In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 05-579V
(To be published)

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	*	Special Master Corcoran
HOLLY AUSTIN, *parent of* K.A., *a minor*,	*	
	*	Filed: May 15, 2018
	*	
Petitioner,	*	Decision without Hearing;
	*	Dismissal; Diphtheria Tetanus
v.	*	acellular-Pertussis ("DTaP")
	*	Vaccine; Encephalopathy;
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Developmental Regression; Autism.
	*	
Respondent.	*	
	*	
* *		

Robert Krakow, Law Office of Robert J. Krakow, P.C., New York, NY, for Petitioner.

Ann D. Martin, U.S. Dep't of Justice, Washington, DC, for Respondent.

DECISION GRANTING MOTION TO DISMISS CASE[1]

On May 27, 2005, Holly Austin, on behalf of her son, K.A., filed a petition seeking compensation under the National Vaccine Injury Compensation Program ("Vaccine Program").[2] In it, Mrs. Austin alleged that a number of childhood vaccines (the Diphtheria Tetanus acellular-Pertussis ("DTaP"), Hepatitis B ("Hep. B"), and Pneumococcal vaccines that K.A. received on

[1] This Decision will be posted on the United States Court of Federal Claims's website, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public. *Id.*

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

July 28, 2003; the Influenza ("flu") vaccine he received on December 15, 2003; the Hib vaccine he received on June 1, 2004; and the DT vaccine he received on June 8, 2004) caused K.A. to experience an encephalopathic reaction (accompanied by increased seizure activity), later manifesting as developmental regression, and ultimately evolving into an autism spectrum disorder ("ASD"). Petition at 1-2. Years later, Mrs. Austin filed an Amended Petition in August 2017, altering her allegations in an effort to exclude autism as the complained-of injury, and arguing instead that K.A. had merely experienced seizure activity following each of the vaccinations listed above, along with developmental regression following the June 8, 2014 DT vaccination. Am. Pet. (ECF No. 123) at 1-2.

After the parties filed expert reports, and based upon my initial review of the case record in light of the disposition of similar cases previously adjudicated in the Vaccine Program, I proposed that the matter be decided without holding an evidentiary hearing, and I invited the parties to brief the substantive merits of Petitioner's claim. To that end, Respondent filed a motion to dismiss, dated October 12, 2017 (ECF No. 127) ("Mot."), to which Petitioner responded on November 14, 2017 (ECF No. 129) ("Opp.").

Having completed my review of the evidentiary record and the parties' filings, I hereby **GRANT** Respondent's Motion for a Ruling on the Record Dismissing the Case, and **DENY** Petitioner's request for compensation. As discussed in greater detail below, the record does not support Petitioner's contention that K.A. suffered an encephalopathy, that he experienced any non-transient reaction at all to the relevant vaccines, or that the vaccines caused his seizure activity. In addition, the claim recycles causal theories involving autism as a vaccine injury that have been universally rejected in the Vaccine Program.

I. FACTUAL BACKGROUND

Birth and Early Medical History

K.A. was born via spontaneous vaginal delivery on January 24, 2003, following a normal pregnancy. Ex. 3 at 1.[3] No concerns or complaints were raised during the pregnancy, labor, or delivery. *Id.* Birth weight, head circumference, and length were all within the normal limits. Ex. 2 at 1, 65. K.A.'s hearing and neonatal screens were also normal, and he was discharged one day later. *Id.* at 6, 13, 31.

As the contemporaneous medical records reveal, K.A.'s health in his first year of life was characterized by the kind of illnesses that many otherwise-healthy infants experience. For example, K.A. was seen on several occasions for a variety of infections (including ear infection,

[3] Petitioner's exhibits in this case are referenced numerically, while Respondent's exhibits are referenced alphabetically.

URI, and perioral cyanosis). *See, e.g.*, Ex. 3 at 9 (1/25/2003, possible jaundice and observed as "jittery" in office); Ex. 8 at 47 (4/23/2003, diagnosed with conjunctivitis in left eye and constipation), 47 (5/2/2003, diagnosed with an ear infection and an upper respiratory infection), and 51 (5/11/2003, possible sinusitis). In addition, K.A. was treated for oral thrush on June 25, 2003, and pharyngitis on July 8, 2003. Ex. 14 at 20-22. Apart from these minor health problems, the records from K.A.'s well-child appointments generally indicated that he was progressing normally from a developmental standpoint.

Receipt of Vaccinations and Subsequent Medical History

K.A. received his first Hep. B vaccination on February 5, 2003, according to his vaccination record. Ex. 14 at 2. On March 27, 2003, at his two-month well-child visit, he received his initial round of childhood vaccinations, including the DTaP, Hep B (second dose), Hib, Pneumovax, and IPV vaccines. Ex. 8 at 26-28; Ex. 14 at 2-3. K.A. received a second round of vaccinations (including DTaP, IPV, Hib, and Pneumococcal) at his four-month well-child visit on June 13, 2003, and a third round on July 28, 2003 (including DTaP, Hep B, and Pneumovax). Ex. 8 at 26, 29; Ex. 14 at 2, 6. The medical records reference no adverse reactions following receipt of any of these vaccinations.

On July 12, 2003 (about two weeks after K.A.'s last June pediatric visit), Mrs. Austin went to the emergency room at Franklin Memorial Hospital in Bangor, Maine, complaining that K.A. had experienced an apparent "acute life-threatening event," including an episode of starring and limpness. Ex. 11 at 2. According to the treater's notes, Petitioner went to pick him up around 6:00 p.m. that evening, and he immediately turned blue and went limp for a period lasting "a few seconds to minutes." *Id.* at 4. She then placed him down on the floor, and he eventually opened his eyes and became responsive. *Id.* at 4.

K.A was subsequently transferred to Eastern Maine Medical Center (also in Bangor), and was placed on cardiorespiratory monitoring throughout his admittance. Ex 11 at 2, 45-47. Testing noted no abnormalities in his chest cavity or lungs. *Id.* at 5-6. Treaters also conducted an upper GI series, which was normal, and noted no further limpness episodes during his hospital stay. *Id.* at 2-3, 19-20. As there was some evidence of a cough associated with the episode, treaters questioned whether Petitioner had observed any reflux, and also did not rule out an ear infection. After monitoring K.A., and determining that his vitals remained stable, treaters discharged him with a final diagnosis of an unspecified "acute life-threatening event," presumptive gastrointestinal reflux, and left otitis media. *Id.* at 2. He was proscribed Zantac for any future problems. *Id.* K.A.'s treatment plan also included a Good Start formula diet, Tylenol for temperature increases, and Amoxicillin for his on-going ear infection. *Id.* at 6. Summary notes indicated that K.A.'s symptoms did not include muscle twitching or spasm activity. *Id.* at 4.

Two weeks later, on July 28, 2003, K.A. had his six-month well-child visit at Penobscot Pediatrics, and received his third round of vaccinations (including DTaP, Hep B, and Pnuemovax). Ex. 8 at 2; Ex. 14 at 2. Once again, no developmental problems were noted. More specifically, K.A.'s pediatrician, Dr. Elizabeth Trefts, noted that K.A. was alert and happy. Ex. 8 at 30; Ex. 14 at 5. Dr. Trefts's notes indicated K.A.'s parents had reported that K.A. had been taken to the emergency room on July 12th for an "episode" which included limpness and a change in color. *Id.* The record from this appointment stated that K.A. was diagnosed with reflux and treated with Zantac. *Id.* No further episodes of limpness or change in color were noted in the intervening period. *Id.*

Acute Onset Seizures

On that same day, a few hours later, K.A. experienced acute onset seizures. Ex. 11 at 55; Ex. 4 at 11-14. K.A. was taken back to Eastern Main Medical Center and admitted to the pediatric intensive care unit for cardiopulmonary monitoring. Ex. 11 at 53. During the admittance process, K.A. experienced an additional four to eight seizures that were observed by various medical staff members. *Id.* He was treated with Fosphenytoin[4] and Phenobarbital during the course of his hospitalization. *Id.* An EEG and MRI completed during the visit were both normal. *Id.* Treater notes also indicated that K.A.'s parents voiced some concern that Moxifloxacin (antibiotic taken by K.A.'s mother while breast-feeding) had caused him to develop seizures. *Id.* However, medical staff explained to her that they "could not link the two together." *Id.*

During his hospitalization, K.A. was evaluated by Dr. James Sears, a neurologist. Ex. 11 at 63. Dr. Sears noted that K.A.'s earlier acute life-threatening event from July 12th, when viewed in conjunction with his current episode, supported a diagnosis of partial seizures. *Id.* at 64. According to Dr. Sears, the later episode was similar to the earlier one two weeks before – seizures lasting 30-60 seconds, accompanied by eye deviation, blank stares, brief twitching, and irregular breathing. *Id.* Dr. Sears noted that K.A.'s EEG showed prominent artifacts and indicated some focal slowing, but no abnormal activity. *Id.* He also opined that K.A.'s MRI showed normal intracranial activity. *Id.* Dr. Sears treated K.A. with Fosphenytoin and maintenance Phenobarbital, with a transition into Dilantin. *Id.* at 65. Dr. Sears also was aware that K.A. had received vaccinations earlier that day, but maintained that the vaccines did "not appear to be [a] substantial element" relating to the seizure episode. *Id.* at 64. K.A. was discharged three days later with after a normal physical exam (which included no seizure activity for three days). *Id.* at 54.

[4] Fosphenytoin is an anticonvulsant drug used to treat epilepsy. *Dorland's Illustrated Medical Dictionary* 736 (32nd ed. 2012) (hereinafter "*Dorland's*"). It can be administered intravenously or intramuscularly. *Id.*

Over the next several months, K.A. seemed to be doing well once again. He returned to Penobscot Pediatrics on August 4, 2003, seven days following his hospitalization. Ex. 8 at 57; Ex. 14 at 25. The attending pediatrician reported that K.A.'s seizure medications were working, although he had displayed some minimal staring-type episodes, and there remained some lingering reflux concerns. Ex. 8 at 57. Following this appointment, K.A. saw Dr. John Hickey of Pine Tree Pediatrics for a nine-month well child visit on October 3, 2003. Ex. 8 at 14. Dr. Hickey assessed K.A. as a healthy nine-month old with no developmental problems. *Id.* K.A. received his third round of the IPV vaccine on October 31, 2003, and no adverse reaction was noted, with no additional seizures occurring in the days and weeks immediately thereafter. Ex. 14 at 2; Ex. 8 at 14.

On December 15, 2003, K.A. received an influenza vaccine during a visit to Pine Tree Pediatrics. Ex. 14 at 2; Ex. 8 at 10. Eight days later, Mrs. Austin called the clinic on December 23, 2003, reporting that K.A. had again experienced seizure activity on the prior night (December 22nd). Ex. 8 at 11. Mrs. Austin described the episode as lasting five seconds, and stated that K.A. did not lose consciousness, although he did convulse. *Id.* Dr. Hickey recommended that K.A.'s mother continue administering his doses of Phenobarbital. *Id.* Later treater records expand this seizure event into a four-part episode, including a December 21st staring spell (lasting approximately ten seconds), a December 22nd generalized seizure (including arm/leg stiffness, clenched jaw, and shaking), a December 26th seizure, and a December 27th seizure. Ex. 5 at 7 (January 6, 2004, appointment with Dr. Stephen Rioux).

2004 Seizure Treatment and Medical Visits

K.A. presented to Dr. Stephen Rioux, a pediatric neurologist at Maine Medical Partners, on January 6, 2004. Ex. 5 at 7. The Austins provided Dr. Rioux a history of K.A.'s seizure disorder, and reported that he had done well on medication since his seizures first began in July 2003. *Id.* Upon examination, Dr. Rioux opined that K.A. likely had a generalized seizure disorder, adding that it was "possible that quinolones[5] transmitted to the child through breast milk, as well as subsequent immunizations lowered the seizure threshold and may have been responsible for the timing of the child's seizures." *Id.* However, Dr. Rioux also noted that it was "*unlikely that either of these interventions or agents are responsible* for his ongoing seizure difficulties." *Id* (emphasis added). Dr. Rioux recommended that K.A. remain on Phenobarbital for the time being and schedule a follow-up appointment in six months. *Id.* During a follow-up visit on May 6, 2004, he also (at Petitioner's request) proposed that the schedule for future vaccines be spread out temporally to reduce the possibility that any single vaccine might *trigger* a seizure (although, as indicated above, the record does not suggest that Dr. Rioux believed that any vaccine was causal of K.A.'s larger seizure activity). Ex. 5 at 7; Ex. 8 at 20-21.

