

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Case No. 09-684V

Filed: March 7, 2014

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MELISSA DAVIS & CECIL DAVIS, SR.,
As the Parents and Natural Guardians of
C.D., an Infant,

Petitioners,

v.

SECRETARY OF HEALTH
AND HUMAN SERVICES,

Respondent.

* * * * *

PUBLISHED

Special Master Dorsey

Entitlement; Decision without
a hearing; Ruling on the record;
Pediarix; Prevnar; PedvaxHIB;
Encephalopathy; Seizures;
Congenital disorder of glycosylation;
Mutation of *PIGT* gene.

Mark Theodore Sadaka, Englewood, NJ, for petitioners;

Justine Elizabeth Daigneault, U.S. Department of Justice, Washington, DC, for respondent.

DECISION ON ENTITLEMENT¹

I. Introduction

On October 9, 2009, Melissa Davis and Cecil Davis, Sr. (“petitioners”), as the parents and natural guardians of C.D., an infant, filed a petition for compensation under the National Vaccine Injury Compensation Program (“the Program”²), alleging that numerous vaccinations, including Pediarix,³ Prevnar,⁴ and PedvaxHIB,⁵ that C.D. received on October 18, 2006, caused

¹ This Decision was originally filed on February 19, 2014. On March 5, 2014, petitioners requested a redaction. The motion was granted in an Order filed on March 7, 2014. In the reissued version, the minor child’s birth date is redacted and this footnote is changed to reflect the redaction. The remainder of the Decision is unchanged.

² The Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C. §§ 300aa-10 *et seq.* (hereinafter “Vaccine Act” or “the Act”). Hereafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

³ Pediarix consists of diphtheria-tetanus-acellular-petussis (“DTaP”), hepatitis B, and inactivated polio virus (“IPV”) vaccines. See Centers for Disease Control & Prevention, Pediarix Vaccine: Questions and Answers, <http://www.cdc.gov/vaccines/vpd-vac/combo-vaccines/pediarix/faqs-hcp-pediarix.htm>.

her to suffer and continue to suffer from “vaccine induced encephalopathy with seizures and loss of muscle control, which were either ‘caused-in-fact’ by the above-stated vaccinations, or, in the alternative, significantly aggravated by the above-stated vaccinations.” Petition at 1. Petitioners subsequently clarified that C.D. “developed [a] vaccine-induced seizure disorder after receiving the second doses of Pediarix, PedvaxHIB, and Prevnar vaccines on October 18, 2006.” Amended Petition (“Am. Pet.”) at 1. The medical records and other information in the record, however, do not support a finding that petitioners are entitled to compensation under the Program.

Under the Program, petitioners may not receive compensation based solely upon their claims. In order to receive compensation, the petition must be supported by either medical records or the opinion of a qualified physician which proves a causal relationship. See § 300aa-13(a)(1). Here, the medical records do not support petitioners’ claims, so a medical opinion is required. Id. Petitioners have offered the opinion of Dr. Garrett C. Burris, a pediatric neurologist. See Petitioners’ Exhibit (“Pet’rs’ Ex.”) 16. But, as described in detail below, Dr. Burris’ opinion fails to provide support for the elements necessary to prove causation. Additionally, petitioners have failed to provide any expert report or opinion addressing the fact that C.D. has been diagnosed with an inherited genetic disorder, which is a “congenital disorder of glycosylation with two different mutations in the *PIGT* gene.” Pet’rs’ Ex. 42 at 1.⁶ For these reasons, and the reasons discussed below, petitioners have failed to demonstrate that they are entitled to compensation.

II. Procedural History

Petitioners filed their action for compensation on October 9, 2009, and the case was initially assigned to Special Master Dee Lord. Petitioners filed numerous medical records related to C.D.’s birth and medical care on January 6 and February 10, 2010. Respondent filed her Report pursuant to Vaccine Rule 4(c), in which she stated that medical personnel of the Division of Vaccine Injury Compensation had reviewed the petition and medical records and concluded

⁴ Prevnar 13 is a pneumococcal conjugate vaccine, also known as PCV 13. See Centers for Disease Control & Prevention, Pneumococcal Vaccination, available at <http://www.cdc.gov/VACCINES/vpd-vac/pneumo/default.htm>.

