouth, and her eyes rolled back in her head while she was holding her." *Id.* at 2. T.M. now had a fever of 102.2 degrees, but was awake, alert, and crying but consolable. *Id.* at 4. She was discharged home the same day with impression of febrile seizure. *Id.* at 5–6.

The next day, T.M. was evaluated by the Illinois Bureau of Early Intervention. Ex. 3 at 34–47; Ex. 26 at 1. Petitioner reported that T.M. "had a seizure at 8 months due to shots and at 10 months due to a cold," with subsequent additional seizures "every two months." Ex. 3 at 34. She also stated a family history of some developmental problems, having a brother with autism and a nephew with ADHD. *Id.* T.M.'s results showed a 54% cognitive delay, 54% receptive language delay, 76% expressive language delay, 58% gross motor delay, 54% fine motor delay, 31% social/emotional delay, and 31% self-help delay. *Id.* at 47. She was recommended for therapies in cognitive development, physical development, and language/speech. *Id.* at 43.

T.M. was subsequently taken to a pediatric neurologist, Dr. Lubov Romantseva, on May 8, 2014, for evaluation of her seizures and developmental delays. Ex. 2 at 10. The history provided at this examination reported that T.M.'s seizures began at "9 months of age, (4 months prior to presentation) with seminology of eyes locking, body stiffening, cyanotic lips, and tonic clonic activity in all extremities lasting 15 to 20 min[utes] in duration with no focal Todd's Phenomenon[6] afterwards, but a several hour period where she was not back to her normal self." *Id.* at 15. It was also reported that about a few times a day T.M. would turn her head to one side with her eyes shifting in the other direction, and that she would then become unresponsive. *Id.* Other

[6] "Todd's Phenomenon" (also referred to as Todd paralysis) is defined as a "hemiparesis or monoparesis lasting for a few minutes or hours, or occasionally for several days, after an epileptic seizure." *Todd paralysis,* Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=96185&searchterm=Todd+paralysis (last visited Sept. 23, 2022).

developmental plateaus and regressions in specific behaviors were also discussed. *Id.* Dr. Romantseva noted that T.M. had displayed normal eye and hearing exams in the past. *Id.* A repeat EEG was ordered, lab work for inborn errors of metabolism, and continued EI therapies, plus a prescription of low dose Keppra (an anti-epileptic drug). *Id.* at 17. T.M.'s diagnoses were epileptic seizures, febrile seizures, staring spell, and developmental delay. *Id.* at 10.

A week later, T.M. had a well-child check on May 13, 2014. Ex. 3 at 76–80. In review of developmental milestones, it was reported that T.M. was able to do a precise pincer grasp, bang blocks together, look for dropped or hidden items, feed herself, and wave goodbye. *Id.* at 77–78. She was still showing gross motor delays, however, and some hesitancy in obtaining the full range of six-month vaccines was stated. *Id.* at 79. Nevertheless, T.M. at this time did receive her first doses of several vaccines, including varicella and MMR. *Id.* at 3, 80. Two months later, in July 2014 another EEG was performed, revealing normal results except for some evidence of slow wave activity. Ex. 2 at 42–43.

By the fall of 2014, T.M. began showing some improvement developmentally, although overall deficits remained. At an early September well-child pediatric visit, for example, T.M. was reported to be rolling more frequently, and getting up on her knees but not crawling. Ex. 3 at 71, 74. It was also reported, however, that she was not saying any words, communicating through crying, and not reaching, and she was receiving many different kinds of developmental-oriented therapies. *Id*. The treating pediatrician, Dr. Lester Hockenberry, specifically stated at this time that "I do not believe her delay is in any way related to vaccination," but noted that Petitioner was "waiting to hear back from her lawyer before additional vaccines are given." *Id.*

That same month T.M. returned to see Dr. Romantseva for evaluation of both her prior febrile seizures and developmental delay (with regression noted to have begun around one year, or March 2014). Ex. 2 at 53. At this time, T.M. had been tolerating her anti-seizure medication, and had experienced no additional seizures since March (although the aforementioned records suggest a febrile seizure occurred in May). *Id.* at 75. Dr. Romantseva reported global developmental delays but also acknowledged some improvement. *Id.* at 76. And Dr. Romantseva reiterated the need for diagnostic lab work to determine the source of T.M.'s delays and seizures, since the summer EEG had yielded normal results (although labs performed at this time were mostly inconclusive). Ex. 2 at 61, 63–64, 76.

During the second half of December 2014, T.M. was again taken to the ER after the occurrence of a seizure. Ex. 9 at 4–6. Her father reported at this time, however, that he had forgotten to administer T.M.'s Keppra that morning. *Id.* at 5. The seizure lasted ten minutes until it resolved on its own—T.M. was crying loudly, had muscle stiffness, and was not focusing on her parents post seizure. *Id.* The ER physician noted that T.M. had a history of cerebral palsy (although

the record reviewed in this case does not support this diagnosis) and epilepsy. *Id.* T.M. returned to baseline by the time she was examined, and subsequently discharged the same day. *Id.* at 6.[7]

Treatment in 2015 to Present and Efforts at Diagnosing Seizure Activity

T.M.'s concurrent developmental delay and seizure activity has continued on from early 2015 to the present. *See, e.g.*, Ex. 28 at 7–8 (T.M. provided bilateral ankle braces for motor activity in May 2015, revealed little spasticity); Ex. 13 at 22 (follow-up with rehab specialist in September 2015 to evaluate secondary effects of braces) and 24–25 (assessed at that time with "mild [cerebral palsy] vs autism"); Ex. 15 at 10–11 (October 2015 visit, prescribed bilateral knee immobilizers and a gait training device). She did not learn to walk until she was almost four years old, with a stiff and wide gate. Ex. 27 at 45 (February 2017 visit to Dr. Romantseva). She continues to be nonverbal, and still needs diapers. *Id.*; Ex. 58 at 15, 21 (August 2019 pediatric well-check visit).

Treatment of developmental issues reveals no heightening or specific worsening, although the overall trajectory is not improving. Nor have any treaters directly linked T.M.'s developmental issues with her prior seizures or vaccinations. At the same time, some did start to consider whether an autism diagnosis might be appropriate. *See, e.g.*, Ex. 15 at 45–46 (Dr. Romantseva referring T.M. for an autism evaluation in November 2015); Ex. 59 at 20–23 (August 2019 visit with Dr. Romantseva, again referring to autism as potentially explanatory for developmental issues).

2016 saw a more forceful recurrence of seizure activity that was less apparent in the prior year. Thus, T.M. was again taken to the ER in May 2016 while experiencing an ongoing, active afebrile seizure. Ex. 14 at 5, 13. In the course of T.M.'s subsequent hospitalization, however, Petitioner reported that T.M. had been congested for several days, had experienced multiple contacts with others suffering from colds, and had also missed her morning Keppra dose. Ex. 24 at 51. Dr. Romantseva saw T.M. in connection with this event, and the records set forth her impression—a "3 year old girl with focal epilepsy, language and developmental delays, idiopathic diplegia who presented with status epilepticus last night, likely triggered by a combination of viral illness and missed medication dose." *Id.* at 60. And in October, T.M. had another seizure resulting in overnight hospitalization. Ex. 29 at 47, 53. Such seizure activity has been afebrile, and has continued on an intermittent basis—although in many instances the seizure seemed to have an explanation. *See, e.g.*, Ex. 27 at 45 (January 2017 seizure—afebrile but proposed by Dr. Romantseva to be attributable to recurrent ear infections); Ex. 59 at 21 (August 2019 visit to Dr. Romantseva referencing seizure that occurred in December 2018, but attributing it in part to fact

[7] At a January 2016 pediatric visit focused on following up from this occurrence, it was noted in the record that the seizure had been milder than prior events. Ex. 13 at 6. But it was also recorded that Petitioner had been "advised by previous physician not to continue DTaP at this time," (although the December 2015 seizure was proximately attributed to the missed Keppra dose and not a vaccine). *Id.* at 7.

that T.M. had been off her Keppra medication for two weeks prior, while alternative treater diagnoses were explored).

T.M. has thus continued to receive medication and other treatments aimed at control of further seizures. *See e.g.*, Ex. 12 at 30–31 (increase in Keppra dose in May 2015). But testing to evaluate their possible etiology continued to yield normal results, and has failed to result in an explanation associated with any underlying brain injury that may have occurred close-in-time to the December 2013 vaccinations at issue. Ex. 15 at 26–27 (November 2015 EEG); Ex. 24 at 18–19 (March 2016 brain MRI results deemed unremarkable pre and post-contrast); Ex. 14 at 32–33 (normal CT scan in May 2016, performed in wake of ER visit due to afebrile seizure). Lab studies also continue to yield negative results. Ex. 58 at 39, Ex. 59 at 105.

At most, an August 2016 ambulatory EEG (performed at Dr. Romantseva's direction) yielded (for the first time) abnormal results due to "regional epileptiform discharges." Ex. 24 at 294. However, no seizures or focal slowing occurred during the test, and ultimately the results were termed consistent with "an active epileptogenic source in the midline parieto-occipital region" of the brain. *Id*. Notably, this test result was obtained *more than two and one-half years* after vaccination—and after T.M. had experienced several prior seizures, with at least two directly attributed to a missed Keppra dose, as discussed above.

