In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 19-1750V
Filed: October 14, 2025

COURTNEY NINA and PEDRO NINA, as parents and natural guardians of K.N., a minor,

Petitioners,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Vanessa Lee Brice, Colling Gilbert Wright & Carter, Orlando, FL, for petitioners.
Mary Eileen Holmes, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On November 12, 2019, petitioners filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] on behalf of their daughter, K.N. (ECF No. 1.) Petitioners allege that K.N. experienced seizures and resulting developmental delay caused by her April 11, 2018 vaccinations, which included diphtheria-tetanus-acellular-pertussis ("DTaP"), influenza ("flu"), pneumococcal conjugate ("Prevnar-13"), polio, rotavirus, and Hemophilus Influenza B ("Hib") vaccines. (*Id.*) On July 19, 2024, petitioners filed a motion for a ruling on the written record. (ECF No. 94.) In the motion, they contend that K.N.'s condition constitutes either a Table Injury of encephalopathy relative to the DTaP vaccine or, alternatively, an injury caused-in-fact by the DTaP vaccination. (*Id.*)

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

[2] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

For the reasons discussed below, I now find that petitioners are *not* entitlement to compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury or death; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a causal link between the vaccination and the injury. § 300aa-11(c)(1); § 300aa-13(a)(1)(A)-(B).

In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination unless it is affirmatively shown that the injury was caused by some factor other than the vaccination.[3] § 300aa-13(a)(1)(A)-(B); § 300aa-11(c)(1)(C)(i); § 300aa-14(a). Pertinent to this case, "encephalopathy or encephalitis" may be Table Injuries if they occur within 72 hours of a DTaP vaccine. 42 C.F.R. § 100.3(a)(II)(B).

Table Injury cases are guided by a statutory "Qualifications and aids in interpretation" ("QAI"), which provides more detailed explanation of what should be considered when determining whether a petitioner has actually suffered an injury listed on the Vaccine Injury Table. § 300aa-14(b). Within the QAI, encephalopathy requires that an "acute encephalopathy occurs within the applicable time period and results in a chronic encephalopathy." 42 C.F.R. § 100.3(c)(2). For a child less than 18 months of age (K.N. received the vaccinations at issue at about six months of age), acute encephalopathy "is indicated by a significant decreased level of consciousness that lasts at least 24 hours" and, if following a seizure,[4] "cannot be attributed to a postictal state." 42 C.F.R. § 100.3(c)(2)(i)(A). However, seizures in themselves are not sufficient

[3] Of note, the Secretary of Health & Human Services has authority under the Vaccine Act to amend the Vaccine Injury Table. § 300aa-14(c)(1). Petitions in the program are resolved based on the Table as it existed at the time the petition was filed. § 300aa-14(c)(4). Thus, this case is governed by the Vaccine Injury Table that was in effect from March 21, 2017 to January 2, 2022, while the current Table became effective on January 3, 2022. However, none of the changes that became effective on January 3, 2022 are significant to this case.

[4] "Seizure includes myoclonic, generalized tonic-clonic (grand mal), and simple and complex partial seizures, but not absence (petit mal), or pseudo seizures. Jerking movements or staring episodes alone are not necessarily an indication of seizure activity." 42 C.F.R. § 100.3(d)(5).

to constitute a diagnosis of acute encephalopathy and shall not be viewed as the first symptom or manifestation of an acute encephalopathy absent other evidence. 42 C.F.R. § 100.3(c)(2)(i)(D). Moreover, "[t]he following clinical features in themselves do not demonstrate an acute encephalopathy or a significant change in either mental status or level of consciousness: Sleepiness, irritability (fussiness), high-pitched and unusual screaming, poor feeding, persistent inconsolable crying, bulging fontanelle, or symptoms of dementia." 42 C.F.R. § 100.3(c)(2)(i)(C). Instead, "decreased level of consciousness" is indicated by decreased or absent response to environment, decreased or absent eye contact, or inconsistent or absent responses to external stimuli. 42 C.F.R. § 100.3(d)(4)(i)-(iii). A chronic encephalopathy "occurs when a change in mental or neurologic status, first manifested during the applicable Table time period as an acute encephalopathy or encephalitis, persisted for at least 6 months from the first symptom or manifestation of onset."[5] 42 C.F.R. § 100.3(d)(1)(i).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1278-79; *Hines*, 940 F.2d at 1525. Under that standard, petitioners must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. They need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition at issue and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, petitioners must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[.]" *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Ultimately, petitioners must satisfy what has come to be known as the *Althen* test, which requires: (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. *Id*.

[5] "Individuals who return to their baseline neurologic state, as confirmed by clinical findings, within less than 6 months from the first symptom or manifestation of onset or of significant aggravation of an acute encephalopathy or encephalitis shall not be presumed to have suffered residual neurologic damage from that event; any subsequent chronic encephalopathy shall not be presumed to be a sequela of the acute encephalopathy or encephalitis." 42 C.F.R. § 100.3(d)(1)(ii).

A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human 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. § 300aa-13(b)(1). A petitioner may also rely upon circumstantial evidence. *See Althen*, 418 F.3d at 1280. The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3(b)(1). Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case, including having the discretion to decide cases without an evidentiary hearing. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). 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." § 300aa-13(b)(1). The special master is required to consider the entirety of the evidentiary record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

Petitioners filed an affidavit, as well as medical and school records between November of 2019 and May of 2021. (ECF Nos. 1, 9, 21-30, 41-43, 49-52.)[6] Respondent filed his Rule 4 Report, recommending against compensation, in January of

[6] Petitioner's counsel's filings and exhibit designations are difficult to follow. In the interest of clarity, all evidence filed by petitioner will be referenced by its ECF number and pagination.

2021. (ECF No. 45.) Respondent argued that the record supported neither a Table Injury nor causation-in-fact, suggesting that the evidence was limited to the fact that K.N.'s seizures and developmental delay followed the vaccinations placed at issue. (*Id*. at 9-11.)

Petitioners subsequently filed an expert opinion by pediatric neurologist Marcel Kinsbourne, M.D. (ECF No. 54), with supporting materials (ECF No. 55). In response, respondent filed reports by immunologist Andrew MacGinnitie, M.D., Ph.D., and pediatric neurologist Gregory Holmes, M.D. (ECF Nos. 62-63; Exs. A-D; *see also* ECF Nos. 64-67 (supporting materials).) Thereafter, I gave the parties preliminary guidance in connection with a Rule 5 status conference. (ECF No. 68.) Petitioners then filed updated medical records (ECF No. 71), two additional affidavits (ECF Nos. 74-75), a medical article (ECF No. 76), and two reports from Dr. Kinsbourne, representing separate responses to each of respondent's two expert reports (ECF No. 77 (regarding Dr. MacGinnitie's report) and ECF No. 78 (regarding Dr. Holmes's report). Respondent responded with supplemental reports by Drs. Holmes and MacGinnitie. (ECF No. 80; Exs. E-F.) Petitioners subsequently filed some of the medical literature cited in Dr. Kinsbourne's supplemental reports. (ECF Nos. 96-97.)

An entitlement hearing was set to commence in June of 2024. (ECF No. 83.) However, Dr. Kinsbourne passed away prior to the scheduled hearing and the hearing was therefore cancelled. (ECF No. 92.) I provided petitioners an opportunity to determine how they wished to proceed, but cautioned that if requesting an opportunity to secure a new expert, they would need to substantiate that need. (*Id*.) Because both parties had the opportunity to respond to the preliminary guidance provided in my Rule 5 Order, I advised that permitting petitioners to secure a different, testifying expert, would likely result in undue delay. (*Id*.) Ultimately, petitioners opted to resolve entitlement based on the existing record. (ECF No. 93.) Petitioners filed a motion for a ruling on the record on July 19, 2024. (ECF No. 94.) Respondent filed his response on September 10, 2024. (ECF No. 95.) Petitioners did not file any reply.

In light of the above, I have concluded that the parties have had a full and fair opportunity to develop the record and that it is appropriate to resolve this case without an entitlement hearing. S*ee Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (citing *Simanski v. Sec'y of Health & Human Servs*., 671 F.3d 1368, 1385 (Fed. Cir. 2012)); *see also* Vaccine Rule 8(d); Vaccine Rule 3(b)(2).

III. Factual History

K.N. was born in September of 2017 without complications. (ECF No. 26-1, p. 14.) She was a generally well child and received early vaccinations without issue. (ECF No. 22-1, pp. 1, 97-116.) However, beginning at about four months of age, it was noted that K.N. was not rolling, an expected developmental milestone. (*Id.* at 105-06.) She received her four-month routine vaccinations without issue. (*Id.* at 106-07.)

At her six-month checkup on March 28, 2018, K.N. was suffering an upper respiratory infection. (ECF No. 22-1, pp. 97-102.) Accordingly, her six-month vaccinations were postponed until her infection resolved. (*Id*. at 100.) It was noted that she could not roll on either side or pull to sit up without a head lag. (*Id*. at 98.) However, K.N.'s mother submitted an affidavit in which she stated that K.N. "made cooing sounds prior to being 5 months old, and once she was about 5 months old, she began to babble" and further averred that "[K.N.] was meeting all her developmental milestones when compared with her older brothers." (ECF No. 74-1, p. 1.)

K.N. received her six-month vaccinations – the vaccinations at issue in this case – two weeks later on April 11, 2018. (ECF No. 22-1, pp. 1, 95-96.) No physical exam was documented at that time. K.N. received her third dose of DTaP, Hib, Prevnar 13, polio, and rotavirus, as well as a seasonal flu vaccination. (*Id.*) According to K.N.'s mother, that evening was "uneventful." (ECF No. 74-1, p. 1.) However, the next morning, K.N.'s grandmother recalled that during breakfast K.N. "began flailing her arms, moving her head side-to-side, and screaming. She began to jerk her left arm back and forth uncontrollably." (ECF No. 75-1, p. 1.) "The first scream and jerking motions lasted a few seconds and then [K.N.] stopped. From that point on, the screaming and jerking motions in both of her arms and legs became intermittent with each session lasting approximately 30 seconds or more." (*Id*. at 1-2.)

K.N. presented to her pediatrician's office on April 12, 2018. (ECF No. 22-1, pp. 92-94.) She cried continuously through the exam and had several jerking movements in her legs lasting about 10 seconds. (*Id.* at 92-93.) The pediatrician suspected a seizure and recommended K.N. be taken to the hospital for further testing. (*Id*. at 93-94.) K.N. was admitted to the hospital later that day. (*Id*. at 84-85.) Although her temperature had been recorded as normal at the pediatrician's office (*Id*. at 92), it was 100.4 degrees upon her initial exam at the hospital when measured rectally (*Id*. at 85-86). The physical exam was otherwise unremarkable, though K.N. cried and screamed when moved. (*Id*.) K.N. was assessed with a new onset seizure and low-grade fever "likely secondary to vaccination" given the lack of any indicators of an infectious cause. (*Id*. at 86; ECF No. 24-1, p. 80.)

K.N. remained hospitalized from April 12 to April 15, 2018. (ECF No. 24-1, p. 50.) A lumbar puncture showed normal results.[7] (ECF No. 22-1, pp. 87, 90-91.) A CT

[7] Lumbar puncture can *sometimes* be considered a surgical intervention within the meaning of the Vaccine Act. *Leming v. Sec'y of Health & Human Servs*., 98 F.4th 1107, 1112-13 (Fed. Cir. 2024) (defining surgical intervention as any "surgical act or measure for diagnostic or therapeutic purposes taken to prevent harm of a patient or to improve the health of a patient"); *Galvan v. Sec'y of Health & Human Servs*., No. 20-313V, 2020 WL 4593163, at *11-13 (Fed. Cl. Spec. Mstr. July 6, 2020) (explaining that needle-based procedures can be, but are not necessarily, surgical), *mot. for rev. denied*, 151 Fed. Cl. 789 (2021); *Spooner v. Sec'y of Health & Human Servs*., No. 13-159V, 2014 WL 504728, at *12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014) (distinguishing lumbar puncture performed in the emergency department from lumbar puncture performed in an operating room under general anesthesia). In this case, however, K.N.'s procedure was performed bedside in the emergency department by a non-surgeon with only local anesthesia. Accordingly, there is not preponderant evidence that it constituted an act or measure of the surgical variety. *Elvira ex rel. D.E. v. Sec'y of Health & Human Servs.*, No. 17-531V, 2024 WL 4966035,

scan was also unremarkable. (*Id.* at 89.) However, a brain MRI showed "[s]ubcortical and right trigonal white matter abnormality" along with "terminal zones of myelination" and "[s]lightly more prominent focus of white matter signal abnormality in the left peritrigonal region." (ECF No. 22-1, pp. 81, 604.) She had a second cluster of seizures on April 13, 2018.[8] (ECF No. 25-1, p. 298.) An EEG[9] showed numerous electroclinical seizures lasting 10-60 seconds. (*Id.* at 452.) K.N. was diagnosed with focal seizures with apparent onset over the midline right central region. (*Id*. at 299.) Throughout her hospitalization, K.N. was treated with clonazepam, Keppra, and phenobarbital.[10] (ECF No. 22-1, p. 80.) She responded to treatment and was seizure free by April 14, 2018. (*Id*.) She was discharged after 48 hours of being stable and seizure free with a recommendation for a follow-up EEG in two weeks. (*Id*. at 82-83.)

At an April 18, 2018 follow up with her pediatrician, K.N. was noted to still be seizure free and she had begun to roll over at home. (ECF No. 22-1, pp. 67-68.) On physical exam she was "slightly hypotonic," but she vocalized well and was able to reach with her upper extremities. (*Id*. at 68-69.) She was diagnosed with both a seizure disorder and gross motor delay. (*Id*. at 69.) Physical therapy was recommended and K.N.'s parents were advised to hold further pertussis or flu vaccination pending genetic and metabolic testing. (*Id*.)

K.N. returned to the neurologist on April 25, 2018. (ECF No. 22-1, p. 59.) She had no new seizures or behavior changes and was continuing to take Keppra and phenobarbital without complications. (*Id*.) Her physical exam was normal, and she demonstrated all appropriate milestones for her age (sitting with support, reaching for objects using both hands, rolling over, babbling, making eye contact, reciprocal smiling,

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at *15-16 (Fed. Cl. Spec. Mstr. Nov. 6, 2024) (finding that a bedside lumbar puncture did not constitute a surgical intervention for purposes of § 300aa-11(c)(1)(D)).

