In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Case No. 10-032V
Filed: August 10, 2017

STEVEN FORREST *and* NICOLE FORREST, *on behalf of E.M.F.*, Petitioners, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	PUBLISHED DECISION Special Master Thomas L. Gowen Denial of Entitlement; Hepatitis B ("Hep B") Vaccine; Inactivated Polio Vaccine ("IPV"); Diphtheria-Tetanus-Acellular Pertussis ("DTaP") Vaccine; Haemophilus Influenza ("Hib") Vaccine; Pneumococcal Conjugate Vaccine ("PCV"); Sudden Infant Death Syndrome ("SIDS").

Jessica W. Hayes, Murray Law Firm, New Orleans, LA, for petitioners.
Heather L. Pearlman & Ryan D. Pyles, United States Department of Justice, Washington, DC, for respondent.

DECISION ON ENTITLEMENT[1]

On January 14, 2010, Steven Forrest and Nicole Forrest ("petitioners"), as the representatives of the estate of their deceased minor child, E.M.F., filed a petition under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Vaccine Program").[2] Petitioners allege that E.M.F. died on January 14, 2008, as a result of receiving the Diphtheria-Tetanus-acellular-Pertussis ("DTaP"), inactivated polio ("IPV"), haemophilus B influenza ("Hib"), Pneumococcal Conjugate ("PCV"), and Rotavirus vaccinations on January 10, 2008. *See* Petition (ECF No. 1) at 1-2.

[1] Pursuant to the E-Government Act of 2002, *see* 44 U.S.C. § 3501 note (2012), **because this decision contains a reasoned explanation for the action in this case, I intend to post it on the website of the United States Court of Federal Claims.** The court's website is at http://www.uscfc.uscourts.gov. Before the decision is posted on the court's website, each party has 14 days to file a motion requesting 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). "An objecting party must provide the court with a proposed redacted version of the decision." *Id.* **If neither party files a motion for redaction within 14 days of the date this decision is filed, the decision will be posted on the court's website without any changes.**

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to 34 (2012). All citations in this decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

After carefully analyzing and weighing all of the evidence and testimony presented in this case in accordance with the applicable legal standards, I find that petitioners have not met their legal burden. Petitioners have failed to provide preponderant evidence that the vaccinations E.M.F. received on January 10, 2008, caused her death. Accordingly, petitioners are not entitled to compensation.

I. BACKGROUND

A. Procedural History

On January 14, 2010, petitioners filed a petition for compensation on behalf of their deceased minor daughter, E.M.F., pursuant to the Vaccine Act. The petition alleged that vaccinations administered on January 10, 2008 were the cause-in-fact of E.M.F.'s death on January 14, 2008. Petition at 1-2. Petitioners filed medical records on April 12, 2010. Exhibits 1-10 (ECF Nos. 8-9). On June 8, 2010, respondent filed a Rule 4(c) Report advising against compensation. Respondent's Report (ECF No. 10) at 8. Respondent argued that petitioners "failed to proffer a reliable medical or scientific opinion or theory supporting their petition, and have failed to make a prima facie case for entitlement to compensation." *Id.* Thereafter, petitioners filed three expert reports from Dr. John Shane (ECF Nos. 31, 40, 52). Respondent filed two reports from Dr. Sara Vargas and two reports from Dr. Hart Lidov (each expert's first report is at ECF No. 45; each expert's second report is at ECF No. 66). Both petitioners' affidavits were filed on October 24, 2013. Affidavits (ECF No. 60).

The case was reassigned to me on March 4, 2014. Petitioners requested and were granted several extensions of time to file an additional expert report. On September 29, 2014, petitioners submitted the expert report of pathologist Dr. Laurel Waters, along with her curriculum vitae and supporting medical literature. Exhibits 25-28 (ECF No. 80).

During a telephonic status conference on October 22, 2014, petitioners stated that they would not move forward with Dr. Shane as an expert. Order (ECF No. 84). Respondent indicated that removing Dr. Shane would narrow the issues going forward and limited the need for Dr. Lidov, who was primarily responding to Dr. Shane. *Id.* On December 3, 2014, petitioners filed Dr. Waters's second report. Exhibit 31 (ECF No. 87). On March 24, 2015, after settlement efforts proved unsuccessful, I set dates for an entitlement hearing. Hearing Order (ECF No. 95). On November 9, 2015, petitioners submitted additional medical literature in support of their position. Exhibits 40-42 (ECF No. 101). On November 30, 2015, petitioners filed supplemental affidavits from Steven Forrest, Elmer Darwin, and Dianne Darwin. Exhibits 43-45 (ECF Nos. 104-106). The parties filed separate prehearing submissions and a joint prehearing submission in preparation for the entitlement hearing. (ECF Nos. 98, 99, 102).

The entitlement hearing was held on December 7, 2015, in New Orleans, Louisiana. *See* Transcript ("Tr.") filed December 29, 2015 (ECF No. 109). Petitioners Mr. and Ms. Forrest appeared as fact witnesses. Dr. Waters also testified on behalf of petitioners. Dr. Vargas testified on behalf of respondent. On March 15, 2016, petitioners filed a post-hearing brief and an addendum from Dr. Waters intended to "supplement [her] initial report." Petitioners' Post-Hearing Brief (ECF No. 112). On April 29, 2016, respondent filed his post-hearing brief, in which he argued I should not consider Dr. Waters's addendum or alternatively, I should allow respondent and Dr. Vargas to "respond to any new arguments made therein." Respondent's Post-Hearing Brief (ECF No. 114). I granted respondent's request to respond to any new arguments raised by Dr.

Waters. Order filed June 23, 2016 (ECF No. 115). On August 23, 2016, respondent filed a third report from Dr. Vargas in response to Dr. Waters. Exhibit R (ECF No. 117). This matter is now ripe for adjudication.

B. Standards for Adjudication

The Vaccine Program was created to compensate injuries and deaths associated with vaccines administered within the United States. § 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 Vaccine 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).

Petitioners' burden of proof is by a preponderance of the evidence. § 300aa-13(a) (1). Under this standard, petitioners must demonstrate that it is more likely than not that a vaccine caused the injury. *Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). If petitioners satisfy this burden, they are entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the injury is "due to factors unrelated to the administration of the vaccine." § 300aa-13(a) (1) (B).

To receive compensation under the Vaccine Program, petitioners must prove either: (1) a "Table injury" – i.e., a specific injury within a specific period of time following the administration of a vaccine listed on the Vaccine Injury Table – or (2) that the vaccine caused the injury. *See* §§ 300aa-13(a) (1) (A), 300aa-11(c) (1); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). Petitioners must show that the 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 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).

In the present case, petitioners do not allege a Table injury, so they must argue that the vaccines E.M.F. received were the cause-in-fact of her death. To do so, they must establish by a preponderance of the evidence each of the three prongs in the test detailed in *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005). Namely, petitioners must establish: (1) a medical theory causally connecting the vaccines and E.M.F.'s injury; (2) a logical sequence of cause and effect showing that the vaccines was the reason for the injury; and (3) a showing of a proximate temporal relationship between the vaccines and the injury. *Althen*, 418 F.3d at 1278.

Further, the causation theory must relate to the injury alleged. Thus, petitioners must provide a reputable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioners cannot establish entitlement to compensation based solely on their assertions. Rather, their claim must be supported either by medical records or by the opinion of a medical doctor. § 300aa-13(a) (1).

In determining whether petitioners are entitled to compensation, the special master shall consider all material contained in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 300aa-13(b) (1) (A). The special master must weigh the submitted evidence and the testimony of the parties' experts and rule in petitioners' favor when the evidence weighs in their favor. *See Moberly*, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence"); *Althen*, 418 F.3d at 1280 (emphasizing that "close calls" are resolved in petitioners' favor).

Another important aspect of the causation-in-fact case law under the Vaccine Act concerns the factors that a special master may consider in evaluating the reliability of expert testimony and other scientific evidence relating to causation issues. In *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579 (1993), the Supreme Court listed certain factors that federal trial courts should utilize in evaluating proposed expert testimony concerning scientific issues. In *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999), the Federal Circuit ruled that it is appropriate for special masters to apply the *Daubert* factors while evaluating the reliability of causation-in-fact theories actually presented in Vaccine Program cases. In addition, 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 v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1362 (Fed. Cir. 2000)). 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 v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)).

C. Summary of Relevant Facts[3]

E.M.F.'s mother, Ms. Nicole Forrest, is a registered nurse and a clinical manager of the intensive care unit at West Jefferson Medical Center in Louisiana, a position she has held since 1996. Tr. 9. Ms. Forrest stated that while she was pregnant with E.M.F., she did not drink, smoke, or have any health problems, and that she received consistent prenatal care. Tr. 11.

E.M.F. was born on August 28, 2007, at forty-weeks gestation, weighing six pounds, eight ounces. Exhibit 1 at 17. E.M.F. had a physical exam on August 30, 2007, and was discharged from the hospital in good health on September 1, 2007. Joint Pre-Hearing Submission at 1.

E.M.F.'s pediatrician Dr. Vigour found her to be a well child at visits on September 4, 2007 and September 15, 2007. Joint Pre-Hearing Submission at 1. On September 29, 2007, Dr. Vigour saw E.M.F. for her one-month check-up. Exhibit 4 at 9. At that visit, Dr. Vigour noted that E.M.F. was spitting up her formula after feeding and diagnosed her as a well child with reflux. Exhibit 4 at 9. A few weeks later, on October 13, 2007, E.M.F. was brought to the pediatrician for a rash on her face and was diagnosed with baby acne. Exhibit 4 at 9.