[5] Quinolone is a term used to define a group of synthetic antibacterial agents (or antibiotics). *Dorland's* at 1567. Examples include nalidixic acid, cinoxacin, rosoxacin, and fluoroquinolones. *Id.*

Over the next two months, K.A. was brought back to his pediatricians for various ailments (although not to treat seizures). On February 12, 2004, for example, K.A. was seen by Dr. Hickey at Pine Tree Pediatrics for an ear check due to restlessness and crying during the night. Ex. 8 at 13. He was assessed with ear pain and teething. *Id.* Roughly a month later, on March 4, 2004, K.A. presented again to Dr. Hickey for his one year well-child visit. *Id.* at 14. Upon evaluation, Dr. Hickey noted that K.A. displayed no developmental problems. *Id.* On March 10, 2004, K.A. presented again to Penobscot Pediatrics for a five-day history of fever of up to 104.4 degrees, a cough, and cold symptoms. *Id.* at 12. K.A.'s treating pediatrician diagnosed him with a URI and possible respiratory syncytial virus. *Id.* Two weeks later, on March, 29, 2004, K.A. was diagnosed with otitis media and viral pharyngitis. Ex. 14 at 30.

On April 26, 2004, K.A. was seen for his fifteen-month well-child visit at Penobscot Pediatrics. Ex. 14 at 8. K.A. was due to receive additional vaccines during this visit, but his parents requested that they not be given pending Dr. Rioux's evaluation. *Id.* Office notes recorded at this time regarding K.A.'s history were somewhat contradictory of the actual medical history as set forth above. Thus, the record from the April 26th visit states that K.A.'s first seizure occurred two weeks *prior* to his six-month vaccinations, and that the cause of his second was "s/p vaccines". *Id.* These notes also indicated (correctly) that K.A. had "4-5 seizures following [the] flu vaccine" and "seizure #3 . . . temporarily related to [the] flu vaccine," although the record does not set forth the actual time interval between (other than recording the first post-flu vaccine seizure to have begun on December 21st). *Id.* at 2, 8; *see also* Ex. 5 at 7; Ex. 8 at 10-11. No developmental problems were otherwise noted during this visit.

K.A.'s next set of records from Penobscot Pediatrics, dated May 24-28, 2004, include telephone messages between Mrs. Austin and the doctor's office concerning her requests to change K.A.'s immunization schedule. Ex. 14 at 42. According to the notes, Petitioner wanted K.A. to receive one vaccination at a time per Dr. Rioux's recommendation, and wholly eliminate the DTaP vaccination from his schedule. *Id.* Following these requests, K.A.'s vaccine record indicated that he received the IPV vaccine on May 25, 2004, a third dose of Hib on June 1, 2004, and the DT vaccine on June 8, 2004. *Id.* a 2, 31. Notes from the DT vaccine administration indicated that K.A. did not experience any post-vaccination seizure activity in the office. *Id.* at 31. Following the DT vaccination, K.A. presented to his pediatrician on June 11, 2004, with a two-day history of congestion and fussiness. *Id.* at 32. He was diagnosed with a URI. *Id.* Office notes also indicated that K.A. presented with a small, two centimeter lump on his thigh at the DT vaccination injection site. Ex. 14 at 32.

Return of Seizure Activity and Reported Developmental Problems

On July 5, 2004 (about one month following receipt of the DT vaccine), Petitioner called Penobscot Pediatricians and reported that K.A. had experienced additional seizures. Ex. 14 at 43. The attending pediatrician instructed her to consult K.A.'s neurologist, as well as have him evaluated for developmental delays. *Id.* at 44. K.A. thereafter presented to a pediatrician, Michael Ross, four days later, on July 9, 2004. Ex. 14 at 35-36. Dr. Ross's notes indicated that K.A. had increased seizure activity during the previous weekend while on a therapeutic level of Phenobarbital. *Id.* at 35.

The records from this post-seizure July 2004 treatment visit now set forth – for the first time in K.A.'s medical history – issues with K.A.'s development. The Austins reported that K.A. had developed a vocabulary of six to seven words, but had not spoken any words over the previous three weeks (or since the middle of June), and had displayed a "steady decrease back to wordless mumbling." Ex. 14 at 35. Several concerning behaviors were also reported at this time, including decreased attention span, failure to regard faces, constant looking away, lingering blank looks, frequent head and ear rubbing, aimless wandering, and unsteady gait. *Id.* In addition, the Austins reported that K.A. had experienced three distinct seizure episodes and several episodes of blank staring the weekend prior. *Id.* Upon examination, Dr. Ross assessed K.A. with "seizure disorder and significant developmental delay." *Id.* at 36. Dr. Ross recommended that K.A.'s parents schedule a repeat EEG, orthopedic consultation, and a hearing assessment, as well as evaluation for developmental problems. *Id.*

K.A. subsequently returned to Dr. Rioux (his neurologist) for a follow-up visit on July 22, 2004. Ex. 5 at 5-6; Ex. 8 at 20. Dr. Rioux confirmed the reports from the visit earlier that month with Dr. Ross that K.A.'s developmental status had begun to regress, and noted possible concerns for autism. Ex. 5 at 6. Dr. Rioux was unsure if the latest episodes were attributable to K.A.'s seizures, but he conducted a repeat EEG (which was normal) and ordered additional testing. *Id.* According to Dr. Rioux's notes, Mrs. Austin specifically stated her view that K.A.'s developmental setbacks were associated with the vaccinations he had received, although Dr. Rioux assured her that no scientific basis existed for a vaccine-induced autism injury. *Id.*

K.A. was next evaluated at Penobscot County Child Development Services ("CDS") in Bangor, Maine, on July 28, 2004. Ex. 11 at 238-47. His reported symptoms at this time included loss of language, lack of eye contact, poor responsiveness, and unsteady gait. *Id.* K.A. was thereafter referred to the Maine General Medical Center's Developmental Evaluation Clinic ("DEC") in Waterville, Maine, and evaluated on September 9, 2004. Ex. 25 at 1-25. Anne Uecker, Ph.D., a licensed psychologist, determined that K.A. exhibited all six critical indicators for autism on the Modified Checklist for Autism in Toddlers. *Id.* at 11-12. She also found that K.A. was at the eleven-month level on the Bayley Scales of Infant Development. *Id.* DEC's

assessment for K.A. also included a seizure disorder, severe receptive and expressive language delays, severe articulation delay, mild gross motor delay, internal tibial torsion and femoral anteversion, but ruled out Landau-Kleffner syndrome, and Fragile X syndrome. *Id.* at 2-3. DEC evaluators recommended speech, occupational and physical therapy, an orthopedic follow-up, a hearing evaluation, and continued monitoring and treatment for autism. *Id.* at 3-4.

In August, at his neurologist's recommendation K.A. was taken to an orthopedic surgeon, Dr. James Greene, for an evaluation of unsteady gait, plus tripping and falling when walking. Ex. 12 at 1-2. Upon examination, Dr. Greene found that K.A. had excellent circulation in extremities and normal muscle strength throughout. *Id.* at 3. Dr. Greene assessed K.A. with femoral anteversion, internal tibial torsion, physiologic genu varum (bow-leggedness), and a seizure disorder (all of which Dr. Greene stated were not unusual for a child of K.A.'s age and would generally resolve in a couple of years). *Id.* Overall, Dr. Greene recommended additional physical therapy, with a follow-up visit in six months for another evaluation. *Id.*

The Austins took K.A. thereafter to a new pediatrician, on September 14, 2004, at Norumbega Medical Pediatrics in Bangor, Maine, for his eighteen-month visit. Ex. 6 at 2. K.A.'s health history at this time now officially included autistic regression, and noted that he continued to take medication for his seizure disorder. *Id.* Upon evaluation, the treating pediatrician observed that K.A. had some receptive language, but no words. *Id.* No physical problems were otherwise noted, and his overall assessment was consistent with K.A.'s past treaters: autism and a seizure disorder. *Id.*

Treatment in 2005 and Beyond

In the twelve-plus years thereafter, K.A. has received many therapeutic interventions and treatments for his autism - including speech, physical therapy, occupational therapy, dietary restrictions, hyperbaric oxygen, chelation, methylcobalamin injections, and supplements. *See generally* Ex. 10 (dietary restrictions and supplements), 17 (speech therapy), 20 (occupational therapy), 22 (chelation), and 24 (occupational and physical therapy); *see also* Ex. 21 at 66 (speech therapy); Ex. 4 at 1, 6-7 (methylcobalamin injections); Ex. 26 (pediatric visit and lab testing); Ex 32 (occupational and educational therapy). While the Austins have received a variety of proposals for the etiology of K.A.'s condition, none of the records suggest a plausible connection between the vaccinations at issue and his subsequent medical and/or developmental problems.

On March 3, 2005, for example, K.A. was evaluated by Dr. Cheryl Garganta in the genetics department at the metabolic clinic at Tufts Medical Center in Boston, Massachusetts, to determine if his autism had a metabolic etiology. Ex. 4 at 10. As the history from this evaluation indicates, the Austins themselves attributed his neurologic condition and autism symptoms to his

vaccinations. *Id.* Dr. Garganta, however, noted that K.A. had received his vaccinations at an age where children who were later diagnosed with autism tended to first experience regression. *Id.* at 12. Dr. Garganta ordered that K.A be tested for Fragile X syndrome, amino acid disorders, organic acidemias, mitochondrial dysfunction, disorders of creatinine synthesis and transport, disorders of pyruvate and pyrimidine metabolism, congenital disorders of glycosylation, and Angelman syndrome (all of which returned normal results apart from an deficiency in carbohydrate transferring). *Id.* at 12-13.

Dr. Garganta ultimately could not identify, based on K.A.'s normal lab test results, any particular adverse genetic condition from which K.A. suffered that could be deemed to have a metabolic component. Ex. 4 at 3. Certainly, it appeared to her from K.A.'s history that he was not suffering from any progressive condition that had developmental regression as a side effect; as she noted, K.A. continued to gain motor milestones, and his regression in language and social interaction could not therefore be deemed a sign of a neurodegenerative disorder. *Id.* Overall, Dr. Garganta opined that K.A.'s health problems were unrelated to his immunizations, although she recommended that K.A.'s parents schedule a follow-up appointment in a few years, as advances in testing would likely occur within that time period. *Id.* at 13.

K.A. returned to Dr. Garganta for follow-up visits on August 10, 2006, and again on March 3, 2010. Ex. 4 at 2-8. Dr. Garganta again ordered multiple lab tests (including plasma lactate, serum uric acid, RBC folate, hermatocrit, prolactin, urine organic acids, and lactyl-lactate) *Id.* at 1, 3-4, 6-8. Overall, as the notes from this visit indicated, Dr. Garganta once again concluded that K.A. did not display any medical symptoms associated with a mitochondrial disease other than autism (indicating a low likelihood of a primary mitochondrial disease, which would be far more debilitating and progressive in character). *Id.* at 3. Although she could not rule out the possibility that K.A. suffered from some kind of mitochondrial dysfunction or cerebral folate deficiency, K.A.'s symptoms did not in her opinion warrant any invasive procedure. *Id.* at 4. Dr. Garganta specifically declined to conduct a lumbar puncture or muscle biopsy due to the lack of clinical marker evidence. *Id.* She recommended that K.A. reschedule follow-up appointments every few years. *Id.* at 5.

On July 10, 2008, K.A. presented to Dr. Wendy Smith, a geneticist at Maine Pediatric Specialty Group. Ex. 18 at 1. Upon evaluation, Dr. Smith found minimal small joint hyper extensibility, but no dysmorphic features. Dr. Smith assessed K.A. with autism, and no clear syndromic diagnosis. *Id.* at 4. Dr. Smith recommended that K.A. be tested via comparative genomic hybridization (or microarray), which is used to detect small variants that may offer some clinical, genetic significance in patients with mental retardation, developmental delays, and autism. *Id.* at 5. Dr. Smith also recommended that K.A. continue his current course of therapy treatment. *Id.* No follow-up appointment was recommended.