[8] K.N.'s multiple seizure clusters are considered one seizure event, which is considered a "complex" seizure because it included multiple events over 24 hours. (ECF No. 78-1, pp. 7-8.)

[9] An electroencephalogram (EEG) features "a recording of the potentials on the skull generated by currents emanating spontaneously from the nerve cells in the brain" with fluctuations in potential being seen in the form of waves that correlate well with, and are used to diagnose, different neurologic conditions. *Electroencephalogram*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=15813 (last visited Sept. 24, 2025).

[10] Clonazepam is a benzodiazepine that is orally administered and used as an anticonvulsant in the treatment of myoclonic seizures. *Clonazepam*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=10139&searchterm=clonazepam (last visited Sept. 24, 2025). Keppra is the trademark name for levetiracetam, another orally administered anticonvulsant that is used "as an adjunct in the treatment of partial and myoclonic seizures and idiopathic generalized epilepsy." *Keppra*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=26816 (last visited Sept. 24, 2025); *Levetiracetam*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=28136 (last visited Sept. 24, 2025). Finally, phenobarbital is "a long-acting barbiturate" that is orally administered and "used as a sedative, hypnotic, and anticonvulsant." *Phenobarbital*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=38484 (last visited Sept. 24, 2025).

and tracking). Her EEG was normal. (*Id*. at 59, 61-62.) A metabolic work-up was normal, but a potential genetic etiology was still a consideration. (*Id*. at 63.) The neurologist diagnosed "localization-related (focal) (partial) epilepsy and epileptic syndromes with complex partial seizures, without mention of intractable epilepsy." (*Id*. at 62.) K.N. was to begin weaning off Keppra. (*Id*. at 63.)

As of a May 25, 2018 follow-up, K.N. was weaning off Keppra and her parents reported no new seizures. K.N. was meeting her motor and language milestones. (ECF No. 22-1, p. 43.) K.N. had a normal exam and continued weaning from Keppra was recommended. (*Id*. at 45-47.) Her diagnosis remained the same. (*Id*. at 47.)

K.N. had her nine-month well child visit with her pediatrician on July 13, 2018. (ECF No. 22-1, p. 33.) She was still seizure free. (*Id.* at 34.) She was expected to be off Keppra by the end of June but was expected to remain on phenobarbital for another six months. (*Id.*) She was able to sit briefly, hold herself up on her hands and knees, scoot backward, and say "mama" and "dada." (*Id.*) However, on physical exam, she had decreased truncal and lower extremity muscle tone. (*Id.* at 36-37.) The pediatrician was concerned regarding K.N.'s gross motor development, and physical therapy and developmental testing was therefore recommended. (*Id.* at 35.) The pediatrician planned to complete a VAERS form. (*Id.* at 37.) At a physical therapy evaluation on July 17, 2018, K.N. was assessed as having a two-month delay in gross motor development. (*Id.* at 29-30.)

On August 2, 2018, K.N. presented to the emergency department for seizures occurring in the context of a one-day history of an upper respiratory infection with fever. (ECF No. 22-1, pp. 18-21.) She was experiencing episodes of eye twitching and screaming with arm and leg stiffening lasting "seconds" and occurring every two minutes. (*Id*. at 18.) K.N. had been treated with clonazepam at home after the seizure had lasted more than five minutes. She had weaned off of Keppra by this time, but was still taking phenobarbital. (*Id*.) K.N. tested positive for rhinovirus/enterovirus and was assessed with a viral upper respiratory infection with fever and breakthrough seizures. (*Id*. at 18-19.) She remained hospitalized from August 3-5, was treated with a bolus of phenobarbital, and was discharged after being seizure free for 36 hours. (*Id.*) She was to continue phenobarbital and to add a higher dosage of clonazepam if any seizure lasted for more than five minutes. (*Id*. at 19.) K.N. followed up with her pediatrician the day after her discharge. (*Id.* at 22-24.) She was diagnosed with seizures with a provoking factor and a viral upper respiratory infection. (*Id*. at 24.) K.N. had a neurology follow up on August 10, 2018. (*Id.* at 12.) She remained seizure free following the recent hospitalization. (*Id*.) Her physical exam was normal, and the neurologist likewise concluded she had experienced breakthrough seizures in the setting of her infection. She remained off Keppra and continued on phenobarbital and clonazepam. (*Id.* at 14-16.)

K.N. had her twelve-month well visit with her pediatrician on October 1, 2018. (ECF No. 22-1, p. 6.) K.N. remained seizure free and her genetic test results showed no abnormalities. (*Id*. at 7.) She was enrolled in an Early Steps intervention program

and continued physical therapy. She was making progress with her motor development, but was "still not babbling consonant sounds." (*Id*.) Her physical exam was normal, except for "slightly" low muscle tone. (*Id.* at 8-9.) The pediatrician continued to delay vaccinations pending guidance from the neurologist. (*Id*. at 9.) Later that month, K.N. had a follow up with a new neurologist. She was doing well developmentally and with no regression. (ECF No. 41-1, pp. 29-32.) She was to continue her existing medications. (*Id.*)

K.N. returned to the pediatrician on November 2, 2018, for a sick visit. She had a temperature of 100.5 and was coughing and congested. She had been experiencing "mild seizures" in the context of five days of intermittent fever. (ECF No. 22-1, pp. 2-4.) It was confirmed that the neurologist had indicated that K.N. had no restriction from vaccination. (*Id*. at 2-3.) In pertinent part, K.N. had no physical or neurologic deficits on exam, and her diagnosis remained unchanged. (*Id.* at 3-4.) A history of fever was again recorded during her fifteen-month well visit on January 2, 2019. (*Id.* at 151-54.) She was reportedly doing much better with her gross motor development. She was babbling, cruising, walking when holding onto hands, pointing, and saying "dada." (*Id*. at 152.) Her exam was normal, except for decreased truncal tone. (*Id.* at 153-54.) She followed up several weeks later for her vaccinations, receiving a Prevnar booster on January 25, 2019, and a fourth dose of Hib on February 11, 2019. (*Id.* at 149-50.)

On February 13, 2019, K.N. completed a Florida First Steps evaluation at the age of sixteen months. (ECF No. 27-1, p. 8.) She had age-appropriate social skills, but was delayed in expressive and receptive language skills. (*Id.*) She was eligible for assistance due to developmental delays in communication, adaptive skills, and gross motor skills. (*Id*. at 8-10.) K.N. had her eighteen-month well visit on April 1, 2019. (ECF No. 22-1, p. 144.) At this time, she was instructed to begin weaning phenobarbital over the course of the next three and a half months. K.N.'s ongoing early intervention and physical therapy was discussed. It was noted that K.N. was doing better with receptive language skills than expressive. (ECF No. 22-1, p. 144.) K.N. had a normal physical and neurologic exam, and she was newly assessed as having an expressive language delay in addition to her unchanged seizure disorder and gross motor delay. (*Id.* at 146-47.)

The remainder of K.N.'s medical records need not be summarized at length. K.N. continued to make progress with interventions for her developmental delays; however, she did remain delayed. As of July 17, 2019, she was diagnosed with a mixed receptive and expressive language disorder. (ECF No. 23-1, pp. 1-3.) She entered Pre-K in August of 2020 with an individualized education plan ("IEP") to address her developmental delays. (ECF No. 43-1; ECF No. 41-1, p. 6; ECF No. 49-1, pp. 16-17.) Around this time, a comprehensive epilepsy panel was negative for any evidence of a pathogenic variant. (ECF No. 50-1, pp. 59-63.) She received subsequent vaccinations, including a fourth dose of DTaP, without adverse reactions. (ECF No. 22-1, pp. 142, 147.) And she remained seizure free, except during febrile illnesses. (ECF No. 41-1, pp. 1, 15.)

IV. Summary of Expert Opinions

a. Petitioners' Expert, Neurologist Marcel Kinsbourne, M.D.[11] – Initial Report

Dr. Kinsbourne noted that, prior to the vaccinations at issue, K.N.'s development was mostly recorded as normal, though she was not yet rolling over at six months of age and could not pull to sit without head lag. (ECF No. 54, pp. 1, 5.) He observed that, although her six-month vaccinations were initially delayed by two weeks due to illness, her temperature was normal at the time the vaccines were administered on April 11, 2018. (*Id.* at 1.) Thereafter, about 16-17 hours post-vaccination, K.N. experienced the onset of the first of three "clusters of brief seizures,"[12] with the third set of seizures being observed in the primary care provider's office on April 12. (*Id.* (citing Ex. 2, p. 92).) By the time K.N. was seen at the emergency department, she had a temperature of 100.4, which was attributed to her vaccinations, and an abnormal EEG showed epileptiform discharges localized in the midline and right central region of the cortex, as well as numerous electroclinical seizures. (*Id.* at 2.) However, K.N. was "alert and responsive" between seizures. (*Id.*) Antiepileptic drugs suppressed the episode for four months, until a recurrence was provoked by an upper respiratory infection. (*Id*. at 5-6.) Although K.N.'s gross motor delay began prior to the vaccinations at issue, "her main deficit is in language development, which presented subsequently and was still apparent when she was three years old." (*Id.* at 6.)

According to Dr. Kinsbourne, K.N.'s seizures were "Rolandic seizures," which are focal and brief (from 1-3 minutes) centrotemporal seizures. (ECF No. 54, p. 6.) The location of K.N.'s seizures within her brain, her prior developmental delay, age at onset, and progression to tonic-clonic seizures, are all consistent with benign epilepsy with

[11] Dr. Kinsbourne's curriculum vitae was not filed into the record of his case; however, his credentials are well known in the program and to the undersigned. *See, e.g. Rodela v. Sec'y of Health & Human Servs.*, No. 17-236V, 2024 WL 2034220 (Fed. Cl. Spec. Mstr. Apr. 5, 2024). Dr. Kinsbourne received his bachelor's degree from Oxford University in 1952, his medical degree from Oxford in 1952, his master's degree from Oxford University in 1956, and his doctorate degree from Oxford in 1963. He also received an American medical license from the state of North Carolina in 1967. In his clinical capacity, Dr. Kinsbourne served as a house physician and/or house surgeon at six different hospitals in London, with a focus on pediatrics and neurosurgery in his most recent positions. He also served as registrar and senior registrar for the Neurology Department at United Oxford Hospitals and as a registrar for the Neurology Department at the Hospital for Sick Children in London. Dr. Kinsbourne held numerous academic positions, including lecturer in experimental psychology at Oxford University, professor of pediatrics and neurology at Duke University Medical Center, and professor of pediatric neurology at the University of Toronto. Most recently, he had served as a senior fellow at the Center for the Study of Aging and Human Development at Duke University, an adjunct professor of neurology at Boston University School of Medicine, a research professor at the Center for Cognitive Studies at Tufts University, and a professor of psychology at New School University. Dr. Kinsbourne published over 400 articles on pediatrics and psychology. He also published his own book on child psychology, as well as edited eight different books on pediatrics and psychology. *Id.* at *9 n.11.

[12] Dr. Kinsbourne characterized the seizures as follows: "Initially, the seizures consisted of twitching of the left arm and leg and repetitive blinking. Then the twitching became bilateral." (ECF No. 54, p. 1.)

centrotemporal spikes ("BECTS"), which is also known as Rolandic epilepsy. (*Id.* (citing K.P. Vinayan et al., *Educational Problems with Underlying Neurophyschological Impairment Are Common in Children with Benign Epilepsy of Childhood with Centrotemporal Spikes (BECTS)*, 14 SEIZURES 207 (2005) (ECF No. 55-18); Meng-Han Tsai et al., *Clinical Genetic Study of the Epilepsy-Aphasia Spectrum*, 54 EPILEPSIA 280 (2013) (ECF No. 55-17); Chrysostomos P. Panayiotopoulos et al., *Benign Childhood Focal Epilepsies: Assessment of Established and Newly Recognized Syndromes*, 131 BRAIN 2264 (2008) (ECF No. 55-13)).) Additionally, having associated K.N.'s seizure disorder with a deficit in language development, Dr. Kinsbourne indicated that there is an "epilepsy-aphasia spectrum" of disorders that ranges from severe epileptic encephalopathy to mild childhood epilepsy with centrotemporal spikes. (*Id*. at 7.) Along that spectrum, Dr. Kinsbourne suggested that Landau-Kleffner syndrome ("LKS") is a "well-documented condition that combines a seizure disorder with language delay or deficit" and has a varied presentation, though he stopped short of explicitly opining that K.N. suffered LKS specifically. (*Id*. at 6-7 (citing Gerry A. Stafanatos et al., *Acquired Epileptiform Aphasia: A Dimensional View of Landau-Kleffner Syndrome and the Relation to Regressive Autistic Spectrum Disorders*, 8 CHILD NEUROPHYSIOLOGY 195 (2002) (ECF No. 55-15)).)

According to Dr. Kinsbourne, while the mechanism by which BECTS can cause language disorders is not known, a study by Kagitani-Shimono et al., demonstrates that the epileptic spikes in BECTS have a negative influence on responsiveness to auditory language comprehension in the language-associated cortices and, thus, can change the functional development of the language network. (ECF No. 54, pp. 7-9, 13 (citing Kuriko Kagitani-Shimono et al., *Abnormal Cortical Activation During an Auditory Word Comprehension Task in Benign Childhood Epilepsy with Centrotemporal Spikes: A Magnetoencephalographic Study*, 87 EPILEPSY & BEHAV. 159 (2018) (ECF No. 55-9)); *see also* Ramya Ghantasala & Gregory L. Holmes, *Benign Rolandic Epilepsy: Widespread Increases in Connectivity in a Focal Epilepsy Syndrome*, 21 EPILEPTIC DISORDERS 567 (2019) (Ex. C, Tab 18); René M.H. Besseling et al., *Aberrant Functional Connectivity Between Motor and Language Networks in Rolandic Epilepsy*, 107 EPILEPSY RSCH. 253 (2013) (ECF No. 55-2); Sara Bulgheroni et al., *Verbal Dichotic Listening Performance and Its Relationship with EEG Features in Benign Childhood Epilepsy with Centrotemporal Spikes*, 79 EPILEPSY RSCH. 31 (2008) (ECF No. 55-3).) In that regard, Dr. Kinsbourne stated that "[K.N.]'s Rolandic seizures proved to be mild and were rapidly controlled by anti-epileptic treatment. The language impairment that followed was typical also, but not mild at all, implicating executive language and to a somewhat lesser degree, receptive language." (*Id*. at 7.) According to Dr. Kinsbourne, the fact that K.N. had not yet developed speech is immaterial to the ability of her condition to cause aphasia. (*Id*. at 8-9.)