[3] Although I considered the record as a whole to reach my decision, this section reviews only the most relevant facts. A more detailed recitation of the facts may be found in respondent's Rule 4(c) Report and the parties' pre-hearing submissions.

On October 29, 2007, E.M.F. was seen for her two-month check-up. Exhibit 4 at 8. Dr. Vigour noted that E.M.F. continued to have reflux, but otherwise her examination was normal. Exhibit 4 at 8. At that visit, E.M.F. received her first DTaP, pneumococcal, Hib, IPV, and rotavirus vaccinations. Exhibit 4 at 8. Ms. Forrest stated that about two hours after the vaccinations, E.M.F. began to develop a fever, which went up to 103° Fahrenheit. Tr. 16-17; *see also* Tr. 55 (testimony of Mr. Forrest, concurring that E.M.F. had a fever "that afternoon, by the time [Ms. Forrest] brought her home"). That night, E.M.F. was fussy, irritable, and vomiting when she ate. Tr. 16. Ms. Forrest also noticed some inflammation and redness at the site of administration on E.M.F.'s thigh. Tr. 16. Ms. Forrest called the pediatrician's office and was instructed to give Tylenol to treat the fever. Tr. 16. Ms. Forrest stated that the elevated temperature lasted for several days after receipt of the vaccines. Exhibit 12 at 1. The gastrointestinal difficulties with vomiting and constipation began after receipt of the vaccines, and continued intermittently until E.M.F.'s death on January 14, 2008. Ms. Forrest's Affidavit (ECF No. 60-1).

Mr. Forrest, who was a nursing student at the time, also observed these symptoms, and "could have just seen [them] as GI, - you know - other than the fever." Tr. 63. But he thought the most notable changes were "neuro." *Id.* Specifically, during her first two months of life, E.M.F. "seemed like a happy child" and "was always smiling." *Id.* But after receiving her first vaccinations, within two days (by October 31, 2007), E.M.F.'s behavior changed. *Id.* She "wasn't acting like herself." Tr. 63. She did not try to climb out of her car seat. Tr. 64-65. She also spent a lot of time on her back, and did not try to turn onto her front. Tr. 64-65.

On November 12, 2007, the pediatrician recorded a statement from Ms. Forrest that E.M.F. was constipated and spitting up. Exhibit 4 at 7. Neither Mr. Forrest nor Ms. Forrest remembered whether this was a telephone call or whether they brought E.M.F. to the pediatrician in person. Tr. 35, 80.

On November 19, 2007 at approximately 12:40 a.m., E.M.F. was brought to the emergency room at Slidell Memorial Hospital. Exhibit 6 at 2. The complaints included a one-day history of high fever and congestion. Joint Pre-Hearing Submission at 2. The emergency room records note a rectal temperature of 102.1° Fahrenheit. Exhibit 6 at 2. Bloodwork results included an automated differential of 39.1% neutrophils (lower than the normal range of 42.2 - 75.2%) and 52.5% lymphocytes (higher than the normal range of 21.0 - 51.0%). Exhibit 6 at 8. The absolute cell count for lymphocytes was 4.0 cells per cubic milliliter (higher than the normal range of 1.2- 3.4 K/ul). Exhibit 6 at 8.

E.M.F. was discharged from Slidell with instructions to be taken directly to West Jefferson Medical Center (where her mother, petitioner Ms. Forrest, worked as a nurse). Ex. 6 at 5. E.M.F. was admitted to West Jefferson with an assessment of "fever, vomiting, and rule out sepsis". Joint Pre-Hearing Submission at 2. Upon discharge on November 24, 2007, the diagnosis was resolved acute gastroenteritis, GE reflux, and a slight dilation of the lower pole collecting system of the right kidney. Joint Pre-Hearing Submission at 2.

Ms. Forrest stated that after discharge from the hospital on November 24, 2007, E.M.F. had some increased congestion and needed more frequent suctioning. Tr. 20. On November 26, 2007, Ms. Forrest took E.M.F. to a follow-up with Dr. Vigour. Tr. 20; Exhibit 4 at 7. Dr. Vigour noted that E.M.F. had a fever of between 100.0 - 100.7° Fahrenheit and had been vomiting. Exhibit 4 at 7. A chest x-ray performed that day showed a "patchy infiltrate noted in the left perihilar region and to

a lesser extent right lower lobe infrahilar region," leading to a radiologic impression of pneumonia. Exhibit 3 at 9. Nasal washing tested negative for influenza A, influenza B, para-influenza types 1-3, adenovirus, and respiratory syncytial virus. Exhibit 3 at 6-8.

A record from the pediatric clinic dated November 29, 2007, notes that petitioner had nasal congestion and a rash on the elbow, but no fever. Exhibit 4 at 6. The rash was treated with hydrocortisone cream and moisturizer. Exhibit 4 at 6.

On December 3, 2007, E.M.F. was seen by a pediatric gastroenterology and nutrition specialist, Dr. Khoshoo, at West Jefferson Medical Center. Exhibit 5 at 1. He recorded that E.M.F. presented with a history of post-prandial regurgitation without much irritability, but she did not have any recurrent respiratory illnesses. Exhibit 5 at 1. Dr. Khoshoo recommended discontinuing all medication and "treat[ing] her very conservatively with small volume thickened feeds, positioning, and avoidance of cigarette smoke." Exhibit 5 at 1. Ms. Forrest testified that over the next month, E.M.F. had continuing respiratory issues, but stopped having fevers and began to return to her normal self. Tr. 24-25.

There do not appear to be any medical records for E.M.F. from December 3, 2007, until January 10, 2008, when she had a four-month check-up with Dr. Vigour. Tr. 22; Exhibit 4 at 6. On January 10, E.M.F. had constipation, nasal congestion, and a rash on her elbows and forearms. She was diagnosed with constipation and dermatitis and prescribed 1% hydrocortisone cream. Exhibit 4 at 6. It is difficult to tell if the doctor did a complete physical examination at this appointment, as the various systems -genital, eyes, ENT, heart, lungs, and abdomen - are just checked without any notation. *Id.* E.M.F. received her second DTaP, pneumococcal, Hib, IPV, and rotavirus vaccinations that day. *Id.* Dr. Vigour's chart record provides that the appointment began at 10:36 a.m. and ended at 11:45 a.m. *Id.*

Ms. Forrest stated that E.M.F.'s reaction to the second vaccines was "similar... but more intense" than her reaction to the first vaccines. Tr. 26. "Immediately" after the vaccines, E.M.F. began crying and developed a fever; she did not sleep that afternoon or that night. Tr. 26-27. Her parents began alternating doses of Motrin and Tylenol, gave her a cool bath, and drove her around in the car, but she still stayed up the entire night. Tr. 67. Ms. Forrest's and Mr. Forrest's first affidavits state that on the evening of January 10, 2008, E.M.F. had an elevated temperature of 102° Fahrenheit. Affidavits (ECF Nos. 60-1, 60-2). At the hearing, Mr. Forrest stated that her fever was "between 103 and 105." Tr. 67, 85. Ms. Forrest said that the fevers fluctuated off and on and that E.M.F. was not eating. Tr. 26.

The next morning, on Friday, January 11, 2008, E.M.F. was "still very sick." Tr. 27. Ms. Forrest was scheduled to work a three-day shift at the hospital. Tr. 68. They decided together that Mr. Forrest would monitor E.M.F. and "if there were any other persisting problems, [he] would bring her to the ER." Tr. 68.

Over the weekend, Mr. Forrest stayed with E.M.F. Tr. 71. He took her rectal temperature every four hours. *Id.* On January 11 and 12, 2008, E.M.F. had a periodic fever that went down when she received Motrin or Tylenol, and then went back up. Tr. 68-70. She was vomiting, as she had been doing since late October 2007, but in greater volume. Tr. 69. Mr. Forrest assumed that she did not like the formula. Tr. 68. These symptoms "began tapering off" in the morning of Sunday, January 13, 2008. Tr. 70. That evening, Mr. Forrest told his wife over the phone that

E.M.F. seemed to be doing better and he would like to bring her to their new babysitter the next day, January 14, 2008, so that he could go to school. Tr. 72.