⁵ PedvaxHIB is a Haemophilus influenzae type b vaccine which prevents meningitis (an infection of the covering of the brain and spinal cord), pneumonia (lung infection), epiglottitis (a severe throat infection), and other serious infections caused by a type of bacteria called Haemophilus influenza type b. See Centers for Disease Control & Prevention, Hib Vaccination, available at <http://www.cdc.gov/Vaccines/vpd-vac/hib/default.htm>.

⁶ Petitioners have filed another petition for compensation on behalf of their other child, C.D.J, in which they allege that the same vaccines C.D. received caused the same injuries C.D.J suffered. See Davis v. Sec’y of Health & Human Servs., No. 10-615V. A similar Decision has been issued in that case. See Davis v. Sec’y of Health & Human Servs., No. 10-615V, Decision, filed February 19, 2014.

that the case was not appropriate for compensation under the Program. Respondent's Report ("Resp't's Rep't"), filed April 8, 2010, at 2.

On August 11, 2011, petitioners filed the expert report of Dr. Garrett Burris. Pet'rs' Ex. 16. On February 1, 2012, petitioners filed a supplemental report by Dr. Burris. Pet'rs' Ex. 18. On June 11, 2012, respondent filed the expert report of Dr. Gerald V. Raymond. Respondent's Exhibit ("Resp't's Ex.") A.

On July 13, 2012, petitioners were ordered to file an amended petition to clarify their allegations, which they did on August 6, 2012. Am. Pet., filed Aug. 6, 2012. In their amended petition, petitioners allege that C.D. developed a vaccine-induced seizure disorder five days after receiving the second doses of Pediarix, PedvaxHIB, and Prevnar vaccines on October 18, 2006. Am. Pet. at 2. Petitioners further allege that when C.D. received her second set of vaccinations, "she had an undiagnosed condition that is generally termed as a 'metabolic disorder'" and that persons with "metabolic disorders" are more susceptible to vaccine-related injury. Id. Petitioners allege that as a "direct result of receiving the second doses of Pediarix, Prevnar, and PedvaxHIB, C.D. suffered and continues to suffer from grand mal seizures to the extent that she is currently on medication for epilepsy." Id. at 3.

On August 28, 2012, this case was reassigned to Special Master George Hastings. A status conference was held on November 29, 2012. During that conference, counsel for petitioners indicated that while they had filed an expert report, they would likely be switching experts in the near future. See Order, filed Nov. 30, 2012, at 1. On January 28, 2013, petitioners filed a status report, stating that they had made "continuous efforts" but had been unable to find a new expert. See Status Report, filed Jan. 28, 2013, at 1.

On February 22, 2013, the case was reassigned to the undersigned. A status conference was held on March 28, 2013. As a result of that status conference, petitioners were ordered to file any outstanding medical records by March 29, 2013, and to file an expert report by May 31, 2013. Order, filed Mar. 28, 2013. On May 31, 2013, petitioners filed an unopposed motion for an extension of time until July 30, 2013, to file an expert report. That motion was granted. Order, filed May 31, 2013. On July 30, 2013, petitioners filed another motion for an extension of time until August 29, 2013, to file an expert report. That motion was again granted. Order, filed July 30, 2013. Petitioners did not file an expert report by August 29, 2013, as ordered. Instead, petitioners requested a status conference. See Status Report, filed Aug. 29, 2013, at 2.

Subsequently a status conference was held on September 17, 2013. During the status conference, counsel for petitioners stated that petitioners intended to file a motion for a decision on the record. See Order, filed Sept. 18, 2013. Petitioners were ordered to obtain and file all outstanding medical records from the National Institutes of Health ("NIH") and follow-up visits related to genetic testing by October 17, 2013. Id. On October 17, 2013, petitioners filed a motion for judgment on the administrative records. Motion, filed Oct. 17, 2013. On October 31, petitioners filed a letter from the NIH Undiagnosed Diseases Program, reporting on C.D.'s genetic tests results. Pet'rs' Ex. 42. Petitioners also filed a status report, stating that both C.D. and her younger brother had received a confirmed diagnosis of a "congenital disorder of

glycosylation with two different mutations in the *PIGT* gene. This diagnosis is an inherited autosomal recessive disease.” Status Report, filed Oct. 31, 2013, at 1.