On November 3, 2010, K.A. presented to another pediatric neurologist, Dr. Peter Morrison at Maine Medical Partners, for an autism evaluation. Ex. 15 at 1. As the record from this visit reveals, Dr. Morrison had agreed to evaluate K.A. due to his history of seizures (even though autism was outside his scope of practice). *Id.* After obtaining a brief history of K.A.'s seizure symptoms, Dr. Morrison noted the Austins's concern that K.A. had developed a mitochondrial disorder (leading to autism) due to his exposure to antibiotics in breast milk and thimerosal in his immunizations. *Id.* at 2. Upon evaluation, however, Dr. Morrison generally opined that autism cannot be linked to a specific cause. *Id.* at 1. He further noted that Petitioner expressed a concern that K.A. had a cerebral folate deficiency, but stated his view that this type of deficiency was typically not related to regressive autism. *Id.* Dr. Morrison did not completely exclude the possibility that K.A. had some underlying mitochondrial dysfunction, but he noted that he could not find any systematic signs during his exam. *Id.*

Updated records filed between December 2014 and February 2015 include various additional pediatric visits and specialty visits. *See generally* Ex. 28 (pediatric visit for lab testing); Ex. 30 (same), Ex. 32 (occupational and educational therapy). None are any more supportive of the conclusion that K.A.'s autism was vaccine-related. For example, in February 2015, K.A. was seen by another geneticist and mitochondrial specialist, Dr. Fran Kendall[6] of Virtual Medical Practices, LLC, in Atlanta, Georgia. Ex. 29. at 1. Dr. Kendall reviewed various tests and labs during the visit (including CBC, CMP, CPK, T4, TSH, coenzyme, Q10, carnitine, vitamin D, and urine amino acid) in order to determine if K.A. displayed clinical symptoms of a mitochondrial disease. *Id.* at 4. Upon evaluation, Dr. Kendall opined that K.A.'s autism (along with general fatigue and constipation), did not suggest that he was experiencing an underlying mitochondrial disorder. *Id.* In response to Petitioner's concerns that K.A.'s condition was vaccine-caused, Dr. Kendall stated that "no clear proof of causation" existed between the two. *Id.*

II. EXPERT REPORTS

A. Dr. Yuval Shafrir

Dr. Shafrir prepared two expert reports for Petitioner, both of which attempt to establish a link between K.A.'s regression/autism and his vaccinations. *See* Report, dated Nov. 23, 2015, field as Ex. 34 (ECF No. 94-1) ("Shafrir First Rep."); Report, dated May 3, 2016, filed as Ex. 35 (ECF No. 105-1) ("Shafrir Second Rep."). According to Dr. Shafrir, K.A. suffered from an autoimmune encephalopathy (manifesting as acute onset seizures) that evolved into an autistic syndrome as a result of receiving the combination of vaccinations outlined above.

[6] Dr. Kendall has testified on behalf of other petitioners claiming autism as a vaccine injury. *See, e.g., Murphy v. Sec'y of Health & Human Servs.*, No. 05-1063V, 2016 WL 3034047 (Fed. Cl. Spec. Mstr. Apr. 25, 2016), *mot. for rev. den'd*, 128 Fed. Cl. 348 (2016); *Holt v. Sec'y of Health & Human Servs*., No. 05-0136V, 2015 WL 4381588 (Fed. Cl. Spec. Mstr. June 24, 2015), *mot. for rev. den'd*, 132 Fed. Cl. 194 (2017).

As his CV indicates, Dr. Shafrir is a pediatric neurologist at Sinai Hospital in Baltimore Maryland. *See* Shafrir CV, filed as Ex. 36 (ECF No. 122). Dr. Shafrir received his medical degree from the Tel Aviv University Sackler School of Medicine in Israel, where he also completed a pediatric residency. *Id.* at 1. Upon his arrival in the United States, he completed an additional pediatric residency at Cornell University Medical College and two fellowships, one in pediatric neurology from Washington University Medical Center in St. Louis, Missouri, and one in pediatric neurophysiology from Miami Children's Hospital. *Id.* at 1-2. Dr. Shafrir is board certified in pediatric neurology, clinical neurophysiology, and epilepsy. *Id.* at 2. He has published over ten peer-reviewed articles and has served on faculty at various medical institutions around the United States, including Georgetown University School of Medicine, the University of Oklahoma School of Medicine, and the United Services University of Health Sciences, in addition to his most recent position the University of Maryland School of Medicine. *Id.* at 3, 4-5.[7]

Dr. Shafrir opined that the DTaP vaccine K.A. received on July 28, 2003, triggered seizures and "started an encephalopathic process, which was exacerbated by his . . . influenza vaccination [on December 15, 2003] and greatly exacerbated by his . . . DT vaccination [on June 8, 2004]." Shafrir First Rep. at 45, 38.

Dr. Shafrir's First Expert Report

Dr. Shafrir's report began with an overview of K.A.'s health course and regression following his vaccinations. He pointed specifically to K.A.'s immediate onset of seizure activity following the July 28, 2003 DTaP vaccination as direct evidence of an encephalopathy. Shafrir First Rep. at 36. Dr. Shafrir did not deem his conclusion undermined, however, by the fact that K.A.'s EEG and MRI findings (conducted during his hospitalization) were both normal, or that K.A. had presented to the hospital without fever (or any other symptoms consistent with an encephalopathic reaction). *Id.* He also identified an instance in the record where K.A.'s treating pediatrician recommended that K.A.'s future vaccinations be spread out over time as this "may reduce the risk of seizures." *Id.* at 37 (Ex. 8 at 21). Apart from these factual references, Dr. Shafrir offered no additional discussion of any purported evidence of encephalopathy in K.A.'s health record. The remainder of his discussion of K.A.'s records center on the temporal association between K.A.'s DT vaccination the following year (on June 8, 2004), and his onset

[7] Dr. Shafrir has testified on behalf of petitioners asserting autism as a vaccine injury on numerous occasions. *See, e.g.*, *Cunningham v. Sec'y of Health & Human Servs.*, No. 13-482, 2016 WL 4529530 (Fed. Cl. Spec. Mstr. Aug. 1, 2016), *mot. for rev. den'd*, 2017 WL 1174448 (Fed. Cl. Jan. 25, 2017); *R.V. v. Sec'y of Health & Human Servs*., No. 08-504V, 2016 WL 3882519 (Fed. Cl. Spec. Mstr. Feb. 19, 2016), *mot. for rev. den'd*, 127 Fed. Cl. 136 (2016); *Lehner v. Sec'y of Health & Human Servs.,* No. 08-554V, 2015 WL 5443461 (Fed. Cl. Spec. Mstr. July 22, 2015). His credibility on the topic has been sharply questioned by other special masters. *See, e.g*, *Cunningham*, 2016 WL 4529530, at *15-16.

of regression around two weeks later (which he also labeled as encephalopathic in origin). *Id.* at 37; Shafrir Second Rep. at 5.

Dr. Shafrir analogized K.A.'s alleged vaccine injury, autism, to epilepsy, arguing that their neurologic origins and course are congruent. Shafrir First Rep. at 38. Dr. Shafrir asserted that an association between seizures, epilepsy, and autism has in fact been observed in several medical articles. *Id.*; *see, e.g.*, E. Saemundsen, et al., *Autism Spectrum Disorders in Children with Seizures in the First Year of Life—A Population-based Study*, 48 Epilepsia 1724 (2007), filed as Ex. 34 (ECF No. 94-5) (cohort study concluding that prevalence of ASD is higher in children with history of seizures in first year of life); J. Lugo, et al., *Early-Life Seizures Result in Deficits in Social Behavior and Learning*, 256 Exp. Neuro. 74, 78 (2014), filed as Ex. 34 (ECF No. 94-9) (animal laboratory study concluding that early-life seizures result in significant reduction in social behavior in mice); P. Bernard, et al., *Behavioral Changes Following a Single Episode of Early-Life Seizures Support the Latent Development of an Autistic Phenotype*, 44 Epilepsy & Behavior 78, 78 (2015), filed as Ex. 34 (ECF No. 94-10) (animal laboratory study concluding that early-life seizures resulted in permanent physiological and behavioral changes in rat models, consistent with clinical and experimental ASD).[8] While Dr. Shafrir acknowledged that K.A.'s medical history does not support a diagnosis of epilepsy, he nevertheless maintained that K.A.'s seizure condition is connected to his autism diagnosis. Shafrir First Rep. at 38.[9]

Dr. Shafrir next considered a genetic component associated with both epilepsy and autism, and the degree to which it may establish an autoimmune basis for autism. As Dr. Shafrir explained, abnormalities in the Caspr2 gene[10] have been suggested to have a causal relationship with both autism and epilepsy in infants. Shafrir First Rep. at 38-39; *see* K. Strauss, et al.,

[8] Dr. Shafrir also relied on two papers drawing an association between autism and seizure disorders. *See* M. Matsuo, et al., *Characterization of Childhood-onset Complex Partial Seizures Associated with Autism-Spectrum Disorder*, 20 Epilepsy & Behavior 527 (2011), filed as Ex. 34 (ECF No. 94-6) (cohort study concluding that ASD is "very common" in patients with childhood development of complex seizures); P. Bernard, et al., *Early Life Seizures: Evidence for Chronic Deficits Linked to Autism and Intellectual Disability Across Species and Models*, 263 Exp. Neurol. 72, 76 (2015), filed as Ex. 34 (ECF No. 94-8) (reviewing recent developments concerning the association between ELS and autism, and concluding the causal relationship is still in its infancy).

[9] Dr. Shafrir's report also discusses literature evidencing signs of epileptic discharge following DTaP and DT vaccinations. Shafrir First Rep. at 41; S. Nouna, et al., *Adverse Effects on EEG and Clinical Condition After*, 32 Acta Paediatrica Japonica 357 (1990), filed as Ex. 34 (ECF 96-8) (Nouna). Dr. Shafrir asserted that Nouna described the appearance of epileptic discharges in the EEG of children with a history of seizure disorder after DTaP vaccination (who had a normal EEG prior to vaccination). Shafrir First Rep. at 41. As noted above, however, K.A.'s EEG is not consistent with an epilepsy diagnosis, and he has never received such a diagnosis either.

[10] Caspr2, also known as contactin associated protein like 2 (CNTNAP2), is a gene responsible for coding proteins which function in the vertebrate nervous system as cell adhesion molecules and receptors. Caspr 2 proteins also mediate interactions between neurons and glia during nervous system development and are involved in localization of potassium channels. *See CNTNAP2: Contactin Associated Protein Like 2*, NCBI, https://www.ncbi.nlm.nih.gov/gene/26047 (last accessed on May 3, 2018).

Recessive Symptomatic Focal Epilepsy and Mutant Contactin-Associated Protein-Like 2, 354 New Eng. J. Med. 1370 (2006), filed as Ex. 34-11 (ECF No. 95-2); D. Obregon, et al., *Potential Autoepitope within the Extracellular Region of Contactin-Associated Protein-Like 2 in Mice*, 4 Bit. J. Med. & Med. Research 416 (2014), filed as Ex. 34-13 (ECF No. 95-4) ("Obregon").[11] In Obregon, researchers identified the Caspr2 protein as a target for autoantibodies found in the serum of autistic children. Shafrir First. Rep. at 39. Obregon's authors used protein sequence databases to show homology between several amino acid sequences in infectious agents and the Caspr2 protein (including three segments of five to six amino acids in the filamentous hemagglutinin in the Bordetella pertussis vaccine) – thereby suggesting an autoimmune component to these conditions. *See, e.g*, G. Lilleker, et al., *VGKC Complex Antibodies in Epilepsy Diagnostic Yield and Therapeutic Implications*, 22 Seizures 776 (2013), filed as Ex. 34-14 (ECF No. 95-5). However, it does not appear that Dr. Shafrir has opined that a gene mutation theory is applicable in the present matter, or even that K.A. had the Caspr2 mutation (let alone any genetic disorder at all relevant to his vaccine injury claim).

As additional support for the autoimmune character of the injury in question, Dr. Shafrir relied on a variety of case reports and studies that he maintained associate DTaP or other vaccines with various antibody-mediated autoimmune encephalopathies[12] – and in particular anti-NMDA-receptor encephalitis.[13] First Shafrir Rep. at 40; S. Irani, et al, *N-methyl-D-aspartate Antibody Encephalitis: Temporal Progression of Clinical and Paraclinical Observations in a Predominantly Non-paraneoplastic Disorder of Both Sexes*, 133 Brain 1655, 1655-67 (2010),

[11] In his supplemental report, Dr. Shafrir noted that Obregon included a "significant error." Shafrir Second Rep. at 7. Specially, the filamentous hemagglutinin protein does not contain certain amino acid sequences (CSSR2 and CSSR3) as Obregon purports to show. *Id.* However, Dr. Shafrir indicated in his supplemental report that he did not rely exclusively on these two sequences in opining on molecular mimicry in the present context. *Id.* Thus, in his view Obregon's other statements relating to homology between the components in the DTaP vaccine and human proteins (other than CSSR2 and CSSR3) remain reliable. *Id.*

[12] Dr. Shafrir also cited literature discussing the causal relationship between autoimmune encephalitis in the context of anti-VGKC (voltage-gated potassium channel proteins) autoantibodies, and the capacity for such an encephalitis to in turn produce developmental regression. First Shafrir Rep. at 42, 43-44. R. Dhamija, et al., *Neuronal Voltage-gated Potassium Channel Complex Autoimmunity in Children*, 44 Pediatric Neurol. 275, 275 (2011), filed as Ex. 43 (ECF No. 97-4) ("Dhamija"). Dhamija was conducted by the Mayo Clinic and involved twelve patients, all with positive anti-VGKC antibodies; one patient was a twenty-nine month old who suffered autistic regression following receipt of the pertussis vaccine. According to Dr. Shafrir, the Dhamija authors concluded that the regression was the direct result of the cross-reaction. Shafrir Rep. at 43. However, Dhamija specifically attempts to document autoimmunity targeting VGKC complexes in the sera of children. More specifically, the authors conclude that larger case studies are needed to investigation the association between autism/developmental regression and the presence of VGKC antibodies. The paper does not otherwise suggest the vaccine was itself causal. Dhamija at 1, 6.