II. Hearing Testimony

A. *Melodie Weaver*

Ms. Weaver, T.M.'s mother and the Petitioner, was the first testifying witness. *See generally* Tr. at 5–29. She lives with T.M. and her two siblings, aged "twelve, eight, and five." Tr. at 5. She recalled that her pregnancy with T.M. "started off fairly normal, full term, didn't need any medication, vaginal birth." *Id.* at 6. She was not in labor long, and there were no complications. *Id.*

Ms. Weaver recalled T.M. to have been "[a] typical happy baby, chunky, because I breastfed, and was meeting her milestones a little bit slower. . .it was like a little bit of delay, but everything other than that was pretty normal." Tr. at 6. These delays, she specified, were more physical in manifestation, such as "wanting to pull up or, like, use her body." *Id.* at 7. Ms. Weaver testified that they continued to allow T.M. to have more tummy time to support continual meetings of her milestones. *Id.* All of these events occurred at T.M.'s four-month checkup. *Id.*

Ms. Weaver then addressed the December 2013 appointment when the relevant vaccines were administered. T.M. was now "gaining a little bit more skills, like just doing a little bit more—a little bit more active, interacting with [them], things like that." Tr. at 7. Nevertheless, Ms. Weaver

recalled discussing with medical treaters that T.M. was displaying some concerning problems like weakness. *Id.* at 9. At this appointment T.M. was referred to developmental assessment. *Id.*

That same evening, T.M. had "a high fever, and so [they] administered, like, Tylenol, the cold rags, just like normal procedures, how you try to bring down a fever, and it subsided enough for us to think that, you know, we would go ahead and lay her down and everything would be okay the next day." Tr. at 10. Later, however, T.M.'s fever spiked again, and Ms. Weaver was awoken when she saw "the whole bassinet just shaking," and she heard "gagging." *Id.* at 11. T.M. now appeared to be experiencing a seizure, with her "arms extended out, shaking, seemed to be unconscious, and, like, struggling to breathe, eyes rolling up to the top of her head, drooling." *Id.* Petitioner recalled calling 911, with T.M. subsequently being transported to the hospital. *Id.* at 12–13.

After an overnight stay at the hospital, T.M. was brought home, and Petitioner recalled that T.M. was at that time "lethargic and, like, lazy, and didn't want to participate in her daily activities." Tr. at 13. This lethargy continued until T.M.'s follow-up appointment. *Id.* By this time, T.M. seemed developmentally stagnant. *Id.* Ms. Weaver also noted that the treaters "kept referencing the vaccine can cause febrile seizures, and in a baby that young, it is common." *Id.*

Ms. Weaver then recounted T.M.'s second seizure from March 2014, stating that T.M. was in her back seat in her car seat, and then displayed a "stiff body, eyes rolling up, drooling out the mouth, like the whole car seat just like shaking because of the throbbing motion, like—it looks like she's in pain." Tr. at 15. Ms. Weaver then took her back to the hospital, where treaters attributed a "low-grade fever" as causal of the seizure. *Id.* at 17.

Petitioner did not herself witness T.M.'s third seizure (from early May 2014). Tr. at 17. By this time, Petitioner recalled, T.M.'s developmental issues were more pronounced, and thereafter T.M. began to have to relearn things like "[r]olling, getting up to crawl stance, engaging to our voices and things like that, all of that had eventually stopped, and she stopped interacting with us." *Id.* at 19.

Later, Dr. Romantseva proposed a seizure treatment plan for T.M., with a focus on medication to control seizure activity and help Petitioner care more effectively for T.M. when seizures did manifest. Tr. at 21. T.M. still had seizures while on the medication, but had not experienced any (as of the trial date) for two years. *Id.* All of the testing resulted in normal determinations, and thus had not provided any insight to Petitioner's knowledge about potential explanations for T.M.'s condition. *Id.* at 22. One rehab specialist, however, had proposed that cerebral palsy might explain it. *Id.* T.M. had also been taken to an autism specialist, although she was never diagnosed with autism and her tentative cerebral palsy diagnosis was also withdrawn,

leaving only a diagnosis of developmental delay. *Id.* at 22–23. T.M. has otherwise received '[p]hysical therapy, occupational therapy, and speech." *Id.* at 23.

Currently, T.M. is "still very dependent." Tr. at 24. She requires "hand-over-hand assistance to just be able to eat, and [they] clothe her, and [they] have to dress her. She's not potty-trained yet. She's nonverbal." *Id.* T.M. can feed herself somewhat using a utensil, but will return to "fist feeding." *Id.* T.M. can also walk at "a slower speed and as long as the ground is, like, at one level, like it's very restricted." *Id.* at 25. Her mental capacities and attention span also seem to Petitioner to have improved over time, in comparison to where she was. *Id.* at 26.

B. *Petitioner's Experts*

1. Dr. Mahbubul Huq, M.B.B.S., Ph.D.

Dr. Huq, a pediatric neurologist and clinical geneticist, submitted one report and testified for the Petitioner. *See generally* Tr. at 30–204, 363–65; Report, dated Aug. 25, 2021, filed as Ex. 60 (ECF No. 42-1) ("Huq Rep."). He proposed that T.M.'s overall seizure disorder was attributable to the vaccines she received in December 2013, and that her preexisting developmental delay was in turn worsened by her seizure activity.

Dr. Huq received his medical degree from Dhaka Medical College in Bangladesh and his Ph.D. in medical science at Tokushima University in Japan. Tr. at 31–32; Curriculum Vitae, filed Jan. 24, 2022, filed as Ex. 122 (ECF No. 65-2) ("Huq CV") at 1. He completed his residency in pediatrics and his fellowship in pediatric neurology at Wayne State University and the Children's Hospital of Michigan. Tr. at 32. He did a clinical and post-doctoral fellowship in genetics at Baylor College and a medical fellowship in genetics at the University of British Columbia in Vancouver. *Id.*; Huq CV at 1. Dr. Huq previously worked as a professor of pediatrics and neurology at Wayne State University and as a clinical geneticist at the University of British Columbia. Tr. at 32–33; Huq CV at 2. He is currently a professor of pediatrics at Central Michigan University and a professor of neurology at Wayne State University. Tr. at 32. He is a member of the American Academy of Neurology and is licensed to practice in the state of Michigan. *Id.* at 33.

Dr. Huq's research focuses on "genetics of metabolic disorder, autism, Tourette's syndrome" and he has also published papers focusing on the genetics of epilepsy. Tr. at 34; Huq CV at 21–30. In addition to research, Dr. Huq teaches clinical courses to graduate students, medical residents, and fellows, and he has lectured on advanced genetics at Wayne State University. Tr. at 35; Huq CV at 7–8. He currently has seven half-day clinics, is on in-patient service for six to eight weeks a year and spends weekends as an attending physician in a community hospital as a consultant. Tr. at 35. Dr. Huq estimates he sees 250–300 patients a year, 30% of which have epilepsy. *Id.* at 35–36. He estimates another 30% of his patients have developmental delays. *Id.* at

36. He is involved with all aspects of treatment, including genetic testing (close to 100% of his epileptic patients undergo genetic testing). *Id*. at 36.

Dr. Huq's testimony began with consideration of the overall medical record. He described Ms. Weaver's pregnancy with T.M. as uncomplicated, but noted the evidence of pre-vaccination concerns about T.M.'s development. Tr. at 38–40, 126; Huq Rep. at 2–3, 11; Ex. 4 at 8. In fact, T.M. was understood to be displaying developmental lag at the December 2013 pediatric visit when she received the vaccines at issue, although Dr. Huq did not find the record was detailed enough to gain insight into the extent of this lag. Tr. at 38–40, 363–64; Huq. Rep. at 2–3; Ex. 8 at 4–5. In his experience, developmental lag occurs when a child fails to meet expected milestones, although because of the wide variation in the population for childhood development, catch-up is possible (although ultimately progress can only be discerned in retrospect). Tr. at 40, 126. As a result, when a child is suspected of being delayed, further assessment, therapy, and genetic testing is important, as most developmentally delayed children he treats improve with such intervention (unless there is an underlying neurodegenerative condition). *Id.* at 40–41, 137, 364–65; Huq Rep. at 3.

Dr. Huq next addressed T.M.'s first seizure the day after the administration of her six-month vaccines in December 2013. Tr. at 42; Ex. 8 at 4, 6. He deemed it best classified as a febrile seizure—common to children (since they occur in approximately 2–4% of the pediatric population), although their ultimate cause is not well understood. Tr. at 58. Dr. Huq proposed, however, that underlying genetic factors (evidenced by tentative findings linking defined areas of the chromosome to the development of febrile seizure) as well as certain polymorphisms in inflammation-related genes might likely explain why some children tended to experience febrile seizures when others do not. *Id.* at 65–66. Infections by certain viruses (herpesvirus 6, influenza, pneumococcus infection, etc.) may also lead to febrile seizures. *Id.* at 66, 188.

Dr. Huq defined T.M.'s first seizure as "complex," since it lasted more than 30 minutes. Tr. at 42–44, 64–65; Ex. 7 at 59. As he explained, the term complex refers to "[a]nything atypical, such as if it is a focal seizure, if it is longer than 15 minutes, or if there are multiple occurrences within the same illness or 24 hours." *Id.* at 65. A simple seizure, by contrast, reflects "whole-body convulsion, less than 15 minutes." *Id.* Dr. Huq maintained there was no persuasive scientific authority establishing which children were more at risk for complex versus simple seizures. *Id.* at 95. He pointed to one study finding that some children who develop febrile, complex seizures may have underlying hippocampal malnutrition, but agreed the medical record did not in this case suggest that was true of T.M. *Id.* at 95–96; D. Lewis et al., *Do Prolonged Febrile Seizures Produce Medial Temporal Sclerosis? Hypotheses, MRI Evidence and Unanswered Questions*, 135 Progress in Brain Research 263 (2002) filed as Ex. 87 (ECF No. 47-9).