Mr. Forrest stated that on the morning of January 14, 2008, he was "a little bit rushed," but E.M.F. did not seem sick. Tr. 72-73. She did not have a fever. Tr. 73. Mr. Forrest brought her to their new babysitter for the first time. Exhibit 22 at 3; Tr. 28 ("it was her first day there"). Before that day, Mr. and Ms. Forrest had met the babysitter at her home twice and had given her written instructions in advance. Tr. 86-89. The instructions stated that E.M.F. was being medicated with "gas drops 0.3 as needed but not more than twice daily, teething tablets or Orajel as needed." Exhibit 7 at 16. They also directed the babysitter to "make sure [E.M.F.] eats her jar food twice daily and a 4 oz. bottle every 3 to 4 hours. She will let you know when she is hungry." *Id.*

Mr. Forrest stated that he dropped off E.M.F. at about 8:40 a.m., on his way to classes that began at 9:00 a.m. Tr. 72-73. However, according to the police incident report, the babysitter stated that Mr. Forrest dropped off E.M.F. a little later - "between 900 and 930 hrs." Exhibit 22. The babysitter stated that Mr. Forrest told her when and what to feed E.M.F. Exhibit 30 at 14:45-18:40.[4] She said that Mr. Forrest also mentioned that E.M.F. "got a little fussy with gas from her feedings." *Id.* at 33:05-33:24. According to the babysitter, Mr. Forrest left without filling out a contract or saying goodbye. *Id.* at 14:45-18:40. The babysitter did not notice that he left, because she was holding E.M.F. and cleaning up after the six other children in her care. *Id.* at 14:45-18:40. After Mr. Forrest left, the babysitter placed E.M.F. in a swing in the living room, fed her, burped her, then placed her back in the swing. *Id.* at 19:10-20:08. The babysitter noticed that E.M.F.'s eyes were red and that she looked tired and ready for a nap. *Id.* at 20:50-21:10. However, E.M.F. was not crying. *Id.* at 21:10 (the babysitter told another child that E.M.F. was "not crying, she's laughing. She's happy.") At about 11:00 a.m., the babysitter placed E.M.F. on her back in a portable crib, in a bedroom at the end of the hall. *Id.* at 21:10-22:20. She left the door "cracked open" and took another baby into the kitchen to be fed. *Id.* at 22:20-23:50. The babysitter brought the other baby back into the bedroom, laid him down for a nap, and then went into the kitchen again to feed the other children. *Id.* at 23:50- 24:50. At approximately 11:30 a.m., the babysitter went back into the bedroom and observed that E.M.F. was still on her back and appeared to be sleeping. *Id.* at 26:00-26:50. The babysitter took care of the other children and then ate lunch. *Id.* at 26:50-27:45.

When the babysitter went into the bedroom again, she found E.M.F. lying on her stomach and what appeared to be spit-up by her face, on the mattress. *Id.* at 27:45-28:30. The babysitter picked up E.M.F. and saw that she was not breathing and that blood appeared to be coming from her nose. *Id.* at 28:30-29:10. At 12:34 p.m., the babysitter called 911. Exhibit 9 at 1. While waiting for EMS to arrive, the babysitter performed CPR. Exhibit 30 at 29:20-31:20. When the paramedics arrived, they recorded that E.M.F. was a "baby girl [with] no signs of life and vomit (sic) and blood on [her] shirt." Exhibit 9 at 1. The paramedics ventilated her, resumed CPR, and administered epinephrine and atropine while transporting her to the hospital. Exhibit 9 at 1. Upon arrival to the hospital, E.M.F. had no blood pressure, no pulse, and a temperature of 88.8° Fahrenheit. Exhibit 7 at 3. E.M.F. was pronounced dead at 1:14 p.m. Exhibit 7 at 5. There does not appear to have been a police reconstruction of the events.

[4] The police detective interviewed the babysitter in the presence of her attorney on the day of E.M.F.'s death. No transcript was made of the interview but I have reviewed the videotape of that interview. The time entries noted represent minutes and seconds elapsed on the videotape and not time of day.

Dr. Peter R. Galvan performed an autopsy on January 15, 2008. Exhibit 11. He found no evidence of injury or trauma. *Id.* at 4. The heart was noted to have a 3-millimeter atrial septal defect. *Id.* at 4. The right lung weighed 100 grams and the left lung weighed 80 grams. *Id.* at 5. Pulmonary cut surfaces of the lungs demonstrated congestion. *Id.* at 5. The microscopic report noted "mild congestion; intra-alveolar microphages"[5] in the lungs. *Id.* at 7. It also noted "mild congestion" in the kidneys and "mild portal triaditis" in the liver. *Id.* at 7. The final diagnosis was atrial septal defect of heart and the cause of death was sudden infant death syndrome. *Id.* at 3.

D. Sudden Infant Death Syndrome[6]

Sudden infant death syndrome is defined as "the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history."[7] SIDS is temporally associated with sleep, "leading to the premise that it occurs during sleep or transitions between sleep and waking." Exhibit 17 at 306.

SIDS is a leading cause of infant mortality, with an overall incidence of 0.57 per 1,000 live births. Exhibit 17 at 306. "SIDS peaks at 2-4 months of age, and 90 percent of cases occur under 6 months of age, suggesting that development mechanisms play a major role in pathogenesis." Exhibit 17 at 306.

In 1994, Dr. Hannah C. Kinney, Dr. James J. Filiano, and their colleagues proposed a framework for SIDS called the Triple Risk Model, which they illustrated in the diagram below.[8]

[5] The medical examiner's report uses the term "microphages" but Dr. Vargas testified that that must be a typographical error as there are no microphages in the lung. The term should have been macrophages. Tr. 190.

[6] This summary introduces the basic concept of SIDS. It draws from one book chapter and one article filed by petitioners and Dr. Waters. For a longer explanation of SIDS and relevant medical literature, see my ruling on entitlement in *Boatmon v. Sec'y of Health & Human Servs.*, No. 13-611V (Fed. Cl. Spec. Mstr., July 10, 2017).

[7] Exhibit 17, R. Folkerth & H.C. Kinney, *Disorders of the Perinatal Period: Sudden Infant Death Syndrome*, in Greenfield's Neuropathology (8th ed. 2008) at 306.

[8] Exhibit 17, citing J.J. Filiano & H.C. Kinney, *A Perspective on Neuropathologic Findings in Victims of the Sudden Infant Death Syndrome*, 65 Biol. Neonate 194 (1994).

As shown on the diagram, SIDS is thought to occur when: (1) an infant in a critical development period; (2) possessing an underlying vulnerability; (3) encounters an exogenous stressor.

The first risk factor is the critical developmental period, thought to be the first year of life. The second risk factor is fulfilled by the infant possessing some inherent vulnerability. Many SIDS deaths have been characterized by: (i) maternal and pregnancy-related risk factors, such as pre-natal exposure to tobacco and alcohol; (ii) neonatal abnormalities in neurological or autonomic function; (iii) post-neonatal abnormalities in crying, cardiac, and ventilatory patterns, and state organization; and (iv) subtle CNS and/ or systemic abnormalities. Exhibit 17 at 309. The "most robust and specific" neurologic hypothesis, which has been "confirmed in several independent data sets and laboratories" is that up to 70% of SIDS infants studied have a brainstem abnormality in the medullary serotonergic (5-hydroxytryptamine [5-HT]) system.[9] This area of the brain "plays a key role in coordinating many respiratory, arousal, and autonomic functions and, when dysfunctional, might prevent normal protective responses to stressors that commonly occur during sleep." Exhibit 34 at 7. A defective 5-HT system may fail to respond to exogenous stressors, such as disruptions to the infant's breathing, and therefore causes SIDS. Ex. 17 at 310. Researchers are also investigating genetic mutations to the serotonin transporter, cardiac channelopathies, the autonomic nervous system, and other areas. Exhibit 34 at 8. These "require more study to determine their importance." Exhibit 34 at 8.

Third, the infant must encounter one or more "exogenous stressors" which, combined with the aforementioned two risk factors, lead to death. Exhibit 17 at 310. Kinney et al. have emphasized that SIDS is likely to be multifactorial, and often an infant whose death is classified as SIDS has at least two exogenous stressors. Perhaps the most widely recognized exogenous stressor is the prone sleep position. Exhibit 17 at 306. The mechanism is unknown, but "may involve rebreathing of expired gases with hypoxia, hypercapnia or asphyxia, upper airway obstruction, or impaired arousal thresholds in the prone position that hamper efforts to turn the head. Other possible explanations include compromised upper airway reflexes, hyperthermia due to heat trapping in the face-down position, or altered sensory/vestibular influences on blood pressure." Exhibit 17 at 306-7.

II. EXPERT OPINIONS AND CAUSATION ANALYSIS[10]

A. *Althen* Prong One: Petitioners' Medical Theory

Under the first *Althen* prong, petitioners must set forth a medical theory explaining how the vaccines could have caused E.M.F.'s death. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1375 (Fed. Cir. 2009); *Pafford*, 451 F.3d at 1355-56. Petitioners' theory of causation must be informed by a "sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548; *see also Veryzer v. Sec'y of Health & Human Servs.*, 98 Fed. Cl. 214, 223 (2011) (noting that under § 300aa- 13(b) (1) and Vaccine Rule 8(b) (1), special masters must consider only evidence that is both "relevant" and "reliable"). If petitioners rely upon a medical opinion to support their theory, the

[9] Exhibit 34, American Academy of Pediatrics – Task Force on Sudden Infant Death Syndrome, *SIDS and Other Sleep Related Infant Deaths: Expansion of Recommendations for a Safe Infant Sleeping Environment*, 128 Pediatrics 1030 (2011).

[10] As stated above, to reach my decision, I considered the record as a whole, including all aspects of the experts' opinions and their supporting medical literature. This section only reviews the materials that were most relevant to my decision.

basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. *See Broekelschen*, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); *Perreira v. Sec'y of Health & Human Servs.*, 33 F.3d 1375, 1377 n. 6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it") (citing *Fehrs v. United States*, 620 F.2d 255, 265 (Ct. Cl. 1980)).

1. Petitioners' Expert Dr. Laurel Waters

Dr. Laurel Waters is board-certified in pediatric pathology, anatomic pathology, clinical pathology, and nuclear medicine. Tr. 109. She graduated from the University of California at Berkeley in 1974 with one major in nutrition and food science, and a second major in biochemistry. Tr. 108-09. Dr. Waters earned her medical degree from the University of California at Davis in 1978. Tr. 109. She has completed a residency in anatomic and clinical pathology and a fellowship in pediatric pathology. Tr. 109-112; Exhibit 32 at 1. Dr. Waters has worked as a pathologist at several hospitals. Exhibit 32 at 1-3. She testified that she last performed autopsies "routinely" while working as an associate pathologist at Children's Hospital in Oakland, California from 1995-2000. Tr. 121. She has done pediatric autopsies "periodically, independently" for other pathologists in the area, most recently "a few years" ago. Tr. 121. At the entitlement hearing in December 2015, Dr. Waters indicated that she had been a clinical assistant professor in pathology at UC Davis since 2014. Tr. 114; *see also* Exhibit 32 at 1. Dr. Waters stated that the frequency of her teaching was "variable." Tr. 115.