Dr. Kinsbourne opined that three of K.N.'s vaccinations – DTaP, Prevnar-13, and flu – are known to be capable of inducing seizure activity in susceptible children. (ECF No. 54, p. 10.) However, he presented data only for pertussis-containing vaccinations, some publications relating to whole cell pertussis-containing vaccines, which are not at issue in this case, and some pertaining to the later acellular pertussis vaccines. (*Id*. at

10-12 (citing Dennis A. Conrad & Hal B. Jensen, *Using Acellular Pertussis Vaccines for Childhood Immunization: Potential Benefits Far Outweigh Potential Risk*, 105 POSTGRADUATE MED. 165 (1999) (ECF No. 55-6); Nicole 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 (2003) (ECF No. 55-11); Lisa A. 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) (ECF No. 55-8; *see also* Ex. F, Tab 4); Weibke Hellenbrand et al., *The Epidemiology of Pertussis in Germany: Past and Present*, BMC INFECTIOUS DISEASES, Feb. 2009 (ECF No. 55-4); Sarah von Spiczak et al., *A Retrospective Population-Based Study on Seizures Related to Childhood Vaccination*, 52 EPILEPSIA 1506 (2011) (ECF No. 55-14); CENTER FOR DISEASE CONTROL & PREVENTION, NO. RR-7, PERTUSSIS VACCINATION: USE OF ACELLULAR PERTUSSIS VACCINES AMONG INFANTS AND YOUNG CHILDREN: RECOMMENDATION OF THE ADVISORY COMMITTEE ON IMMUNIZATION PRACTICES (ACIP) (1997) [hereinafter MMRW] (ECF No. 97-11); CDC Vaccine Information Statement for DTaP (not filed); Yuelian Sun et al., *Risk of Febrile Seizures and Epilepsy After Vaccination with Diphtheria, Tetanus, Acellular Pertussis, Inactivated Poliovirus, and* Haemophilus Influenzae *Type B*, 307 JAMA 823 (2012) (ECF No. 55-16); Gregory L. Holmes & Yehezkiel Ben-Ari, *The Neurobiology and Consequences of Epilepsy in the Developing Brain*, 49 PEDIATRIC RSCH. 320 (2001) (ECF No. 55-7)).) Based on this, Dr. Kinsbourne opined that there is "widely accepted evidence that there is a risk of seizures after DTaP within a three-day risk interval after vaccination." (*Id*. at 10.) He contended that the transition from whole cell to acellular pertussis "radically reduced" the frequency of post-vaccination seizures, but that, "[n]evertheless, such seizures still occur." (*Id*.)

Further to this, Dr. Kinsbourne opined that a DTaP-caused seizure (excepting benign febrile seizures) can usher in epilepsy. (ECF No. 54, p. 12 (citing Holmes & Ben-Ari, *supra*, at ECF No. 55-7).) He contended that "[s]eizures facilitate more seizures," because they produce IL-1β, which is a pro-inflammatory cytokine that it itself epileptogenic. (*Id*. (citing Ye Wang et al., *Interictal Cytokine Levels Were Correlated to Seizure Severity of Epileptic Patients: A Retrospective Study on 1218 Epileptic Patients*, 13 J. TRANLAT'L MED. 378 (2015) (ECF No. 55-19)).) Thus, Dr. Kinsbourne opined:

> When a vaccination triggers a seizure, the seizure is not stamped with its vaccine origin, any more than it would be labeled with whatever infectious process might have brought the seizure about. Regardless of what triggered the seizure, if it lasts longer than 15 minutes, or by some accounts, 10 minutes, is focal or repeats within 24 hours, it is well known to harbor a significant risk of recurrence, which can rise to the level of epilepsy. That is, the vaccine-provoked complex partial seizures can launch epileptogenesis.

(*Id*.)

b. Respondent's Expert, Neurologist Gregory Holmes, M.D.[13] – Initial Report

Dr. Holmes did not agree with Dr. Kinsbourne's diagnostic opinion that K.N. suffered an epilepsy-aphasia syndrome. (Ex. C, p. 13.) He explained that K.N. suffered from complex febrile seizures. Because her documented seizures were associated with fever, her condition does not meet the medical definition of epilepsy.[14] (*Id*.) Moreover, K.N.'s overall clinical course does not support Dr. Kinsbourne's assessment. K.N.'s developmental delay pre-dated her febrile seizures and, although she is at higher risk of developing non-febrile seizures in the future, she had a normal EEG and is not being treated with chronic anti-seizure medications. (*Id*. at 13, 20 (citing ECF No. 50-1, p. 58).)

Furthermore, Dr. Holmes indicated that "K.N. clearly had no features of BECTS."[15] (Ex. C, p. 16.) He opined that "infants and toddlers do not develop BECTS,"

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[13] Dr. Holmes received his medical degree from the University of Virginia School of Medicine in 1974, before going on to complete an internship and residency in pediatrics at Yale University School of Medicine in 1975 and 1976, respectively, as well as a residency in pediatric neurology at the University of Virginia School of Medicine in 1979. (Ex. D, p. 1.) From there, Dr. Holmes accepted a position as an assistant professor of neurology and pediatrics at the University of Connecticut Health Center. (*Id.* at 2.) He was promoted to associate professor before moving on to Medical College of Georgia, followed by Harvard Medical School where he was finally promoted to professor of neurology. (*Id.*) Then, in 2002, he accepted a position as a professor of neurology and pediatrics at Dartmouth Medical School. (*Id.*) However, since 2013, Dr. Holmes has worked as a professor of pediatrics and a professor and Chair of neurological sciences at Lancer College of Medicine at the University of Vermont. (*Id.*) He is board certified in pediatrics, clinical neurophysiology, and psychiatry and neurology with special competence in child neurology, and he maintains an active medical license in Vermont. (*Id.*) In his research capacity, Dr. Holmes has published 314 peer-reviewed research papers, 142 review articles, 78 books and chapters, 62 book reviews, and 396 abstracts, among other publications. (*Id.* at 33-110.)

[14] He explained that "[e]pilepsy is defined as i) at least two unprovoked (or reflex) seizures occurring more than 24 hours apart; ii) one unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years; or iii) diagnosis of an epilepsy syndrome." (Ex. C, p. 13 (citing Robert S. Fisher et al., *Epileptic Seizures and Epilepsy: Definitions Proposed by the International League Against Epilepsy (ILAE) and the International Bureau of Epilepsy (IBE)*, 46 EPILEPSIA 470 (2005) (Ex. C, Tab 1); Robert S. Fisher, *Redefining Epilepsy*, 28 CURRENT OP. NEUROLOGY 130 (2015) (Ex. C, Tab 2); Robert S. Fisher et al., *A Practical Clinical Definition of Epilepsy*, 55 EPILEPSIA 475 (2014) (Ex. C, Tab 3)).)

[15] He explained that "BECTS is an idiopathic localization-related (i.e. focal) electroclinical syndrome that has an annual incidence of approximately 21 per 100,000 in children younger than 15 years of age . . . . This common childhood epileptic syndrome is characterized by focal seizures with a seizure semiology consisting of unilateral facial sensorimotor symptoms, oropharyngolaryngeal symptoms, speech arrest, and hypersalivation." (Ex. C, p. 16 (citing J. Heijbel & M. Bohman, *Benign Epilepsy of Children with Centrotemporal EEG Foci: Intelligence, Behavior, and School Adjustment*, 16 EPILEPSIA 679 (1975) (Ex. C, Tab 15); Ramya Ghantasala & Gregory L. Holmes, *Benign Rolandic Epilepsy: Widespread Increases in Connectivity in a Focal Epilepsy Syndrome*, 21 EPILEPTIC DISORDERS 567 (2019) (Ex. C, Tab 18); P. Loiseau & M. Beaussart, *The Seizures of Benign Childhood Epilepsy with Rolandic Paroxysmal Discharges*, 14 EPILEPSIA 381 (1973) (Ex. C, Tab 19); M. Beaussart & R. Faou, *Evolution of Epilepsy with Rolandic Paroxysmal Foci: A Study of 324 Cases*, 19 EPILEPSIA 337 (1978) (Ex. C, Tab 20); Gregory L. Holmes, *Rolandic Epilepsy: Clinical and Electroencephalographic Features*, 6 EPILEPSY RSCH.

indicating that onset occurs between ages 3 to 13 years. (*Id*. (citing Paul A.D. Bouma et al., *The Course of Benign Partial Epilepsy of Childhood with Centrotemporal Spikes: A Meta-Analysis*, 48 NEUROLOGY 430 (1997) (Ex. C, Tab 16); Petra M.C. Callenbach et al., *Long Term Outcome of Benign Childhood Epilepsy with Centrotemporal Spikes: Dutch Study of Epilepsy in Childhood*, 19 SEIZURE 501 (2010) (Ex. C, Tab 17)).) And, in any event, BECTS only results in language loss if it includes Electrical Status Epilepticus of Sleep ("ESES"), which K.N. never experienced. (*Id*. (citing Gregory L. Holmes & Peirre-Pascal Lenck-Santini, *Role of Interictal Epileptiform Abnormality in Cognitive Impairment*, 8 EPILEPSY & BEHAV. 504 (2006) (Ex. C, Tab 28); Pinar Gencpinar et al., *Electrical Status Epilepticus in Sleep (ESES)/Continuous Spikes and Waves During Slow Sleep (CSWS) Syndrome in Children: An Electroclinical Evaluation According to the EEG Patterns*, 61 EPILEPSY & BEHAV. 107 (2016) (Ex. C, Tab 29); C.S. Tassinari et al., *Encephalopathy with Electrical Status Epilepticus During Slow Sleep or ESES Syndrome Including the Acquired Aphasia*, 111 CLINICAL NEUROPHYSIOLOGY S94 (2000) (Ex. C, Tab 30)).) Although Dr. Kinsbourne cited Tsai et al. for the proposition that K.N. could have developed BECTS despite being in infancy, Dr. Holmes explained that the subjects within the Tsai study were not similar to K.N. and that "all probands had abnormal EEG studies with typical centrotemporal epileptiform discharges activated by sleep," which K.N. never had, and that, while the authors did note seizure onset as early as 6 months of age, they did not provide data regarding whether cognitive regression occurred. (*Id*. at 16-17.) At six months of age, and with no language development, it is impossible to state that K.N.'s seizures resulted in either a plateauing or regression of language development. (*Id.* at 17.) Similarly, Dr. Holmes noted that K.N. is likely too young to have developed LKS, which typically begins with a loss of language skills at around five years of age. (*Id.*) Moreover, LKS typically shows repetitive spikes, sharp waves, and spike-and-wave activity in the temporal region or parieto-occipital regions, bilaterally. (*Id.*) Thus, "K.N. clearly has no features of LKS." (*Id*. at 18.) Her "seizure semiology, clinical course and EEG findings are totally inconsistent with the diagnosis." (*Id*.)

Dr. Holmes also disagreed with several points underlying Dr. Kinsbourne's theory of causation. He opined:

> Since the DTaP vaccine can cause fever, albeit at low rate, it is possible that in rare cases febrile seizures could occur following immunization. However, the outcome of febrile seizures is no different if an immunization resulted in the fever than if the seizure was caused by another infection. Further, there is no evidence that a febrile seizure would result in an epilepsy-aphasia syndrome in a 6-month-old child.

(Ex. C, p. 16 (citations omitted) (citing Michael E. Pichichero, *Acellular Pertussis Vaccines: Towards an Improved Safety Profile*, 15 DRUG SAFETY 311 (1996) (Ex. C, Tab

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SUPPLEMENT 29 (1992) (Ex. C, Tab 21); Gregory L. Holmes, *Benign Focal Epilepsies of Childhood*, 34 EPILEPSIA S49 (1993) (Ex. C, Tab 22); Gregory L. Holmes, *Clinical Spectrum of Benign Focal Epilepsies of Childhood*, 41 EPILEPSIA 1051 (2000) (Ex. C, Tab 23)).)

12); Deborah G. Hirtz et al., *Seizures Following Childhood Immunizations*, 102 J. PEDIATRICS 14 (1983) (Ex. C, Tab 13)).)

Based on his own review of the relevant epidemiology, Dr. Holmes opined that "[t]he link between DTaP and seizures of any type is weak," noting in particular the conclusion of the Institute of Medicine ("IOM")[16] Committee to Review Adverse Effects of Vaccines, which indicated it had "limited confidence" in the epidemiologic evidence of a link between DTaP and seizures. (Ex. C, p. 14.) Of the studies cited by Dr. Kinsbourne, Le Saux et al., Conrad and Jenson, and Jackson et al. did not include any control group and did not assess for epilepsy. (*Id*. at 14-15 (discussing Le Saux et al., *supra*, at ECF No. 55-11; Conrad & Jensen, *supra*, at ECF No. 55-6; Jackson et al., *supra*, at ECF No. 55-8).) And, while von Spiczak et al. did examine epilepsies, it likewise did not include any control. (*Id.* at 15 (discussing von Spiczak et al., *supra*, at ECF No. 55-14).) Sun et al. did find a small increased risk of febrile seizures, but it was only evidenced on "day 0," meaning the day of vaccination. (*Id.* (discussing Sun et al., *supra*, at ECF No. 55-16).) K.N.'s seizure, which occurred the day following vaccination, occurred at a time when Sun et al. had not found an increased risk of seizures. (*Id*.)

Moreover, Dr. Holmes opined that Dr. Kinsbourne "mischaracterizes the literature" with respect to his contention that "seizures may cause more seizures." (Ex. C, p. 18 (quoting ECF No. 54, p. 12).) He explained that,

> [e]ven though seizures, like other secondary experiences, change the brain, it does not mean that a seizure or series of seizures render the brain "epileptic." Evidence from multiple sources regarding the nature and natural history of seizures and epilepsy in humans has repeatedly demonstrated that in most cases the occurrence of seizures itself does not influence the long-term outcome of epilepsy.