[13] Anti-NMDA-receptor encephalitis is an autoimmune, neurologic disease where the body creates antibodies against the NMDA receptors in the brain, disrupting signaling patterns in the brain and causing brain inflammation. Symptoms can include a paranoia, aggression, speech problems, movement disorders, and autonomic dysfunction. *See Anti-NMDAR Encephalitis: What is Anti-NMDA Receptor Encephalitis*, Cent. for Autoimmune Neurology, Perleman Sch. Med. U. Penn., https://www med.upenn.edu/autoimmuneneurology/nmdar-encephalitis.html (last accessed on May 3, 2018).

filed as Ex. 34 (ECF No. 96-9) (thirteen-year-old female received DTaP vaccine one day prior to seizure onset); J. Dalmau, et al., *Clincal Experience and Laboratory Investigations in Patients with Anti-NMDAR Encephalitis*, 10 Lancet Neuro. 63, 66 (2011), filed as Ex. 34 (ECF No. 96-10) (two patients out of four hundred developed anti-NMDAR encephalitis after the flu vaccine and one patient developed symptoms after a receipt of a DTaP booster vaccination). Indeed – Dr. Shafrir maintained that reliable literature actually establishes a causal connection between NMDA-receptor encephalitis and autistic regression. *See* O. Scott, et al., *Anti-N-Methyl-D-Aspartate (NMDA) Receptor Encephalitis: An Unusual Cause of Autistic Regression in a Toddler*, J. Child Neuro. (2013), filed as Ex. 34 (ECF No. 97-5) ("Scott") (case report finding that 33-month-old patient with NMDAR encephalitis developed autistic syndrome, but concluding *only* that NMDAR encephalitis was a possible cause, and noting that further studies will be needed). Although Dr. Shafrir acknowledged that autoimmune encephalitis in children is "exceedingly rare . . . as there are only a small number of cases in which autoimmune encephalitis led to autistic syndrome[,]" he maintained that the phenomenon is still significant and supportive of his theory. Shafrir First Rep. at 44. There is, however, no evidence herein that K.A. possessed the autoantibodies associated with NMDA encephalitis (or *any other* known autoantibody-driven encephalitis), and he certainly was never so diagnosed.

Dr. Shafrir next attempted to propose a specific mechanism by which the DTaP vaccine could have produced an initial encephalopathic reaction in K.A. – molecular mimicry. As the literature filed in support of this concept explains, molecular mimicry is a process occurring when the body is exposed to a foreign antigen (whether via infection or the components of a vaccine) that resembles, or "mimics," a self-structure, resulting in a cross-reaction when antibodies produced by the immune system to attack the foreign antigen also mistakenly attack the self. *See* N. Agmon-Levin, et al., *Vaccines and Autoimmunity*, 5 Perspectives 648 (2009), filed as Ex. 24 (ECF No. 96-5) ("Agmon-Levin"); Shafrir First Rep. at 40. Dr. Shafrir proposed that the immune attacks occurring in K.A.'s brain were "probably related to an increase in the level of antibodies against brain proteins," in combination with K.A.'s weakened state as a result of earlier vaccinations. *Id.*

In support of the above, Dr. Shafrir relied on literature exploring the possibility that molecular mimicry was the applicable mechanism by which a specific formulation of the flu vaccine caused a narcolepsy outbreak in Europe. M. Partinen, et al., *Increased Incidence and Clinical Picture of Childhood Narcolepsy Following the 2009 H1N1 Pandemic Vaccination Campaign in Finland*, 7 PlosOne E33723 (2012), filed as Ex. 34 (ECF No. 96-4) ("Partinen"). Partinen discussed protein sequence fragments common to both the flu virus and the H1N1 flu vaccine, hypothesizing that these fragments could generate a cross-reactive immune response to a self receptor in the brain linked to narcolepsy based on similarities between the two. Partinen did not, however, involve the DTaP vaccine—the vaccine implicated in Dr. Shafrir's theory –

and the article by its own terms involves a distinguishable condition that only shares with autism its neurologic character.[14]

Dr. Shafrir also attempted to establish protein sequential homology between components of the DTaP vaccine and human protein structures in the body. First Shafrir Rep. at 39-40; D. Kanduc, et al., *Peptide Cross-Reactivity: The Original Sin of Vaccines*, Frontiers Bioscience, filed as Ex. 34-15 (ECF No. 95-6) ("Kanduc"). Kanduc examined the tetanus toxins in the DTaP vaccine and determined sufficient identity existed between sequential fragments of those vaccine components and multiple genes and channel receptors in the body (including sodium channels, and the KCMA1, CAC1C, GBRB2, and NLGN3 genes). Shafrir First Rep. at 39. Researchers have also analyzed the pertussis bacterium and found a large amount of homology between the pertussis hemagglutinin protein component and several synaptic proteins (for example SCN8A). *Id.* at 39-40. Dr. Sharfri acknowledged that Kanduc did not analyze actual sequences in human patients or animal models, but he maintained the study lends support for a theory based on molecular mimicry reactivity and vaccine injuries at large. *Id.* at 40.

In addition to offering molecular mimicry as a possible mechanism, Dr. Shafrir maintained that the pertussis vaccine is generally a strong inducer of brain inflammation. Shafrir First Rep. at 41; S. Lassman, et al., *Induction of Type 1 Immune Pathology in the Brain Following Immunization Without Central Nervous System Autoantigen in Transgenic Mice with Astrocyte-Targeted Expression of IL-12*, 167 J. Immunol. 5485 (2001), filed as Ex. 34-25 (ECF No. 96-7). Lassman analyzed the peripheral immune system stimulation in mice (using the pertussis toxin) and found that mice immunized with the toxin developed neurological symptoms. Lassman at 5-7**.** Although Dr. Shafrir acknowledged that the mice used in the experiment had an "engineered" immune system, he maintained that the study should not be discounted as unpersuasive in support of a vaccine-induced pertussis encephalopathy. Shafrir First Rep. at 41.

Dr. Shafrir concluded his first report by discussing the extent to which genetically-determined immune dysfunction could provide a fertile environment for the development of autism. Shafrir First Rep. at 44; R. Sacco, et al., *Genome-Wide Expression Studies in Autism Spectrum Disorders: moving from Neurodevelopment to Neuroimmunology Genomics, Proteomics, and the Nervous System*, 2 Adv. Neurobio. 469 (2011), filed as Ex. 38 (ECF No. 97-10); P. Goines, et al., *Cytokine Dysregulation in Autism-Spectrum Disorders (ASD): Possible*

[14] Dr. Shafrir's supplemental report additionally included some discussion of the role inflammatory cytokine production can play with regard to vaccine-induced encephalopathies in the DTaP/epilepsy context. Shafrir Second Rep. at 3-4. He appears to have offered it, however, mainly in support of Petitioner's argument that K.A.'s onset was medically acceptable, rather than to establish some other mechanistic basis by which the DTaP vaccine could cause encephalopathy – and I do not find that the limited evidence he offers relating to the above was sufficient from a preponderant basis in any event.

Role of the Environment, 36 Neurotoxicol. Teratol. 67-81 (2013), filed as Ex. 37 (ECF No. 97-9). Because, he opined, "the only documented effective therapy for autism is immunosuppressive therapy," an exaggerated immune response must in turn have some relationship with autism. Shafrir First Rep. at 44; M. Chez, et al., *Immune Therapy in Autism: Historical Experience and Future Directions with Immunomodulatory Therapy*, 7 Neurotherapeutics 293 (2010), filed as Ex. 34 (ECF No. 98-5) ("Chez"); F. Duffy, et al., *Corticosteroid Therapy in Regressive Autism: A Retrospective Study of Effects on the Frequency Modulated Auditory Evokes Response (FMAER), Language, and Behavior*, 14 BMC Neuro. 1 (2014), filed as Ex. 34 (ECF No. 98-6) ("Duffy"). These studies, however, simply discuss the effectiveness of steroid treatment as it relates to autism, and do not conclude or explore the thesis on behalf of which they have been invoked. Moreover, K.A. has not been shown to have a dysfunctional immune system – let alone one caused by genetic mutation or similar factors.

Dr. Shafrir's Supplemental Expert Report

Dr. Shafrir's supplemental report mainly sought to address the report of Respondent's expert, Dr. Gregory Holmes. In response to Dr. Holmes's critiques of the proffered causation theory in this case, Dr. Shafrir argued that it was not his intent to opine that a vaccine could cause autism *directly*, but instead to demonstrate that (based upon literature cited in his earlier report) vaccinations could cause autoimmune phenomena, presumably with developmental injuries as a secondary effect. Second Shafrir Rep. at 5. While acknowledging the literature he offered involved patients with clinical pictures different from K.A.'s herein, Dr. Shafrir nevertheless maintained that "newly emerging" concepts in the field of pediatric neurology connected together into a plausible theory of causation. *Id.*

Dr. Shafrir also attempted to clarify statements in his first report relating to the nature of immune dysfunction in autism. Shafrir Second Rep. at 6. In his view, autoimmunity is a major factor underlying the pathophysiology and risk factors involved in autistic syndrome. *Id.* For support, Dr. Shafrir relied on the papers cited in his original report, but also submitted for consideration additional literature published after he wrote his initial report in this case, all of which, he argued, establish a reliable association between autism and an increased susceptibility for autoimmune disorders, although all conclude that additional research is necessary to determine the causal nature.[15]

[15] *See, e.g.*, M. Careaga, et al., *Immune Endophenotypes in Children with Autism Spectrum Disorder*, 15 Biol. Psychiatry 738, 738-60 (2015), filed as Ex. 35 (ECF No. 106-7) (study examining immune responses in male children, fifty with autism and sixteen without, and determining that autistic patients experienced greater cytokine production/greater behavioral impairment after lipopolysaccharide stimulation); C. McDougle, et al, *Toward an Immune-Mediated Subtype of Autism Spectrum Disorder*, 18 Brain Research 72, 72-92 (2015), filed as Ex. 35 (ECF No. 106-8) (paper discussing the relationship between familial autoimmune disorders and autism, post-mortem neuroimaging studies, present animal model work in ASD, and immunotherapy drug treatment for ASD, and suggesting that future research could help define the role of immune factors and inflammation in ASD); O. Zerbo, et al., *Immune-Mediated Conditions in Autism Spectrum Disorders*, 46 Brain Behav. Immun. 232, 232-36 (2015), filed

Dr. Shafrir further maintained that K.A.'s medical record supported his contention that K.A. had experienced an "immune attack" on the brain (a point Dr. Holmes endeavored to refute) resulting in encephalopathy. Shafrir Second Rep. at 4. In so doing, Dr. Shafrir admitted that he could identify no evidence in K.A.'s medical records supporting his contention that K.A. suffered from immune dysfunction, or that his purported encephalopathy had been caused by an autoimmune process in the first place. *Id.* at 5, 7. But he maintained that the lack of evidence of an autoimmune response could be attributable to the fact that none of K.A.'s treaters ever looked for it. *Id.* More broadly, Dr. Shafrir argued that K.A.'s medical record demonstrated a repeated series of neurological reactions to separate vaccinations at different times, even though he could not pinpoint the abnormality that caused each. *Id.* at 6. K.A., in his view, had experienced several encephalopathic episodes following his immunizations, which he deemed "compatible with [an autoimmune reaction] possibility." *Id.* at 7.