In 2000, Dr. Waters founded PerinatalPath, where she currently works as a medical legal consultant and expert witness. Exhibit 32 at 1. Her practice has been primarily in the medical legal consulting arena since 2000. Exhibit 32 at 1; Tr. 116-18. I admitted Dr. Waters as an expert in pathology and pediatric pathology. Tr. 122-23.

Dr. Waters stated that she formed her opinion after reviewing E.M.F.'s medical records, autopsy reports and slides, medical literature, and various reports by other experts, and then interviewing Mr. and Ms. Forrest over the phone. Tr. 127. Dr. Waters opined that SIDS is a diagnosis of exclusion and that past studies have not established whether or not vaccines may cause certain deaths that are currently categorized as SIDS. She theorized that vaccines may cause these deaths by causing elevated temperatures that act as an exogenous stressor in the recognized "Triple Risk Model" for SIDS. She also opined, with more detail, that vaccines could prompt a "type IV anamnestic hypersensitivity response" leading to death. Tr. 128. She stated that when E.M.F. received her first set of vaccines, she had a fairly significant reaction. E.M.F. had a fever, indicating that the initial exposure to the antigens provided a release of lymphokines that would cause a reaction like that. Then, when the second set of vaccines were administered, "it could be an anamnestic response, which is much, much bigger than the initial response." Tr. 128.

Dr. Waters stated that SIDS "is a term used when an infant who was previously well unexpectedly dies." Exhibit 25 at 13. These deaths occur "primarily in early infancy with 95% occurring between one and nine months of age," most frequently between "two to four months." Exhibit 25 at 13. She stated that SIDS is a "diagnosis of exclusion." Exhibit 25 at 15. An autopsy and death scene investigation are necessary to rule out possible causes of death, including "homicide, asphyxiation, congenital heart disease, other serious structural abnormalities, infectious causes, and inborn errors of metabolism." Exhibit 25 at 15. Once these are ruled out, the cause of death is recorded as SIDS. Exhibit 25 at 15. However, she said the term SIDS is not universally

accepted because it implies a syndrome that is well understood and has clear diagnostic criteria. Dr. Waters and others would prefer to say sudden unexpected death of an infant ("SUDI") or sudden unexpected infant death ("SUID"). Exhibit 31 at 4, Tr. 123.

Despite arguing that SIDS is not understood and merely a "diagnosis of exclusion," Dr. Waters acknowledged "agreement on a basic model," – the Triple Risk Model – in which "a vulnerable infant at a critical developmental period suffers from exogenous stressors." Exhibit 25 at 13. Dr. Waters stated that all three aspects of this model are still being studied. Exhibit 25 at 13. However, prone sleeping has been identified as an external stressor. The "Back-to-Sleep" campaign in the 1990s, which encouraged placing infants to sleep on their backs, was followed by a significant decrease in the SIDS incidence rate. Exhibit 25 at 13. Dr. Waters also stated that SIDS may be caused by hyperthermia, caused by high external temperatures, overdressing, or "recent febrile illness." Exhibit 25 at 13. She stated that fevers are commonly seen after immunizations. Exhibit 25 at 13. She also stated that SIDS infants' lungs often – but not always – have congestion, edema, hemorrhages, macrophages, and inflammation. Exhibit 25 at 13-14.

Dr. Waters opined that a small subset of the deaths attributed to "SIDS" are caused by vaccinations. Tr. 162. She stated this has not been proven yet, because past studies were "poorly designed." Exhibit 25 at 14. She cited a 2003 Institute of Medicine report[11] that reviewed a number of controlled studies on vaccination and SIDS from "a position of neutrality." Exhibit 25 at 14. This report "found the evidence inadequate to accept or reject a causal relationship between SIDS and DTaP… HiB, HepB, OPV, and IPV." Exhibit 25 at 14. Dr. Waters stressed that the IOM report was inconclusive and that it recommended further study.

Dr. Waters stated that past studies were poorly designed because they compared children that had been vaccinated with children who had never been vaccinated. Exhibit 25 at 17-18. She opined that "the never-vaccinated group made a flawed comparison in that it was made up of individuals from socioeconomic categories already identified as being at increased risk for SIDS, specifically those whose parents were poorer and having children at an earlier age." Exhibit 25 at 17; *see also* Tr. 128-29. Dr. Waters further opined that future studies should control for these socioeconomic and age factors, and focus on the temporal association between vaccination and death. Exhibit 25 at 17. Dr. Waters stated that one study by Walker[12] was designed well and supported her theories. Exhibit 25 at 17. This 1987 study was of immunized and non-immunized infants who died in infancy. It included all apparently-healthy infants of birthweight greater than 2500 grams born between 1972-83 in hospitals affiliated with the Group Health Cooperative of Puget Sound (GHC), who subsequently used GHC medical services and had retrievable records. The Walker study's key finding was that there was a 7.3-fold elevation in the risk for SIDS in the first four days following DTP immunization within the first year of life. Walker also reported that the group of children who were old enough to receive DTP but never received it had a mortality rate "more than six times higher than those who had been immunized." Exhibit 19 at 945.

[11] Exhibit 28, Institute of Medicine, *Immunization Safety Review: Vaccinations and Sudden Unexpected Death in Infancy* (K. Stratton et al., eds. 2003).

[12] Exhibit 19, A. Walker et al., *Diphtheria-Tetanus-Pertussis Immunization and Sudden Infant Death Syndrome*, 77 American Journal of Public Health 945 (1987).

Dr. Waters also filed a 2014 study by Maturri which found that 11.8% of deaths categorized as SIDS occurred within seven days of a hexavalent vaccination.[13] Maturri stated that the study "does not prove a causal relationship between the hexavalent vaccination and SIDS," but hypothesized that "vaccine components could have a lethal outcome in vulnerable babies." Exhibit 39 at 1. Maturri recommended that deaths shortly following hexavalent vaccinations be "appropriately investigated and submitted to a post-mortem examination, particularly of the autonomic nervous system to objectively evaluate the possible causative role of the vaccine in SIDS." Exhibit 39 at 1. Dr. Waters also cited several studies of simultaneous sudden infant deaths in twins shortly after vaccination, to "lend weight to an association" between vaccinations and death. Exhibit 25 at 6.[14] Based on these studies, Dr. Waters opined that certain SIDS deaths are caused by vaccines. Exhibit 25 at 4. She stated that "there are no specific markers we know at this time." Exhibit 25 at 4. However, she believes that "vaccinations are eventually going to be found – be proven to be" the cause of a small subset of SIDS cases. Tr. 162.

Dr. Waters suggested that vaccinations can cause elevated temperatures, which act as an exogenous stressor in the Triple Risk Model of SIDS. Exhibit 25 at 4; Tr. 131-32. However, she did not discuss this theory in detail. Her primary theory is that vaccinations cause an anamnestic type IV delayed hypersensitivity response that leads to death.

Dr. Waters filed an excerpt from a book by Roger Byard that discusses whether various antigens cause increased immunoreactivity.[15] It provides: "An alternative approach suggests that SIDS infants are in fact immunologically 'too' competent, and that allergic responses to a wide variety of materials are responsible for their unexpected death. Many substances have been proposed, including a variety of intrauterine pathogens, fungal organisms, house dust mite (*Dermatophagoides pteronyssinus*), and cow's milk protein." Exhibit 33 at 2. Byard writes that studies have not consistently demonstrated elevated levels of these materials in SIDS infants, and have "cast doubt" on the theory of immunoreactivity. Exhibit 33 at 2. Byard does not discuss vaccines as the source of antigens that possibly cause this fatal immune response.[16]

Other than the Byard excerpt, Dr. Waters did not file any other medical literature about how antigens in general, or vaccines in particular, may cause a heightened immune system response leading to death. Tr. 153. She primarily presented this theory in her own reports and testimony. Dr. Waters stated that the first administration of an antigen can trigger an immune response, and that this response may not be very severe. Tr. 130. However, "when the antigen is seen again, there's a more rapid response that can be of a much greater magnitude than the initial response because you already have primed cells." Tr. 128. This subsequent response is "anamnestic." Tr. 129, Exhibit 31 at 3. She testified that this is a cell-mediated response, mostly involving T cells and

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[13] Exhibit 42, L. Maturri et al., *Sudden Infant Deaths Following Hexavalent Vaccination: A Neuropathologic Study*, 21 Current Medicinal Chemistry 941 (2014).

[14] Citing Exhibit. 36, Y. Balci et al., *Simultaneous Sudden Infant Death Syndrome*, 14 Journal of Forensic Legal Medicine 87 (2007); Exhibit 37, S.C. Roberts, *Vaccination and Cot Deaths in Perspective*, 62 Archives of Disease in Children 754 (1987).

[15] Exhibit 33, Roger W. Byard, *Sudden Death in the Young* (3rd ed. 2010) at 592-593.