T.M.'s second febrile seizure, in March 2014, was not an uncommon occurrence either (although Dr. Huq felt it was rare for a child to experience more than four such febrile seizures). Tr. at 49; Ex. 2 at 21. But in the following two years, her tempo of seizures increased. Although T.M. was taking antiepileptic medication, she experienced "close to ten seizures" through 2015 and 2016, some without fevers, and Dr. Huq felt they were for the most part epileptic in origin. Tr. at 56, 117–18; 157–58; Huq Rep. at 2–3. He admitted, however, that some could have been merely "breakthrough" seizures attributable to a missed anti-seizure medicine dose or some other factor (although he contended that going off a medicinal course did not guarantee a seizure reaction). Tr. at 117, 118–22, 123. Dr. Huq did not categorize T.M.'s seizures overall as intractable, but he would not say they were easy to control either. *Id.* at 57, 118, 123–24.

Because T.M. did not appear to have any brain malformation or genetic cause that could explain her subsequent epilepsy, Dr. Huq proposed that the initial seizure was the most likely cause of her subsequent seizures, and he attempted to explain how this could be so. Tr. at 203–04. A child's immature brain is more susceptible to alterations, as neural networks are still forming. *Id.* at 97; Y. Ben-Ari & G. Holmes, *Effects of Seizures on Developmental Processes in the Immature Brain*, 5 Lancet Neurology 1055 (2006), filed as Ex. 20 (ECF No. 8-10) ("Ben-Ari & Holmes"). As a result, children who experience febrile seizures are two to three times more likely to develop epilepsy—with an even higher risk for those who experience prolonged complex seizures, which are more likely to harm the brain. Tr. at 66–67, 90–91, 116; Huq Rep. at 8.

The concept of "lowering the seizure threshold" as a result of successive injury to the brain explains the causal association of initial seizures to subsequent events. Tr. at 94, 96, 201–02; Huq Rep. at 8. After an initial "extrinsic insult," less may be required for a susceptible individual[8] to experience more seizure activity. Tr. at 95; Ben-Ari & Holmes at 1056. Thus, the initial seizure itself could set the stage for future aberrant responses to similar stimuli, leading to more seizures. Tr. at 91. S. Shinnar et al., *MRI Abnormalities Following Febrile Status Epilepticus in Children: The FEBSTAT Study*, 79 Neurology 871 (2012), filed as Ex. 100 (ECF No. 49-4) (children with a first febrile seizure that lasted longer than ten minutes were more likely to have abnormal development compared to those with a brief simple febrile seizure).

Animal studies, Dr. Huq noted, also corroborated the fact that "seizures can induce changes that will make the network even more susceptible to have a seizure." Tr. 101–03; Huq Rep. at 11; G. Smith et al., *Early-Life Status Epilepticus Induces Long-Term Deficits in Anxiety and Spatial Learning in Mice*, 4 Int. J. Epilepsy 36 (2017), filed as Ex. 101 (ECF No. 49-5) (demonstrating the effect a single occurrence of a status epilepticus can have on long-term impairments such as anxiety disorders and spatial learning behaviors); K. Chen et al., *Febrile Seizures in the Developing Brain Result in Persistent Modification of Neuronal Excitability in Limbic Circuits*, 5 Nat.

[8] Susceptibility, Dr. Huq maintained, referred to "a pre-existing lowered threshold for [handling] any kind of insult." Tr. at 192–93.

Medicine 888 (1999), filed as Ex. 23 (ECF No. 9-3) ("Chen") (when an immature or developing brain is exposed to hyperthermia-induced seizures, persistent modifications in neuronal excitability are present in the limbic system).

Evidence from the medical record, however, suggested that the foregoing theory might not apply to T.M.—since there was no proof, from around the time of the first and second seizures, that her brain *had* been harmed. As Dr. Huq admitted, several tests run on T.M. in the timeframe after the first seizure produced normal results. Tr. at 47–48 ("[a] viral respiratory panel that was normal. The CT head was normal, chest x-ray was normal. They have her broad-spectrum antibiotic, like ciprofloxacin and vancomycin, routine blood count, too"), 173. Dr. Huq also noted that the need for intubation was not *per se* evidence of anything abnormal about the first seizure. *Id.* at 46.

More significantly, the results of an EEG performed at that time were deemed normal. Ex. 7 at 68, 83, 88. And T.M. underwent several EEGs throughout her treatment process, without consistent determinations or early evidence of brain malformation or erratic activity. Tr. at 141–49; Huq Rep. at 2–3. An EEG, Dr. Huq acknowledged, can not only confirm the presence of seizure activity, but can also establish the diagnosis of epilepsy in 30-40% of cases. Tr. at 138–39. T.M.'s EEGs were normal up until May 7, 2016 (over *two years* post-vaccination), when she underwent a continuous monitoring[9] EEG which showed diffuse slowing (with another EEG in August producing slightly different results but still confirming the presence of epilepsy). Tr. at 141–43, 145–47; Ex. 24 at 293. Nor did any MRIs ever confirm any kind of brain harm after the first seizure. Ex. 24 at 18–19.

Dr. Huq nevertheless attempted to diminish the significance of the relevant EEG findings. He noted that routine EEGs identify abnormalities in only 30% of cases, and thus a "normal" EEG reading did not rule out the possibility of harm to the brain due to seizure activity. Tr. at 45, 139. Indeed, even some of the later "normal" EEG results in the years after T.M.'s first seizure confirmed her epilepsy. *Id.* at 148–49. Nor did normal MRI imaging results disprove his theory. Although MRIs can reveal brain injury caused by epilepsy, or the presence of some underlying brain malformation that could encourage seizure activity, Dr. Huq would not expect to see these neuronal changes until many years later (if ever). *Id.* at 93, 150; Huq Rep. at 6. Thus, a "clean" MRI did not mean no brain issues were present. Tr. at 151–52, 169, 173; Huq Rep. at 3. And even though T.M.'s treating physicians did not perform additional MRIs on T.M. after March 16, 2016, this was because her seizures were not intractable, and in any event MRIs require sedation which is harmful for the developing brain, further diminishing their use under the circumstances. Tr. at 174.

[9] Continuous monitoring EEGs take longer than routine EEGs and are typically video recorded. Tr. at 143.

With the foregoing as backdrop, Dr. Huq went on to propose how T.M.'s December 2013 vaccinations could have triggered an overall seizure disorder.[10] First, he explained generally how vaccinations interact with the human immune system. When vaccines are administered, they are recognized by pattern recognition receptors. Tr. at 76. The activated immune cells spread throughout the body, which creates (by design) inflammation reflective of the innate immune response. *Id.* at 76–77, 160, 196; Huq Rep. at 6. In particular, vaccine antigens attract locally-present immune cells which secrete pro-inflammatory cytokines intended to further the overall immune response (including "teaching" the immune system to recognize the relevant antigen presented by a vaccine in the future). Tr. at 76; Huq Rep. at 6, 8.

Vaccination (especially in the relevant context, where several vaccines were administered on a single occasion) thus encourages an inflammatory reaction. Tr. at 63, 79–80, 82, 109, 159, 197; Huq Rep. at 4. The presence of such pro-inflammatory cytokines has been linked to the development of febrile seizures. Tr. at 68, 70–71; Huq Rep. at 4, 6, 8–10; C. Dubé et al., *Interleukin-1β Contributes to the Generation of Experimental Febrile Seizures*, 57 Ann. Neurology 152 (2004), filed as Ex. 40 (ECF No. 26-10); A. Vezzani et al., *The Role of Inflammation in Epileptogenesis*, 69 Neuropharmacology 16 (2013), filed as Ex. 112 (ECF No. 70-2) ("Vezzani"). This likely occurs, Dr. Huq maintained, because the cytokines cause excitation and inhibition in the overall neural environment. Tr. at 69. Indeed, the very context of inflammation itself could be contributory to seizures. *Id.* at 73–74, 81–82; Huq Rep. at 6, 8–9.[11]

Thus, the upregulation of pro-inflammatory cytokines occurring in the context of T.M.'s vaccination likely caused her to have a fever after getting vaccinated, which in turn induced her initial seizure. Tr. at 63, 68, 71–72, 189–190, 197–99; Huq Rep. at 5, 10; And this initial seizure, coupled with localized inflammation caused by the vaccine, may have induced systemic changes that could "produce an epileptic state in the brain and cause subsequent recurrent seizure." Tr. at 64, 69, 73, 109. Dr. Huq emphasized that a consensus has formed that febrile status can cause epilepsy, despite prior uncertainty. *Id.* at 194.

Besides the more general association with vaccination-caused febrile seizures, Dr. Huq noted that certain vaccines (including those received in December 2013 by T.M.) have been more directly associated with febrile seizures (and possibly epilepsy more generally after an initial febrile seizure). Certain inactivated vaccines (like DTaP) are known to increase the risk of febrile seizure within 24 hours, while live vaccines (such as the MMR) can increase the risk within five to fourteen days. Tr. at 78; Huq Rep. at 4. And Dr. Huq noted the existence of case reports and small studies linking vaccines to febrile seizures. Tr. at 82–83; Huq Rep. at 5; Y. Sun et al., *Risk*

[10] Dr. Huq did generally admit that only in about 40% of cases is the cause of early-onset epilepsy identified. *Id.* at 58.