(*Id.* at 18-19.) Dr. Holmes acknowledged that in some syndromes deterioration is progressive; however, in those instances, it is the underlying syndrome, rather than the seizures, that results in deterioration. (*Id*. at 19.) Although "extremely prolonged" seizures can be damaging, any broader notion that childhood seizures beget further seizures is incompatible with the fact that greater than 60% of cases of childhood epilepsy go into complete remission. (*Id*. (citing Anne T. Berg & Karen Rychlik, *The Course of Childhood-Onset Epilepsy Over the First Two Decades: A Prospective, Longitudinal Study*, 56 EPILEPSIA 40 (2015) (Ex. C, Tab 48)).) If Dr. Kinsbourne's assertion were correct, then childhood epilepsy would generally be progressive and the

[16] The Institute of Medicine (known as the National Academy of Medicine since 2015) is the medical arm of the National Academy of Sciences. The National Academy of Sciences ("NAS") was created by Congress in 1863 to be an advisor to the federal government on scientific and technical matters (*see* An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863)), and the Institute of Medicine is an offshoot of the NAS established in 1970 to provide advice concerning medical issues. When Congress enacted the Vaccine Act in 1986, it directed that the IOM conduct studies concerning potential causal relationships between vaccines and illnesses. *See* § 300aa-1 note.

number of remissions "quite low." (*Id.*) Dr. Holmes disputed Dr. Kinsbourne's assertion that a "vaccine-provoked complex partial seizure can launch epileptogenesis," suggesting that Dr. Kinsbourne equated risk with causation. (*Id.* at 19-20.) Although a small percentage of children with febrile seizures go on to develop afebrile seizures, this risk is not as great as the risk for children with developmental delays. (*Id.* at 19 (citing Karin B. Nelson & Jonas H. Ellenberg, *Predictors of Epilepsy in Children Who Have Experienced Febrile Seizures*, 295 NEJM 1029 (1976) (Ex. C, Tab 52); John F. Annegers et al., *Factors Prognostic of Unprovoked Seizures After Febrile Convulsions*, 316 NEJM 495 (1987) (Ex. C, Tab 53)).) Rather than the febrile seizures being causal of epilepsy, Dr. Holmes indicated that febrile seizures are thought to be "an age-specific marker for a predisposition to epilepsy." (*Id*. at 20 (citing Shlomo Shinnar et al., *The Risk of Seizures Recurrence After a First Unprovoked Afebrile Seizures in Childhood: An Extended Follow-Up*, 98 PEDIATRICS 216 (1996) (Ex. C, Tab 57)).)

c. Respondent's Expert, Immunologist Andrew MacGinnitie, M.D., Ph.D.[17] – Initial Report

Dr. MacGinnitie deferred to Dr. Holmes with respect to the type of seizures or seizure disorders that may be at issue. (ECF No. A, p. 6.) He noted that "the diagnosis of seizures in the setting of a fever does not seem to be in doubt in this case with onset within 24 hours of K.N. receiving her 6-month vaccinations." (*Id*.) However, he raised a number of points as counseling against Dr. Kinsbourne's opinion that the vaccines were causally implicated in K.N.'s condition.

Dr. MacGinnitie challenged Dr. Kinsbourne's reliance on elevated IL-1β as a mechanism by which one seizure may lead to the generation of additional seizures. (ECF No. A, p. 6.) In contrast to Dr. Kinsbourne's citation to Wang et al., which examined post-seizure cytokines in epilepsy, Dr. MacGinnitie cited a study by Choi et al., which showed that a first febrile seizure did not significantly increase IL-1β. (*Id*. (citing Jieun Choi et al., *Increased Levels of HMGB1 and Pro-Inflammatory Cytokines in Children with Febrile Seizures*, 8 J. NEUROINFLAMMATION, no. 135, 2011, at 1 (Ex. C, Tab 1)).) Further, animal model studies have shown that IL-1 is safe and effective as an adjuvant without significant adverse effects, including seizures. (*Id*. at 7 (citing Charles A. Dinarello, *Interleukin-1 in the Pathogenesis and Treatment of Inflammatory Diseases*,

[17] Dr. MacGinnitie received his Ph.D. in pathology and medical degree from Pritzker School of Medicine at the University of Chicago in1996 and 1998, respectively, before going on to complete a residency in pediatrics at Boston Combined Residency Program in 2001, as well as a fellowship in allergy/immunology at the Children's Hospital Boston and a clinical fellowship in pediatrics at Harvard Medical School in 2004. (Ex. B, p. 1.) From there, Dr. MacGinnitie accepted a position as an assistant professor in pediatrics at the University of Pittsburgh. (*Id.*) He moved on to Harvard Medical School in 2011, where he was eventually elevated to associate professor of pediatrics. (*Id.* at 1-2.) During that time, Dr. MacGinnitie also maintained a clinical position as an attending physician in pediatrics allergy/immunology, first at Children's Hospital of Pittsburgh of UPMC and then at Children's Hospital Boston. (*Id.* at 2.) He is board certified in pediatrics, as well as allergy and immunology, and he maintains an active medical license in Massachusetts. (*Id.* at 10-11.) He has published 42 peer-reviewed articles; 3 reviews, chapters, monographs, and editorials; 1 clinical guideline; and 1 abstract, poster presentation, and exhibits. (Id. at 12-17.)

117 BLOOD 2011 (Ex. A, Tab 3); Herman F. Staats & Francis A. Ennis, Jr., *IL-1 Is an Effective Adjuvant for Mucosal and System Immune Responses When Coadministered with Protein Immunogens*, 162 J. IMMUNOLOGY 6141 (1999) (Ex. A, Tab 4); Michael A. Egan et al., *A Comparative Evaluation of Nasal and Parenteral Vaccine Adjuvants to Elicit Systemic and Mucosal HIV-1 Peptide-Specific Humoral Immune Responses in Cynomolgus Macaques*, 22 VACCINE 3774 (2004) (Ex. A, Tab 5); William M. Gwinn et al., *Effective Induction of Protective Systemic Immunity with Nasally Administered Vaccines Adjuvanted with IL-1*, 28 VACCINE 6901 (2010) (Ex. A, Tab 6)).) Dr. MacGinnitie also opined that the acellular pertussis vaccine is not associated with an increased risk of seizures. (*Id*. at 8-9.) In addition to challenging Dr. Kinsbourne's reliance on the Le Saux et al., Conrad and Jenson, and Sun et al. studies (*Id.*), Dr. MacGinnitie cited a study by Huang et al. as finding no increased risk of seizures after acellular pertussis vaccination (*Id*. at 9-10 (citing Wan-Ting Huang et al., *Lack of Association Between Acellular Pertussis Vaccine and Seizures in Early Childhood*, 126 PEDIATRICS e263 (2010) (Ex. A, Tab 15; *see also* Ex. F, Tab 2))). He also cited mouse model study that found that whole cell pertussis, but not acellular pertussis, induced seizures and elevated levels of IL-1β in the central nervous system. (*Id.* at 7 (citing Sheila Donnelly et al., *Whole-Cell but Not Acellular Pertussis Vaccines Induce Convulsive Activity in Mice: Evidence of a Role for Toxin-Induced Interleukin-1β in a New Murine Model for Analysis of Neuronal Side Effects of Vaccination*, 69 INFECTION & IMMUNITY 4217 (2001) (Ex. A, Tab 2)).)

Dr. MacGinnitie opined that vaccination is only a "minor" immune stimulus. (ECF No. A, pp. 7-8.) Absent ongoing immune dysfunction (which would otherwise be present over years), K.N.'s vaccinations represent an immune stimulus "similar to what the human immune system is routinely exposed to." (*Id*. at 8.) In that regard, Dr. MacGinnitie noted that none of K.N.'s treating physicians opined that K.N.'s seizures were caused by her vaccinations and she received subsequent vaccinations without recurrence of her seizures. (*Id*.) Like Dr. Holmes, Dr. MacGinnitie also stressed that K.N.'s developmental delay pre-dated her seizures. (*Id*. at 10.)

d. Dr. Kinsbourne's Supplemental Reports[18]

Dr. Kinsbourne presented two supplemental reports, one responding to Dr. MacGinnitie's initial report (ECF No. 77-1) and one responding both to Dr. Holmes's report and the undersigned Rule 5 Order (ECF No. 78-1).

[18] Petitioners initially failed to file any of the literature accompanying Dr. Kinsbourne's supplemental report. They later filed some of the literature after prompting. (ECF Nos. 96-97.) However, some of the literature cited by Dr. Kinsbourne has never been filed (also including some of the literature cited in Dr. Kinsbourne's initial report). In some instances, the same literature has been filed by respondent, and citation is made to respondent's filings where possible. Statements contained in Dr. Kinsbourne's supplemental reports that are supported by citations to medical literature that has not otherwise been filed into the record have still been considered, but have been credited only as Dr. Kinsbourne's *ipse dixit*.

i. Response to Dr. MacGinnitie

Dr. Kinsbourne sought to challenge Dr. MacGinnitie's reliance on the study by Choi et al. Noting that Dr. MacGinnitie relied on one specific finding within the study (pertaining to a first febrile seizure), Dr. Kinsbourne characterized the evidence drawn from Choi et al. as "scant." (ECF No. 77-1, p. 1.) He suggested that "massive medical literature" contradicts Dr. MacGinnitie's assertion, though he provided no accompanying citation. (*Id.* at 3.) Dr. Kinsbourne noted that Dr. MacGinnitie had suggested that, absent elevated IL-1β, a first febrile seizure could not be implicated as the cause of a lower seizure threshold. (*Id.* at 1.) He responded that "[i]f this were so, seizures triggered by infections (and vaccinations) would lack the means to trigger further seizures and be 'isolated' (i.e., they could not 'beget' more seizures)." (*Id.*) Dr. Kinsbourne felt this would be "counterintuitive" and suggested that it is counter to "widely accepted medical literature" that indicates that neuroinflammation has a "bidirectional relationship" with seizures. (*Id.* (citing Costagliola, et al., *Targeting inflammatory mediators in epilepsy: A systematic review of its molecular basis and clinical applications*, 11 FRONTIERS IN NEUROLOGY 13 (2022); 741244 (not filed).) Dr. Kinsbourne felt it notable that, despite questioning the causal role of IL-1β, Dr. MacGinnitie did not offer any alternative mechanism for ictogenesis. (*Id.* at 3.) In any event, Dr. Kinsbourne charged that the Choi et al. finding cited by Dr. MacGinnitie is inapposite because K.N. suffered three clusters of seizures in one day, meaning that her seizures were "dramatically recurrent" and the Choi study did find that recurrent seizures generated increased IL-1β. (*Id.* at 2.)

Dr. Kinsbourne agreed that, in general, vaccination is a minor immune stimulus. (ECF No. 77-1, p. 3.) However, he suggested that genetic variants, such as SCN1A (which is not at issue in this case), explain why in some cases "the vaccinations transform a preexisting clinically harmless susceptibility into clinically evident seizures." (*Id*.) He suggested that K.N. had an unidentified genetic risk factor that left her susceptible to experience seizures following vaccination. (*Id*.) He otherwise opined that "[h]appily, adverse reactions to vaccinations are rare. As such they elude epidemiology because epidemiology is rarely sufficiently powered to reveal signals from rare events." (*Id*. at 4.)

ii. Response to Dr. Holmes (and Rule 5 Order)

In response to Dr. Holmes, Dr. Kinsbourne devoted significant attention to seeking to dismiss the conclusions of the IOM based on the notion that the evidentiary standard utilized by the IOM is higher than what is required in the VICP.[19] (ECF No. 78-

[19] Dr. Kinsbourne is correct that special masters have previously observed that the IOM employs a standard for finding causation that is higher than what is required by petitioner's burden of proof. *E.g.*, *Raymo v. Sec'y of Health & Human Servs.*, No. 11-0654V, 2014 WL 1092274, at *21 n.39 (Fed. Cl. Spec. Mstr. Feb. 24, 2014). Accordingly, IOM reports and findings are typically approached with caution and generally not treated as dispositive. *Porter v. Sec'y of Health & Human Servs.*, 663 F.3d 1242, 1252 (Fed. Cir. 2011) (noting the special master's comment that "IOM reports are favored, although not dispositive, in the Vaccine Act Program," then affirming special master's decision). However, Dr.

1, pp. 1-2.) Dr. Kinsbourne equated reliance on epidemiology to determine whether rare events were caused by vaccination as "accumulat[ing] a heap of false negatives." (*Id*. at 2.) However, Dr. Kinsbourne also reiterated his view that the studies by Sun et al., Le Saux et al., Conrad and Jenson, and von Spiczak et al. support the DTaP vaccine as a cause of seizures. (*Id*. at 3-4; *see also* Steven Rosenthal et al., *The Safety of Acellular Pertussis Vaccine vs Whole-Cell Pertussis Vaccine: A Postmarketing Assessment*, 150 ARCHIVES PEDIATRICS & ADOLESCENT MED. 457 (1996) (ECF No. 97-12); MMWR, *supra*, at ECF No. 97-11).) In particular, Dr. Kinsbourne opined that studies comparing the whole and acellular pertussis vaccines are valuable and that they show that "both cause the same adverse reactions. The nature of the adverse responses has not changed during the transition from whole cell pertussis vaccine to acellular pertussis, but only their frequency." (*Id*. at 5.) Additionally, Dr. Kinsbourne opined that other studies have shown that the pneumococcal conjugate, Hib, and flu vaccines can also cause seizures and, further, that simultaneous administration increases the risk of seizures. (*Id*. at 5-6 (citing Shirley V. Wang et al., *Determining Which of Several Simultaneously Administered Vaccines Increase Risk of an Adverse Event*, 43 DRUG SAFETY 1057 (2020) (ECF No. 97-18; *see also* Ex. F, Tab 1); Neal A. Halsey et al., *The Safety of Influenza Vaccines in Children: An Institute for Vaccine*

Kinsbourne is incorrect to imply that this means the IOM reports should be entirely disregarded. As I explained in a prior decision,

> [S]pecial masters apply the preponderant evidence standard to the record as a whole, not specific pieces of evidence in isolation. The IOM report is not dispositive, but nor does it need to be dispositive to constitute relevant evidence that must be evaluated in reaching a determination based on the record as a whole.