Respondent filed a response to petitioners’ motion for judgment on the administrative record on January 17, 2014. Respondent argued that Dr. Burris’ “opinion regarding the genetic nature of [C.D.]’s condition is simply incorrect” and that “there is no reliable evidence that any of [C.D.]’s clinical manifestations were caused or exacerbated by any of the immunizations that she received.” Respondent’s Response to Petitioner’s Motion for a Decision on the Record (“Resp’t’s Resp.”), filed Jan. 17, 2014, at 18. Rather, respondent claimed that all of C.D.’s “symptoms...are presentations of [her] ...genetic disorder.” Resp’t’s Resp. at 16. Thus, respondent asserted that, “[o]n the present record, petitioners have clearly failed to meet their burden of proving by preponderant evidence that [C.D.]’s vaccinations either caused her injury or significantly aggravated her preexisting condition.” Id. at 1.

This case is now ripe for a ruling on petitioners’ motion for judgment on the record.

III. Summary of Relevant Medical Records and Other Filed Exhibits

C.D. was born prematurely at 31 weeks gestation on [redacted]. Pet’rs’ Ex. 1 at 1; Pet’rs’ Ex. 2 at 2. C.D. remained in the hospital for six weeks after she was born. Pet’rs’ Ex. 2 at 3. During her time in the hospital, C.D. suffered from respiratory distress syndrome, apnea,⁷ bradycardia,⁸ heart murmur, and anemia. Id. at 3-7. She was placed in an oxihood and required mechanical ventilation. Id. at 3-4. C.D. was discharged from the hospital on June 24, 2006. Id. at 3.

In July 2006, C.D. was diagnosed with an upper respiratory infection (“URI”) when she was seen in the Northwest Medical Center of Benton County. Pet’rs’ Ex. 12A at 6. It was noted that C.D.’s “eyes roll back” and that she “throws her head back” a lot at home. Id. at 2. On October 18, 2006, C.D. was seen for her well child check and to establish care by Carl Engmann, M.D. Pet’rs’ Ex. 7B at 181-82. Dr. Engmann observed that C.D. had lagging gross motor development, because she was unable to hold her head up or roll front to back. Id. at 182. C.D. was noted to be experiencing dystonia⁹ and esotropia,¹⁰ but not seizures or a seizure disorder. Id. On the same date, C.D. received her second DTaP, Hepatitis B, Hib, Pneumococcal, Pediarix, Prevnar, Pedvax, and IPV vaccinations. Pet’rs’ Ex. 3 at 1; Pet’rs’ Ex. 7B at 182.

⁷ Apnea is a “cessation of breathing.” Dorland’s Illustrated Medical Dictionary (32d ed. 2012) (“Dorland’s”) at 116.

⁸ Bradycardia is “slowness of the heartbeat, as evidenced by slowing of the pulse rate to less than 60.” Dorland’s at 245.

⁹ Dystonia is “dyskinetic movements due to disordered tonicity of muscle.” Dorland’s at 582.

¹⁰ Esotropia is “strabismus in which there is manifest deviation of the visual axis of an eye toward that of the other eye, resulting in diplopia.” Dorland’s at 648.

Approximately five days later, on October 23, 2006, an ambulance was called and C.D. was sent to the emergency room at North West Medical Center after “her first documented seizure.” Am. Pet. at 2; Pet’rs’ Ex. 12A at 11-14. Upon arrival to the emergency room, C.D. experienced another seizure, for which she was treated with Valium. Pet’rs’ Ex. 12A at 15. A chest x-ray showed that “bilateral interstitial opacities suggest[ed] pneumonia.” Id. at 40. Phenobarbital was administered to treat C.D.’s epilepsy. Id. at 83. C.D. was discharged on October 24, 2006, with anti-seizure medication and a recommendation for follow-up neurology consultation. Id. at 11-14.

On November 1, 2006, C.D. was seen by a pediatric neurologist at Arkansas Children’s Hospital, W. David Walters, M.D. Pet’rs’ Ex. 11A at 12-13. C.D. was diagnosed with partial complex seizures, hypotonia,¹¹ and developmental delay. Id. at 13; Pet’rs’ Ex. 11O at 1045-46. Dr. Walters also noted that C.D. had an elevated ammonia level during her previous hospitalization. Pet’rs’ Ex. 11A at 12. However, an EEG performed on November 1, 2006, was “essentially within normal limits for age.” Pet’rs’ Ex. 11O at 1051. During another visit on November 28, 2006, a brain MRI revealed that myelination¹² was delayed, even for a significantly premature infant, and the corpus callosum¹³ was thin and poorly myelinated. Pet’rs’ Ex. 11A at 26. C.D. was noted to have a chromosomal abnormality. Id. at 16.