[11] Dr. Huq also noted that anti-inflammatory treatments often proved effective for ameliorating epilepsy—although it was not evident in this case that T.M. received them. Tr. at 75, 171.

of Febrile Seizures and Epilepsy after Vaccination with Diphtheria, Tetanus, Acellular Pertussis, Inactivated Poliovirus, and Haemophilus Influenzae Type B, 307 JAMA 823 (2012), filed as Ex. 55 at 6 (ECF No. 32-3) ("Sun") (finding that the relative risks of febrile seizures were increased on the day of the first and second vaccinations, but the absolute risks were low). Although no studies have explicitly associated the DTaP vaccine with subsequent epilepsy, Dr. Huq maintained a connection had not been ruled out either. Tr. at 181; Huq Rep. at 7. Indeed, the DTaP vaccine is contraindicated when seizure activity occurs after receipt of certain doses. Tr. at 89; *General Recommendations on Immunization*, 60 Centers for Disease Control and Prevention 1 (2011), filed as Ex. 119 (ECF No. 70-9).

Next, Dr. Huq attempted to set forth how T.M.'s epilepsy could have exacerbated her preexisting developmental issues, leading to a "more profound delay." Tr. at 62, 64. Developmental impairment generally is more likely in a child under two years old experiencing recurrent seizures than if the onset occurs in an older child. Tr. at 99; Huq Rep. at 11; S. Haut et al., *Susceptibility of Immature and Adult Brains to Seizure Effects*, 3 Lancet Neurology 608 (2004), filed as Ex. 81 (ECF No. 47-3) ("Haut") (explaining that age is a significant factor when analyzing the susceptibility of the brain to injuries induced by a seizure(s)); B. Hermann et al., *The Neurodevelopmental Impact of Childhood-Onset Temporal Lobe Epilepsy on Brain Structure and Function*, 43 Epilepsia 1062 (2002), filed as Ex. 82 at 8 (ECF No. 47-4) (finding that the presence of recurrent seizures in the developing brain appears to be associated with an adverse effect on both brain structure and function). Haut thus observed "a consensus that seizures affect brain function." Tr. at 108; Haut at 614.

Dr. Huq conceded that T.M. already was displaying develop mental delay *before* her seizure activity began, although its cause could not be identified.[12] Tr. at 124–26; 127–32. But she was not experiencing a progressively worsening condition pre-vaccination, as might be seen in connection with other kinds of disease processes associated with developmental loss. Tr. at 105–06, 110. He thus disagreed with Respondent's expert, Dr. John Zempel, that T.M.'s developmental delays at eight months of age (or right before the December 2013 vaccinations) were moderate to severe. *Id.* at 363. By the time of her third seizure in May 2014, however, T.M.'s developmental problems were more pronounced and global in nature, as evidenced by treater statements in the medical record. Tr. at 52–54; Ex. 7 at 241. T.M.'s subsequent visit to the neurologist noted continued loss of milestones. Ex 2 at 75.

[12] Dr. Huq noted that there were no identified genetic or brain-structural causes of T.M.'s developmental delays, nor could they be attributed to anything during pregnancy or her early childhood. Tr. at 59; Huq Rep. at 4. T.M.'s initial developmental issues also could have been "evidence of . . . a neurometabolic disorder, where the disease is progressive and neuronal loss is happening," or some other infectious process. Tr. at 51–52. Though extensive testing performed on T.M. showed no signs of neurodegenerative or progressive disease, Dr. Huq noted that a whole genome test had never been performed. Tr. at 111, 133–36, 191. It thus could not on this record be definitively concluded T.M. did *not* have an underlying neurodegenerative condition (although Dr. Huq proposed that this was unlikely). *Id.* at 111, 133–36, 191–92. A metabolic condition was also proposed at one time as explanatory. *Id.* at 56.

Dr. Huq therefore proposed that T.M. had experienced some pre-vaccination developmental lag, but after the "insult" of vaccination she settled at "a lower state of development." Tr. 105–06, 110. The ongoing seizures were contributing to this environment of developmental limitation. *Id.* at 55. Seizures are known to have "detrimental" effects on brain function. *Id.* at 107–08; Haut at 614–15. Seizures may also lead to further "development of epilepsy, cognitive impairments, and psychiatric impairments." Tr. at 108; Huq Rep. at 11. It was likely, Dr. Huq argued, that T.M.'s seizures "significantly aggravated whatever . . . propensity she might have for delayed development." Tr. at 110, Huq Rep. at 11.

On cross-examination, however, Dr. Huq conceded that it was possible that the causal relationship ran "the other way," with T.M.'s seizure disorders being a *product* of her preexisting developmental issues, rather than the former only exacerbating the latter. Thus, he admitted that "preexisting neurological dysfunction will increase the risk of febrile [seizure] status" Tr. at 194, 196; *see also* Haut at 613 ("[t]here are few data on the effects of a single brief seizure. Most studies are in patients with epilepsy, which introduces the possibility that *abnormalities are present before the seizure happens*") (emphasis added).

Finally, Dr. Huq addressed the timeframe between T.M.'s vaccinations and her first seizure, deeming it consistent with the short interval for initial inflammatory changes (which could occur within 24 hours to a few days after vaccination). Tr. at 112, 199. He noted that filed medical literature supported the acceptability of onset of a febrile seizure following the DTaP vaccine in such a timeframe. Tr. at 112–13. A broader state of epilepsy would occur within weeks, months, or even years after the first seizure, though Dr. Huq admitted that the longer the timeframe, the less probable an association existed. Tr. at 113–14, 199–200. But T.M.'s overall course herein was reasonably consistent with the first seizure being causal of what followed. Tr. at 113–14, 199–200.

2. Dr. Marcel Kinsbourne

Dr. Kinsbourne, a pediatric neurologist, submitted two reports for the Petitioner in support of the opinion that T.M.'s vaccinations were causal of her epilepsy injury. *See generally* Report, dated Sept. 22, 2017, filed as Ex. 30 (ECF No. 25-1) ("Kinsbourne Rep."); Report, dated Apr. 9, 2018, filed as Ex. 47 (ECF No. 30-1) ("Supp. Kinsbourne Rep."). Although Dr. Kinsbourne did not testify at trial, Petitioner has not formally disclaimed his opinions, and I therefore will summarize them below.

Dr. Kinsbourne received his medical degree from Oxford University in England, along with his Bachelor of Arts, and his Master of Arts. Curriculum Vitae, dated Jan. 24, 2022, filed as Ex. 123 (ECF No. 65-3) ("Kinsbourne CV") at 1. He then received his M.D. from the State of North Carolina. *Id.* After his schooling, Dr. Kinsbourne did several years of different post-doctoral

training in neurology, pediatrics, and chest diseases. *Id.* at 1–2. He is a member of the American Board of Pediatrics and Royal College of Physicians. *Id.* at 2. Dr. Kinsbourne was previously a professor of psychology, professor of pediatrics, lecturer in neurology, adjunct professor of linguistics and cognitive science, adjunct professor of occupational therapy director of the behavioral neurology department at the Eunice Kennedy Shriver Center, and other positions related to neurologic and cognitive studies. *Id.* at 2–3. Dr. Kinsbourne has been on several editorial boards, professional societies, and administrative assignments. *Id.* at 4–6. His research has considered pediatric disorders, developmental delays and factors, cerebral deficiencies, learning disabilities, therapies, and epilepsy. *Id.* at 6–39.[13]

Dr. Kinsbourne's first report began with a short recitation of the factual background history of T.M.'s illness. Kinsbourne Rep. at 1–3. He then highlighted the fact that the DTaP vaccine (which T.M. received in December 2013) has been determined to trigger seizure activity in infants. *Id.* at 3; Le Saux et al., *Decrease in Hospital Admissions for Febrile Seizures and Reports of Hypotonic-Hyporesponsive Episodes Presenting to Hospital Emergency Departments Since Switching to Acellular Pertussis Vaccine in Canada: A Report From IMPACT*, 112 Pediatrics e348, e349–50 (2003) filed as Ex. 53 (ECF No. 32-1) ("LeSaux"). In fact, Dr. Kinsbourne purported that "some 80 percent of DTaP provoked seizures occur within a day of the administration of the vaccine," as medical scientific authorities have recognized (like the Centers for Disease Control), although the report itself does not substantiate this specific figure. Kinsbourne Rep. at 3.

In explaining how a vaccine could produce a seizure, Dr. Kinsbourne highlighted the interplay between vaccination and the human immune system. Vaccines trigger an immediate, innate immune response, with the goal of secondarily stimulating an adaptive, memory response that will allow effective reactions in the future to the relevant presenting viral or bacterial antigens. Kinsbourne Rep. at 4; D. van Duin et al., *Triggering TLR Signaling in Vaccination*, 27 Trends in Immunology 49 (2006), filed as Ex. 44 (ECF No. 27-4). This activation causes pattern recognition and the (initial) release of proinflammatory cytokines (specifically IL-1β). Kinsbourne Rep. at 4–5. But the proinflammatory cytokines can, in turn, establish a neuroinflammatory environment in which seizures are more likely, due to a "lowering" of the threshold for them. *Id.* at 5; J. Choi et al., *Cellular Injury and Neuroinflammation in Children with Chronic Intractable Epilepsy*, 6 J. Neuroinflammation 1 (2009), filed as Ex. 37 (ECF No. 26-7) (findings suggest that active neuroinflammation and cellular injury could play a pathogenic role or be a consequence of epilepsy in children).[14]

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[13] *See Holmes v. Sec'y of Health & Hum. Servs.*, 08-185V, 2011 WL 2600612, at *2 (Fed. Cl. Spec. Mstr. April 26, 2011).