Bangerter v. Sec'y of Health & Human Servs., No. 15-1186V, 2022 WL 439535, at *20 n.30 (Fed. Cl. Spec. Mstr. Jan. 18, 2022). Numerous prior cases have demonstrated that special masters may account for IOM findings in reaching their decisions. *See, e.g.*, *Crutchfield v. Sec'y of Health & Human Servs.*, 125 Fed. Cl. 251, 262 (2014) (noting that "it was appropriate for the special master to consider the medical literature presented, including the IOM report" and that "the court often has relied on the findings of the Institute of Medicine."); *see also Isaac v. Sec'y of Health & Human Servs.*, 108 Fed. Cl. 743, 772-73 (2013) (affirming the special master's reliance on findings of the IOM), *aff'd per curiam*, 540 F. App'x 999 (Fed. Cir. 2013); *Cedillo v. Sec'y Health & Human Servs.*, No. 98-916V, 2009 WL 331968, at *93-94 (Fed. Cl. Spec. Mstr. Feb. 12, 2009) (finding it is "quite appropriate" to rely on IOM reports as one item of evidence concerning causation), *mot. for rev. denied*, 89 Fed. Cl. 158 (2009), *aff'd*, 617 F.3d 1328 (Fed. Cir. 2010); *Rodriguez v. Sec'y of Health & Human Servs.*, 67 Fed. Cl. 409, 410 (2005) (affirming the chief special master's reliance on IOM report regarding vaccine causation of an injury); *Althen v. Sec'y of Health & Human Servs.*, No. 00-170V, 2003 WL 21439669, at *11 n.28 (Fed. Cl. Spec. Mstr. June 3, 2003) ("Due to the IOM's statutory charge, the scope of its review, and the cross-section of experts making up the committee reviewing the adverse events associated with vaccines, the court considers their determinations authoritative and subject to great deference."), *rev'd on other grounds*, 58 Fed. Cl. 270 (2003) (citing IOM reports frequently in support of various scientific propositions), *aff'd*, 418 F.3d 1274 (Fed. Cir. 2005); *Terran v. Sec'y of Health & Human Servs.*, 41 Fed. Cl. 330, 337 (1998) (affirming special master's reliance on conclusions of IOM), *aff'd*, 195 F.3d 1302 (Fed. Cir.1999); *Cucuras*, 993 F.2d at 1529 (noting that the special master had placed "a great deal of weight" on an IOM report in reaching a decision, then affirming the special master's decision); *Stroud v. Sec'y of Health & Human Servs.*,113 F.3d 1258 (Fed. Cir. 1997) (unpublished table decision) (affirming the special master's reliance upon an IOM report that neither party filed as evidence); *Ultimo v. Sec'y of Health & Human Servs.*, 28 Fed. Cl. 148, 152 (1993) (finding it proper for a special master to rely on IOM report).

Safety White Paper, 33 VACCINE F1 (2012) (ECF No. 97-6; *see also* Ex. F, Tab 3)).) Thus, he opined that it is significant that K.N. received the Prevnar and flu vaccines at the same time she received the DTaP vaccine at issue. (*Id*.)

Dr. Kinsbourne indicated that "[v]accines have no role whatever in determining the nature of the seizure disorder or epileptic encephalopathy that they trigger. That depends on whatever predisposition the patient might have." (ECF No. 78-1, p. 7.) Thus, he opined that vaccines can "trigger," rather than "cause," epilepsies. (*Id*. (citing Ingrid E. Scheffer, *Vaccination Triggers, Rather Than Causes, Seizures*, 15 EPILEPSY CURRENTS 335 (2015) (ECF No. 97-13)).) He opined that a focal complex partial seizure can "launch" epileptogenesis. (*Id*. at 7-8.) Although Dr. Holmes opined that K.N.'s history does not meet the definition of epilepsy because her seizures were febrile, Dr. Kinsbourne suggested that K.N. "is still within the age range during which fevers mostly accompany seizures," seeming to imply that Dr. Holmes's acknowledgement that she is at high risk of developing non-febrile seizures later is some indication that she is already epileptic. (*Id*. at 6-7.) Nonetheless, he explicitly conceded that K.N. did not meet the criteria for epilepsy and instead is better characterized as having suffered multiple febrile seizures. (*Id*.) However, Dr. Kinsbourne also opined that, subject to EEG confirmation, K.N.'s reported staring spells would constitute atonic seizures separate and apart from her febrile seizures. (*Id*. at 8.)

Quoting a paper by Holmes and Noebels from 2016, Dr. Kinsbourne defined epileptogenesis as "the process by which a brain network that was previously normal is functionally altered toward increased seizure susceptibility, thus developing an enhanced probability to generate epileptic seizures." (ECF No. 78-1, p. 10 (quoting Gregory L. Holmes & Jeffrey L. Noebels, *The Epilepsy Spectrum: Targeting Future Research Challenges*, COLD SPRING HARBOR PERSPS. MED., July 2016, at 1 (ECF No. 97-8)).) Therefore, he opined that "[w]hen a sequence of seizures or epilepsy begins with a seizure that was provoked by an infection or vaccination, the onset seizure is taken in the [VICP][20] to be the trigger for the whole ensuing sequence. It does not qualify as a one/off independent and isolated event." (*Id*. at 11.) Because Dr. Holmes was one of the two authors of the Holmes and Ben Ari article he initially cited, Dr. Kinsbourne suggested that Dr. Holmes has contradicted himself in contending that human evidence does not support the notion that seizures beget seizures. (*Id*. at 8-9.) Dr. Kinsbourne quotes the following from a 2006 paper by Holmes and Ben Ari:

[20] Notably, Dr. Kinsbourne did not specify what prior program experience he was invoking. However, Dr. Holmes subsequently observed that the assertion is incorrect, indicating that "it has been decided in numerous cases that it is the underlying condition that is responsible for the epilepsy, rather than a seizure precipitated by a vaccination." (Ex. F, p. 5.) In my prior Rule 5 order, I had acknowledged that one prior case had found Dr. Kinsbourne credible in opining that a vaccine-related febrile seizure can "beget" subsequent seizures. *Fuller ex rel. B.F. v. Sec'y of Health & Human Servs*., No. 15-1470V, 2019 WL 7576382, at *16 (Fed. Cl. Spec. Mstr. Dec. 17, 2019). In that case, similar to this case, Dr. Holmes had testified that seizures can increase the risk of subsequent seizures, but do not necessarily result in epilepsy and further noted that only prolonged or frequent seizures affect the neuronal network. *Id*. at *18. The special master favored Dr. Kinsbourne's opinion because she had determined that the initial seizure was a prolonged complex seizure. *Id*. at *14, *18. However, special masters are not bound by the prior decisions of other special masters or of the Court of Federal Claims. *E.g. Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998), *aff'd*, 191 F.3d 1344 (Fed. Cir. 1999).

> [I]nfants and children are at high risk for seizures compared with adults. Although most seizures are benign and result in no long-term consequences, increasing experimental animal data strongly suggest[s] that frequent or prolonged seizures in the developing brain result in long lasting sequelae. Such seizures may intervene with developmental programs and lead to inadequate construction of cortical networks rather than neuronal cell loss. As a consequence, the deleterious actions of seizures are strongly age-dependent . . . .

(*Id*. at 10 (quoting Yehezkel Ben-Air & Gregory L. Holmes, *Effects of Seizures on Developmental Processes in the Immature Brain*, 5 LANCET NEUROLOGY 1055 (2006) (Ex. C, Tab 42)).) Although Dr. Holmes had suggested that Dr. Kinsbourne's theory is incompatible with the known rates of epilepsy remission among children, Dr. Kinsbourne disclaimed any opinion that K.N.'s seizures predisposed her to a "permanent" epileptic state. (*Id*. at 11.) He suggested that it is unknown why some epilepsies go into remission and others do not. (*Id.*) Given that the chronicity of epilepsy is related to a number of factors, including age, he opined that long-term prospects are not relevant to causation. (*Id*.) Thus, he opined that K.N.'s initial seizure "clearly did heighten her seizure susceptibility." (*Id*.)

Regarding K.N.'s specific diagnosis, Dr. Kinsbourne criticized Dr. Holmes for essentially concluding that K.N.'s condition is unexplained. (ECF No. 78-1, p. 12.) He contended that Dr. Holmes relied on outdated information in discounting the likelihood that K.N.'s condition constitutes LKS, especially with respect to age of onset. (*Id*. at 12-14 (citing Thierry Deonna & Elaine Roulet-Perez, *Early-Onset Acquired Epileptic Aphasia (Landau-Kleffner Syndrome, LKS) and Regressive Autistic Disorders with Epileptic EEG Abnormalities: The Continuing Debate*, 32 BRAIN & DEV. 746 (2010) (ECF No. 97-4); Tsai et al., *supra*, at ECF No. 55-17).) Specifically, Dr. Kinsbourne opined that K.N. suffered an "intermediate epilepsy-aphasia disorder" ("IEAD"), which he placed on a spectrum between BECTS at the mild end and LKS at the severe end. (*Id*. at 12-13.) Dr. Kinsbourne asserted that a direct role for epilepsy as a cause of aphasia is supported. (*Id*. at 14.) Although K.N. did have preexisting gross motor delay, he indicated that "I can find no medical literature that characterizes isolated mild gross motor delay as a risk factor for seizures or epilepsy," suggesting that Dr. Holmes is wrong to imply that K.N.'s developmental delay is a complete explanation for her seizure disorder, especially absent cognitive delays. (*Id*. at 15.) According to Dr. Kinsbourne, K.N.'s presentation qualifies as an epileptic encephalopathy as defined by the International League Against Epilepsy Commission report because it is a condition in which the epileptic activity itself contributes to cognitive impairment beyond the underlying pathology. (*Id*. at 15-16 (discussing Carl E. Stafstrom & Eric H. Kossoff, *Epileptic Encephalopathy in Infants and Children*, 16 EPILEPSY CURRENTS 273 (2016) (ECF No. 97-15)).) That is, epileptic encephalopathy is a slowing or regression of development that is primarily due to seizures. (*Id.* at 16 (citing Stafstrom & Kossoff, *supra*, at ECF No. 97-15).) However, whereas Dr. Holmes would place the onset of any such encephalopathy at the onset of K.N.'s gross motor delay, Dr. Kinsbourne opined

that K.N.'s condition was not indicative of any encephalopathy until after her vaccination. (*Id.*) He opined that gross motor delay is not a precursor to delayed language development and that K.N.'s language difficulty can be better correlated to seizure activity emanating from the right central cortex. (*Id.* at 16-17.) He opined that "[p]relinguistic babbling is in continuity with the beginnings of speech." (*Id.* at 17.)

Ultimately, Dr. Kinsbourne concluded:

> This relatively early onset of seizure activity was due to its premature activation by the vaccinations that she received when she was only six months old. It is medically reasonable to conclude that had she not received her six month vaccinations, her seizures, if at all, would have started later on during her childhood, with correspondingly far less severe repercussions for her language and cognitive development.

(ECF No. 78-1, pp. 19-20.)

e. Dr. Holmes's supplemental report

In response to Dr. Kinsbourne's further discussion of the relevant epidemiology, including his challenge to the IOM report, Dr. Holmes acknowledged that it can be difficult for epidemiology to capture rare events. However, given how many infants throughout the world receive multiple doses of DTaP every year, he opined that it remains revealing that the various studies have not detected a credible signal. Dr. Holmes countered that "Dr. Kinsbourne fails to consider the real likelihood that the DTaP vaccine does not cause epilepsy." (Ex. F, p. 2.) Dr. Holmes questioned the relevance of studies by Wang et al., Huang et al., and Halsey et al., as further cited by Dr. Kinsbourne, because Dr. Kinsbourne has not contended that K.N. suffered a post-vaccination febrile seizure, but has instead opined that the DTaP vaccine caused an epileptic-aphasia syndrome. (*Id*. at 1-2 (discussing Wang et al., *supra*, at Ex. F, Tab 1; Huang et al., *supra*, at Ex. F, Tab 2; Halsey et al., *supra*, at Ex. F, Tab 3).)

To the extent Dr. Kinsbourne opined that K.N.'s disorder was merely "triggered" by, rather than "caused" by, her vaccination, Dr. Holmes opined that this explanation "conveniently eliminates any need for determining the pathophysiological basis of vaccine-mediated injury." (Ex. F, p. 3.) Dr. Holmes agreed that vaccines can cause fevers but noted that the risk of DTaP-related febrile seizure is "miniscule." (*Id*. (citing Jackson et al., *supra*, at Ex. F, Tab 4).) Therefore, he explained that fever, regardless of cause, can trigger a febrile seizure in a child predisposed to febrile seizures. However, K.N. had seizures associated with non-vaccine related fevers. "Dr. Kinsbourne has not provided a mechanism by which a vaccine-induced febrile seizure results in an epileptic-aphasia syndrome in a six-month-old infant." (*Id*.) Although Dr. Kinsbourne cited Holmes and Ben-Ari for the proposition that seizures beget seizures, Dr. Holmes submitted that Dr. Kinsbourne misinterpreted the paper. (*Id*. at 4 (discussing Ben-Ari & Holmes, *supra*, at Ex. C, Tab 42; Holmes & Ben-Ari, *supra*, at ECF No. 55-7).) He explained that "[t]here is no evidence from the clinical and

experimental literature that a single febrile seizure can lead to epilepsy." (*Id*.) In the paper at issue, the authors observed that seizures result in functional changes in the brain that can lead to excitability; however, "[t]he increased excitability cannot be extrapolated to suggest that increased excitability equates to epilepsy, a condition characterized by recurrent unprovoked seizures." (*Id.*) Dr. Holmes asserted that the idea that "seizures beget seizures," as invoked by Dr. Kinsbourne, "was rejected by the medical community decades ago. (*Id.*) "The available human data strongly suggest that seizures do not beget seizures and that epilepsy in humans is usually not a progressive disorder." (*Id*. at 5 (citing Anne T. Berg & Shlomo Shinnar, *Do Seizures Beget Seizures?: An Assessment of the Clinical Evidence in Humans*, 14 J. CLINICAL NEUROPHYSIOLOGY 102 (1997) (Ex. F, Tab 12)).)