In early 2007, C.D. continued to experience seizures and low muscle tone, and she also suffered from respiratory syncytial virus (“RSV”), bronchiolitis, URIs, and gastroesophageal reflux. Pet’rs’ Ex. 7B at 166, 172-75. C.D. started receiving speech, physical, and occupational therapies for her developmental delays and hypotonia. See, e.g., id. at 168. C.D. also had difficulty swallowing. Pet’rs’ Ex. 36.4 at 158. On June 20, 2007, C.D. received her third and final dose of PedvaxHIB. Pet’rs’ Ex. 3 at 1. Subsequently two different EEGs performed on July 23 and August 29, 2007, showed that she suffered from epilepsy. Pet’rs’ Ex. 11H at 546; Pet’rs’ Ex. 11O at 1047.

Dr. Walters suspected that a mitochondrial disorder might be the cause of C.D.’s seizures. Pet’rs’ Ex. 11A at 31-32. Dr. Walters noted that C.D. suffered from persistent low muscle tone, “numerous other minor anatomic anomalies,” and “marked gross motor delays” without regression. Id. at 48. Dr. Walters recommended “mitochondrial disease workup.” Id. at 49. C.D. was thereafter tested for mitochondrial disorder pursuant to Dr. Walters’ recommendation. Pet’rs’ Ex. 11B at 92; Pet’rs’ Ex. 11G at 450.

On August 25, 2007, C.D. was admitted to the Northwest Medical Center for grand mal seizures. Pet’rs’ Ex. 12B at 107, 140. She was transferred to Arkansas Children’s Hospital for

¹¹ Hypotonia is “a condition of diminished tone of the skeletal muscles, so that they have diminished resistance to passive stretching and are flaccid.” Dorland’s at 907.

¹² Myelination is “the act of furnishing with or taking on myelin; formation of a myelin sheath.” Dorland’s at 1218.

¹³ Corpus callosum is “an arched mass of white matter [in the brain], found in the depths of the longitudinal fissure, composed of three layers of fibers, the central layer consisting primarily of transverse fibers connecting the cerebral hemispheres.” Dorland’s at 417.

evaluation of seizures and a diagnosis of viral meningitis. Pet'rs' Ex. 11A at 58; Pet'rs' Ex. 11I at 612, 645. C.D. was discharged on August 31, 2007, and was noted to have a "suspected mitochondrial disorder." Pet'rs' Ex. 11A at 44.

On September 21, 2007, a muscle biopsy for myopathy with possible mitochondrial disease did not reveal any recognizable abnormalities. Id. at 66; Pet'rs' Ex. 11G at 450. In a follow-up visit, Dr. Walters noted that C.D.'s organic acids were normal, but that she had persistently high lactate/pyruvate level. Pet'rs' Ex. 11B at 129. On November 29, 2007, a Chromosomal Microarray Analysis¹⁴ report revealed that there were "[n]o abnormalities [] detected for the regions included on the current version of the Chromosomal Microarray." Pet'rs' Ex. 8 at 1.

In early 2008, while C.D. was hospitalized at Arkansas Children's Hospital for intractable seizures, she failed a swallow study and a gastrostomy tube was placed for feeding. Pet'rs' Ex. 11B at 123; Pet'rs' Ex. 11D at 225. By May 2008, C.D. continued to suffer from global developmental delay, hypotonia, eye turning, and seizures. Pet'rs' Ex. 7A at 24; Pet'rs' Ex. 7B at 148. She was functioning at about a four-month-old age level. Pet'rs' Ex. 7A at 24.

On August 6, 2008, an Arkansas Children's Hospital's report suggested that the cause of C.D.'s developmental delay and seizures had still not been identified. Pet'rs' Ex. 11D at 286. C.D. was started on a ketogenic diet, which is high in fat and low in carbohydrates. Id. On August 19, 2008, during a follow-up visit with Dr. Stephen G. Kahler for both C.D. and her younger brother, C.D.J., it was observed that C.D. and her brother shared similar clinical presentations. Pet'rs' Ex. 11E at 323. Dr. Kahler noted that the family history suggested similar autosomal recessive genetic condition. Id. Subsequently, a MitoMet oligonucleotide microarray ("CGH") analysis¹⁵ showed that no negative results and no abnormalities were detected. Pet'rs' Ex. 8 at 2; Pet'rs' Ex. 11F at 420.