Dr. Shafrir particularly took issue with Dr. Holmes's assertion that K.A.'s seizure disorder began *before* the July 28, 2003 vaccinations. Shafrir Second Rep. at 1. In so arguing, Dr. Shafrir maintained that one of K.A.'s treaters, Dr. Rioux, had erred in his characterization of K.A.'s seizure disorder (when conducting an EEG during a July 22, 2004 office visit) as "ongoing," when in fact K.A. had experienced no ongoing seizures until after K.A.'s receipt of the flu vaccine in December 2003. *Id.* at 1. Further, Dr. Shafrir contended that Dr. Rioux incorrectly labeled K.A.'s July 22, 2004 sleeping-state EEG report as normal. *Id.* According to Dr. Shafrir, the report was conducted during wakefulness only, and thus could not be read as Dr. Holmes proposed. *Id.*

Finally, Dr. Shafrir attempted to bulwark the concept that the timeframe of K.A.'s injuries was medically acceptable, addressing Dr. Holmes's brief criticism of Petitioner's assertion that an autoimmune reaction is possible within hours of a vaccination.[16] Shafrir Second Rep. at 3. In his view, a period of several hours is a sufficient period of time for an immune reaction to occur. *Id.* For support, Dr. Sharfir listed a host of articles discussing an acceptable timeframe for an immune response. *Id., citing* D. Skowronski, et al., *Injection-site Reactions to Booster Doses of Acellular Pertussis Vaccine: Rate, Severity, and Anticipated Impact*, 112 Pediatrics E453, E456 (2003), filed as Ex. 35 (ECF No. 105-10) ("Skowronski") (DTaP injection-site reaction can occur in two to three hours following vaccination); N. Matin, et al.,

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as Ex. 35 (ECF No. 106-9) (case-control study suggesting that children with autism have elevated presence of specific immune-related comorbidities, but concluding that more research is needed to determine the type of association that exists between the two); L. Matelski, et al., *Thinking Outside the Brain*, 67 J. Autoimmun. 1, 1-7 (2016), filed as Ex. 35 (ECF No. 106-10) (paper discussing, among other things, the evidence of the immune system's role in ASD, including maternal infection during pregnancy, familial autoimmunity, immune cell anomalies found in children with ASD).

[16] It does not appear that Dr. Shafrir addressed symptom onset in his first report, apart from a passing conclusory reference that onset occurred within an "expected" timeframe. Shafrir First Rep. at 45.

Epilepsy and Innate Immune System: A Possible Immunogenic Predisposition and Related Therapeutic Implications, 11 Human Vaccines & Immunotherapeutics, 2021, 2023 (2003), filed as Ex. 35 (ECF No. 106-1) (innate immune system reaction, such as increased glial and microglial cells can produce pro-inflammatory cytokines within minutes of onset of seizures); Y. Kashiwagi, et al., *Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influensae Type B (Hib), and 7-Valent Pneumococcal (PCV7) Vaccines*, 10 Human Vaccines Immunotherapeutics 677, 677-85 (2014), filed as Ex. 35 (ECF No. 106-5) (cytokine production began six hours after stimulation with vaccinations, including DPT, Hib, and PVC7). He did not, however, provide any basis derived from his own experience to explain why the facts of this case were consistent with such literature, nor did he go into detail as to his specific views on this causation factor (apart from opining that the literature cited above is sufficient to support his contention that an autoimmune reaction can occur within hours of a vaccination).

B. Dr. Gregory Holmes

Dr. Holmes provided one expert report for Respondent. Dr. Holmes is board certified by the American board of Psychiatry and Neurology with a special qualification in child neurology. Report, dated Feb. 2, 2016 (ECF No. 102-1) (Ex. A) ("Holmes Rep.") at 1; ECF No. 102-2 (Ex. B) ("Holmes CV"). He received his bachelor's degree from Washington and Lee University and his medical degree from the University of Virginia. Holmes CV at 2. Following medical school, Dr. Holmes completed two residencies, one in pediatrics from Yale University School of Medicine and another in neurology from the University of Virginia. *Id.* Currently, Dr. Holmes is a professor of neurological sciences and pediatrics, and chair of the neurological sciences department at the University of Vermont College of Medicine in Burlington, Vermont. Holmes Rep. at 1. His expertise is focused on pediatric neurology with an emphasis in pediatric epilepsy. *Id.* Previously, Dr. Holmes held positions at both Dartmouth-Hitchcock Medical Center and Children's Hospital in Boston. Holmes CV at 3.

Dr. Holmes has demonstrated expertise in the fields of child neurology and epilepsy. He has served on the editorial board of multiple peer-reviewed journals, including the *Journal of Epilepsy* and the *Journal of Child Neurology*. Holmes CV at 8. He has also served as a primary investigator during multiple clinical evaluations involving antiepileptic drugs. *Id*. at 4-5. Today, Dr. Holmes routinely serves as a lecturer for the American Academy of Clinical Neurophysiology and the American Academy of Neurology, and he has also published extensively in the field of pediatric neurology. *See generally* Holmes CV.

Dr. Holmes asserted that Dr. Shafrir had provided no compelling causation theory that vaccines can cause autism. Holmes Rep. at 22.[17] In particular, he dismissed the scientific and

[17] Indeed, Dr. Holmes stated that he found Dr. Shafrir's overall opinion somewhat confusing, noting that it was not

medical literature offered in support of the argument that vaccines, through the mechanism of molecular mimicry, can cause autoimmune phenomena as speculative at best, or inapplicable to the present case (for example, Partinen, which involved flu vaccine-caused narcolepsy and not autism). *Id.* at 22. Dr. Holmes also emphasized that molecular mimicry is a pathological mechanism that can more easily be triggered by an infectious agent than a vaccine. *Id.*; M. De Martino, et al., *Vaccines and Autoimmunity*, 26 Int'l J. Immunopathol. & Pharmacol. 433, 433 (2013), filed as Ex. A-31 (ECF No. 118-1) (concluding that vaccines are not a source of autoimmune diseases, but rather that infectious agents are the primary trigger of autoimmune mechanisms).

Dr. Holmes next attacked the basis for Dr. Shafrir's opinion that autoimmune encephalitis caused K.A.'s autism. Holmes Rep. at 23. One article Dr. Shafrir heavily relied upon for this argument (R. Dhamija, et al., *Neuronal Voltage-gated Potassium Channel Complex Autoimmunity in Children*, 44 Pediatric Neurol. 275, 275 (2011), filed as Ex. 43 (ECF No. 97-4) ("Dhamija")), for example, did not implicate any vaccine as the cause of the patient's encephalopathy, but rather asserted that the condition could be caused by a familial history or viral illness. *Id.*; Dhamija at 6-7. Dr. Holmes also dismissed literature discussing the developmental regression of a 33-month-old patient who was found to have anti-NMDA receptor antibodies in his cerebrospinal fluid, noting that the autoimmune response therein was not considered to have been vaccine-mediated. Holmes Rep. at 23-24; *see* Scott at 691-94. While Dr. Holmes agreed that autoimmune encephalitis can in rare circumstances lead to autistic behavior in a small number of cases, K.A.'s medical history did not establish he had experienced an autoimmune encephalitis at any time. *Id.*[18]

Dr. Holmes also discussed the relationship between seizures, epilepsy, and autism emphasized by Dr. Shafrir. Even though autism does not have a clear etiology, the prevalence of autism in individuals with epilepsy is 22.2 percent higher than in the general population, and Dr. Holmes acknowledged that children who experience seizures early in life also develop ASDs at a higher rate. Holmes Rep. at 20; *see* A. Selassie, et al., *Epilepsy Beyond Seizure: A Population-Based Study of Comorbidities*, 108 Epilepsy Research 305, 305 (2014), filed as Ex. A-4 (ECF No. 115-4). However, he denied that this created a causal relationship between the two, suggesting instead that pathophysiological properties shared between the two "far more likely" explained the symptoms K.A. experienced. Holmes Rep. at 21.

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clear from Dr. Shafrir's report if he was arguing that K.A. had developed autism due to a toxin in one of his vaccines, a poor immune response, or via molecular mimicry. Holmes Rep. at 21-22.

[18] Dr. Holmes similarly contested Dr. Shafrir's basis for his argument that the pertussis vaccine is a strong inducer of brain inflammation, and could therefore encourage encephalopathy. Holmes Rep. at 23. Although Dr. Holmes agreed with Dr. Shafrir's assertion that a pertussis vaccine-induced encephalopathy was possible (albeit rare), he maintained his view that no record evidence indicated that K.A. had ever experienced any kind of immune

In response to Dr. Shafrir's discussion of immune dysfunction and autoimmunity in autism, Dr. Holmes countered that the medical literature in fact did not support the conclusion that autism is an autoimmune disorder. First Holmes Rep. at 24. Thus, Dr. Holmes dismissed Dr. Shafrir's reasoning that autism must be autoimmune because the only documented effective therapy for autism is immunosuppressive steroid therapy. *Id.*; M. Chez, et al., *Immune Therapy in Autism: Historical Experience and Future Directions with Immunomodulatory Therapy*, 7 Neurotherapeutics 293 (2010), filed as Ex. A-38 (ECF No. 118-8) (stating that neither immunization nor thimerosal exposure has been conclusively linked to autism); F. Duffy, et al., *Corticosteroid Therapy in Regressive Autism: A Retrospective Study of Effects on the Frequency Modulated Auditory Evoked Response (FMAER), Language, and Behavior*, 14 BMC Neurol. 70 (2014), filed as Ex. A-39 (ECF No. 118-9). Rather, Dr. Holmes argued that there are many other effective treatments having nothing to do with immunology, such as behavioral therapies. *Id.*; *see* D. Granpeesheh, et al., *Applied Behavior Analytic Interventions for Children with Autism: A Description and Review of Treatment Research*, 21 Ann. Clin. Psychiatry 162 (2009), filed as Ex. A-40 (ECF No. 118-10). Overall, Dr. Holmes maintained that the usefulness of treating ASD patients with steroids "does not indicate that autism is a disorder of autoimmunity." Holmes Rep. at 24.

In Dr. Holmes's view, K.A.'s autism disorder could not be associated with any of the vaccines he received. Holmes Rep. at 24. Rather, Dr. Holmes attributed K.A.'s immediate symptoms to his seizure disorder (which he opined began approximately *two weeks prior* to K.A.'s July 28, 2013 vaccinations), with the autism symptoms that K.A. later displayed in 2004 reflective of a typical ASD clinical course. *Id.* at 21, 22. In so arguing, Dr. Holmes acknowledged that K.A. had experienced an additional cluster of seizures following the vaccines received on July 28, 2013, but maintained that "onset of afebrile seizures within hours of the vaccination would make an autoimmune cause of the seizures untenable." *Id.*

The record otherwise, in Dr. Holmes's view, established that the July 28th seizures were not reflective of an encephalopathic reaction to vaccination (for example, K.A.'s EEG and MRI scan conducted that same day produced normal results). First Holmes Rep. at 20. Dr. Holmes also maintained that the medical record did not support Dr. Shafrir's assertion that K.A. had experienced an autoimmune encephalopathy and/or recurring "immune attacks" on the brain as a result of any of his vaccinations, pointing out that there was no evidence of neurologic injury or deterioration, and that none of K.A.'s treaters ever proposed that he had suffered an encephalopathy. Holmes Rep. at 21-22.

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dysfunction reflective of such a process. *Id*.

III. PROCEDURAL HISTORY

As noted above, this action (originally assigned to former Special Master Hastings) was initiated in May 2005, nearly thirteen years ago. Petition at 1. Petitioner filed this claim in conjunction with the Omnibus Autism Proceedings ("OAP")[19], resulting in a long hiatus in the progress of this matter while the OAP was concluded. *See* Order, dated Mar. 15, 2010 (ECF No. 11). The case was formally separated from the OAP on January 12, 2011, in light of Petitioner's expressed desire to proceed on an alternate theory. *See* Order, dated Jan. 12, 2011 (ECF No. 15); Order, dated Sept. 30, 2011 (ECF No. 18). Petitioner filed the majority of the medical records associated with this case in February 2012 (ECF No. 27).

Petitioner's original counsel withdrew from the case on August 7, 2012 (ECF No. 38). After counsel's withdrawal, Special Master Hastings directed Petitioner to file an expert report on or before March 4, 2013 (ECF No. 40). Thereafter, Petitioner made numerous requests for extensions of time to file the expert report. *See* ECF Nos. 41, 44, 47. In the interim, Respondent filed his Rule 4(c) Report in September 2013 (ECF No. 49) contesting the propriety of compensation.

On January 9, 2014, Petitioner obtained new counsel. ECF Nos. 52-53. That same day, Special Master Hastings again directed Petitioner to file a status report within thirty days,

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[19] In the OAP, thousands of petitioners' claims that certain vaccines caused autism were joined for purposes of efficient resolution. A "Petitioners' Steering Committee" was formed by many attorneys who represent Vaccine Program petitioners, with about 180 attorneys participating. This group chose "test" cases to represent the entire docket in the OAP, with the understanding that the outcomes in these cases would be applied to cases with similar facts alleging similar theories.