Dr. Holmes charged that Dr. Kinsbourne's assertion that focal complex partial seizures can launch epileptogensis is entirely unsupported. (Ex. F, p. 4.) He asserted that, apart from febrile status epilepticus potentially leading to status epilepticus, there is no evidence that complex febrile seizures cause epilepsy. (*Id*. (citing Leena R. Mewasingh et al., *Current Understanding of Febrile Seizures and Their Long-Term Outcomes*, 62 DEV. MED. & CHILD NEUROLOGY 1245 (2020) (Ex. F, Tab 9); Anne Berg et al., *Childhood-Onset Epilepsy With and Without Preceding Febrile Seizures*, 53 NEUROLOGY 1742 (1999) (Ex. F, Tab 10); Rod C. Scott*, Consequence of Febrile Seizures in Childhood*, 26 CURRENT OP. PEDIATRICS 662 (2014) (Ex. F, Tab 11).) Dr. Holmes raised Annegers et al.[21] as indicating that an association between complex febrile convulsions and partial (or focal) seizures may be due to the presence of an underlying brain disease, rather than reflecting a causal relationship. (*Id.* (citing John F. Annegers et al., *Factors Prognostic of Unprovoked Seizures After Febrile Convulsions*, 316 NEJM 463 (1987) (Ex. F, Tab 8)).) He quoted the authors as explaining that:

> Several investigators have suggested that complex febrile seizures are causally related to temporal lobe epilepsy. Although there is some support in studies of animals for an association between prolonged seizures and cell death, the conditions required to maintain convulsions are extreme; seizures must continue considerably longer than is usual with prolonged febrile convulsions in humans, and a direct causal sequence has therefore yet to be demonstrated. We would like to posit an alternative explanation: the tendency to have complex febrile convulsions reflects preexisting brain disease that is also responsible for the subsequent development of partial epilepsy. According to this explanation, complex febrile seizures are not causally related to partial unprovoked seizures but rather indicate the preexisting brain abnormality underlying both.

(*Id*. (quoting Annegers et al., *supra*, at Ex. F, Tab 8).) In this case, K.N. was diagnosed with a complex febrile seizure because she experienced recurrent, brief (less than 60 second) seizures. She did not have febrile status epilepticus. (*Id*.)

[21] Dr. Holmes described this paper as "seminal" and felt it was the likely source for the uncredited statistic within Dr. Kinsbourne's supplemental report that children with relevant risk factors have a 5-10% change of developing epilepsy later in life. (Ex. F, p. 4.)

Finally, Dr. Holmes asserted that Dr. Kinsbourne effectively understated the degree to which the medical records reflect K.N. to have been developmentally delayed prior to the vaccinations at issue. (Ex. F, p. 5.) Noting that at six months K.N. was not rolling from prone to supine and vice versa, as well as not pulling to sit without head lag (citing ECF No. 24-2, pp. 38, 40), Dr. Holmes explained that "[d]evelopmental assessments in 6-month-old infants are heavily weighted to gross motor skills because of the limited repertoire of behaviors in a 6-month-old, and here K.N. demonstrated gross motor delays on the day of vaccination." (*Id.*) Furthermore, "delays in social cognition, language and fine motor skills are very difficult to assess in younger infants and the milestone testing is repeated at subsequent well visits in a better attempt to capture true delays." Therefore, Dr. Holmes opined that "Dr. Kinsbourne's reliance on a lack of documented cognitive and fine motor deficits at six months[] is misplaced." (*Id*.)

f. Dr. MacGinnitie's Supplemental Report

Dr. MacGinnitie acknowledged that Choi et al. found that recurrent febrile seizures did show elevated cytokine levels as Dr. Kinsbourne had stressed; however, he suggested that K.N.'s seizures would not constitute "recurrent" seizures as couched by the paper's stated methods. (Ex. E. p. 1) Thus, he stood by his initial interpretation. Moreover, absent evidence that IL-1β preceded the first febrile seizure, it does not stand as a potential mechanism to explain how a vaccine could trigger seizures. (*Id*.) Although Dr. Kinsbourne cited Costagliola et al. for the proposition that cytokines perpetuate seizures, Dr. MacGinnitie stressed that this study examined patients with febrile infection-related epilepsy syndrome ("FIRES"), which is a specific syndrome characterized by refractory status epilepticus and "very different" from K.N.'s presentation. (*Id*. at 2.) Although Costagliola et al. discusses IL-1β blocking medicines as treatment, these medications are not approved by the FDA for treatment of epilepsy. (*Id.*)

V. Analysis

K.N. suffered a febrile seizure within 24 hours of her April 11, 2018 vaccinations and the underlying fever was in turn attributed to her vaccinations by her treating physicians. (ECF No. 22-1, p. 84; ECF No. 24-1, p. 80; Ex. C, pp. 13, 16; Ex. A, p. 6.) Moreover, respondent's expert agrees that it is possible that vaccinations can cause febrile seizures. However, as respondent argues in his motion response (ECF No. 95, p. 26), if petitioners are unable to causally connect K.N.'s April 12, 2018 seizure episode to her later seizures and/or developmental delay, then the April 12, 2018 seizure standing alone does not represent a compensable injury in the program.[22] *Accord Fiorello v. Sec'y of Health & Human Servs.*, No. 17-1869V, 2024 WL 4133302 (Fed. Cl.

[22] The statutory severity requirement is also discussed in note 7, *supra*, noting K.N.'s lumbar puncture did not constitute a surgical intervention, and note 29, *infra*, explaining that K.N.'s treatment with anti-seizure medication does not preponderantly demonstrate ongoing residual effects of her alleged injury given that she was not epileptic.

Spec. Mstr. Aug. 12, 2024) (finding a brief isolated episode of an unspecified chronic immune disorder, even if vaccine related, is not compensable absent evidence demonstrating the episode to be a cause of, rather than merely a manifestation of, the broader condition), *mot. for rev. denied*, 175 Fed. Cl. 375 (2025); *Vinesar v. Sec'y of Health & Human Servs.*, No. 18-440V, 2023 WL 5427935, at *31-32 (Fed. Cl. Spec. Mstr. July 28, 2023) (finding that a first post-vaccination febrile seizure was not the cause or trigger of a child's Dravet Syndrome), *mot. for rev. denied*, 170 Fed. Cl. 681 (2024), *appeal filed*, No. 24-1787 (Fed. Cir. May 6, 2024).

Petitioners present two arguments as to why K.N.'s April 12, 2018 seizure may be causally connected to her broader presentation. First, they argue that K.N. suffered an encephalopathy consistent with the requirements of the Vaccine Injury Table. (ECF No. 94, p. 5.) Second, they argue that K.N.'s initial post-vaccination seizure was a febrile partial complex seizure, which ultimately has the capacity to result in an impairment in language development. (*Id*.) Thus, they contend that the DTaP vaccine caused-in-fact K.N.'s condition by triggering the seizure that represented the onset of the seizure disorder that impaired her language development, specifically Rolandic epilepsy or BECTS based on Dr. Kinsbourne's accompanying opinion. (*Id*. at 5-6.)

For the reasons discussed below, neither argument is persuasive, and petitioners are therefore not entitled to compensation despite the close temporal relationship between K.N.'s vaccinations and her first seizure.

a. Table Encephalopathy

Although it was not alleged in the petition, petitioners' motion for a ruling on the written record contends that "K.N. was diagnosed as having a white matter encephalopathy following seizures which occurred in less than 24 hours after administration of the vaccines, including DTaP and Hib on April 11, 2018." (ECF No. 94, p. 5.) Petitioners argue that "[t]he onset of the seizure activity and corresponding encephalopathy fall within the timeframe described in the Vaccine Injury Table." (*Id*.)

Petitioners cite to K.N.'s April 13, 2018 MRI as demonstrating a white matter abnormality (ECF No. 1, pp. 2-3; ECF No. 94, p. 4); however, that MRI also indicated no acute intracranial abnormality and petitioners have not pointed to any medical opinion within the medical records supporting the presence of an encephalopathy based on the white matter abnormality. Nor did petitioners' expert, Dr. Kinsbourne, raise any such possibility. (ECF Nos. 54, 77-1, 78-1.) Instead, the medical records characterize the white matter abnormality as "nonspecific." (ECF No. 25-1, p. 3; ECF No. 24-1, p. 50.) Dr. Kinsbourne did characterize K.N.'s longer term developmental delay as an epileptic encephalopathy (ECF No. 78-1, pp. 15-17); however, even accepting this as an opinion favoring the presence of a chronic encephalopathy, Dr. Kinsbourne never suggested that K.N.'s initial post-vaccination presentation constituted an acute encephalopathy

consistent with the requirements for a Table encephalopathy.[23] In fact, Dr. Kinsbourne explicitly invoked the existence of a Table encephalopathy as evidence of what the DTaP vaccine is capable of causing without opining that K.N. herself suffered an injury consistent with the Vaccine Injury Table. (ECF No. 54, p. 11.)

Although it is undisputed that K.N.'s seizures began within 24 hours of her DTaP vaccination, the QAI provides that seizures in themselves are not sufficient to constitute a diagnosis of acute encephalopathy and shall not be viewed as the first symptom or manifestation of an acute encephalopathy absent other evidence. 42 C.F.R. § 100.3(c)(2)(i)(D). An acute encephalopathy requires a significantly reduced level of consciousness lasting at least 24 hours. 42 C.F.R. § 100.3(c)(2)(i)(A). Here, however, petitioners have not pointed to any evidence suggesting that K.N. experienced a reduced level of consciousness as part of her initial presentation. In fact, Dr. Kinsbourne specifically remarked, based on his review of the medical records, that "[i]n between seizures, [K.N.] was alert and responsive." (ECF No. 54, p. 2.)

Therefore, there is not preponderant evidence that K.N. suffered a Table encephalopathy.

b. Causation-In-Fact

i. Medical Theory of Causation (*Althen* Prong One)

Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004)). Such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *See Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378 (Fed. Cir. 2009) (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [their] theory." *Boatmon*, 941 F.3d at 1359 (quoting *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 (Fed. Cir. 2010)). "While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Id.* (quoting *Knudsen*, 35 F.3d at 548-49).

With regard to their theory of causation, petitioners' motion for a ruling on the record is no more specific than to argue that "K.N.'s seizures began within 16 hours of administration of the vaccines described above. DTaP can cause/trigger seizures. In predisposed children these seizures may impair language development." (ECF No. 94,

[23] Dr. Holmes also agrees on respondent's behalf that K.N. experienced a diffuse encephalopathy. However, he places the presence of that encephalopathy prior to the vaccination(s) at issue based on the presence of gross motor delay. (Ex. C, pp. 13-14.)

p. 5.) However, petitioners incorporate Dr. Kinsbourne's report into their argument by reference. (*Id.*) Dr. Kinsbourne, in turn, more specifically opined that K.N.'s impaired language development was due to Rolandic epilepsy, or BECTS, that was initiated by K.N.'s April 11, 2018 vaccinations and related febrile seizure and resulted in "intermediate epilepsy-aphasia disorder." (ECF Nos. 54, 78-1.)

Although the experts devoted significant attention to debating whether the DTaP vaccine can cause seizures, this is ultimately of little significance. Dr. Holmes agreed that "[s]ince the DTaP vaccine can cause fever, albeit at a low rate, it is possible that in rare cases febrile seizures could occur following immunization."[24] (Ex. C, p. 16 (citation omitted).) In that regard, Dr. Kinsbourne's opinion was explicit in embracing an initial febrile seizure as part of his theory (ECF No. 78-1, p. 6) and he seemingly indicated that it was immaterial to his theory how the vaccine caused the initiating seizure, so long as that seizure was complex (*Id*. at 7-8). Additionally, it has been accepted in at least some prior cases that febrile seizures can be the first "domino" that results in a chronic seizure disorder. *E.g.*, *Weaver v. Sec'y of Health & Human Servs*., 164 Fed. Cl. 608, 614 (2023); *see also Fuller*, 2019 WL 7576382, at *16.[25] The key remaining question, however, is under what circumstances can a febrile seizure be so implicated. *E.g.*, *Vinesar*, 2023 WL 5427935, at *31-32 (finding that a first post-vaccination febrile seizure was not the cause or trigger of a child's Dravet Syndrome). Not all seizure disorders are the same and, as Dr. Holmes explains, "[e]ven though seizures, like other sensory experiences, change the brain, it does not mean that a seizure or series of seizures will render the brain 'epileptic[,]' . . . in most cases the occurrence of seizures itself does not influence the long-term outcome of epilepsy." (Ex. C, pp. 18-19.)

As a general matter, respondent's experts are persuasive in stressing that, even accepting the epidemiology cited by Dr. Kinsbourne as supporting the DTaP vaccine as the cause of isolated seizures, these studies would still not suffice to support any further contention of DTaP vaccine as a cause of seizure disorders or epilepsy. (Ex. C, pp. 14-16, 18-19; Ex. A, pp. 9-11.) Dr. Kinsbourne cited Conrad and Jenson for the proposition that the change from whole cell to acellular pertussis reduced the frequency, but not the nature of, adverse events following vaccination. (ECF No. 54, p. 10 (citing Conrad & Jenson, *supra*, at ECF No. 55-6).) However, while this paper did identify isolated seizures as potential "moderately severe" adverse reactions to both whole cell and acellular pertussis vaccination, the paper specified that there has been no accepted relationship between whole cell pertussis vaccination and either encephalopathy or epilepsy. (*Id.* at 2-3, 5.) Sun et al. found a small increased risk of febrile seizures on the day of the first two administrations of DTaP-IPV-Hib vaccination, but explicitly found no increased risk of epilepsy. (Sun et al., *supra*, at ECF No. 55-16, p. 1.) Jackson et al. identified seven children experiencing post-DTaP seizures, six of which were febrile.

[24] Dr. Holmes has likewise agreed in prior cases before the undersigned that the DTaP vaccine can cause febrile seizures. *Bangerter v. Sec'y of Health & Human Servs.*, No. 15-1186V, 2022 WL 439535, at *15, n.23 (Fed. Cl. Spec. Mstr. Jan. 18, 2022).

[25] However, special masters are not bound by the prior decisions of other special masters or of the Court of Federal Claims. *Hanlon*, 40 Fed. Cl. at 630.