In May 2009, C.D. was admitted for a comprehensive evaluation at the NIH. Pet'rs' Ex. 9 at 23. During the evaluation, C.D. underwent multiple tests and was seen by an ophthalmologist, an audiologist, a gastroenterologist, a physiatrist, an occupational therapist, a physical therapist, a dermatologist, and a neurogeneticist. Id. at 23-28. C.D. was diagnosed with profound global developmental delay, epileptic encephalopathy, hypotonia, brain abnormalities, and hearing loss. Id. at 28-29. A metabolic genetics laboratory report on October 07, 2009, for fragile X and organic acid metabolic screenings did not detect any abnormalities. Pet'rs' Ex. 11A at 4-5.

¹⁴ Chromosomal Microarray Analysis (CMA) is a "new molecular cytogenetic test designed to detect losses or gains representing deletions or duplications for a wide array of clinically significant regions of human genome." Pet'rs' Ex. 8 at 1.

¹⁵ "Comparative genomic hybridization is performed on a custom oligonucleotide microarray to assess for copy number changes in the mitochondrial genome and/or nuclear gene(s) of interest based upon the indication for testing. This method can simultaneously detect mtDNA depletion and deletions, and can estimate both the deletion breakpoints and the percentage of deletion heteroplasmy in the mtDNA." Pet'rs' Ex. 8 at 2.

Neurology clinic notes dated June 6, 2013, by Dr. Tonya M. Balmakund, described C.D.'s family history as "remarkable for a sibling with similar diagnosis." Pet'rs' Ex. 39 at 7. On July 25, 2011, Dr. Balmakund and C.D.'s mother corresponded by email, and Dr. Balmakund explained that C.D. and C.D.J.'s condition was not due to any alternative acquired disease, and that the "children are word for word out of texts for several metabolic and neurodegenerative diseases." See Davis v. Sec'y of Health & Human Servs., No. 10-615V, Pet'rs' Ex. 14.1 at 75-76.¹⁶

On October 31, 2013, petitioners filed a letter dated October 29, 2013, from Ms. Gretchen A. Golas, C.R.N.P., and Dr. Cynthia J. Tifft, Director, NIH Pediatric Undiagnosed Diseases Program. The letter states, in pertinent part, the following:

This letter is to confirm our recent phone conversation regarding the molecular diagnosis of a congenital disorder of glycosylation¹⁷ with two different mutations in the *PIGT* gene for both your children, C.D. and [C.D.J.]... Both C.D. and [C.D.J.] have inherited an autosomal recessive disease called a congenital disorder of glycosylation involving mutations in the *PIGT* gene. The complex pathway of glycosylation involves many different genes participating in the biochemical process of adding and removing sugars to/from proteins. *PIGT* is a gene that has been only very recently identified (May 2013) in the medical literature as an additional cause of a type of congenital disorders of glycosylation with the feature of intellectual disability.

Pet'rs' Ex. 42 at 1.

Petitioners subsequently filed a copy of a medical article referenced in the above letter.¹⁸ The article reports on the "novel autosomal recessive syndrome, characterised by distinct facial features, intellectual disability, hypotonia and seizures, in combination with abnormal skeletal, endocrine and ophthalmologic findings." Pet'rs' Ex. 43 at 1. The author examined four patients with a similar phenotype, using whole exome sequencing and identified a "homozygous mutation, c.547 A>C ... in *PIGT*." Id. Further study revealed that this mutation was the cause of a "novel autosomal recessive intellectual disability syndrome." Id. Patients with *PIGT* mutations have clinical findings which include hypotonia, mild microcephaly, impaired motor and cognitive development, severe motor and intellectual disability, abnormal brain imaging, seizures, initially normal EEG with subsequent development of seizures, impaired vision, renal abnormalities, skeletal abnormalities, cardiac abnormalities, mild dysmorphic facial features, and global cerebral atrophy. Id. at 5-7. The authors conclude that they have found "strong evidence that a syndrome of intellectual disability, hypotonia, seizures, and skeletal and ophthalmologic findings seen in the patients of this study is caused by mutations in *PIGT*." Id. at 7.

¹⁶ This letter was filed by petitioners on December 6, 2011, in petitioners' other child, C.D.J.'s, case. See Davis v. Sec'y of Health & Human Servs., No. 10-615V.

¹⁷ Glycosylation is "the formation of linkages with glycosyl groups." Dorland's at 794.