[14] Dr. Kinsbourne also argued (despite the fact that T.M.'s initial seizure was unquestionably febrile in origin) that the pertussis toxoid has been shown to have its own pathologic path to triggering seizure activity through neuronal excitation. Kinsbourne Rep. at 5; J. Choi & S. Koh, *Role of Brain Inflammation in Epileptogenesis*, 49 Yonsei Med J.

Further support for the argument that febrile seizures could increase neuronal excitability was identified from articles involving epilepsy patients. *See, e.g.*, Chen at 6 (suggesting that early-life febrile seizures can lead to continuous effects on neuronal excitability due to the long-lasting nature of presynaptic hippocampal interneuronal terminals); R. Badawy et al., *On The Midway to Epilepsy: Is Cortical Excitability Normal in Patients with Isolated Seizures?*, 24 Int'l J. Neural Systems 1430002-1 (2014), filed as Ex. 32 (ECF No. 26-2) ("Badawy") (patients with isolated seizures compared to controls had increased cortical excitability which suggested the presence of a mild degree of cortical hyperexcitability). Dr. Kinsbourne acknowledged, however, that excitability did not definitively mean that a patient would develop epilepsy, as noted in some of these articles. *See* Badawy at 6 (cortical excitability is disrupted in patients with a single seizure, and that such disruption can persist even with a lack of recurring seizures).

In addition, Dr. Kinsbourne sought to explain how T.M.'s seizure activity might have negatively impacted her developmental problems. He opined (based on a reading of the medical record) that T.M.'s seizures were localized to the midline parieto-occipital region of the brain, and he proposed it was likely that "a hyperexcitable network in that region" had some association with her established, pre-vaccination development issues. Kinsbourne Rep. at 4. Medical literature had observed generally a connection between childhood seizure disorders/epilepsy and progressive development issues. *Id.* at 6; Ben-Ari & Holmes at 1056, 1060. And the kinds of seizure disorders associated with developmental delay were often triggered by an initial event. T. Baram & C. Hatalski, *Neuropeptide-Mediated Excitability: A Key Triggering Mechanism for Seizure Generation in the Developing Brain*, 21 Trends Neuroscience 471 (1998), filed as Ex. 33 (ECF No. 26-3) ("Baram & Hatalski") ("[s]urprisingly, the majority of developmental seizures are not spontaneous but are provoked by injurious or stressful stimuli"). Thus, Dr. Kinsbourne opined that T.M.'s developmental delay was likely aggravated by her vaccine-induced epilepsy/seizure disorder. Kinsbourne Rep. at 6.

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1 (2008), filed as Ex. 36 (ECF No. 26-6). While the whole-cell pertussis vaccine (which has been supplanted by vaccines containing the acellular pertussis toxoid) has more demonstrated adverse capacity, there is still "an adequate amount of pertussis-specific antigens to provoke a protective antibody response in the recipient"—sufficient in turn to cause seizures. Supp. Kinsbourne Rep. at 2; Le Saux at e351–52; L. Jackson et al., *Retrospective Population-Based Assessment of Medically Attended Injection Site Reactions, Seizures, Allergic Responses and Febrile Episodes after Acellular Pertussis Vaccine Combined with Diphtheria and Tetanus Toxoids*, 21 Pediatric Infectious Disease J. 781 (2002), filed as Ex. 52 (ECF No. 31-5).

My determination that vaccines "can cause" febrile seizures, purely through the innate reaction they provoke, and that this occurred in T.M.'s case, obviates the need to consider closely the reliability or persuasiveness of this aspect of Petitioner's case. I also note that although Dr. Huq indicated in his testimony that he embraced Dr. Kinsbourne's opinion, he did not primarily rely upon the pertussis component issue in theorizing that the vaccines T.M. received could have caused her seizures. Tr. at 184 ("that is not my main hypothesis"). I give Dr. Huq's opinion somewhat more weight than Dr. Kinsbourne's overall.

Dr. Kinsbourne's supplemental report addressed some of Respondent's arguments, but also added detail to his prior expert opinion. He agreed that he largely relied on circumstantial evidence that vaccines can trigger seizures, although he deemed this sufficient for present circumstances. Supp. Kinsbourne Rep. at 1. He reiterated his argument that even if the acellular pertussis-containing vaccines presented less risk of associated seizure, the risk had not been eliminated. *Id.* at 2–3. He maintained that the possession of an antecedent development delay, as was true here, was actually associated with longer-lasting (and hence damaging) seizures. *Id.* at 4. And he questioned the supposition (unsupported by evidence) that T.M.'s epilepsy either predated vaccination, or had some to-date unidentified relationship with those problems independent of vaccination. *Id.* at 5–6.

C. *Respondent's Expert—Dr. John Zempel*

Dr. Zempel, a pediatric epileptologist, submitted three reports and testified for the Respondent in support of the argument that T.M.'s vaccinations were not causal of her larger seizure disorder or epilepsy. *See generally* Tr. at 205–362; Report, dated Feb. 1, 2018, filed as Ex. A (ECF No. 29-1) ("Zempel Rep."); Report, dated June 12, 2018, filed as Ex. C (ECF No. 34) ("Second Zempel Rep."); Report, dated Oct. 16, 2021, filed as Ex. D (ECF No. 53-1) ("Third Zempel Rep.").

Dr. Zempel attended the University of Wisconsin-Madison, obtaining a B.S. in Molecular Biology. Tr. at 205; Curriculum Vitae, filed Feb. 2, 2018, filed as Ex. B (ECF No. 29-8) ("Zempel CV") at 1. He then become part of the Medical Scientist Training Program at Washington University, attaining his M.D. and PhD in Neurobiology. Tr. at 205; Zempel CV at 1. Dr. Zempel remained at Washington University for his internship and residency in pediatrics. Zempel CV at 2. Following this he did a residency in adult neurology, a residency in child neurology, a pediatric epilepsy fellowship, and finally a clinical neurophysiology fellowship. *Id.* at 2. He was later hired by Washington University as a faculty member, progressing from assistant, associate, to full-time professor in neurology and pediatrics. Tr. at 206. Dr. Zempel is board certified in psychiatry and neurology, child neurology, clinical neurophysiology, and pediatrics. Zempel CV at 2–3.

Dr. Zempel presently works in a weekly outpatient clinic "specializ[ing] in the care of children with intractable epilepsy." Tr. at 206. Half of his time is devoted to inpatient services, between the neurology floor, ICU consultation service, emergency room, and neonatal neurology service. *Id.* He is also the medical director of the EEG Laboratory in the Clinical Neurophysiology Laboratory, where pediatric seizures are evaluated for treatment. *Id.* at 206–07. Dr. Zempel also performs many second opinions and gets referrals from neurologists, with a focus on cases of intractable epilepsy. *Id.* at 207–08. He is a member of the American Epilepsy Society and the Child Neurology Society. *Id.* at 209. Dr. Zempel has published numerous articles on neuroscience and epilepsy, but none on febrile seizures. *Id.* at 209; Zempel CV at 5–8.

Dr. Zempel's analysis began with an overview of the different types of seizures—and he noted at the outset that not all seizures "fit" into a diagnosis of epilepsy. Provoked seizures "can be associated with stroke, head injury, hypoglycemia, infection, and variety of triggers," such as independent environmental stimuli (a fever, stress, sleep deprivation, etc.). Tr. at 210–211. An unprovoked seizure, by contrast, does not need a stimulus to occur. *Id.* at 210. Epilepsy can be characterized by a high probability of recurrent *unprovoked* seizures. *Id.* at 210–11.

A febrile seizure, Dr. Zempel explained, is a classic kind of provoked seizure. Tr. at 211. "Simple" febrile seizures usually last less than 15 minutes, have no focal features, are generalized tonic clonic,[15] and will occur on one side of the body. *Id.* at 212. A complex febrile seizure, however, will usually last longer than 15 minutes—although some that are focal can also be deemed complex despite their length, and having more than one seizure in a 24-hour period may suggest the presence of complex seizures. *Id.* Regardless, children who experience a febrile seizure of any kind are not necessarily displaying a presenting symptom of something larger, and "only a fraction of cases go on to develop epilepsy." *Id.* at 211, 217.