(Jackson et al., *supra*, at ECF No. 55-8, p. 3.) "On follow-up none of those six children had neurologic abnormalities or subsequent afebrile seizures documented. Two had recurrent febrile seizures that were not associated with DTaP vaccine." (*Id*.) Le Saux et al. found a 79% reduction in febrile seizures with the introduction of the acellular pertussis vaccine. (Le Saux et al., *supra*, at ECF No. 55-11, p. 2.) However, as Dr. Holmes explained, this study did not compare the decreased rate of febrile seizures against any background rate and did not examine epilepsies or seizure disorders. (Ex. C, pp. 14-15.) Importantly, von Spiczak et al. surveyed the national German database of adverse events following immunizations for both seizures and epilepsies. (von Spiczak et al., *supra*, at ECF No. 55-14, p. 1.) However, like Le Saux, the paper included no case control or background rate against which to compare the findings. Thus, the findings are merely descriptive. Nonetheless, citing prior studies, the authors specifically note that the risk of epilepsies is not elevated following DTaP vaccination "even though epilepsy may present with a seizure following vaccination." (*Id*. at 1-2.) The authors posited that "genetic epilepsy syndromes might masquerade as vaccination-related epilepsies." (*Id*. at 2.)

Absent broader epidemiologic support (which petitioners are not obligated to present), Dr. Kinsbourne's theory otherwise turns on the specific mechanism of causation he presented, which is often summarized as "seizures beget seizures." On this record, its assertion is based primarily on two separate concepts raised by Dr. Kinsbourne. First, he contends that severe and prolonged seizures may affect the developing brain such that a child enters epileptogenesis. Second, he contends that studies have shown that seizures are mediated by the pro-inflammatory cytokine IL-1β, which is itself a product of seizures, suggesting the potential for an inflammatory feedback loop. As noted above, this theory has been accepted in prior cases. *E.g.*, *Weaver*, 164 Fed. Cl. at 614; *Fuller*, 2019 WL 7576382, at *16. However, critical to petitioners' showing under *Althen* prong one, Dr. Kinsbourne is not persuasive in seeking to apply these concepts to the BECTS he posits as the explanation for K.N.'s clinical presentation.

Whereas "epilepsy" in general is a broad clinical syndrome that can encompass a number of differing presentations, BECTS is a specific disorder. While it is not completely understood, there is a body of literature addressing its pathophysiology. None of that literature, at least as filed in this case, implicates vaccines as a cause of BECTS. Nor have any other triggering factors been clearly identified. Instead, the literature discusses BECTS as a condition that is age specific in onset and with a significant genetic component. (Bouma et al., *supra*, at Ex. C, Tab 16, pp. 2, 6; Callenbach et al., supra, at Ex. C, Tab 17, pp. 1-2; Tsai et al., *supra*, at Ex. C, Tab 31, pp. 6-7.) As Dr. Holmes explains, ultimately, BECTS is considered idiopathic. (Ex. C, pp. 16-18.)

Furthermore, while it is true that BECTS is a type of seizure disorder that can potentially be damaging, it is the centrotemporal spikes that are considered the "hallmark" of BECTS. (Panayiotopoulos et al., *supra*, at ECF No. 55-13, p. 3.) However, the frequency, location, and persistence of the centrotemporal spikes does

not correlate to or determine the frequency and severity of seizures. (*Id.*; *see also* Bouma et al., *supra*, at Ex. C, Tab 16, p. 6.) Nor does the pattern of seizure activity dictate the course of the condition. For example, in one study cited by Dr. Holmes, "patients with multiple seizures in the first month after onset did not have significantly longer active disease periods than patients who had isolated ones . . . . [and] [n]o predictive factors could be identified for the course of the epilepsy." (Callenbach et al., *supra*, at Ex. C, Tab 17, p. 5.) Literature filed in this case indicates that while the epilepsy associated with BECTS is more typically benign (*Id.* at 2, 4-5; Bouma et al., *supra*, at Ex. C, Tab 16, p. 7.), there is now "considerable evidence" that the centrotemporal spikes seen as part of the condition play a role in its cognitive comorbidities independent of seizure activity (Ghantasala & Holmes, *supra*, at Ex, C, Tab 18, pp. 8-9). Indeed, Dr. Kinsbourne himself conceded that he did not know how BECTS can cause an aphasia syndrome. (ECF No. 54, p. 8 (quoting Ghantasala & Holmes, *supra*, at Ex, C, for the proposition that "[t]he mechanism of the nexus between Rolandic/BECTS seizures and language disorders is not definitely known.")).

In contrast to cases where the concept that "seizures beget seizures" has been accepted, BECTS is more readily compared to prior cases that have addressed whether the DTaP vaccine can cause a distinct form of seizure disorder known as infantile spasms. For example, in *Bangerter v. Secretary of Health & Human Services*, Dr. Kinsbourne sought to implicate a child's post-vaccination seizure as the beginning of infantile spasms. Dr. Holmes persuasively explained, however, that the seizures that occur in the context of infantile spasms are not provoked seizures and that the course of infantile spasms is determined, not by the seizure activity, but by the presence of hypsarrhythmia, a separate and distinct feature of infantile spasms. 2022 WL 439535, at *21. Moreover, the development of hypsarrhythmia occurs over weeks to months. *Id*. Thus, Dr. Kinsbourne was not persuasive in contending that a vaccine could cause the condition by triggering the first seizure. *Id*. Here, Dr. Kinsbourne similarly has not substantiated that the seizures that occur in BECTS are provoked, that the first seizure that occurs is indicative of the onset of the condition, or that it is the pattern of seizure activity, as opposed to the independent presence of centrotemporal spikes, that explains the course of the condition. Given these shortcomings, Dr. Kinsbourne has not substantiated that the "seizures beget seizures" concept is relevant to the condition he alleges is present.

Additionally, Dr. Kinsbourne sought to buttress his opinion that seizures beget seizures by demonstrating that production of IL-1β is a byproduct of seizures, which is itself a contributor to seizures. (ECF No. 54, p. 12.) This is potentially significant because IL-1β is a potential causal factor in the development of fevers. (Gwinn et al., *supra*, at Ex. A, Tab 6, pp. 6, 11.) Thus, implicating IL-1β in the generation of seizures could help to explain how fever can be implicated not just as the cause of the first febrile seizure, but of subsequent seizures as well. (ECF No. 54, p. 12 (citing Wang et al., *supra*, at ECF No. 55-19).) For this proposition he cited a study by Wang et al., which measured cytokine levels among 1,218 epilepsy patients. (Wang et al., *supra*, at ECF No. 55-19, p. 4.) However, while Wang et al. did indicate that several cytokines may be potential biomarkers of seizure activity, the authors explained that a causal role for the

cytokines in the development of seizures remained a matter of speculation. (*Id.* at 12.) Moreover, IL-1β was not among the cytokines the authors proposed as biomarkers of epilepsy. (*Id.* at 2.) Instead, they proposed IL-6, IFN-γ, and IFNλ3 as biomarkers.[26] (*Id.*) With respect to IL-1β, the study found that IL-1β correlated to seizure severity in the context of extratemporal lobe epilepsy. (Wang et al., *supra*, at ECF No. 55-19, p. 12.) However, this finding was equivocal in that it was observed only when seizure activity was measured by one of the several metrics examined (*i.e*., the Veterans Administration Seizures Frequency and Severity Rating Scale score). (Wang et al., *supra*, at ECF No. 55-19, p. 12.) Overall, the Wang study is not strong evidence as support for Dr. Kinsbourne's theory.

For all these reasons, petitioners have not met their burden of proof under *Althen* prong one.

ii. Logical sequence of cause and effect (*Althen* prong two)

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 1326-27; *Grant*, 956 F.2d at 1147-48. Medical records are generally viewed as particularly trustworthy evidence. *Cucuras*, 993 F.2d at 1528. However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. *See* § 300aa-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 & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) ("[T]here 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."). A petitioner may support a cause-in-fact claim through either medical records or expert medical opinion. § 300aa-13(a). The special master is required to consider all the relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler*, 88 F.4th at 963 (citing *Hines*, 940 F.2d at 1528).

Petitioners argue that K.N.'s DTaP vaccine "did trigger febrile partial complex seizures" and that her language development was impaired as a result. (ECF No. 94, p. 5.) Petitioners contend that, although K.N. had pre-language babbling prior to the vaccination, this babbling stopped after the seizures began. They urge that "[n]o alternate causes of the onset of the seizures or of the language delays are in evidence." (*Id*.) Importantly, however, "[a]lthough probative, neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149).

[26] In his first report, Dr. Kinsbourne specifically highlighted IL-1β as being epileptogenic while also invoking pro-inflammatory cytokines broadly (ECF No. 54-1, p. 12), but in his response to Dr. MacGinnitie, he stated that "[t]he notion that febrile seizures occur without a rise in the level of IL-1beta contradicts a massive medical literature, and Dr. MacGinnitie offered no alternative mechanism of ictogenesis" (ECF No. 77-1, p. 3).

In this case, although K.N.'s treating physicians attributed her initial seizure to a fever that was in turn caused by her vaccination (ECF No. 22-1, p. 86; ECF No. 24-1, p. 80), there is no treating physician opinion documented in the medical records that would support the further contention that K.N. suffered a vaccine-caused epilepsy that resulted in the delay in her language development. For that proposition, petitioners rely exclusively on Dr. Kinsbourne's opinion. However, there are several reasons why Dr. Kinsbourne's opinion is not persuasive.

Dr. Kinsbourne has based his opinion on the presence of BECTS and an epilepsy-aphasia syndrome. However, neither of these diagnoses were ever reached by any treating physician and Dr. Holmes has persuasively explained why these diagnoses do not fit K.N.'s presentation. Specifically, Dr. Holmes explained that K.N.'s young age, seizure semiology, clinical course, EEG findings (particularly, the lack of centrotemporal spikes), and lack of ESES are "totally inconsistent" with a diagnosis of either BECTS or a related epilepsy-aphasia. (Ex. C, pp. 16-18.) For example, Dr. Holmes noted that LKS typically features an inability to understand or express speech "after having achieved early developmental milestones" but that K.N. did not experience language regression as she "never acquired normal language." (*Id.* at 17-18.) Accordingly, Dr. Holmes opined that K.N.'s "clinical course and EEG findings are not at all compatible with the diagnosis of the epilepsy-aphasia syndrome." (Ex. F, p. 3.) Because Dr. Kinsbourne's ultimate causal opinion is premised on a diagnosis that is not preponderantly supported, it is entitled to little to no weight.[27] Although Dr. Kinsbourne did discuss the concept that "seizures beget seizures," which could otherwise apply outside the context of BECTS, his ultimate causal opinion as applied to the facts of this case cannot be divorced from his erroneous diagnostic assessment. BECTS is the basis by which Dr. Kinsbourne purported to explain why K.N.'s own seizure presentation constituted an epilepsy and why K.N.'s alleged epilepsy in turn explained her delayed language development. None of K.N.'s medical records otherwise attribute her language delay to her seizures.

[27] *Burns v. Sec'y of Health & Human Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (holding that "[t]he special master concluded that the expert based his opinion on facts not substantiated by the record. As a result, the special master properly rejected the testimony of petitioner's medical expert."); *see also Rickett v. Sec'y of Health & Human Servs.*, 468 F. App'x 952, 958 (Fed. Cir. 2011) (holding that "it was not error for the Special Master to assign less weight to Dr. Bellanti's conclusion regarding challenge-rechallenge to the extent it hinged upon Mr. Rickett's testimony that was inconsistent with the medical records"); *Dobrydnev v. Sec'y of Health & Human Servs.*, 566 F. App'x 976, 982-83 (Fed. Cir. 2014) (holding that the special master was correct in noting that "when an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert's opinion" (quoting *Dobrydneva v. Sec'y of Health & Human Servs.*, No. 04-1593V, 2010 WL 8106881, at *9 n.12 (Fed. Cl. Spec. Mstr. Oct. 27, 2010), *rev'd*, 98 Fed. Cl. 190 (2011), *rev'd sub nom. Dobrydnev v. Sec'y of Health & Human Servs.*, 566 F. App'x 976, 982-83 (Fed. Cir. 2014)) (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp*., 509 U.S. 209, 242 (1993))); *Bushnell v. Sec'y of Health & Human Servs.*, No. 02-1648V, 2015 WL 4099824, at *12 (Fed. Cl. Spec. Mstr. June 12, 2015) (finding that "because Dr. Marks' opinion is based on a false assumption regarding the onset of J.R.B.'s condition, and the incorrect assumption of a 'stepwise regression' after each vaccine administration, it should not be credited").

In any event, Dr. Kinsbourne's theory that "seizures beget seizures" – that is, that K.N.'s first seizure rendered her epileptic – is likewise unsupported insofar as K.N. never experienced any unprovoked seizure. Instead, as Dr. Holmes explained, K.N.'s subsequent seizures are explained by separate febrile episodes. (Ex. C, p. 13.) Thus, in his supplemental report, Dr. Kinsbourne conceded that K.N.'s seizure disorder "did not meet the formal definition for epilepsy" and that "currently she can only be considered as having had multiple complex febrile seizures." (ECF No. 78-1, p. 6.) Whether a vaccine-caused febrile seizure can launch epileptogenesis is irrelevant absent demonstration that K.N. was actually in an epileptic state.[28] Dr. Holmes agreed that K.N.'s febrile seizures coupled with her developmental delay, put her at statistically increased risk of developing non-febrile seizures *in the future*, but explained that this has not been the nature of her condition to date.[29] (Ex. C, p. 13.)