The Petitioners' Steering Committee ultimately chose six test cases to present two different theories regarding autism causation. The first theory alleged that the measles portion of the MMR vaccine precipitated autism, or, in the alternative, that MMR plus thimerosal-containing vaccines caused autism, while the second theory alleged that the mercury contained in thimerosal-containing vaccines could affect an infant's brain, leading to autism.

The first theory was rejected in three test case decisions, all of which were subsequently affirmed. *See generally Cedillo v. Sec'y of Health & Human Servs*., No. 98-916V, 2009 WL 331968 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *mot. for review den'd*, 89 Fed. Cl. 158 (2009), *aff'd*, 617 F.3d 1328 (Fed. Cir. 2010); *Hazlehurst v. Sec'y of Health & Human Servs*., No. 03-654V, 2009 WL 332306 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *mot. for review den'd*, 88 Fed. Cl. 473 (2009), *aff'd*, 605 F.3d 1343 (Fed. Cir. 2010); *Snyder v. Sec'y of Health & Human Servs*., No. 01-162V, 2009 WL 332044 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 88 Fed. Cl. 706 (2009).

The second theory was similarly rejected. *Dwyer v. Sec'y of Health & Human Servs*., No. 03-1202V, 2010 WL 892250 (Fed. Cl. Spec. Mstr. Mar. 12, 2010); *King v. Sec'y of Health & Human Servs*., No. 03-584V, 2010 WL 892296 (Fed. Cl. Spec. Mstr. Mar. 12, 2010); *Mead v. Sec'y of Health & Human Servs*., No. 03-215V, 2010 WL 892248 (Fed. Cl. Spec. Mstr. Mar. 12, 2010).

After the OAP's conclusion, a total of 11 lengthy decisions by special masters, the judges of the U.S. Court of Federal Claims, and the panels of the U.S. Court of Appeals for the Federal Circuit had unanimously rejected the petitioners' claims. These decisions found no persuasive evidence that the MMR vaccine or thimerosal-containing vaccines caused autism. The OAP proceedings concluded in 2010.

updating the Court on the status of the expert report (originally requested in March 2013). *See* ECF No. 52. Petitioner continued to request extensions of time to file an expert report from January 2014 to November 2015, finally filing Dr. Shafrir's first report on November 23, 2015. Thereafter, Respondent filed an expert report from Dr. Holmes on February 18, 2016, with Dr. Shafrir's supplemental report filed on May 4, 2016.

The case was reassigned to me on April 19, 2017 (ECF No. 109). On May 5, 2017, I held a status conference with the parties after reviewing the case record and each side's expert reports in greater detail. By this point, it was evident that Petitioner's causation theory relied on the determination that K.A. had experienced a vaccine-induced encephalopathic reaction that was subsequently exacerbated by additional vaccinations, and that resulted in seizures plus eventual developmental regression (and an autism diagnosis). But it was equally evident that the medical record (which contained the most probative and reliable evidence as to K.A.'s history) did not support Petitioner's contentions. I thus informed Petitioner that I had serious concerns about the claim's viability, in light of both the history of Program claims asserting a similar theory, as well as my own experience deciding similar cases in which vaccinations were alleged to have been linked to an autism injury.

Given the above, during the May 2017 conference I proposed that, in lieu of a hearing, the parties brief Petitioner's claim, and I set a schedule for so doing in a Non-PDF Order, dated May 5, 2017. Over several months, the parties filed their respective briefs, as referenced at the outset of this decision, on August 14, 2017, and October 12, 2017, respectively. Along with her brief, Respondent filed a Motion for Ruling on Record. ECF No. 127. Prior to filing her brief in support of her claim, Petitioner filed an Amended Petition on August 11, 2017 (ECF No. 123). In it, Petitioner specifically deleted autism as an alleged injury, and instead asserted only a vaccine-induced developmental regression (accompanied by increased seizure activity) as the alleged vaccine-caused injuries.

IV. Parties Respective Arguments

Petitioner's Brief

In her opening brief, Petitioner maintains that she has established preponderant evidence in support of her causation-in-fact claim under each of the three prongs set forth by the Federal Circuit in *Althen v. Sec'y of Health & Human Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005). First, Petitioner proposes that Dr. Shafrir has offered a reliable theory establishing that vaccines can cause autoimmune encephalopathies, later capable of producing developmental regression (and ultimately evolving into an autism diagnosis). Mem. at 50-51. For the second, "did cause" prong, Petitioner argues that K.A.'s medical records support a causal connection between the July 28,

2013 vaccination and onset of his seizures. Mem. at 52. In particular, Petitioner cites to one instance in the medical record where a treating pediatrician made a reference to spreading out future vaccinations over a period of time to reduce possible immune system stimulation and risk of further seizure activity (Ex. 8 at 2), which she argues shows that K.A.'s treaters were concerned about the relationship between his seizures and additional vaccinations. *Id.* at 52-53. Petitioner otherwise relies on the fact that K.A. experienced seizures post-vaccination on two occasions, followed by developmental regression after a third-vaccination to establish a logical cause and effect sequence. *Id.* at 53.

Finally, Petitioner contends that the timeframe in which K.A.'s vaccine reaction and subsequent developmental problems occurred was medically appropriate. Mem. at 53. She notes that Dr. Shafrir's report (showing that the medical concept of anamnesis[20] does not prescribe a timeframe for sensitization reactions, for example) supports the conclusion that four hours is an acceptable timeframe for an onset of seizures following vaccination, as in K.A.'s case. *Id.* at 54. Furthermore, Petitioner argues that this same concept would support an onset of seizures within six days of K.A.'s flu vaccine, and onset of developmental regression within a month of K.A.'s DT vaccine. *Id.*

Petitioner also asserts that a hearing should be held, to permit a closer examination of the facts and expert opinions set forth herein, especially because (in her view) the causation theory asserted herein differs from those examined in the OAP. Mem. at 46-47. It also appears from Petitioner's brief that she is concerned that K.A.'s autism diagnosis, coupled with the case's former inclusion in the OAP, will be overemphasized in rendering a decision. *Id.* Furthermore, Petitioner argues that the medical facts of the present case (described as "three episodes of post-vaccine seizures, coupled with dramatic regression after [a] third") merit the kind of detailed evaluation more likely at a hearing. *Id.* at 48.

Respondent's Brief

Respondent contests the adequacy of Petitioner's showing so much that he maintains reasonable basis for the claim is in question. First, Respondent argues that the medical record does not support Petitioner's contention that K.A. suffered any encephalopathy post-vaccination. Mot. at 6. Rather, K.A. developed a seizure disorder in 2003 prior to vaccination (which he was subsequently treated for), and later developed autism in 2004. *Id.*

Second, Respondent proposes that Petitioner's causation theory is deficient, as it improperly (and broadly) concludes that vaccines can cause an autoimmune reaction via

[20] Anamnesis, or immunologic memory, refers to "the elevated immune response following a secondary or tertiary administration of immunogen to a recipient previously primed or sensitized to the immunogen (i.e. the secondary response)." *Illustrated Dictionary of Immunology* 39 (3rd ed. 2009), filed as Ex. 35 (105-8).

molecular mimicry that will eventually result in developmental regression or an autism diagnosis. Mot. at 6-7. In fact, Dr. Shafrir's theory (based primarily on literature suggesting only *possible associations* between vaccine-induced encephalopathies manifesting as ASD) has been fully adjudicated and determined to be unreliable in numerous other cases. Mot. at 5, 8-13; *see, e.g.*, *R.V. v. Sec'y of Health & Human Servs.*, No. 08-504V, 2016 WL 3882519 (Fed. Cl. Spec. Mstr. Feb. 19, 2016), *mot. for review den'd,* 127 Fed. Cl. 136 (2016). Respondent argues that such cases are not factually distinguishable in any meaningful sense, and that Petitioner's theory represents "an effort to avoid the unfavorable precedent facing petitioners who attempt to advance a case alleging that vaccines cause autism . . . ." Mot. at 13.

Finally, Respondent proposes that Dr. Shafrir lacks sufficient expertise on the immunologic or molecular biologic issues implicated in his theory to even opine reliably as to the issues in the present matter. Mot. at 8-9.

Petitioner's Reply

On Reply, Petitioner asserts that Respondent has "distorted the facts" in K.A.'s medical records as they apply to his initial, purportedly pre-vaccination seizure event. Reply at 10. K.A.'s treating doctor, Petitioner maintains, actually described this event as merely an "acute life-threatening event" (with a presumption of gastrointestinal reflux), and not a seizure, and that only one treater later characterized it as a seizure during subsequent treatment visits. *Id.* (citing Ex. A at 20). Otherwise, Petitioner maintains that K.A.'s onset of seizures (following his vaccinations between June 2003 and December 2003),[21] followed by his onset of autistic features post-vaccination in 2004, "infuses [P]etitioner's case with credibility." *Id.* at 14. Petitioner repeats her earlier concerns that Respondent overemphasizes K.A.'s autism diagnosis in her causation analysis (as opposed to the seizure evidence). *Id.* at 3. Overall, Petitioner continues to assert that the present case is distinguishable from other autism cases based on the medical facts, and that Respondent's reliance on K.A.'s autism diagnosis as relevant to the case herein should be disregarded. *Id.* at 3-7.

IV. APPLICABLE LEGAL STANDARDS

A. Petitioner's Burden in Vaccine Program Cases

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury" – i.e., an injury falling within the Vaccine Injury Table –

[21] Petitioner's Reply also repeats her earlier arguments that K.A.'s medical record clearly documents evidence of encephalopathy, including "loss of balance, weakness or numbness in part of [K.A.'s] body, blurred speech or loss of speech, ataxia, cognitive impairment, agitation, [and] sleeplessness." Mot. at 14. However, Petitioner cites no record support for these assertions.

corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006).[22] In this case, Petitioner does not assert a Table claim.

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

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim (which is the kind of claim asserted in this matter), a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

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

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

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Human Servs.*, 121 Fed. Cl. 230, 245 (2015) ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)). But this does not negate or reduce a petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & 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. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record – including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742,

749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 136 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd,* 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *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 also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id*. at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Factual Determinations

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

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical

records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Human Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs*., No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs*., 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms. It is equally unlikely that pediatric neurologists, who are trained in taking medical histories concerning the onset of neurologically significant symptoms, would consistently but erroneously report the onset of seizures a week after they in fact occurred").

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

However, there are situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Human Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Human Servs*., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In

determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y Health & Human Servs*., 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records over contrary testimony, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc*., 509 U.S. 579, 594-96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742-45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be

"based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 Fed. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339).

D. Consideration of Medical Literature

Both parties relied on a few pieces of medical and scientific literature in this case in support of their respective positions. I have reviewed all of the medical literature submitted in this case, although my decision does not discuss each filed article in detail. *Moriarty v. Sec'y of Health & Human Servs.*, No. 2015-5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted).

E. Determination to Resolve Case without Hearing

I have opted to decide entitlement in this case based on written submissions and evidentiary filings, including the expert reports filed by each side. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers rather than via evidentiary hearing, where (in the exercise of their discretion) they conclude that the former means of adjudication will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The choice to do so has been affirmed on appeal. *See Hooker v. Sec'y of Health & Human Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Human Servs.,* 38 Fed. Cl. 397, 402-03 (1997) (special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Human Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Ct. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

After careful review of the expert reports, medical records, and the arguments of both sides, and taking into account my own experience resolving similar claims (as well as numerous parallel decisions from other Vaccine Act cases), I conclude that Petitioner has not established preponderant evidence in favor of her claim.

A. K.A. Did Not Experience a Post-Vaccination Encephalopathy.

Petitioner's claim depends on a fact finding that K.A. suffered from an encephalopathy prior to his alleged regression and/or seizures and other symptoms – making that a threshold matter for resolution. *See Broekelschen*, 618 F.3d at 1346 (when an injury or diagnosis is disputed, and "the proposed injuries differ significantly in their pathology," the special master may "first find which of [the] diagnoses was best supported by the evidence presented in the record before applying the *Althen* test so that the special master could subsequently determine causation relative to the injury"). The facts from the medical records, however, do not support the conclusion that K.A. experienced *any* kind of encephalopathy reaction after his July 28, 2013, vaccinations (or subsequent vaccinations in December 2013 and June 2014). *See* Ex. 11 at 64-65.