Dr. Zempel next discussed epilepsy broadly, defining it to be a condition involving unprovoked seizures occurring randomly and without a clear explanation. Tr. at 214. The diagnosis of epilepsy is usually based on clinical evidence—in particular, proof of two or more unprovoked seizures—and the goal of treatment is to determine a treatment approach to limit the likelihood of future seizures. *Id.* Epilepsy will be deemed "intractable" when antiseizure medications prove ineffective (although this does not mean seizures can never be controlled in such a patient). *Id.* at 215. Its causes are various, but they can include "injury, an injury prenatally, an injury at birth, an injury any time in the rest of your life, including strokes and other—and accidents and trauma can very clearly cause your brain to transition to a state where unprovoked seizures are more likely." *Id.* at 216. Some cases have strong genetic bases, with epilepsy running in families, and it can also be related to a developmental problem. *Id.*

It cannot be assumed, Dr. Zempel opined, that the occurrence of a febrile seizure is evidence of the presence of epilepsy in a larger sense. Relevant medical literature reflects ample debate "over whether you have febrile seizures and epilepsy as separate pieces, [or] whether your febrile seizures are really a presentation of epilepsy with fever," with general agreement that a single febrile seizure is not, by itself, evidence of likely epilepsy. Tr. at 236. Nevertheless, Dr. Zempel admitted that children who experience a *complex* febrile seizure more commonly develop epilepsy than those whose febrile seizures are shorter. *Id.* at 333 Dr. Zempel went on to define "febrile status epilepticus," or a febrile seizure evidencing epilepsy, as "continu[ed] seizure

[15] "Tonic-clonic" is defined as "a spasm or seizure consisting of a convulsive twitching of the muscles." *Tonic-Clonic*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=50263 (last visited Sept. 23, 2022).

activity or lack of return to normal in between [febrile] seizures for more than 30 minutes." *Id.* at 213.

Dr. Zempel agreed that T.M.'s initial seizure was likely a complex febrile seizure (despite some doubts about its length). Tr. at 225–26. And he agreed it was likely vaccine-associated. *Id.* at 358–59. But he disputed the fact that it reflected the first step in a vaccine-triggered epilepsy, observing that the record did not establish that the initial febrile seizure had caused injury to T.M.'s brain. For example, the CT scan conducted at that time detected no injury. *Id.* at 228. More importantly, the EEG (which can reveal brain wave activity) also resulted in normal findings—even though after a "prolonged or intense seizure" EEG readings more commonly reveal abnormal results. *Id.* at 229–31. Although EEGs alone cannot be relied upon to diagnose epilepsy, they can be used to "clinch" the diagnosis. *Id.* at 250.

Subsequent record evidence from the months after the first seizure also revealed no likely brain injury associated with that initial seizure. T.M.'s second and third EEGs (performed July 3, 2014, and November 12, 2015) produced normal results. Tr. at 246–47. Her fourth EEG was comparable in terms of no revealing ongoing seizure activity, although Dr. Zempel observed that (in contrast to the first three) there were some distinctions that could in part be attributed to medications T.M. was by this time receiving. *Id.* at 249–50. Only by the time of the EEG performed about two years after the relevant vaccinations did epileptiform abnormalities appear (and for the first time), thus now establishing that "in the future that there could be recurrent unprovoked seizures." *Id.* at 254–55.

Overall, this aspect of the medical record (reflected in particular by EEG results) did not, in Dr. Zempel's estimation, establish any instance in which T.M. experienced a "catastrophic" seizure capable thereafter of snowballing into a series of ever-more damaging seizures. Tr. at 274–75. And literature offered by Dr. Huq confirmed that some kind of brain-injuring event associated with a specific seizure would need to be demonstrated to attribute subsequent seizure activity to the prior event. *See* T. Salmenperä et al., *MRI Volumetry of the Hippocampus, Amygdala, Entorhinal, Cortex, and Perirhinal Cortex after Status Epilepticus*, 40 Epilepsy Research 155 (2000), filed as Ex. 95 (ECF No. 48-8) (status epilepticus does not regularly lead to damage in the medial temporal lobe structures; instead, the hippocampus is the most severely affected brain region in patients with a history of status epilepticus).

T.M.'s treatment record was also inconsistent with a "seizures begetting seizures" scenario in other ways, Dr. Zempel argued, or at least did not confirm the supposition that this encapsulated T.M.'s experience. For example, no lumbar puncture was performed which could have identified an underlying infectious cause for the initial seizure—an omission that suggested to Dr. Zempel that initial treaters did not suspect the seizure to be more than febrile in nature (although he qualified the strength of this inference). Tr. at 232–33. In fact, T.M.'s neurologist did not conclude

that vaccination could have been causal for T.M.'s epilepsy course. *Id.* at 243. There was otherwise no indication of neuronal injury or cerebral inflammation after the December 2013 seizure, and T.M. quickly returned to a baseline level of health once she was discharged from the hospital. *Id.* at 233–35. And her next seizure (experienced almost three months later) was deemed a simple febrile seizure occurring in the context of evidence of some kind of upper respiratory infection—which by itself could lower a seizure threshold (independent of whether the initial seizure had established circumstances for future seizure events). *Id.* at 236.

More evidence diminishing the centrality of the initial febrile seizure in T.M.'s subsequent history were the results of the MRI performed in March 2016. Its findings were deemed by treaters as unremarkable, which Dr. Zempel interpreted to mean that brain abnormalities associated with epilepsy, like cortical malformation, were not present. Tr. at 261–65.[16] And T.M.'s epilepsy was effectively managed by a single antiseizure medication (and a low dose of it as well), further underscoring that her epilepsy was not particularly acute in nature. *Id.* at 243–44. The instances in which T.M. appeared to suffer seizures after missed medication were also considered by Dr. Zempel as corroborating the likelihood that seizures after the December 2013 febrile seizure event were not the product of it, although Dr. Zempel conceded that seizures were not inevitable simply because a medicine dose was skipped or missed. Ex. 9 at 4–6 (on Dec. 30, 2014, T.M. had a seizure that lasted roughly ten minutes, after her father forgot to administer her Keppra that morning); Tr. at 246.

Also significant to Dr. Zempel's opinion was the record proof of T.M.'s pre-vaccination developmental delay, which he felt overall was more extensive than Petitioner and her experts allowed—and which in turn rebutted the conclusion that her developmental issues had been worsened by T.M.'s concurrent epilepsy. As early as T.M.'s well-child visit on July 15, 2013, treaters observed that her eyes were not the same size, suggesting to Dr. Zempel that her developmental problems already were manifesting. Tr. at 219; Ex. 4 at 8. There were also the missed milestones noted at her December 2013 pediatric visit (when she received the vaccines in question). *Id.* at 220–21. Indeed, he found especially important the Petitioner's contemporaneously-recorded statement from this visit that T.M. was "not doing much," and was also at this time referred to a developmental clinic for evaluation. *Id.* at 221–22; Ex. 8 at 4. All of these factors suggested the presence of a significant problem for the future. *Id.* at 223.

Developmental delays can, Dr. Zempel admitted, relate to epilepsy, since both are signs of underlying brain dysfunction—but more importantly, "[p]reexisting developmental delay prior to the onset of seizures is clearly a strong risk factor for the [later] development of epilepsy." Tr. at

[16] At worst, an incidental cavum septum pellucidum ("the median cleft between the two laminae of the spetum pellucidum") was observed, but Dr. Zempel opined that this was "probably a normal variant." Tr. At 263. *Cavum Septi Pellucidi*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=63907 (last visited Sept. 23, 2022).

224. And the record overall established the existence of "very significant delays," before, and then at, the time of the December 2013 vaccinations. *Id.* at 241–42.

Besides analyzing the record, Dr. Zempel commented on other aspects of the Petitioner's causation theory. He agreed that vaccination activates the immune system with the intent of protecting an individual from illness. Tr. at 270 ("it is not surprising or unexpected that vaccination would activate immune mechanisms since that's precisely what we're trying to do with vaccination"). *Id.* But he denied that there was any direct evidence offered in this case (particularly in the form of epidemiologic proof) that supports the conclusion that "standard childhood vaccination is a risk—a strong risk factor for developing epilepsy." *Id.* at 271. While he accepted that the wild Bordetella pertussis virus is known to precipitate seizures, he denied that this conclusion applied equally to the vaccine pertussis component. *Id.* at 278. And literature offered to substantiate this point more commonly discussed infection as the inciting factor in inflammation (and thus was distinguishable from the circumstances implicated in vaccination). *Id.* at 271–72.

Dr. Zempel also made some specific points about individual items of literature offered by Petitioner's experts. Baram & Hatalski, for example, was cited by Dr. Kinsbourne for the proposition that developmental-related seizures are usually provoked at the outset—a contention which Dr. Zempel deemed to overly simplify a complex question. Tr. at 265. Rather, "the translation between seizures and epilepsy is often a fraught one because many children who have seizures in the neonatal period don't go on to develop epilepsy." *Id.* Ultimately, the association between developmental problems and seizure disorders/epilepsy presented, in Dr. Zempel's opinion, a "chicken or the egg question," in which it was hard to know whether seizures occurred because the patient had an overall susceptibility, or whether the seizure *itself* caused an injury that then made the individual further, or more, susceptible. *Id.* at 265–66.

Dr. Zempel generally agreed that the rat model applied in Chen showed febrile seizures could result in "permanent and persistent modification of excitability." Tr. at 267; Chen at 889. But, he maintained, excitability does not automatically correlate with an increase of epilepsy. *Id.* at 267–68. And while some retrospective studies have noted ties between those who experience intractable temporal lobe epilepsy later in life and a history of prolonged febrile seizures, the inverse (studies finding an association of childhood febrile seizures with the subsequent development of epilepsy) was more difficult to ascertain. *Id.* at 268–69.