Moreover, K.N.'s developmental delay cannot be readily attributed to her seizures given that it predated her initial seizure. Dr. Kinsbourne does not dispute that K.N. demonstrated gross motor delay prior to her seizures, but did seek to distinguish K.N.'s language development from her motor development. However, this is speculative. Dr. Holmes explained that developmental assessments in infancy are "heavily weighted to gross motor skills," while language delays are "very difficult to assess" in infancy. (Ex. F, p. 5.) Moreover, at the age of seizure onset, K.N. had not begun talking, making it impossible to conclude that her seizures resulted in language

[28] After conceding K.N. did not qualify for a diagnosis of epilepsy based on her febrile seizures, Dr. Kinsbourne alternatively raised the fact that K.N. suffered separate staring episodes, seeming to imply that these episodes could represent epileptic seizures. (ECF No. 78-1, p. 8.) However, he acknowledged that such a conclusion would require EEG confirmation, which is lacking. (*Id.*)

[29] K.N.'s initial hospitalization did not record any diagnosis of epilepsy; however, her treating neurologist did subsequently treat her with anti-seizure medication on the assumption that she was epileptic. However, consistent with the expert opinions, once K.N. suffered her second, unrelated episode of febrile seizures, epilepsy was no longer documented in K.N.'s medical records. In *Hernandez*, I previously explained that an extended course of treatment for epilepsy can satisfy the Vaccine Act's severity requirement. *Hernandez v. Sec'y of Health & Human Servs.*, No. 17-0143V, 2023 WL 9186318, at *19-22 (Fed. Cl. Spec. Mstr. Dec. 5, 2023). This is because weaning a patient off of anti-seizure medication involves a clinical judgment as to the likelihood that the underlying epileptogenesis has resolved. *Id.* at *21. A patient is not considered "in remission" from epilepsy until a successful weaning from anti-seizure medication has occurred. *Id.* Here, however, both parties' experts agree with the benefit of hindsight that K.N.'s condition did not constitute epilepsy, indicating that, despite the ongoing treatment with anti-seizure medication, there is not preponderant evidence that she was in an epileptic state. Absent the presence of epilepsy, K.N.'s ongoing treatment does not carry the same significance as in *Hernandez*. The Federal Circuit has explained that, while a course of treatment may constitute a "residual effect," this is only when the treatment is for "a condition that has not resolved, if the patient's somatic condition increases the risk of recurrence." *Wright v. Sec'y of Health & Human Servs*., 22 F.4th 999, 1007 (Fed. Cir. 2022). Thus, for example, the severity requirement was satisfied where a child was restricted from physical activity by his treating physicians following a skull fracture. *H.S. v. Sec'y of Health & Human Servs*., No. 14-1057V, 2015 WL 1588366 (Fed. Cl. Spec. Mstr. Mar. 13, 2015). However, the restriction from physical activity was significant only because "the medical restriction due to increased risk of re-injury constitutes evidence that H.S.'s doctors did not believe his skull fracture had fully resolved in the first instance." *Id*. at *3. Similarly here, the question is not simply whether K.N. was receiving anti-seizure medication, but rather what, if anything, that treatment evidences regarding her underlying condition. Considering the record as a whole, the course of treatment does not preponderantly establish an ongoing epilepsy.

regression. (Ex. C, p. 17.) Dr. Kinsbourne's competing contention, distinguishing the motor and language delays, was primarily based on the particular relationship he had identified between centrotemporal lobe epilepsy, such as BECTS, and aphasia syndromes. However, the literature regarding BECTS does not necessarily support the distinction he has attempted to draw between motor and language delay. Instead, "in [Rolandic epilepsy] a significant correlation has been demonstrated between problems in motor and problems in language development, which suggests a common cerebral origin." (Besseling et al., *supra*, at ECF No. 55-2, p. 2.) Considering the record as a whole, Dr. Holmes is persuasive in contending that K.N. likely suffered a preexisting diffuse encephalopathy that explains her entire developmental picture. (Ex. C, p. 12.)

By contrast, Dr. Kinsbourne has not adequately explained how the epilepsy he posits, which he noted to be "mild" and "rapidly controlled" (ECF No. 54, p. 7), would result in an aphasia syndrome. Dr. Kinsbourne described a spectrum of epilepsy-aphasia syndromes wherein BECTS anchors the mild end of the spectrum, with only mild epilepsy and minimal language disruption, and conditions such as LKS and epileptic encephalopathy, the latter of which he opines is implicated in K.N.'s case, anchor the severe end of the spectrum. (ECF No. 78-1, pp. 12-13, 15-16.) He explains that epileptic encephalopathies are conditions in which developmental slowing or regression result from seizure activity (or other interictal cortical or subcortical activity), rather than being attributable to the underlying cause of the seizures, suggesting that severity of seizure activity should correlate with the severity of the developmental outcome. (*Id.* at 16.) Thus, although he sought to place K.N. somewhere in the middle of that spectrum, opining that her proposed epilepsy-aphasia syndrome was "moderate," he conceded that "[t]he correlation between seizure activity and degree of language impairment in [K.N.]'s epilepsy aphasia syndrome is imperfect in that her seizures are readily amenable to control by anti-epileptic agents, whereas the language disorder is severe and potentially long-lasting." (*Id*. at 19.) Dr. Kinsbourne sought to resolve this tension by opining that K.N.'s young age at the time of onset explains her disproportionately severe developmental outcome. However, he ultimately acknowledged that this distinction in itself renders an epilepsy-aphasia syndrome a poor fit, stating "[K.N.]'s epileptic syndrome can be easily diagnosed as on the epilepsy-aphasia spectrum except for one thing: the early age of onset of her seizures." (*Id*.) In that regard, as discussed above, Dr. Holmes persuasively explained that K.N.'s age should be viewed as precluding such a diagnosis. (Ex. C, pp. 16-18.)

And, finally, to the extent these issues represent a "battle of the experts," Dr. Holmes's opinion is entitled to comparatively greater weight than Dr. Kinsbourne's.[30] In

[30] Where both parties offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen*, 618 F.3d at 1347 (citing *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 3157, 1362 (Fed. Cir. 2000)). 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. 136, 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012) (citing *Cedillo*, 617 F.3d at 1339), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013). Weighing the relative persuasiveness of competing expert testimony,

prior cases, I have explained that Dr. Kinsbourne's credentials do not include any specialized education, training, or expertise in pediatric immunology and that his prior experience in clinical neurology is remote. *Hernandez*, 2023 WL 9186318, at *29-31; *Bangerter*, 2022 WL 439535, at *30-31. *But see Eilan v. Sec'y of Health & Human Servs.*, No. 15-381V, 2021 WL 1085925, at *29-31 (Fed. Cl. Spec. Mstr. Feb. 23, 2021). Apart from his one year of pediatric residency in 1958-1959 and board certification in the 1960s, Dr. Kinsbourne's focus has been neuropsychology.[31] Due to lack of experience in pediatric immunology and seizure disorders, Dr. Kinsbourne's opinions are less weighty when compared to Dr. Holmes and Dr. MacGinnitie.[32] As of the submission of their respective curricula vitae, Dr. Holmes remained a practicing clinical pediatric neurologist with special interest in seizure disorders and epilepsy (Ex. D) and Dr. MacGinnitie remained a practicing pediatric immunologist (Ex. B). Both Drs. Kinsbourne and Holmes have the requisite training in neurology to be accepted as experts. However, Dr. Holmes's clinical and research experience is more up-to-date and more relevant to the issues addressed. As noted in prior decisions, Dr. Kinsbourne had not maintained a clinical practice since the early 1990s. *Hernandez*, 2023 WL 9186318, at *30. Thus, even setting aside Dr. MacGinnitie's added immunology opinion, Dr. Holmes's opinion is clearly entitled to comparatively more weight as between the two neurologists.

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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 (noting that "[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("[T]his 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."). Additionally, in determining whether a particular expert's testimony was reliable or credible, a special master may consider whether the expert is offering an opinion that exceeds the expert's training or competence. *Walton v. Sec'y of Health & Human Servs.*, No. 04-503V, 2007 WL 1467307, at *17-18 (Fed. Cl. Spec. Mstr. Apr. 30, 2007) (otolaryngologist not well suited to testify about disciplines other than her own specialty).

[31] Petitioners neglected to file Dr. Kinsbourne's curriculum vitae; however, as previously noted, he has participated in numerous prior cases in this program and his credentials have been summarized in prior decisions and rulings. *E.g.*, *Eilan*, 2021 WL 1085925, at *13; *Hernandez*, 2023 WL 9185318, at *29-31.

[32] *Martin v. Sec'y of Health & Human Servs.,* No. 15-789V, 2020 WL 4197748, at *7, 31 (Fed. Cl. Spec. Mstr. May 8, 2020) ("Dr. Kinsbourne . . . has no demonstrated research or treatment expertise in the matters in dispute, and he relies on neurology expertise that has not been honed or refined, whether by clinical practice or research, for nearly 30 years."), *mot. for rev. denied*, 158 Fed. Cl. 459 (2020); *Jaafar ex rel. A.M. v. Sec'y of Health & Human Servs.*, No. 15-267V, 2018 WL 4519066, at *3 (Fed. Cl. Spec. Mstr. Aug. 10, 2018) (concluding that petitioner did not establish DTaP vaccination caused infantile spasms and noting that "the most recent phase of [Dr. Kinsbourne's] career has had a shallower connection to pediatric neurology clinical care"); *Holmes v. Sec'y of Health & Human Servs.*, No. 08-185V, 2011 WL 2600612, at *20 (Fed. Cl. Spec. Mstr. Apr. 26, 2011) (questioning Dr. Kinsbourne's "clinical expertise in diagnosing and treating febrile seizures and epilepsy"), *mot. for rev. denied*, 115 Fed. Cl. 469 (2014); *Stone v. Sec'y of Health & Human Servs.*, No. 04-1041V, 2010 WL 1848220, at *8 (Fed. Cl. Spec. Mstr. Apr. 15, 2010) ("The fact that for the past twenty-five years Dr. Kinsbourne has not focused his practice, research or teachings in the field of seizure disorders, . . . significantly limited his ability to offer reliable, persuasive, and cogent testimony in this case."), *rev'd on other grounds*, 95 Fed. Cl. 233 (2010); *Hoskins v. Sec'y of Health & Human Servs.*, No. 15-071V, 2017 WL 3379270, at *5 (Fed. Cl. Spec. Mstr. July 12, 2017).

For all these reasons, petitioners have not met their burden of proof under *Althen* prong two.[33]

iii. Proximate temporal relationship between vaccination and injury (*Althen* prong three)

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorders etiology, it is medically acceptable to infer causation-in-fact." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.*; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *mot. for recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd*, 503 F. App'x 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877, at *26 (Fed. Cl. Spec. Mstr. May 30, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

Petitioners argue that both K.N.'s initial seizure, and her fever, arose "within the risk interval for seizure activity caused by DTaP." (ECF No. 94, p. 6.) Focusing specifically on the study by Sun et al., respondent disagrees with this contention. (ECF No. 95, pp. 24-25.) However, more broadly, respondent also argues that petitioners' failure to present a reliable theory of causation fundamentally prevents them from meeting their burden of proof under *Althen* prong three. (*Id*.)

Respondent's argument based on the Sun article derives from Dr. Holmes's first report. In that report, Dr. Holmes questioned the value of the Sun study as epidemiologic support for Dr. Kinsbourne's theory that the DTaP vaccine can directly cause seizures. (Ex. C, p. 15.) Dr. Holmes noted that, overall, Sun found no increased risk of febrile seizures from 0-7 days post-vaccination. (*Id.*) Thus, an association was not indicated. However, Dr. Holmes acknowledged that one finding within the study suggested that a small increased risk existed on the day of the administration of a first

[33] To the extent I have accepted Dr. Holmes's opinion that K.N.'s predisposition toward febrile seizures is a manifestation of a pre-existing diffuse encephalopathy, I also note in the interest of completeness that the same factors I have identified as undercutting Dr. Kinsbourne's opinion relative to *Althen* prong two would also defeat any claim for significant aggravation as would be analyzed under *Loving* prong five. Without placing a burden on petitioners to demonstrate that K.N.'s condition was worse than it otherwise would have been, it remains the case that petitioners have not preponderantly demonstrated that K.N.'s post-vaccination febrile seizure affected the course of her condition. *Compare Sharpe v. Sec'y of Health & Human Servs.*, 964 F.3d 1072, 1081-82 (Fed. Cir. 2020), *with Locane v. Sec'y of Health & Human Servs.*, 685 F.3d 1375, 1381-82 (Fed. Cir. 2012). In particular, whereas Dr. Kinsbourne opined that a complex febrile seizure can launch epileptogenesis, the evidence in this case instead shows K.N.'s subsequent seizures were triggered by separate febrile episodes. Dr. Holmes persuasively opined that, as a result of her febrile seizures and developmental delay, K.N. is statistically at an increased risk of developing non-febrile epileptic seizures at some point *in the future*, but that has not been the nature of her condition to date.

or second dose. (*Id*.) Yet, contrary to what respondent's argument implies, Dr. Holmes's discussion of the Sun study was limited to discounting the value of the study's finding as evidence relevant to K.N.'s presentation. He did not opine that the Sun study is representative of the appropriate risk period for a post-vaccination fever or febrile seizure. Dr. Holmes otherwise opined that, "[s]ince the DTaP vaccine can cause fever, albeit at a low rate, it is possible that in rare cases febrile seizures could occur following immunization." (*Id.* at 16 (citation omitted).) His supporting citation for this proposition indicates that the relevant risk period for fever occurring post-DTaP vaccine is 48 hours. (Pichichero, *supra*, at Ex. C, Tab 12, p. 1.)

But in any event, petitioners' ability to satisfy the third *Althen* prong with respect to K.N.'s initial seizure is not dispositive for all the reasons discussed above. *Veryzer v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 344, 356 (2011) (explaining that a "temporal relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory causally connecting the vaccine and injury"), *aff'd per curiam sub nom. Veryzer v. United States*, 475 F. App'x 765 (2012); *Hibbard v. Sec'y of Health & Human Servs.*, 698 F.3d 1355, 1364-65 (Fed. Cir. 2012) (holding the special master did not err in resolving the case pursuant to *Althen* prong two when respondent conceded that petitioner met *Althen* prong three). As explained under *Althen* prong one, Dr. Kinsbourne was not persuasive in seeking to establish that vaccines can cause BECTS. Thus, respondent is correct to observe that petitioners cannot satisfy *Althen* prong three on that basis. Moreover, as explained under *Althen* prong two, while K.N.'s treating physicians attributed her first seizure to a fever that was in turn caused by her vaccination, there is not preponderant evidence that K.N. suffered epilepsy. Rather than being explained by ongoing epileptogenesis, her subsequent seizures are attributable to unrelated febrile episodes, which did not occur in temporal association with the vaccinations at issue. Additionally, K.N.'s developmental delays, including both her gross motor and language delay, are more likely than not explained by a diffuse encephalopathy that pre-dated the vaccination at issue.

VI. Conclusion

K.N. and her family have my sympathy for the challenges they have faced. Certainly, even seizures that are ultimately benign are frightening episodes for parents, and it is easy to see why petitioners have themselves perceived K.N.'s April 11, 2018 vaccinations as a cause, not only of the seizure that occurred the next day, but also of her broader condition. I do not doubt petitioners' sincerity in bringing this claim. However, for all of the reasons described above, there is not preponderant evidence that K.N. suffered a vaccine-caused seizure disorder and/or developmental delay causally related to her vaccinations.

Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioners are not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master