[16] These pages from this book discussing SIDS can fairly be categorized as a general description of various theories to explain SIDS causation including infection, immunodeficiency, increased immune reactivity, genetic mutations and toxic exposures. As discussed below, respondent filed another page from the book with the specific heading, "Immunization." *See* below and Exhibit P.

some natural killer cells are involved, and lymphocytes scattered throughout the body as E.M.F. had in the lungs in particular. Tr. 129. In referring to lymphocytes, Dr. Waters may have been addressing the autopsy finding of lymphoid hyperplasia[17] in the lungs.

Dr. Waters stated that antigens can trigger several different responses within different time frames. A "humoral response" producing fever and other "sickness behaviors" may occur within twenty-four hours. Tr. 154. She also stated that "a few days" following exposure, she might expect to see a "reaction at the site." Tr. 158. She did not say whether she expected that this would be a humoral or cellular response, but she compared it to the response in a tuberculosis vaccination. She did not recall if there were any indications of a site reaction in E.M.F.'s case. Tr. 157-58.

Dr. Waters stated that an antigen can trigger increased activity of lymphocytes. Tr. 147. She opined that E.M.F. was exposed to these antigens at the two-month vaccinations and was primed. Then, upon receiving the four-month vaccinations containing these same antigens, E.M.F. experienced an anamnestic response with cellular immunity. Tr. 147. The lymphocytes produce lymphokines, which are chemicals that are reactive and can cause cellular problems throughout the body, according to Dr. Waters. Tr. 147-48.

Dr. Waters stated that one possible immune response is "a type IV hypersensitivity response." During this response, "various lymphocytes including CD4 and CD8 T cells, and NK cells," secrete specific cytokines, called lymphokines. Petitioners' Post-Hearing Brief, Addendum A at 4. The lymphokines "attract more immunoactive cells and cause cellular damage." *Id.* at 4. Dr. Waters stated that lymphokines can cause apoptosis (cell death) "from various different mechanisms. Punching holes in the membrane, in the outer cell membrane and so forth." Tr. 150.

She stated that when an antigen – specifically a vaccine – is injected into the muscle, it is "picked up by the bloodstream and goes throughout the body." Tr. 150. Therefore, it would cause damage on the cellular level, "systemic[ally]" throughout the body. Tr. 150-51. Dr. Waters stated that the apoptosis would not be as "massiv[e]" as the damage caused by chemotherapy or certain other treatments. Tr. 156. She said that the apoptosis alone would not cause a sudden death, but that "a combination of apoptosis and other things" could cause death. *Id.* The apoptosis would be "one of many mechanisms." *Id.* Dr. Waters thought "the actual final mechanism… could be an arrhythmia or there could be a neurological kind of pathway to the death." Tr. 172. Dr. Waters stated that this apoptosis would not necessarily be found in the cell samples taken at autopsy. Tr. 150-51. Therefore, there would be no direct evidence of the type IV hypersensitivity response.

Dr. Waters stated that "with the hypersensitivity reaction, you're going to get a mixture of findings. And you're not necessarily going to get…lymphoid hyperplasia" (discussed in more depth below, in the section on Dr. Vargas's testimony). Tr. 134. However, the hypersensitivity and lymphoid hyperplasia would be "consistent," because they are both immune responses. Tr. 131, 134, 172. Dr. Waters opined, without reference to any literature, that the lymphoid hyperplasia seen on autopsy in E.M.F. helps support the diagnosis or mechanism that the death was actually caused by hypersensitivity. Tr. 131.

[17] Lymphoid hyperplasia is defined as a form of chronic lymphadenitis occurring in an immunologic response, often induced by drugs, and characterized by transformation of T cells to lymphoblasts, endothelial cell hypertrophy and the presence of a mixed leukocyte infiltrate. *Dorland's Illustrated Medical Dictionary at 894* (32d ed. 2012) ("*Dorland's*"). Lymphoblasts are activated lymphocytes that have been transformed in response to antigenic stimulation. *Dorland's* at 1084.

Dr. Waters stated that the type IV hypersensitivity response generally occurs within three days of exposure to the antigen. Tr. 127. However, a "delayed" type IV hypersensitivity response can "take a longer time than the normal three days that's considered an association." Tr. 127. She later stated that the type IV hypersensitivity response "starts at about 48 to 72 to 96 hours and then continues for a while after that." Tr. 129-30.

Dr. Waters discussed a possible element of this immune response, lymphoid hyperplasia (the growth and aggregation of many lymphocytes). Tr. 131. This is a non-specific marker. She stated that "some degree of lymphoid hyperplasia is accepted in SIDS, but it could be that when there's enough lymphoid hyperplasia, it's actually a subset that is caused by an immunological response." Tr. 177; *see also* Exhibit 31 at 3. Dr. Waters acknowledged that lymphoid hyperplasia could be caused by a viral infection, such as pneumonia. Tr. 139. She opined that lymphoid hyperplasia could also be caused by the administration of vaccines. Exhibit 31 at 3. She testified that the lymphoid hyperplasia seen in this case was not very significant, that the finding would be acceptable within a SIDS diagnosis, and that it was not a sufficiently significant finding to be diagnostic as a cause of death. Tr. 177-78.

On direct examination, Dr. Waters's explanation of the type IV hypersensitivity reaction and its possible relationship to vaccines was rather vague and general. On cross-examination, she was able to explain it somewhat more clearly. Dr. Waters said that her theory was that E.M.F. had received the two-month vaccines as scheduled, which primed her system. When E.M.F. received the second dose of these antigens in the four-month vaccines, E.M.F. had an anamnestic response. Tr. 147. Dr. Waters explained that E.M.F.'s response to the second set of vaccines seemed stronger than the response to the first and that an "anamnestic" response "is much, much bigger than the initial response." Tr. 128. On redirect, Dr. Waters said that she was persuaded of the likelihood of an anamnestic response to the second vaccines because the child had an immunological reaction to the first ones. Tr. 174. Dr. Waters continued that the body produced lymphocytes in response to the vaccines, which in turn produced lymphokines. Tr. 174. She listed Interleukin 2 and Tumor Necrosis Factor as lymphokines that have been identified, and explained that she used the term lymphokines to mean a subset of cytokines that are produced by the immune system while cytokines can be produced by multiple body systems. Tr. 148-50. She said that there are many lymphokines and they can wreak havoc in the body. Tr. 148. She suggested that the lymphokines could cause excessive apoptosis and that death could be caused by a combination of apoptosis and "other things." Tr. 154. Dr. Waters did not see evidence of apoptosis on the autopsy slides. She indicated that apoptosis could occur and not be visible on the slides. Tr. 151. Ultimately, on questioning by respondent's counsel and the Court, Dr. Waters testified that there are a number of theories about how SIDS occurs, but that it was not clear how hypersensitivity would cause death. Tr. 160.

Dr. Waters did not file any medical literature in support of the notion that a vaccine-caused type IV hypersensitivity reaction could cause a death classified as SIDS, or initially discuss how long it would take lymphoid hyperplasia to form. In her post-hearing brief, she sought to rebut Dr. Vargas's contention that hypersensitivity type IV is not a recognized disease, by referencing the eighth edition of a well-known pathology textbook.[18] *See* Petitioners' Post-Hearing Brief, Addendum A at 2. Petitioners did not file this reference. The ninth and current edition of that textbook does describe four categories of hypersensitivity reaction and does describe a type IV

[18] Citing V. Kumar et al., *Robbins and Cotran: Pathologic Basis of Disease*) at 205-08 (8th ed. 2010).

delayed reaction as being one that does not occur immediately, such as a Type I or anaphylactic reaction would, and that it is a T-cell-mediated response.[19] The textbook provides that subsequent exposure to an antigen results in the secretion of cytokines. Kumar et al. at 208-11. IFN-γ activates macrophages to produce substances that cause tissue damage and promote fibrosis. IFN-γ also produces IL-17 and other cytokines that recruit leukocytes thus promoting inflammation. The textbook also provides that the classical T-cell-mediated inflammatory reaction is a delayed type hypersensitivity and that CD8+ cytotoxic T lymphocytes specific for an antigen recognize cells expressing the target antigen and kill these cells. *Id.* at 211.

Dr. Waters indicated that the lymphoid hyperplasia could be evidence of a hypersensitivity reaction, but she said that signs and symptoms of a delayed hypersensitivity response are not well explained. Tr. 158. She also acknowledged that the autopsy finding of lymphoid hyperplasia could have been residual of the pneumonia E.M.F. had at the end of November, although she thought that it probably would have cleared by the date of death based on her experience as to how long it takes to recover from pneumonia. Tr. 139. She criticized Dr. Vargas's explanation of this issue (set forth below) but she did not recall if there were "mature" germinal centers seen on the slides, as Dr. Vargas testified, and she did not research whether "mature" germinal centers could form in less than the eight-to-twenty-one days suggested by Dr. Vargas and the exhibit she produced to support that opinion. Tr. 142.

Dr. Waters did not discuss the symptomatology associated with lymphoid hyperplasia, though she did say that most pathologists do not consider lymphoid hyperplasia to be a cause of death. Petitioners' Post-Hearing Brief, Addendum A at 1. She did not consider it a cause of death in this case. Tr. 178. She argued that epidemiological studies of SIDS deaths were not properly controlled and compared vaccinated to not-vaccinated populations which likely skewed the results, a deficiency which has been noted in the literature regarding some of these studies.