Another article, cited by Dr. Kinsbourne as evidence that prolonged seizures can cause epilepsy and development delay, was directly challenged by Dr. Zempel. Tr. at 287; D. Hesdorffer et al., *Distribution of Febrile Seizure Duration and Associations with Development*, 70 Annals Neurology 1, 7 (2011), filed as Ex. 50 (ECF No. 31-3) ("Hesdorffer").[17] Hesdorffer noted that "prolonged febrile seizures are associated with an increased risk of developing epilepsy. Indeed,

[17] The filed copy of Hesdorffer does not have page numbers, so I shall refer to the pages in ECF-filed order.

animal data indicate the duration of febrile seizures is correlated with the severity and duration of subsequent unprovoked seizures, as well as increased risk of cognitive dysfunction." Hesdorffer at 7; Tr. at 287. But Dr. Zempel maintained that this was more an associated than causal relationship, especially with the multitude of factors that he considers cause epilepsy. Tr. at 287–89. In fact, Hesdorffer explicitly observed that developmental delay could itself be a risk factor for epilepsy. Hesdorffer at 8; Tr. at 289.

Dr. Zempel also took issue with some of the items of literature filed by Dr. Huq. One such item, for example, only noted that vaccination could be a risk factor increasing the possibility of seizure—as opposed to a statistically-significant causal factor. Tr. at 292–93, 295; N. Andrews et al., *Post-licensure Comparison of the Safety Profile of diphtheria/tetanus/whole cell pertussis/haemophilus influenza type b vaccine and a 5-in-1 diphtheria/tetanus/whole cell pertussis/haemophilus influenza type b/polio vaccine in the United Kingdom*, 28 Vaccine 7215 (2010), filed as Ex. 65 (ECF No. 45-4) at 7220. Vezzani did not in fact stand for the proposition that vaccination can cause epileptogenesis. Tr. at 303–04; Vezzani at 11 (concluding that a "disease modifying drug that regulates inflammation" would assist in treatment of epilepsy, but without mention of the capacity of vaccines to promote such inflammation in the first place). And Dr. Zempel questioned the weight to be given the Sun article. Even though Sun explicitly sought to determine the risk of febrile seizure and epilepsy after DTaP-IPV-Hib vaccination, it found only a small increased risk of febrile seizures on the day of vaccination for infants at three and five months of age, with no associated risk of epilepsy overall. Sun at 823; Tr. at 304. And he deemed Sun to be particularly robust and involving a large sample. Tr. at 305–06.

III. Procedural History

This claim was initiated in November 2016, and Petitioner filed medical records thereafter with the statement of completion filed in January 2017. (ECF No. 12). Respondent's Rule 4(c) Report was filed on March 6, 2017. (ECF No. 16). Expert reports were filed over the course of the ensuing four years. The case was eventually reassigned to me in March 2021, and after reviewing the pre-hearing briefs filed by both parties, I held a two-day hearing on the matter on February 9-10, 2022. The claim is now ripe for resolution.

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] In this case, Petitioner does not assert a Table claim.

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 and is discussed in turn along with the parties' arguments and my findings.

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.

[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. Appx. 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).

However, the Federal Circuit has *repeatedly* stated that the first prong requires a preponderant evidentiary showing. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1360 (Fed. Cir. 2019) ("[w]e have consistently rejected theories that the vaccine only "likely caused" the injury and reiterated that a "plausible" or "possible" causal theory does not satisfy the standard"); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010). This is consistent with the petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted). If a claimant must *overall* meet the preponderance standard, it is logical that they are also required to meet each individual prong with the same degree of evidentiary showing (even if the *type* of evidence offered for each is different).

Petitioners may offer a variety of individual items of evidence in support of the first *Althen* prong, and are not obligated to 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). No one "type" of evidence is required. 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." *Andreu,* 569 F.3d at 1380. Nevertheless, even though "scientific certainty" is not required to prevail, the individual items of proof offered for the "can cause" prong must *each* reflect or arise from "reputable" or "sound and reliable" medical science. *Boatmon,* 941 F.3d at 1359–60.

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).

However, medical records and/or statements of a treating physician's views 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 also 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 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, 356–57 (2011), *aff'd without opinion*, 475 F. App'x. 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 is a medically acceptable timeframe must also coincide 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.*, 2013 WL 1896173 (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 review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Significant Aggravation Claim*

Where, as here, a petitioner alleges significant aggravation of a preexisting condition, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *Loving v. Sec'y of Health & Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Servs.*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory

> causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the *Loving* test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.,* 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, the Petitioner need not demonstrate an *expected* outcome, but merely that the injured individual's relevant post-vaccination condition was worse than pre-vaccination. *Sharpe*, 964 F.3d at 1081. And a claimant may make out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish alternative cause, where a petitioner's showing is enough to make out a prima facie case, and thereby shift the burden of proof to Respondent). *Id.* at 1083.

C. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(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 v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[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 when compared 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 testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("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 may be required 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.

D. *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).

However, in the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings—e.g., 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 particular 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").

E. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all 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—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 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. Appx. 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. Febrile Seizures and Their Relationship to Seizure Disorders

Both side's experts agreed that vaccines can trigger febrile seizures, as a result of the vaccine's stimulation of the innate immune system (which includes upregulation of pro-inflammatory cytokines specifically associated with fever). Huq Rep. at 4; Tr. at 358–59. This issue is thus uncontested—and I find it is wholly consistent with medical science pertaining to

febrile seizures. But this highlights the larger, and hotly-disputed, question in this case: whether a single febrile seizure, vaccine-caused or not, will *inevitably* lead to some form of epilepsy—and thus whether a vaccine-caused febrile seizure is more likely than not causal of a child's subsequently-manifesting epilepsy.

The Vaccine Program has confronted this question before. Although the results have varied (depending on the specific facts at issue, as well as the relative strength of showing made in each case) it is clear that vaccine-instigated febrile seizures cannot be properly considered causal of all forms of epilepsy, simply because more seizures follow the first event. *See,* e.g., *Caredio v. Sec'y of Health & Hum. Servs.*, No. 17-0079V, 2021 WL 4100294 (Fed. Cl. Spec. Mstr. July 30, 2021), *mot. for review den'd,* __. Fed. Cl. __, 2021 WL 6058835 (2021). In *Caredio*, an infant's autoimmune epilepsy was argued to have been caused by a flu vaccine. The child experienced an initial febrile seizure close-in-time to the vaccination event—and although there was no dispute that the febrile seizure was vaccine-caused, the onset of the child's form of epilepsy was deemed to have occurred slightly later (within two weeks of vaccination). *Caredio*, 2021 WL 4100294, at *2–3, 15. Indeed, the petitioners' causation expert disclaimed any relationship between the febrile seizure and the child's epilepsy, the course of which progressively unfolded in the months thereafter. *Id.* at *12. Although denial of entitlement in *Caredio* turned on a failure to establish that the relevant vaccine "could cause" autoimmune epilepsy, the decision helpfully demonstrates how an initial febrile seizure could have no relationship to a child's subsequently-diagnosed epilepsy.

A different well-reasoned decision, by contrast, *connected* an earlier febrile seizure to epilepsy. *Ginn v. Sec'y of Health & Hum. Servs.,* No. 16-1466V, 2021 WL 1558342 (Fed. Cl. Spec. Mstr. Mar. 26, 2021) (five vaccines, including the flu vaccine, triggered a febrile seizure in four-year-old that contributed/led to development of epilepsy). In *Ginn,* an infant experienced a febrile seizure within 24 hours of receiving several vaccines. Two months later, the child had a second seizure (not identified as febrile), and an EEG performed at this time now revealed the presence of abnormality consistent with epilepsy. *Ginn,* 2021 WL 1558342, at *1–2. The child was thereafter diagnosed with epilepsy. In finding for the petitioners (and in a case where, as here, Dr. Huq served as the claimant's expert), the special master emphasized not only that febrile seizures could propagate further seizure activity, but also that evidence (particularly in the form of the EEG findings from the second seizure event) corroborated that brain changes/damage had occurred after the first seizure—providing sufficient evidence to link the two under the petitioners' causation theory. *Id.* at *8–9.

Thus, the evidence connecting the initial febrile seizure to the child's epilepsy that was missing in *Caredio* was supplied in *Ginn*—underscoring the *importance* of such connective proof for causation purposes. Even though the seizure disorder in *Caredio* was ultimately autoimmune

in nature, the distinction between febrile seizures and later epilepsy remains—as recognized in *Ginn*.

II. Petitioner Has Not Preponderantly Established that T.M.'s Single, Vaccine-Caused Febrile Seizure Lead to or Caused Her Subsequent Seizure Disorder

This claim largely turns on the second *Althen* prong. As discussed above, I can easily determine herein that almost *any* vaccine, alone or grouped with others, could cause sufficient inflammation to trigger a single febrile seizure. In addition, although both experts also agreed that an initial febrile seizure does *not* inevitably mean a child will experience epilepsy (*see, e.g.*, Tr. at 116, 236), Dr. Huq did offer persuasive preponderant evidence in support of the conclusion that as a general matter, children who experience febrile seizures are more likely to develop epilepsy later (although it is not a certainty—and the initial seizure could simply unmask a propensity to seize, rather than be considered the instigating factor). But the medical record *in this case* does not support the conclusion that T.M.'s December 2013 post-vaccination febrile seizure caused her epilepsy.