As I have discussed in prior decisions, although the term "encephalopathy" is less strictly defined in the context of a non-Table claim, it nevertheless is not so elastic as to include *any* possible type of brain injury no matter the degree. *Murphy v. Sec'y of Health & Human Servs.,* No. 05-1063V, 2016 WL 3034047, at *25-26 (Fed. Cl. Spec. Mstr. Apr. 25, 2016), *mot. for review den'd*, 128 Fed. Cl. 348 (2016); *see also Wright v. Sec'y of Health & Human Servs.,* No. 12-423V, 2015 WL 6665600, at *6 (Fed. Cl. Spec. Mstr. Sept. 21, 2015). Thus, even though a petitioner with a non-Table causation-in-fact claim may evade some of the Table's requirements for establishing an encephalopathy (such as that it is both "acute" and "chronic" as defined by the Table's "Qualifications and Aids to Interpretation" ("QAI") (42 C.F.R. § 100.3(b)(2)), a non-Table petitioner will still need to point to reliable evidence from the record establishing that the injured party's symptoms were sufficiently evident and severe to constitute an encephalopathy. *Murphy*, 2016 WL 3034047, at *32 (record did not support contention that child had experienced a post-vaccination encephalopathy).

The decisions of other special masters in non-Table cases have identified the specific kinds of evidentiary factors that would suggest an individual had experienced an encephalopathy. These include evidence of crying, anorexia, insomnia, fever, moodiness, irritability, and/or depression. *See, e.g.*, *Cook v. Sec'y of Health & Human Servs.*, No. 00-331V, 2005 WL 2659086, at *14 (Fed. Cl. Spec. Mstr. Sept. 21, 2005); *Noel v. Sec'y of Health & Human Servs.*, No. 99-538V, 2004 WL 3049764, at *17 (Fed. Cl. Spec. Mstr. Dec. 14, 2004) (non-Table encephalopathy at issue characterized by moaning, high-pitched and eerie crying, and unresponsiveness). While a petitioner might reasonably also seek to include seizures in that partial list, proof of seizures *alone* is generally not considered sufficient to establish an encephalopathy. *See, e.g.*, *Borin v. Sec'y of Health & Human Servs.,* No. 99-491V, 2003 WL 21439673, at *8 (Fed. Cl. Spec. Mstr. May 29, 2003) ("[a] child with no symptoms other than

seizures who is alert, well-appearing, non-toxic, behaving normally, playful, interactive, smiling, and cooing does not have an acute encephalopathy, either Table or non-Table.").

Because of the above, special masters have been reluctant to make a finding of encephalopathy solely where an injured party displays symptoms manifesting *only* as seizures, without something also indicating greater brain dysfunction or the presence of inflammation. *See, e.g.*, *Oliver v. Sec'y of Heatlh & Human Servs.*, No. 10-394V, 2017 WL 747846, at *27 (Fed. Cl. Spec. Mstr. Feb. 1, 2017) (denying entitlement where petitioner experienced seizure within 24 hours post-vaccination, but returned to baseline and experienced no other encephalopathic symptoms), *aff'd*, 133 Fed. Cl. 341, 353 (2017), *appeal docketed*, No. 17-2540 (Fed. Cir. Sept. 13, 2017); *Mohamud v. Sec'y of Health & Human Servs.*, No. 09-812V, 2013 WL 5314611, at * 11 (Fed. Cl. Spec. Mstr. Aug. 30, 2013) (denying entitlement where petitioner experienced seizure within 24 hours of vaccination, but MRI and EEG were normal and no other evidence indicated the presence of brain inflammation).

Here, proof that K.A. experienced a non-Table encephalopathy would need to include medical record evidence detailing severe symptomology (similar to that mentioned above) in addition to seizure activity. But the record in this case establishes only that K.A. developed acute onset seizures immediately following his receipt of the July 28, 2003 vaccinations. The record is subsequently bereft of evidence corroborative of encephalopathy. On the contrary, K.A. appeared healthy and normal both at the time of his July vaccinations (after his arguable first seizure (Ex. 8 at 30; Ex. 14 at 5)), as well as immediately thereafter (Ex. 8 at 57), and then was deemed generally healthy from a neurologic standpoint as well in the months thereafter before his next significant seizure activity in December 2003. In addition, K.A.'s EEG (a test which would more likely than not reveal neurological damage) performed on the same day as his July vaccinations was normal (Ex. 11 at 53), as was his MRI conducted that same day (Ex. 11 at 53). And none of K.A.'s treaters ever proposed that he suffered from an encephalopathic reaction. Indeed, Dr. Spears, K.A.'s treating neurologist, was aware that K.A. had received vaccinations earlier that day, but concluded that they were not likely causative of his condition. Ex. 11 at 64.

Similarly, K.A.'s records surrounding his seizure episode in December 2013 do not allow for the conclusion that he more likely than not suffered any type of encephalopathy following the receipt of the flu vaccine roughly one week prior. Ex. 8 at 11. First, the renewed seizure activity occurred a few days after vaccination rather than immediately (unlike the July 2013 incident), and in response to a different vaccine. Second, K.A. subsequently saw another neurologist, Dr. Rioux, who (like Dr. Sears) opined that K.A.'s seizure disorder was unrelated to his vaccinations. Ex. 5 at 7. Accordingly, this instance, like the July 2013 seizure instances, can only invoke the temporal relationship between vaccine receipt and seizure to establish evidence of encephalopathy.

I also take note of K.A.'s ensuing medical history, between the time of his July 2003 and December 2003 seizure events. Those records generally establish that K.A.'s anti-seizure treatments were effective, and display no larger concerns about his development or status. Ex. 8 at 14, 57; Ex. 14 at 2-5, 25. And the record of later vaccines K.A. received in June 2004 (closer in time to when his developmental problems manifested) is similarly characterized by an absence of evidence that could be interpreted as indicia of encephalopathy, while also being temporally attenuated by *almost a month* from any additional seizure activity. Ex. 14 at 35-36. The overall record simply does not support the conclusion that K.A. ever experienced a post-vaccination encephalopathy at any time.[23]

B. Petitioner Has Not Established a Reliable or Persuasive Causation Theory.

Dr. Shafrir maintained that a component in the DTaP vaccine could initiate an autoimmune process and thereby produce an encephalopathy (somehow encouraged by the other vaccinations K.A. experienced) sufficient to result in developmental regression a year later. Petitioner did not offer direct evidentiary support establishing the capacity of DTaP vaccines to function as alleged, in this or other analogous circumstances. Nor did Dr. Shafrir invoke any persuasive studies involving these vaccines, or others, and their roles in incidents of developmental regression.

Instead, Dr. Shafrir relied on a loose chain of circumstantial propositions in order to establish Petitioner's causation theory. But (and even though circumstantial evidence is wholly acceptable as a general matter in Vaccine Program cases) those propositions were themselves insufficiently supported with reliable scientific evidence required to conclude that the overall theory was plausible. As another special master noted in considering the *Althen* prong one analysis, "[t]he weight to be given an expert's opinion is based in part on the size of the gap between the science and the opinion proffered." *Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 3012), *aff'd*, 108 Fed. Cl. 743 (2013) *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013). That gap is quite wide in this case.[24]

[23] It is instructive to compare the facts of this case with those exceedingly rare cases in which a claimant has established an encephalopathy resulting in ASD-like symptoms (although both involved Table claims in which causation was assumed). In one such instance, the vaccinated child developed a very high fever within 48 hours of vaccination, thereafter displaying crying, sleeplessness, and significant motor problems, all of which were documented in the medical record. *Poling v. Sec'y of Health & Human Servs.*, No. 02-1466V, 2011 WL 678559, at *1 (Fed. Cl. Spec. Mstr. Jan. 28, 2011). In another, the vaccinated child received a multi-virus vaccine and then experienced a seizure on the trip home from the vaccination, followed by a week of noticeably decreased levels of consciousness and lethargy. *Wright v. Sec'y of Health & Human Servs.*, No. 12-423V, 2015 WL 6665600 (Fed. Cl. Spec. Mstr. Sept. 21, 2015). In Petitioner's case, by contrast, there are no records establishing any sort of proximate temporal reaction to the vaccines that would support a finding that K.A. experienced such an encephalopathy.

[24] I also note (as I have in other cases) that Dr. Shafrir lacks the qualifications necessary to offer persuasive expert testimony regarding the propensity of vaccines to cause neurologic injury sufficient to result in autism. *See T.M. v. Sec'y of Health & Human Servs.*, No. 08-284V, 2016 WL 11087157, at *28 (Fed. Cl. Spec. Mstr. Aug. 9, 2016),

For example, certain literature offered in support of Petitioner's theory, like Partinen, not only involved a different vaccine and injury (the flu vaccine and narcolepsy), but also centered on a pathologic process reasonably understood to be autoimmune – something that cannot possibly be said for autism or developmental regression. Partinen at 7-8. In addition, the literature cited with regard to anti-NMDAR or anti-VGKC autoantibody-mediated encephalitis establishes only weak evidence of a possible association between autoimmunity and autism. *See, e.g.*, Dhamija at 6-7. Notably, Petitioner offered no record evidence that relevant testing suggested that K.A. had either of these antibodies in his body (close-in-time to the vaccine or any time thereafter). Similarly, articles such as Agmon-Levin were simply too broad in focus to constitute persuasive support for the theory offered in this case; simply because other vaccines have been linked to other kinds of autoimmune-associated conditions does not mean the same can be concluded to have occurred *in this case*. *See R.K. v. Sec'y of Health & Human Servs.,* No. 03-0632V, 2015 WL 10936124 (Fed. Cl. Spec. Mstr. May 23, 2016) (categorizing Dr. Shafrir's theory as a "one size fits all" approach to autoimmune reactions), *mot. for review den'd*, 125 Fed. Cl. 57 (2016), *aff'd*, 671 F. App'x 792 (2016).

Petitioner's causation theory has an even more fundamental weakness: its striking similarity to theories universally rejected in the Vaccine Program. To date, *every* non-Table claim seeking compensation for autism injuries purportedly related to a vaccine and litigated since completion of the OAP has failed. *See, e.g.*, *Hardy v. Sec'y of Health & Human Servs*., No. 08-108V, 2015 WL 7732603, at *4-5 (Fed. Cl. Spec. Mstr. Nov. 3, 2015) (referencing eleven autism claims unsuccessfully tried, plus six that were rejected (over the petitioners' objections) without trial). I myself have tried several such cases, with the same result. *See, e.g.*, *Anderson v. Sec'y of Health & Human Servs.*, No. 02-1314V, 2016 WL 8256278 (Fed. Cl. Spec. Mstr. Nov. 1, 2016), *mot. for rev. den'd*, 131 Fed. Cl. 735 (2017), *aff'd*, 717 F. App'x 1009 (Fed. Cir. 2018); *T.M. v. Sec'y of Health & Human Servs.*, No. 08-284V, 2016 WL 11087157, at *28 (Fed. Cl. Spec. Mstr. Aug. 9, 2016), *mot. for rev. den'd*, 133 Fed. Cl. 78 (2017); *Murphy*, 2016 WL 3034047, at *25-26; *R.V.*, 2016 WL 3882519.

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mot. for rev. den'd, 133 Fed. Cl. 78 (2017). Although Dr. Shafrir is competent to testify on a wide array of neurological disorders (including autism itself), he is not an immunologist, and has no relevant background experience treating or studying the effects of vaccines on individuals. I have considered his testimony carefully, but it is reasonable to afford it significantly less weight since it comes from a person who lacks the qualifications necessary to advance it. *Daubert v. Merrell Dow Pharm., Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995) ("[o]ne very significant fact to consider is whether the experts are proposing to testify about matters growing naturally and directly out of research they have conducted independent of the litigation, or whether they have developed their opinions expressly for purposes of testifying"). Dr. Shafrir's qualifications and expertise have similarly been called into question by other special masters, and his testimony described as vague and poorly supported – a criticism leveled against him herein as well by Dr. Holmes. *See, e.g.*, *Lehner v. Sec'y of Health & Human Servs.,* No. 08-554V, 2015 WL 5443461 (Fed. Cl. Spec. Mstr. July 22, 2015).

Petitioner argues that the theory offered in this case is qualitatively different. But other petitioners have similarly attempted to recast their claim that a vaccine caused an autism injury as a claim that the vaccine precipitated some form of encephalopathy (more often than not autoimmune in nature) that *later* produced developmental problems (whether or not they are defined as an ASD) due to the resulting neurologic injury. *See, e.g., Cunningham v. Sec'y of Health & Human Servs.*, No. 13-483V, 2016 WL 4529530 (Fed. Cl. Spec. Mstr. Aug. 1, 2016), *mot. for review den'd*, 2017 WL 1174448 (Fed. Cl. Jan. 25, 2017). Such efforts have been correctly understood as seeking to evade the weight of negative precedent involving autism claims – as here. *Cunningham*, 2017 WL 1174448, at *7-8 ("[r]egardless of petitioner's attempt to differentiate this case from other autism cases by creating this second step, the Special Master rightfully classified this case as an autism case").