2. Respondent's Expert Dr. Sara Vargas

Dr. Sara Vargas is board-certified in anatomic and clinical pathology and pediatric pathology. Tr. 181. She received her bachelor's degree from Harvard University in 1988, and her medical degree from the University of Vermont in 1994. Tr. 180; Exhibit B at 2. She then completed a residency in anatomic and clinical pathology at Brigham and Women's Hospital in Boston, Massachusetts. Tr. 180. Subsequently, she completed a fellowship in pediatric pathology at the Children's Hospital in Boston, Massachusetts. Tr. 180. Dr. Vargas now works as a staff pathologist at the Boston Children's Hospital and Brigham and Women's Hospital. Exhibit B at 2. She does a combination of surgical pathology and autopsy pathology at Brigham and Women's. She is also an associate professor at Harvard University. *Id.* at 2. Dr. Vargas did a fellowship specifically in lung pathology at Brigham and Women's Hospital. Tr. 181. She also works with the SIDS research team at the hospital, which is led by Dr. Hannah Kinney, who is a neuropathologist and who relies on lung pathologists such as Dr. Vargas to interpret lung pathology slides. Tr. 182. Dr. Vargas testified that she reviews patient pathology slides and reviews cases one-on-one with pediatric pathology fellows. Tr. 182-83. She performs one or two autopsies each week, primarily on children, and also consults on autopsies that involve difficult lung pathology. Tr. 181. I admitted Dr. Vargas as an expert in clinical, anatomic, and pediatric pathology. Tr. 186.

[19] V. Kumar et al., *Robbins and Cotran: Pathologic Basis of Disease* (9th ed. 2014).

Dr. Vargas endorsed the Triple Risk Model of SIDS and agreed that the underlying mechanism is not fully understood. Exhibit A at 5-6[20]; Tr. 187. She stated that some deaths categorized as SIDS actually could be due to lung disease, but "[t]here are no specific guidelines for precisely how much or which type of lung inflammation precludes a diagnosis of SIDS." Exhibit A at 6.[21] She noted that Byard discusses "focal aggregates of submucosal inflammatory cells . . . within the upper airways, extending on occasion to the alveolar septa" and "focal aggregates of incidental, interstitial chronic inflammatory cells."[22] In what seemed to be a semantic debate with little relevance to the question of whether the vaccines could cause a hypersensitivity reaction resulting in death, Dr. Vargas and Dr. Waters debated whether the death should properly be called SIDS as the medical examiner did, or whether it is more properly called SUDI (Sudden Unexplained Death in Infancy), which would allow for the recognition of the lymphoid hyperplasia present in this case. In her final rebuttal report, Dr. Vargas emphasized that she did not think that the degree of lymphoid hyperplasia in the lungs of E.M.F. was adequate to cause death. Exhibit R at 1. She reiterated her opinion: "Since it is not clear that the degree of lung disease was adequately severe to cause death, the cause of death is best left undetermined, in other words, sudden unexpected death in infancy in the presence of pulmonary interstitial lymphoid hyperplasia. SIDS is the main consideration in the differential diagnosis, but its definitive diagnosis is precluded because of semantic variation over whether the degree of pulmonary lymphoid hyperplasia seen herein is permitted within the case definition of SIDS." *Id.* at 1-2. In short, Dr. Waters and Dr. Vargas agreed that it was unlikely that the lymphoid hyperplasia seen on the autopsy slides of this child was sufficient, by itself, to cause death.

Dr. Vargas brought to bear her considerable background and qualification in the field of pediatric lung pathology in explaining the findings on the autopsy slides in this case. She said that on microscopic examination, there was an accumulation of lymphocytes in the lung that was more than normal and that had been present for some time. She explained when lymphocytes aggregate, they eventually start to form almost a miniature lymph node. They start to form germinal centers and follicles when there is persistent lymphoid inflammation. Tr. 188. She further explained that a germinal center is a cluster of lymphocytes with a specific architecture. The lymphocytes make follicles and the germinal center is the inside part of that follicle. Tr. 189. She said that in this case the lymphoid hyperplasia that was present was potentially the cause of the lungs weighing twice the normal weight, and that the weight of lungs in the presence of lymphoid hyperplasia could help to determine that the lymphoid hyperplasia was substantial. Tr. 189.

Dr. Vargas testified that the fact that E.M.F. had pneumonia diagnosed by x-ray on November 26, 2007, fits very well with the time course for what was seen at autopsy, which is a well-developed lymphoid infiltrate with follicle formation six weeks after the chest x-ray. Tr. 190-91. Critical to the issue in this case, Dr. Vargas testified that lymphoid hyperplasia develops slowly and that what she saw on the slides was a substantial infiltrate with follicle formation, so that she was very confident that the lymphoid hyperplasia was older than four days. Tr. 197. She said that the lymphoid follicle distribution was "bronchocentric," or surrounding the airways in the lungs, which would be consistent with a viral pneumonia. Tr. 199.

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[20] Citing Exhibit G, H.F. Krous et al., *SIDS and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach*, 114 Pediatrics 236 (2004); Exhibit C, Roger W. Byard, *Sudden Death in the Young* (3rd ed. 2010) at 576-77.

[21] A specific diagnosis - for example, a lung infection or pneumonia - as a cause of death would take the death out of the category of SIDS, which is limited to those deaths that are unexplained after complete autopsy and investigation.

[22] Exhibit C at 576-77.

Dr. Vargas disagreed with Dr. Waters's opinion that vaccines may cause deaths categorized as SIDS. Dr. Vargas opined that there was no literature showing that vaccinations are associated with a higher incidence of SIDS or suggesting that vaccinations cause SIDS. Tr. 200. Dr. Vargas stated that the IOM found that "the evidence favors rejection of a causal relationship between some vaccines and SIDS; and that the evidence is inadequate to accept or reject a causal relationship between other vaccines and SIDS." Exhibit O at 3.[23] Therefore, she said that there is no evidence at this time, even if the IOM did recommend future research on the issue.

Dr. Vargas did not know whether any study had examined the relationship between SIDS and the specific combination of DTaP, pneumococcal, haemophilus b, influenza, inactivated polio virus, and rotavirus vaccines. Tr. 232. She said she did not always pay attention to which vaccines were included in each study she reviewed. Tr. 232.

Dr. Vargas discussed the 1987 article by Walker filed by petitioners.[24] She opined that Walker's most significant finding was that the SIDS mortality rate was lower in vaccinated children than in non-vaccinated children. Exhibit O at 3.[25] Dr. Vargas believed the Walker article "supports a lack of association between vaccination and sudden death." Tr. 238. But she also argued that it was "probably not well-controlled" and did not rule out the other possible SIDS risk factors in the non-vaccinated group. Tr. 238.

Dr. Vargas also questioned Walker's finding that the SIDS mortality rate was higher in the first three days following vaccination as compared to later time periods. Tr. 233. She opined that the Walker study – published in 1987 – was not properly controlled and was not guided by the stringent epidemiological methods that have subsequently been adopted. Tr. 233. She also opined that Walker said the study could be "reproduced easily by any number of people," but in the past thirty years, no one has done so. Tr. 234.

Dr. Vargas did not address Maturri's finding of a higher incidence of deaths in the seven days following a hexavalent vaccination, the authors' hypothesis that "vaccine components could have a lethal outcome in vulnerable babies," or their recommendations for further studies.[26] Exhibit O at 4. Dr. Vargas opined only that Maturri had no proof of a causal relationship between vaccinations and SIDS. Exhibit O at 4.

Dr. Vargas alleged that Dr. Waters "misrepresent[ed]" Byard's discussion of vaccinations and sudden death in the young. Exhibit O at 2. Specifically, Dr. Waters filed one paragraph from one page of the book, which summarized several studies about vaccination and SIDS. Exhibit 33 at 2. Dr. Waters then said these studies were poorly designed and not appropriate to determine if a subset of SIDS cases are related to vaccinations. Exhibit 31 at 4. In response, Dr. Vargas filed two

[23] Citing Exhibit 28 at 1; *see also* Exhibit E, Institute of Medicine, *Adverse Effects of Vaccines: Evidence and Causality* 581-82. (K. Stratton et al., eds. 2003)

[24] Exhibit 19, A. Walker et al., *Diphtheria-Tetanus-Pertussis Immunization and Sudden Infant Death Syndrome*, 77 American Journal of Public Health 945 (1987).

[25] Dr. Vargas's representation of the article was in contrast to the author's statement that the "major finding" from the study "is an apparent 7.3 fold elevation in the risk for SIDS in the first four days following immunization with DTP in the first year of life." *Id.* at 947.

[26] Exhibit 42, L. Maturri et al., *Sudden Infant Deaths Following Hexavalent Vaccination: A Neuropathologic Study*, 21 Current Medicinal Chemistry 941 (2014).

full pages from the Byard text, which included the entire discussion of SIDS. Exhibit P at 2.[27] On these pages, Byard devotes another four paragraphs to the topic of vaccinations and sudden death and concludes: "Thus, it has been demonstrated convincingly in many studies from a number of countries that immunization is not causally related to SIDS." Exhibit P at 2. Dr. Vargas that Dr. Waters's other filings did not support a causal relationship between vaccinations and SIDS. Exhibit O at 3.[28] As noted above, the Byard article was a brief summary of multiple proffered theories of SIDS causation and not a scientific study.

Dr. Vargas stated that the normal immune response to vaccinations can include fever. Tr. 203. She did not address Dr. Waters's first theory that vaccine-induced fever may act as an exogenous stressor under the Triple Risk Model.