Specifically, the record does not establish that the initial febrile seizure harmed T.M.'s brain sufficiently to conclude that it likely "explains" what transpired thereafter. On the contrary, persistent testing performed over the next two-plus years, whether in the form of EEGs or MRIs, did not confirm the presence of a seizure-induced brain malformation or injury that could then (under a "seizures beget seizures" theory) be deemed causal of all subsequent seizures. *See, e.g.*, Ex. 7 at 68 (normal EEG readings based on EEG performed on December 11, 2013); Ex. 2 at 42–43 (July 2014 EEG produced normal results with no evidence of epileptiform activity); Ex. 15 at 26–27 (November 2015 EEG produced normal results); Ex. 24 at 18–19 (March 2016 brain MRI deemed to have yielded normal results); Ex. 14 at 32–33 (normal CT scan in May 2016, performed in wake of ER visit due to afebrile seizure). Dr. Huq acknowledged this absence of proof of brain injury. Tr. at 47-48, 141-49, 173.

Only by August 2016 did an EEG yield results that T.M.'s neurologist deemed to corroborate the presence of epileptiform activity. Ex 24 at 294. Dr. Huq reasonably pointed out in reaction that a normal EEG reading did not rule out epilepsy—but the totality of the screening evidence in this case does not establish brain injury close-in-time to the vaccine-caused febrile seizure.

T.M.'s post-vaccination seizure activity after the first, admittedly vaccine-caused febrile seizure, is also not of a character or tempo that would render it likely related to the first event. Thus, T.M.'s second febrile seizure (and second seizure event otherwise) occurred in March 2014—three months after her first febrile seizure, with nothing in the record suggesting an association between the two. Moreover, the second seizure was attributed to a then-existing likely

upper respiratory infection. Ex. 7 at 221–22. T.M. recovered quickly thereafter, suffering no additional seizures for two months. But the May 2014 seizure event was also attributed to a febrile seizure. Ex. 1 at 1–6. Thereafter, for the remainder of that year T.M. received anti-seizure medications, which controlled her seizure activity enough so that her next seizure event—in December 2014 (and thus now a year after the vaccine-related initial first seizure)—was deemed to have been likely caused by a missed Keppra dose. Ex. 9 at 4–6.

This history is not supportive of an association with the initial febrile seizure.[19] Rather, it suggests a non-intractable and treatable epilepsy. And to the extent the continued epileptic activity may have *eventually* damaged T.M.'s brain, such that subsequent EEG and other testing evidence began to confirm more activity, that course cannot be attributed to the first febrile seizure event—and thus not to the December 2013 vaccines either.

Admittedly, the first seizure met the definition of a *complex* seizure given its length. And Dr. Huq credibly established that complex seizures are often associated with brain damage. But that fact must be balanced against T.M.'s quick recovery in December 2013–January 2014, as well as the lack of overall evidence of brain harm discussed above. The *possibility* that a complex seizure could harm the brain or increase neuronal dysfunction, as Dr. Huq maintained, is not *in this case* borne out by the actual medical record.

The fact that T.M.'s seizures occurred in the context of established developmental concerns also weighs against a conclusion that her first seizure caused what followed. The record incontrovertibly establishes that developmental issues were raised with respect to T.M. *before* the December 2013 vaccination event. And Dr. Zempel credibly and persuasively established that medical science supports the conclusion that seizure activity can not only accompany developmental issues (or in some cases cause them), but that it might occur *because* of underlying developmental problems. This is not a case where the injured infant's developmental symptoms all post-date vaccination. Although I do not purport to find that T.M.'s developmental issues *explain* her subsequent seizure activity and epilepsy, Petitioner did not persuasively rebut this kind of evidence, and it therefore further diminishes the possibility that the initial febrile seizure alone explained why T.M. continued to experience seizures afterward.

This is not a case where expert input on either side appreciably "moved the needle" in the direction of one determination over the other. Both experts were sufficiently qualified to offer the opinions they provided, and they largely agreed on the core science in question. Although Dr. Huq did not establish that *all* post-febrile seizure events are necessarily related to the first, his overall interpretation of the record was clear, even if I did not ultimately accept it. And he persuasively

[19] I also note that no contemporaneous treaters opined that T.M.'s epilepsy stemmed from the first febrile seizure. Although treater opinions do not compel a fact finding either way, there is an absence of such evidence in this case in support of Petitioner's claim.

demonstrated that an initial febrile seizure could create the conditions for epilepsy. But *the record in this case* paints a picture of a child likely susceptible to seizure activity generally, but whose course could not be deemed inevitable once the first febrile seizure occurred. T.M.'s *initial* febrile seizure was likely vaccine-caused—but that seizure in turn was not the cause of what followed.[20]

I emphasize again—the contention that a vaccine-caused febrile seizure *could* constitute the first "domino" in a chronic seizure disorder is scientifically reliable. Bulwarked with evidence that a child's brain had likely been harmed by the initial seizure, such a claim could well produce a favorable entitlement decision, as occurred in *Ginn*. But a first, vaccine-caused seizure cannot be deemed responsible for epilepsy that is diagnosed later simply due to its temporal priority, in the absence of record evidence corroborating the presence of brain injury that could credibly be attributed to the vaccine-induced initial seizure. The initial seizure does not "prove" injury to the brain by itself. And the subsequent epileptic events cannot be relied upon either, since arguing that a single initial vaccine-caused seizure had to have caused what comes next amounts to the kind of "post hoc ergo propter hoc" reasoning rejected by the Program when considering causation-in-fact claims. *Pafford v. Sec'y of Health & Hum. Servs*., No. 01-0165V, 2004 WL 1717359, at *9 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied,* 64 Fed. Cl. 19 (2005), *aff'd*, 451 F. 3d 1352 (Fed. Cir. 2006).

III. Petitioner Did Not Establish that T.M.'s Pre-Vaccination Developmental Problems Were Exacerbated by Vaccination

My finding with respect to a lack of sufficient "did cause" proof that the first, vaccine-caused febrile seizure T.M. experienced was causal of her overall epilepsy also bears on Petitioner's claim that her pre-existing developmental delays were exacerbated by the December 2013 vaccinations. For even if the progression of her symptoms *literally* worsened after January 2014 (essentially all the Circuit now requires Petitioners need prove under *Loving*),[21] and even if I assume that a vaccine-induced febrile seizure could cause sufficient brain injury to worsen preexisting developmental issues, I do not also find that *in this case* the first, vaccine-caused febrile seizure was the source of worsening for T.M.—as must be established under *Loving* prong five (the counterpart to *Althen* prong two).

[20] The December 2013 seizure alone is not a basis for compensation even if Petitioner had limited her claim to it, since its sequelae did not last the six months necessary to meet the Act's severity requirement. *Watts v. Sec'y of Health & Hum. Servs.*, No. 17-1494V, 2019 WL 4741748, at *7 (Fed. Cl. Spec. Mstr. Aug. 13, 2019).

[21] In the wake of *Sharpe,* the first three *Loving* prongs can easily be satisfied—and are here. Hence, the record establishes in this case that (a) T.M.'s developmental issues existed pre-vaccination, and (b) those same issues became more pronounced in the following months, as she aged and it became more evident the degree and extent of her developmental delay (*Loving* prongs one and two). The intervening vaccinations in December 2013 predated the time when her developmental issues were more easily observed, making the determination that her delay was aggravated post-vaccination (the third *Loving* prong).

As noted above, the record does not support the conclusion that T.M.'s first seizure in December 2013 harmed her brain, or was otherwise causal of the febrile or breakthrough seizures (after missed anti-seizure medication doses) she experienced later in 2014 and then afterward. In addition, even though I cannot conclude that the same record preponderantly establishes that her preexisting developmental issues were *more likely* causal of her seizure disorder (rather than the other way around), Dr. Zempel's testimony, coupled with some of the filed medical literature, raised reasonable questions about that possibility that Petitioner did not effectively address (other than simply to maintain that febrile seizures in many cases *do* constitute a precursor event to a larger seizure disorder, or may hasten that occurrence due to brain injury—evidence of which is lacking here). Tr. at 219, 265–66; Baram & Hatalski at 1; Ben Ari & Holmes at 1056, 1060.

Thus, this record does not establish that the progression of T.M.'s developmental issues after the first febrile seizure was likely attributable to that seizure. Rather, awareness and understanding of the scope and nature of T.M.'s delay, as its clinical manifestations became more obvious, was occurring concurrently, and contemporaneously, with her slowly-unfolding epilepsy—*not* that the latter was worsening the former. Indeed, it could be concluded from this record (as already mentioned) that T.M.'s epilepsy was a secondary aspect of her developmental problems, or even "caused" by them initially. But this record does not preponderantly establish that a single, vaccine-caused initial febrile seizure aggravated her developmental problems.[22]

CONCLUSION

It is never a happy occasion when a special master denies entitlement to a person who has suffered greatly in the care of a child stricken with a debilitating disease or condition. This is unquestionably the case with respect to Ms. Weaver, who has struggled to help her daughter (and demonstrates great love for her in so doing). But my personal sympathies are not a basis for awarding damages. Rather, I must find that the legal standards for entitlement are met. And unfortunately, they have not been met in this case, since the initial, undoubtedly vaccine-related febrile seizure has not been shown to be the "linchpin" to what followed. Accordingly, I am compelled to DENY entitlement in this case.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[23]

[22] I also note that Petitioner does not argue herein that the December 2013 vaccinations worsened her developmental problems in some other form independent from the seizure activity, *e.g.*, by initiating some kind of autoimmune-mediated process that led to brain damage of some other form of encephalopathic injury. And there is no evidence in this case of any autoimmune injury—certainly T.M.'s epilepsy is not of the autoimmune form.

[23] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.

IT IS SO ORDERED.

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