¹⁸ Pet'rs' Ex. 43, Malin Kvarnung, et al., "A novel intellectual disability syndrome caused by GPI anchor deficiency due to homozygous mutations in *PIGT*," 50 J. MED. GENETICS 8, 521-28 (August 2013).

IV. Expert Opinion

On August 11, 2011, petitioners filed the expert report of Dr. Garrett C. Burris, a child neurologist. Pet'rs' Ex. 16. In his report, Dr. Burris provided a brief summary of medical facts beginning with C.D.'s birth and ending with a short paragraph about C.D.'s admission to the NIH in May 2009. Id. at 1-2. Dr. Burris fails to reference any records after 2009, although his report is dated August 9, 2011. Most notably, there is no reference to C.D.'s genetic mutation in Dr. Burris' report.

Dr. Burris opined that within a reasonable degree of medical certainty, C.D. had vaccine-induced acute encephalopathy, consisting of seizures and "a decreased level of consciousness" occurring "5 days after vaccine administration." Id. at 3. Dr. Burris also opined that C.D. had chronic encephalopathy, "severe global developmental delays" and refractory seizures. Id. Dr. Burris concluded that "[t]he presence of acute encephalopathy and developmental regression with chronic encephalopathy following administration of vaccine is consistent with vaccine induced encephalopathy. There are no other indications of an etiology for this encephalopathy." Id.

In a supplemental expert report, Dr. Burris opined that C.D. had a metabolic disorder which made her more susceptible to developing an abnormal immune response to the vaccinations. Pet'rs' Ex. 18 at 1. Dr. Burris stated that metabolic disorders produce a chronic inflammatory state. Id. Dr. Burris further stated that foreign substances cause antigens and that vaccines elicit an immune response and the production of antibodies. Id. The risk of developing an autoimmune disease increases when vaccines are given to a "patient with a chronic inflammatory condition caused by a metabolic disorder," like C.D.'s. Id.

Dr. Burris identified four theories of autoimmunity: "molecular mimicry, bystander activation, persistent viral infections and fertile field." Id. Generally, Dr. Burris believes that there are two causal pathways of autoimmune epilepsy and encephalopathy – one is "that the body produces auto-antibodies for specific proteins necessary for proper neurological body function." Id. at 2. The second is that an "increased number of antibodies can trigger seizures and encephalopathy." Id. Dr. Burris did not know which causal pathway might be relevant in C.D.'s case. But he concluded that the "record clearly shows the development of seizures within days after vaccination and both children suffer from chronic inflammation caused by the metabolic disorder." Id.

Respondent filed the expert report of Dr. Gerald V. Raymond, a neurologist and clinical geneticist, and the Director of Neurogenetic Research at Johns Hopkins School of Medicine. Resp't's Ex.s A and B at 1. Dr. Raymond has an active clinical practice evaluating children with genetic disorders, including those with "progressive neurodegenerative disorders." Resp't's Ex. A at 4.

In his report, Dr. Raymond stated that C.D. developed respiratory distress after birth, and that there was "suspected sepsis." Id. at 1. C.D. had a lack of eye contact when she was seen in the emergency room at the age of two and a half months; it was noted that her eyes rolled back

and that she threw her head back. Id. at 3. C.D. had other abnormalities including hypertonia and a high arched palate. Id. Dr. Raymond described C.D.'s history of seizures, abnormal EEG findings, hypotonia, and severe global developmental delay. Id. at 2-4. Dr. Raymond noted that MRIs demonstrated "cerebellar atrophy and brainstem atrophy." Id. at 4.

Dr. Raymond explained that C.D. and her brother have a genetic disorder, which is probably autosomal recessive. Id. at 5. Dr. Raymond described the basis for his opinions and provides a brief overview of autosomal recessive genetic disorders and how they occur. Id. He recommended that C.D. have whole exome sequencing testing to determine a genetic diagnosis. Id. at 6. Dr. Raymond concluded that "to a reasonable degree of medical certainty" C.D. has a "genetic epileptic encephalopathy with dysmorphic features that [was] present at the time of birth." Id. Dr. Raymond rejected the notion that C.D.'s injuries were caused by or exacerbated by vaccinations. Id. at 6-7.