Dr. Vargas rejected Dr. Waters's second theory, that vaccines could cause a type IV hypersensitivity response, as being vague and overly simplistic. Dr. Vargas stated that two researchers in the 1960s outlined "four types of hypersensitivity based on the four types of normal immunity in the body." Tr. 201. However, subsequent research has demonstrated that each disease or disorder is complex and involves several types of hypersensitivity. Tr. 201. Therefore, it is not accurate to describe anything as purely a "type IV response." Tr. 201; *see also* Exhibit R at 2.

Dr. Vargas agreed with Dr. Waters's basic explanation of lymphoid hyperplasia, but she went into more detail about the timing. Dr. Vargas opined that it takes longer than a few days to form lymphoid hyperplasia. Exhibit A at 6. Dr. Vargas stated: "While it is true that lymphocytes may migrate to sites of injury/ inflammation within a time period as short as 3-4 days, the amount of time that it would take them to become organized into well-developed lymphoid tissue with mature follicles demonstrating germinal centers, as were present in this case, is significantly longer." Exhibit O at 2.

Dr. Vargas admitted that it was difficult to find data on germinal center formation in humans. Tr. 199. She filed an article by Victora, which discussed germinal center formation in mice.[29] Victora wrote that the germinal center reaction "can vary greatly, depending on the experimental system, from a few weeks to several months or longer." Exhibit R at 2. However, in mouse models, the typical kinetics are: an antigen encounter (days 0-1); antigen-activated B and T cells meeting at the border of the T cell zone and B cell follicle (days 2-4); early germinal center: expansion (days 5-7); mature germinal center: selection (days 8-21); and dissolution (days >21). Exhibit R at 1-2. Dr. Vargas referenced the illustration below to demonstrate that mature germinal centers would take eight to twenty-one days to form and thus would have been forming in response to an antigen prior to the receipt of the second set of vaccines that were administered four days prior to death:

[27] Citing Exhibit 33, Roger W. Byard, *Sudden Death in the Young* (3rd ed. 2010) at 590.

[28] Exhibit 34, American Academy of Pediatrics – Task Force on Sudden Infant Death Syndrome, *SIDS and Other Sleep Related Infant Deaths: Expansion of Recommendations for a Safe Infant Sleeping Environment,* 128 Pediatrics 1030 (2011).

[29] Exhibit R, G.D. Victora, *SnapShot: The Germinal Center Reaction*, 159 Cell 700 (2014).

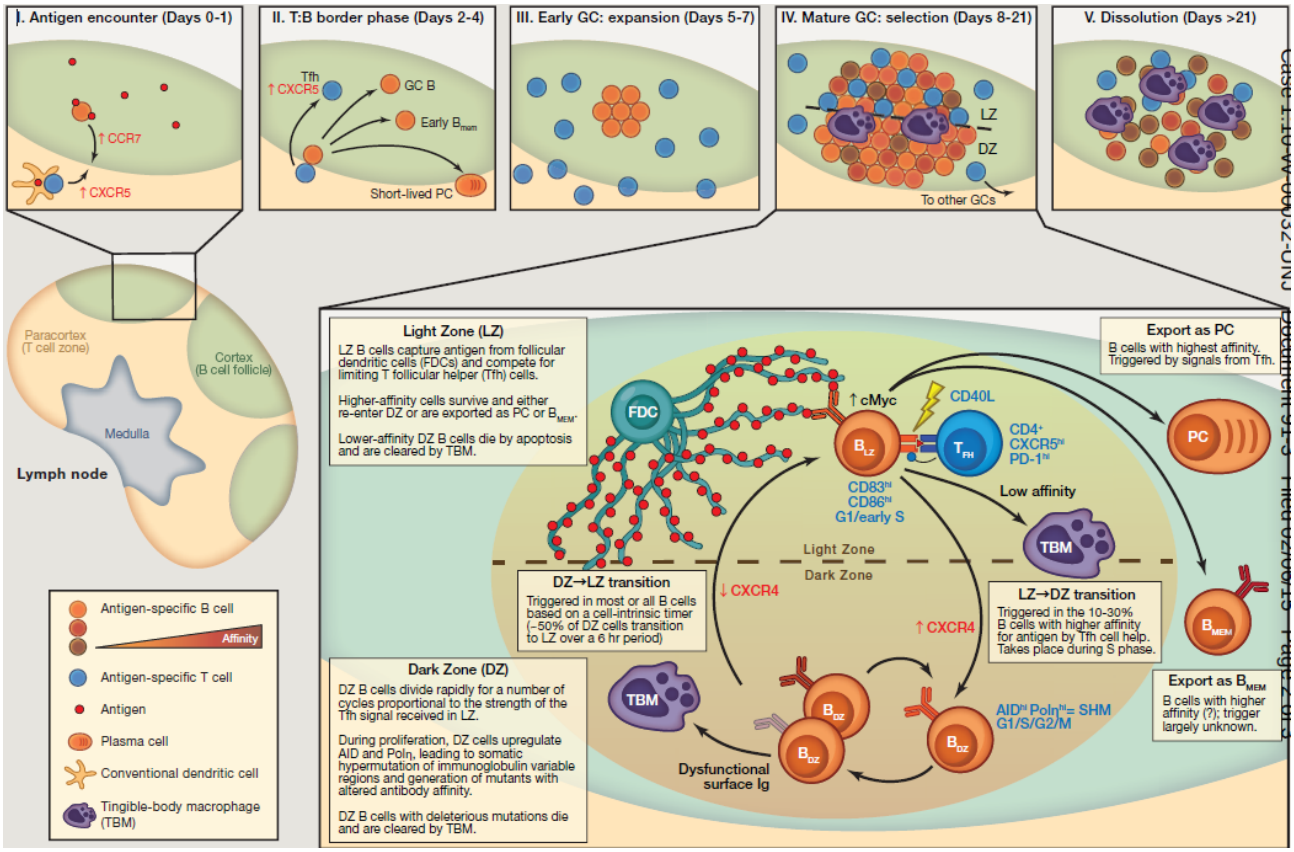

Exhibit R at 1. This exhibit was developed through a study of mice. Dr. Vargas opined that the timeframe for germinal center formation in humans would be "at least somewhat analogous." Tr. 214.

Dr. Vargas also discussed her background and clinical experience with lymphoid hyperplasia in children. She opined that viral infection is the most common cause of lymphoid hyperplasia among children in E.M.F.'s age group. Tr. 192. When a child has a viral infection, he or she will have elevated white blood cells, including lymphocytes. Tr. 237. It takes at least seven to ten days for these lymphocytes to accumulate to the level that is observable on an x-ray, and she believed that these infiltrates were likely present seven to ten days prior to the E.M.F.'s x-ray and diagnosis of pneumonia on November 26, 2007, well before the second set of vaccines were administered. Tr. 222. She said that the medical examiner did not describe from what parts of the lungs the tissue slides were made, and therefore she could not determine if the lymphoid hyperplasia was in the same location as the infiltrates seen on the earlier x-ray. Tr. 221.

Dr. Vargas opined that a child with lymphoid hyperplasia can show symptoms, such as "increased work of breathing, grunting, flaring of the nostrils, [and] wheezing." Tr. 223. However, the child may also be asymptomatic. Tr. 223.

If a child is symptomatic, the child is sent for a chest x-ray, and it detects lymphoid hyperplasia, the treating physician's first course of action will be to prescribe antibiotics or some other treatment. Tr. 195. If some time passes (at least six weeks) and the child is still symptomatic, the physician may order new radiology. Tr. 195-96. If lymphoid hyperplasia is still shown, the treating physician will ask Dr. Vargas to do a lung biopsy. Tr. 196. Therefore, she only ever biopsies lungs that have at least six weeks of symptomatology, and these tend to show mature

germinal centers. Dr. Vargas acknowledged that she might be "biased" due to this clinical experience. However, she maintained that germinal centers take more than a few days to develop in the human body.[30]

Dr. Vargas was asked how long germinal centers can exist before dissolving. Tr. 219. The Victora source suggests that "dissolution" occurs in "days > 21." Exhibit R at 700. Dr. Vargas responded that new germinal centers might be "forming and breaking down all the time," in which case, they would be "persistent." Tr. 219. Therefore, lymphoid hyperplasia with germinal centers would take more than a few days to form, but could persist for months.

3. *Althen* Prong One: Evaluation of the Evidence

As stated above, under *Althen* prong one, petitioners must set forth a reliable medical theory explaining how the vaccines at issue could have caused the alleged injury. *Althen*, 418 F.3d at 1278. While scientific certainty is not required to establish causation under the Vaccine Act, the theory must be supported by a "sound and reliable" medical or scientific explanation. *Althen*, 418 F.3d at 1279; *Knudsen*, 35 F.3d at 548.

Here, petitioners, through their expert Dr. Waters, have failed to establish *Althen* prong one. Dr. Waters presented three different theories, with varying levels of detail. First, she proposed that E.M.F.'s fever may have acted as an exogenous stressor under the Triple Risk Model of SIDS. But she did not thoroughly develop this theory.