All in all, the theory proposed herein is too similar to previously-rejected theories to find it reliable and persuasive – and has significant lapses in its chain of propositions in any event.

C. Petitioner Has Not Established that K.A.'s Vaccines Did Cause Encephalopathy or Seizures Resulting in Autism.

Petitioner's obligation under the second *Althen* prong was to demonstrate a logical sequence of cause and effect connecting the particular facts of her case to her medical theory. *See, e.g., Sturdivant v. Sec'y of Health & Human Servs.*, No. 07-788V, 2016 WL 552529, at *18 (Fed. Cl. Spec. Mstr. Jan. 21, 2016) (discussing that prong two requires a fact-based inquiry into whether the vaccine in question *did* cause the particular injury). But her theory depended on acceptance of her allegation that K.A. suffered from an encephalopathy – a conclusion the record does not support, and thereby rendering it impossible for her to establish that the vaccines K.A. received caused injury.

Even if, however, I had found that K.A. had experienced one or more encephalopathies – or that his seizure disorder was reflective of an encephalopathy – I would still be unable to find based upon the present record that any of the vaccines he received caused that injury. There is no evidence in the record that K.A. was undergoing an autoimmune process of the kind Petitioner's causation theory suggests he should have been experiencing in connection with his alleged encephalopathy. Indeed, Dr. Shafrir acknowledged that (a) K.A.'s clinical picture was different from articles cited pertaining to autoimmune encephalopathies, (b) K.A.'s records do not indicate a history of autoimmune disease, and (c) no medical evidence otherwise exists to establish the autoimmune character of K.A.'s injuries. Ex. 35 at 5, 7.

Moreover, there is credible evidence in the record that K.A. experienced a seizure-like episode *prior* to the series of vaccinations received on July 28, 2003, and which are alleged to have triggered his first encephalopathy. *See* Ex. 11 at 2-12. Although attending physicians labeled this initial episode as an "acute life-threatening event," later-in-time treaters reasonably

took it into account in understanding K.A.'s overall condition, deeming it a seizure episode related to K.A.'s subsequent activity. *See* Ex. 11 at 64; Ex. 14 at 8. The medical record in any Program case must be looked at from an overall standpoint, as evidence that treaters understand at one point may later be viewed otherwise after the passage of time, and after more facts are adduced in the course of treatment. *See, e.g.*, *Bell v. Sec'y of Health & Human Servs.*, No. 13-709V, 2016 WL 8136297, at *24-25 (Fed. Cl. Spec. Mstr. Dec. 1, 2016); *Blackburn v. Sec'y of Health & Human Servs*., No. 10-410V, 2015 WL 425935, at *25 (Fed. Cl. Spec. Mstr. Jan. 9, 2015). Petitioner did not successfully rebut the reasonable inference that the pre-July incident was related to the subsequent seizure series, and that inference casts further doubt on Petitioner's contention that K.A.'s vaccines caused an encephalopathy or seizure disorder (if not completely eliminating the possibility that the July 2003 vaccine initiated a process later resulting in developmental problems and/or autism).

In addition, K.A.'s treaters largely did not opine that he had suffered a vaccine-induced injury (encephalopathic or any other). Admittedly, as Petitioner points out, at least one of K.A.'s treaters (Dr. Rioux) allowed for the vaccination schedule to be adjusted based on parental concerns about possible negative effects (Ex. 14 at 8, 42; Ex. 8 at 20-21; Ex. 5 at 7-8). Thereafter, K.A.'s mother relayed her concerns (along with Dr. Rioux's "recommendation") to K.A.'s pediatrician during subsequent office visits (Ex. 14 at 8, 42). This alone is not sufficiently strong and probative evidence of a *treater* view that the vaccine was causal. Although a treating physician's recommendation to withhold a particular vaccination can be probative evidence of a causal link between the vaccination and an injury sustained, special masters are less likely to find a causal link where the treater does not seem to have a sound scientific rationale – or, as here, offers no explanation at all. *See e.g.*, *Mosely v. Sec'y of Health & Human Servs.,* No. 08-724V, 2015 WL 2354316, at *18 (Fed. Cl. Spec. Mstr. Apr. 27, 2015) (physician's recommendation to withhold vaccination unpersuasive causation evidence where the same treater offered no explanation for his statements); *Arango v. Sec'y of Health & Human Servs.*, No. 09-318V, 2012 WL 4018028, at *21 (Fed. Cl. Spec. Mstr. Aug. 23, 2012) (physician's recommendation to withhold vaccination unpersuasive where the same treater testified that the vaccine was not the cause of the injury), *aff'd*, 109 Fed. Cl. 335 (2013). Here, the statements made by K.A.'s mother to his pediatrician are uncorroborated by evidence of affirmatively-voiced treater opinion that any of the vaccines K.A. received had any connection at all to his injuries,[25] and thus could reasonably be interpreted as a physician acquiescing to a caregiver concern rather than an informed decision bearing strongly on causation.

[25] Although Dr. Rioux's office notes indicate that he could find no precise etiology for K.A.'s seizure disorder, he allowed for the possibility that a combination of antibiotics and vaccinations could have "lowered [K.A.'s] seizure threshold." Ex. 5 at 8. As noted earlier, however, Dr. Rioux ultimately opined that K.A.'s immunizations were not responsible for his on-going seizure difficulties. *Id.*

D. Petitioner Has Not Shown the Timeframe for Development of his Injuries was Medically Acceptable.

The component of Dr. Shafrir's theory addressing the timeframe in which the various vaccines that allegedly injured K.A. would do so is deficient both in specific and general ways. Looking only at the specific support offered for the theory reveals numerous unreliable inconsistencies. Thus, and as Respondent argued, although Dr. Shafrir attempted to bulwark the exceedingly short timeframe between the July 2003 vaccinations and K.A.'s subsequent acute onset seizures (which would, according to Petitioner, provide some initial evidence of the supposed encephalopathy), the scientific evidence provided in support was inconsistent with the aspects of his theory relating to the biologic mechanism that he proposed had caused K.A.'s reaction, molecular mimicry, which his own literature (and/or counter evidence offered by Dr. Holmes) suggested would take somewhat longer. *See* Holmes Rep. at 21; Mot. at 15 n.10. His timing arguments also conflated evidence pertaining to the timeframe in which adaptive immune responses (like an autoimmune process mediated through molecular mimicry) would occur with evidence bearing on the comparatively shorter timeframes involved in innate responses (such as an injection site or hypersensitivity reaction). *See, e.g.*, Skowronski at E456. These inconsistencies are not adequately resolved in Dr. Shafrir's reports.

More generally, however, and relying on incontrovertible facts from K.A.'s medical history, it is impossible to discern an explanation in this case that can credibly and persuasively harmonize the varying response times at issue. Ignoring the initial, potentially-related seizure incident that predated the late-July 2003 vaccinations, K.A. experienced seizures (a) the same day as the July 2003 vaccinations, (b) none after vaccines received in the fall of 2003, (c) approximately a week after the December 2003 flu vaccine was administered, and (d) about a month after two more vaccines were administered in June 2004 on two different occasions. The temporal irregularity of these reactions is not consistent with what would be seen in the context of "challenge-rechallenge," where reactions to a series of vaccines should occur in increasingly shorter intervals. *See e.g.*, *Gramza v. Sec'y of Health & Human Servs.*, No. 15-247, 2018 WL 1581674, at *10, 24 (Fed. Cl. Spec. Mstr. Feb. 5, 2018), *appeal docketed*, No. 15-247 (Fed. Cl. Mar. 7, 2018). Petitioner's *Althen* prong three showing also cannot explain why *no* evidence of developmental regression is seen before July 2004 – a year from the first alleged reaction, and with no evidence in that period that developmental problems had begun. Petitioner's expert and the various pieces of literature filed in connection cannot provide a reasonable, reliable framework that makes scientific sense of this ambiguous and inconsistent fact pattern.

E. This Case was Properly Resolved without a Hearing.

In ruling on the record, I am declining Petitioner's request for a hearing. The choice of how best to resolve this case is a matter that lies generally within my discretion, but given Petitioner's protestations I shall explain my reasoning.

A hearing provides a petitioner with the opportunity to put on live testimony, which aids the special master most in cases where witness (or expert) credibility is at issue, or where there is a need to pose questions to a witness in order to obtain information not contained in, or not self-evident from, the existing filings. *See, e.g.*, *Hooker v. Sec'y of Health & Human Servs.*, No. 02-472V, 2016 WL 3456435, at *21 (Fed. Cl. Spec. Mstr. May 19, 2016) (discussing a special master's discretion in holding a hearing and the factors that weighed against holding a hearing in the matter); *Murphy v. Sec'y of Health & Human Servs.*, No 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991) (no justification for a hearing where the claim is fully developed in the written records and the special master does not need to observe the fact witnesses for the purpose of assessing credibility), *aff'd*, 1991 WL 74931 (Fed. Cl. Apr. 25, 1991) *aff'd*, 968 F.2d 1226 (Fed. Cir. 1992). It may also permit a claimant to expand upon or illuminate points already set forth in paper filings, or respond to unanticipated questions raised in the matter – but again, only where necessary to reach a decision.

Prior decisions have recognized that a special master's discretion in deciding whether to conduct an evidentiary hearing "is tempered by Vaccine Rule 3(b)," or the duty to "afford[] each party a full and fair opportunity to present its case." *Hovey v. Sec'y of Health & Human Servs.*, 38 Fed. Cl. 397, 400-01 (1997) (citing Rule 3(b)), *appeal dismissed*, 135 F.3d 773 (Fed. Cir. 1997). But that rule also includes the obligation of creation of a record "sufficient to allow review of the special master's decision." *Id*. Thus, the fact that a claim is legitimately disputed, such that the special master must exercise his intellectual faculties in order to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes *without* a hearing, and may do so if the record at issue has been sufficiently developed to determine that each side's "full and fair" opportunity has not been abridged.

In this case, live witness testimony was not required in order for me to reach a reasoned decision. The record itself was expansive and contained sufficient evidence upon which to base this decision. As the lengthy procedural history indicates, Petitioner was given ample extensions of time in which to obtain an expert report, and to marshal the arguments she made in opposing Respondent's motion on the record. The flaws in Petitioner's theory and factual arguments were self-evident from review of the medical records and the expert reports submitted, which relied heavily on speculative assertions and statements unsupported by the contemporaneous medical record and not bulwarked by sufficient reliable scientific evidence. And, as noted above, Petitioner's expert offered opinions regarding autism or developmental regressions as a vaccine injury - consistent with theories that have previously been deemed scientifically unreliable and unpersuasive. I have heard Dr. Shafrir testify in a similar context before, and relying on similar scientific evidence, and need not hear him again to understand the logic of his position from the filed written reports. *See, e.g.*, *T.M.*, 2016 WL 11087157, at *10-15; *R.V.*, 2016 WL 3882519, at

*15-20. I simply did not require oral testimony to decide the case. On the contrary: the congruence of the theory espoused herein with numerous, previously-rejected variations on the same theme, plus based upon my own prior experiences with the theory, counseled against expending the time and effort a hearing entails.[26]

At bottom, the most significant issue in deciding whether to hold a hearing is whether the refusal to do so will deprive the claimant of the fair opportunity to prosecute her case. Petitioner here has received such an opportunity. Her counsel has litigated to trial many similar claims involving the same kind of injuries, and is well aware of how such claims have fared; the likely outcome would have been no different had a trial been held, regardless of Petitioner's hopes. A hearing would also have unnecessarily postponed the date by which the matter could be fully resolved. Such circumstances ultimately counseled in favor of resolving the matter on the papers.

CONCLUSION

The record does not support Petitioner's contention that the vaccines K.A. received could, or did, cause his developmental regression, seizure disorder, or autism, nor has she established it more likely than not that he ever suffered from a post-vaccine encephalopathy injury. Petitioner has not established entitlement to a damages award, and therefore I must **DISMISS** her claim.

In the absence of a timely-filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accordance with this decision.[27]

IT IS SO ORDERED.

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

[26] The decision not to hold a hearing because the claim reflected a frequently-litigated theory is not something that would only ever negatively impact a petitioner. The opposite circumstances – where a petitioner asserted a claim that has repeatedly *succeeded* in the past – would motivate me to act in the same manner, and propose to Respondent that either the case be settled or that it too be resolved on the papers.

[27] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.