Dr. Raymond disagreed with the opinions set forth by Dr. Burris. First, Dr. Raymond stated that C.D. did not have acute encephalopathy because she did not have any "alteration of consciousness at the time of onset of seizures beyond the brief events themselves." Id. at 6. Second, Dr. Raymond opined that C.D. does not have various metabolic disorders, but instead, she has a genetic condition. Id. Third, C.D. has demonstrated many times that she responds appropriately to infections, and that she does not have an "abnormal immune response." Id. Fourth, there is no evidence or documentation to suggest that C.D. has had any adverse reactions to vaccines. Id. None of C.D.'s treating physicians has diagnosed her with a vaccine-related disorder and, in fact, her treating physicians have continued to administer vaccinations to C.D.. Id. Fifth, while Dr. Raymond agreed that there are neurological conditions caused by antibodies, such conditions would not explain C.D.'s congenital abnormalities and dysmorphic features. Id. Dr. Raymond opined that "there is substantial evidence that she has exactly the same [genetic] condition as her younger brother." Id.

V. Standard for Adjudication—Causation

The Vaccine Act established the Program to compensate vaccine-related injuries and deaths. § 300aa-10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Human Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. REP. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

To establish causation in fact, a petitioner must show by a preponderance of the evidence that but for the vaccination, the petitioner would not have been injured, and that the vaccination was a substantial factor in bringing about the injury. Cedillo v. Sec'y of Health & Human Servs., 617 F.3d 1328, 1338 (Fed. Cir. 2010); Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352 (Fed. Cir. 1999). Proof of actual causation must be supported by a sound and reliable "medical or scientific explanation that pertains specifically to the petitioner's case, although the explanation need only be 'legally probable, not medically or scientifically certain.'" Moberly v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1322 (Fed. Cir. 2010) (quoting Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994)); see also

Grant v. Sec’y of Health & Human Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) (medical theory must support actual cause). “[A] petitioner must demonstrate the reliability of any scientific or other expert evidence put forth to carry this burden Expert testimony, in particular, must have some objective scientific basis in order to be credited by the Special Master.” Jarvis v. Sec’y of Health & Human Servs., 99 Fed. Cl. 47, 54-55 (2011) (citing Moberly, 592 F.3d at 1322; Cedillo, 617 F.3d at 1339; Terran v. Sec’y of Health & Human Servs., 195 F.3d 1302, 1316 (Fed. Cir. 1999)).

Causation is determined on a case-by-case basis, with “no hard and fast per se scientific or medical rules.” Knudsen, 35 F.3d at 548. A petitioner may use circumstantial evidence to prove the case, and “close calls” regarding causation must be resolved in favor of the petitioner. Althen v. Sec’y of Health & Human Servs., 418 F.3d 1274, 1280 (Fed. Cir. 2005).

To receive compensation under the Program, petitioners must prove either: (1) that C.D. suffered a “Table Injury”—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that C.D. suffered an injury that was actually caused by the vaccine (or vaccines) she received. See §§ 300aa-13(a)(1)(A) and 11(c)(1); Capizzano v. Sec’y of Health & Human Servs., 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Petitioners must show that a vaccine was “not only a but-for cause of the injury but also a substantial factor in bringing about the injury.” Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because petitioners do not allege that C.D. suffered a Table injury, they must prove that a vaccine C.D. received caused her injury. To do so, they must establish, by preponderant evidence: (1) a medical theory causally connecting a vaccine and C.D.’s injury (“Althen Prong One”); (2) a logical sequence of cause and effect showing that a vaccine was the reason for her injury (“Althen Prong Two”); and (3) a showing of a proximate temporal relationship between a vaccinee and her injury (“Althen Prong Three”). Althen, 418 F.3d at 1278; § 300aa-13(a)(1) (requiring proof by a preponderance of the evidence).

a. Althen Prong One

Petitioners failed to submit any expert opinion addressing a theory of how the vaccinations at issue could cause the alleged injuries in a patient with C.D.’s established genetic mutations; that is, mutations in the *PIGT* gene. These genetic mutations have been identified in the medical literature as a cause of congenital disorders of glycosylation with the feature of intellectual disability. Pet’rs’ Ex. 43 at 6. The *PIGT* gene functions to make a specific protein that helps other proteins attach to a sugar-fat structure called a glycolipid (“GPI”). Id. at 4. GPI is found in all cells of the body and is “essential for life.” Id. at 4. A study of four other patients with the same genetic mutation revealed that this mutation was the cause of a “novel autosomal recessive intellectual disability syndrome.” Id. at 1. Patients with *PIGT* mutations have hypotonia, mild microcephaly, impaired motor and cognitive development, severe motor and intellectual disability, seizures, impaired vision, skeletal abnormalities, cardiac abnormalities, mild dysmorphic facial features, and global cerebral atrophy. Id. at 5-7. The authors of the study concluded that there is “strong evidence that a syndrome of intellectual disability, hypotonia, seizures, and skeletal and ophthalmologic findings seen in the patients of this study is caused by mutations in *PIGT*.” Id. at 7.