Second, Dr. Waters suggested that E.M.F.'s apparent reaction after both sets of vaccinations could represent a challenge/re-challenge phenomenon. Dr. Waters pointed to an anamnestic response to the second set of vaccines, which she indicated would be much stronger than the reaction to the first. It is true that E.M.F. suffered fever and various other symptoms after both her two-month and four-month vaccinations, but her father testified that she seemed to be fine on the first day that she was taken to the new babysitter, the day on which she died. The challenge/re-challenge theory generally requires a showing of an enhanced reaction to a second dose of a vaccine that is more pronounced than the initial reaction. The possible reaction to the second set of vaccines included fever and congestion over the weekend. Ms. Forrest described E.M.F.'s reaction to the second vaccinations as similar but more intense than her reaction to the first. Tr. 26. Ms. Forrest said that after E.M.F. received the second vaccinations, she began crying, developed a fever, and did not sleep that afternoon or that night. Tr. 26-27. Mr. and Ms. Forrest alternated doses of Motrin and Tylenol and gave her a cool bath, but she stayed up all night. Tr. 67. At the hearing, Mr. Forrest stated that E.M.F.'s fever was between 103° and 105° Fahrenheit that night. Tr. 67, 85. While Mr. Forrest and Ms. Forrest did report a high fever, crying, and sleeplessness through the first night, there was no hospitalization or medical consultation after the second vaccines. More importantly, E.M.F. appeared to be better by the third day post-vaccination and in good health on

[30] Dr. Waters's two pre-hearing reports did not address germinal centers. At the hearing, Dr. Waters stated that she "hadn't seen the full study" by Victora studying germinal center formation in mice and she "wondered about the methodology and how well that is established." Tr. 141. Dr. Waters did not have any citations or literature to indicate that mature germinal centers could form more quickly in humans. Tr. 142.

Petitioner also filed a post-hearing addendum from Dr. Waters. With regards to the timing of germinal center formation, Dr. Waters notes again that Dr. Vargas may be biased because she examines only lungs with "persistent" lymphoid hyperplasia; Dr. Vargas's only support was the Victora article involving mice; and Dr. Vargas did not have a source for timing in humans. Post-Hearing Brief, Addendum A at 1.

the fourth day, when she was taken to the babysitter. E.M.F. appeared to have passed the symptomatic phase of that reaction by the day she died, as Mr. Forrest testified that the symptoms began tapering off the day before and that she did not seem sick on the morning when he took her to the babysitter. Tr. 72. Dr. Waters did not explain how the re-challenge mechanism could cause death after the reactive symptomatology had passed.

Additionally, about 21 days after the first vaccinations, E.M.F. was taken to Slidell Memorial Hospital with complaints of a one-day history of high fever and congestion. Exhibit 6 at 214-215. Upon examination at that time, she was noted to have fever, rhinorrhea, cough, nausea, and vomiting, and she was diagnosed with acute febrile illness. *Id.* at 215. Later that day, E.M.F. was transferred to West Jefferson Medical Center with an admitting diagnosis of fever, vomiting, and possibly sepsis. Exhibit 2 at 77, 81. E.M.F. remained hospitalized for five days, until November 24, 2007. On November 26, 2007, she was taken to her pediatrician with a fever and vomiting for the past two days. She was diagnosed with fever and on chest x-ray with pneumonia. Exhibit 3 at 198. It appears that the late November symptoms with a diagnosis of pneumonia were more severe than those that occurred in close proximity to both vaccinations, and in Dr. Vargas's opinion the pneumonia was the more likely source of the lymphoid hyperplasia seen on autopsy than were the vaccines administered four days before death.

Dr. Waters's third theory, and her primary focus, was that E.M.F. experienced a type IV delayed hypersensitivity reaction to the four-month vaccinations. She seemed to base this theory on the occurrence of similar symptoms after both sets of vaccinations. However, though she stressed the T-cell nature of the type IV response, she acknowledged on cross-examination that the symptoms immediately after the vaccination, such as fever, were probably a reaction to the "humoral sensitivity which is the antibody sensitivity." Tr. 154. Dr. Waters did not explain why she thought that the vaccines would cause a T-cell response on the heels of a humoral or B-cell response, nor how the initial post-vaccination symptoms which she said were probably a humoral response would be a likely precursor of the T-cell delayed hypersensitivity response. She then admitted that the lymphoid hyperplasia, which she did not find sufficiently significant to mention in her first report but which she later addressed after it was introduced by Dr. Vargas, was the sole piece of evidence in favor of a type IV delayed hypersensitivity response other than the time course. Tr. 14.

Dr. Waters proposed this theory of a hypersensitivity response, but did not supply significant literature to support it. She did produce one book chapter by Drs. Folkerth and Kinney which offers a summary description of SIDS.[31] However, that reference does not mention a hypersensitivity reaction, nor do Dr. Kinney's other more detailed articles on the subject of SIDS causation. While various hypotheses about SIDS causation exist without specific proof of a single cause, this hypothesis seems particularly weak both in terms of its mechanism of action and in terms of its consistency with the underlying facts of this case. I have not found this theory to be persuasive or to be a likely explanation of the cause of this child's death.

[31] Exhibit 17, R. Folkerth & H.C. Kinney, *Disorders of the Perinatal Period: Sudden Infant Death Syndrome*, in Greenfield's Neuropathology (8th ed. 2008) at 306.

B. *Althen* Prong Two: Logical Sequence of Cause and Effect

The second *Althen* prong requires preponderant evidence of a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278. This prong is sometimes referred to as the "did it cause" test; i.e., in this particular case, did the vaccine(s) cause the alleged injury. *Broekelschen*, 618 F.3d at 1345 ("Because causation is relative to the injury, a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case . . . ."); *Pafford*, 451 F.3d at 3. Temporal association alone is not evidence of causation. *See Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992).

Even if I were to accept the theory of a delayed hypersensitivity reaction having occurred in response to the second set of vaccines, based upon the timing of E.M.F.'s unfortunate death and her symptoms after both sets of vaccinations, Dr. Waters completely failed to logically explain how this reaction could cause the child's death. She just said, "There have been a lot of different theories about just how death occurs in this kind of situation. In a sudden death some of them have to do with neurological theories and there's a wide range of actual mechanisms that would be the final pathway." Tr. 160. Dr. Waters was not aware of any literature that linked pulmonary lymphoid hyperplasia to vaccinations. Tr. 161. I asked her specifically whether she could think of a logical explanation for how the amount of lymphoid hyperplasia that was seen on the autopsy would cause the baby to go into cardiac arrest or to stop breathing or anything that would cause death. She answered that the final mechanism is one of those mechanisms that is not explained, that there could be an arrhythmia or there could be a neurological kind of pathway to the death. I asked her further whether she could think of anything that would help us to understand how the lymphoid hyperplasia could activate one of those pathways. She said that during a delayed hypersensitivity reaction, lymphoid hyperplasia is going to be happening all around the body and it could set up an arrhythmia or another more specific final pathway. Tr. 173. Dr. Waters then admitted that the minimal changes that she saw on the slide were not significant and that it seems that there were a few lymphocytes here and there but she was not quite positive on that. Tr. 175. In short, Dr. Waters had no coherent explanation for how the immune response to the vaccines cause a progression from an initial humoral response to a delayed T-cell response to a sudden unexplained death.

What is more, Dr. Waters did not address the issue of E.M.F. being found in the prone position. While the babysitter stated that she put E.M.F. down for her nap on her back, when the babysitter found her later, E.M.F. was lying prone and she had vomited. The prone sleep position is probably the most recognized exogenous risk stressor under the Triple Risk Model of SIDS, and it gave rise to the Back-to-Sleep campaign, as noted by Dr. Vargas. Tr. 208. The prone position is also not proved as a cause of death in this case, but it at least provides a more plausible explanation than Dr. Waters's vague hypersensitivity theory.

C. *Althen* Prong Three: Proximate Temporal Relationship

Under the third *Althen* prong, petitioners must provide "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan*, 539 F.3d at 1352. The acceptable temporal association will vary according to the particular medical theory advanced in the case. *See Pafford*, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. *See, e.g., Veryzer v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 344, 356 (2001) (explaining that "a temporal

relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory causally connecting vaccine and injury").

In consideration of the testimony, I have concluded that Dr. Vargas's experience with and training in pediatric lung pathology should be given more weight in terms of the disagreement as to timing of the development of the lymphoid hyperplasia with mature germinal centers. Dr. Vargas substantiated her contention as to the length of time required for formation of mature germinal centers from her considerable experience in pediatric lung pathology and with the literature source referenced above. Dr. Waters does not have comparable experience with complicated lung pathology and she said that she had not researched the literature on the timing of the development of germinal centers. I have concluded that it is most likely that the lymphoid hyperplasia was present well before the vaccines were administered, as Dr. Vargas testified, and was likely a residual of the pneumonia the child suffered in late November.

Dr. Waters is certainly correct that E.M.F.'s tragic death occurred within a relatively short time period after the administration of the second set of vaccines – four days later. However, the significance of the temporal relationship must be tied to a reasonable theory of how the vaccines could have caused the death and then logically how they did cause the death. A temporal relationship by itself is not sufficient to establish causation, even though it may reinforce a reasonable theory and logical explanation. In this case, neither a persuasive and reliable theory nor a logical explanation of cause and effect were provided leaving the temporal relationship standing alone. Accordingly, petitioners have failed to prove prong three of the *Althen* test.

III. CONCLUSION

I certainly sympathize with the parents in this case who have lost their child, but the Vaccine Act requires that I find that it was more likely than not that the vaccines caused their child's death in order to find them eligible for compensation. Based on the evidence presented in this case, I cannot conclude that petitioners have met this evidentiary burden. Therefore, their claim must be dismissed.

In the absence of a timely-filed motion for review filed pursuant to Vaccine Rule 23, the Clerk of the Court **SHALL ENTER JUDGMENT** consistent with this decision.

IT IS SO ORDERED.

s/Thomas L. Gowen
Thomas L. Gowen
Special Master