Petitioners' expert, Dr. Burris, opined that C.D. has a metabolic disorder that produced a chronic inflammatory state. Pet'rs' Ex. 18 at 1. Dr. Burris stated that the risk of developing an autoimmune disease increases when vaccines are given to a "patient with a chronic inflammatory condition caused by a metabolic disorder," like C.D.'s. Id. But Dr. Burris did not identify any metabolic disorder(s), nor did he set forth facts relevant to this case to support his opinion that a metabolic disorder was at play here. Likewise, Dr. Burris did not cite any facts or evidence from the medical records to show that C.D. has a chronic inflammatory condition.

Dr. Burris identified four theories of autoimmunity: "molecular mimicry, bystander activation, persistent viral infections, and fertile field." Id. Generally, Dr. Burris believes that there are two causal pathways of autoimmune epilepsy and encephalopathy – one is "that the body produces auto-antibodies for specific proteins necessary for proper neurological function." Id. at 2. The second is that an "increased number of antibodies can trigger seizures and encephalopathy." Id. Dr. Burris has not explained how or whether these theories might apply to patients with genetic mutations.

Even if the undersigned were to assume that Dr. Burris' theories of causation satisfied Althen's Prong One, Dr. Burris' reports are deficient as to Prongs Two and Three of Althen, as discussed below.

b. Althen Prong Two

Althen Prong Two requires petitioners to show by a preponderance of the evidence that the vaccinations caused C.D.'s injuries consistent with the medical theory or theories proposed by Dr. Burris. Because Dr. Burris did not address C.D.'s genetic disorder or point to factual support from C.D.'s medical records to support his opinions, petitioners have failed to meet their burden.

Dr. Burris opined that C.D. had a metabolic disorder which produced a chronic inflammatory state. Id. at 1. Dr. Burris stated that the risk of developing an autoimmune disease increases when vaccines are given to "a patient with a chronic inflammatory condition caused by a metabolic disorder," like C.D.'s. Id. Dr. Burris did not, however, refer to any facts from C.D.'s medical records or any other basis to support his opinion that C.D. had a metabolic disorder or a chronic inflammatory condition. Therefore, his opinions are without foundation. "An expert opinion is no better than the soundness of the reasons supporting it." Perreira v. Sec'y of Health and Human Servs., 33 F.3d 1375, 1377 fn. 6 (Fed. Cir. 1994).

Moreover, although Dr. Burris identified four mechanisms of autoimmunity, he failed to articulate how these theories apply to a patient with C.D.'s genetic mutations. Dr. Burris' report does not discuss any logical sequence of cause and effect showing that C.D.'s vaccinations were the reason for her injuries.

Whether or not C.D. is presumed to have a genetic mutation, Dr. Burris' opinions fail Althen Prong Two because they lack factual support or other foundation. Thus, petitioners have failed to provide preponderant evidence of actual causation under Althen Prong Two.

c. Althen Prong Three

Petitioners also failed to prove Althen Prong Three because Dr. Burris' reports are deficient on this issue. Dr. Burris does not address the issue of a proximate temporal relationship between the vaccinations and injury other than to make a conclusory statement that C.D. had acute encephalopathy five days after the administration of the vaccines. Dr. Burris fails to cite any medical facts or evidence to support his conclusion that there existed a temporal proximate relationship between C.D.'s vaccines and her alleged encephalopathy. As such, petitioners have failed to provide preponderant evidence of a proximal temporal relationship between the vaccines and any alleged injury.

VI. Conclusion

For the reasons discussed above, the undersigned finds that petitioners have not established entitlement to compensation and their petition must be dismissed. **Therefore, this case is dismissed for failure to make a prima facie case. The Clerk shall enter judgment accordingly.**¹⁹

IT IS SO ORDERED.

s/ Nora Beth Dorsey
Nora Beth Dorsey
Special Master

¹⁹ Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of notice renouncing the right to seek review.