In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS

No. 06-663V
Filed: March 22, 2012
Published

BRIDGETTE BIGBEE and ROYCE CARTER as Representatives of the Estate of KALEAF CARTER Deceased,	*	
Petitioners,	*	Denial; Diphtheria-tetanus-acellular pertussis, DTaP; Hemophilus influenzae type B, HIB; Hepatitis B, Hep B; Pneumococcal; Death; Cytokine storm or abnormal cytokine response; SIDS; Positional asphyxia
v.	*	
SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Richard Gage, Richard Gage, P.C., Cheyenne, WY, for Petitioners.
Julia Wernett McInerny, U.S. Department of Justice, Washington, D.C., for Respondent.

DECISION[1]

GOLKIEWICZ, Special Master.

The Petition in this case was filed on September 21, 2006. Petition ("Pet."), filed Sept. 21, 2006. Petitioners alleged that their infant son died as a result of the vaccinations he received on May 23, 2005. Petitioners' case was supported by two experts. Unfortunately for petitioners, respondent's two experts proved to be better credentialed, more knowledgeable, highly experienced with infants and gave vastly superior testimony. Petitioners were ultimately unable to present preponderant evidence showing a causal connection between the unfortunate death of their son and the immunizations he received.

PROCEDURAL HISTORY

[1] The undersigned intends to post this decision on the website for the United States Court of Federal Claims, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002). **As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire decision will be available to the public. Id. Any motion for redaction must be filed by no later than fourteen (14) days after filing date of this filing. Further, consistent with the statutory requirement, a motion for redaction must include a proposed redacted decision, order, ruling, etc.**

The Petition and some medical records were filed on September 21, 2006. Additional records were filed on December 12, 2006, and petitioners were granted a subpoena to obtain autopsy slides on March 13, 2007. Notice of Filing, filed Dec. 12, 2006; Order, filed Mar. 13, 2007. Time between November 2006 and September 2009 was spent by petitioners obtaining the autopsy slides and their expert reports. Order, filed Nov., 17, 2006; Petitioners' ("P") Status Report, filed Feb. 21, 2007; Order, filed Nov. 9, 2007; P Status Report, filed Jan. 10, 2008; P Status Report, filed May 9, 2008; P Status Report, filed Aug. 18, 2008; Order, filed Sept. 26, 2008; Order, filed Mar. 9, 2009; P Status Report, filed Apr. 13, 2009; P Status Report, filed May 4, 2009; Order, filed May 18, 2009; P Status Report, filed Jun. 5, 2009; Order, filed Jun. 15, 2009; Order, filed Sept. 1, 2009. The case was converted to electronic filing on April 21, 2009. Notice of Designation of Electronic Case, entered Apr. 23, 2009.

On September 9, 2009, petitioners filed their expert reports from Drs. Levin and Byers. P Ex 10, First Report of Dr. Levin, filed Sept. 9, 2009; P Ex 11, First Report of Dr. Byers, filed Sept. 9, 2009. Thereafter, respondent's Rule 4(c) Report and responsive expert reports from Drs. Gilbert-Barness and McCusker were filed on February 25, 2010. Respondent's ("R") Report, filed Feb. 25, 2010 ("R Report"); R Ex A, First Report, amended, of Dr. Gilbert-Barness, filed Mar. 22, 2010; R Ex D, First Report of Dr. McCusker, filed Feb. 25, 2010.

On July 12, 2010, a Hearing was set for November 11 and 12, 2010. Hearing Order, filed Jul. 12, 2010. Petitioners' filed responsive reports from their experts on September 23, 2010. P Ex 16, Second Report of Dr. Levin, filed Sept. 23, 2010; P Ex 17, Second Report of Dr. Byers, filed Sept. 23, 2010. Respondent filed replies from her experts on October 29, 2010. Medical literature was thereafter filed by both parties.

On November 3, 2010, a status conference was held by the undersigned to consider respondent's concerns regarding a perceived new or broader theory of causation in petitioners' case. Minute Entry, entered Nov. 3, 2010. Although the undersigned did not find a new theory specivically alleged, respondent was granted the opportunity to file additional medical literature, which came in on November 4, 2010. Additional exhibits were also filed by petitioners in anticipation of the upcoming Hearing. The Hearing was held on November 11 and 12, 2010. Minute Entry, entered Dec. 28, 2010. Testimony was taken from petitioner Bridgette Bigbee, Kaleaf's mother. Testimony was also taken from the four experts. Following the Hearing, additional evidence that was discussed at the Hearing was entered into the record. See Order, filed Nov. 28, 2010. Following unsuccessful efforts to informally resolve the matter, post hearing briefs were filed. P Post Hearing Memorandum ("P Memo"); R Post Hearing Memorandum ("R Memo"). As such, this matter is now ripe for this decision regarding entitlement to compensation.

FACTUAL HISTORY

The facts of this case are largely undisputed. Clarification of the events surrounding Kaleaf's death was provided during the November 2010 Hearing. Minute Entry, filed December 28, 2010; Hearing Transcript of Proceedings ("Hr'g Tr."), filed December 6, 2010. Kaleaf was prematurely born[2] on May 23, 2005, and was immediately admitted to the infant intensive care

[2] Premature birth is a risk factor for SIDS. ROBERT M. KLIEGMAN, MD, *et al.*, NELSON TEXTBOOK OF PEDIATRICS 1424, 1428 (19th ed. 2011); see also Hr'g Tr. 223:12-14 ("prematurity is a risk factor for sudden infant death . . .").

unit. Pet. at ¶ 1; P Ex 2 at 2-20. Records indicate his mother had no prenatal care until two weeks prior to his birth[3] and that Kaleaf was between 30 and 36 weeks gestation. See, e.g., P Ex 11 at 2. At the time of birth, Kaleaf's mother was treated for cystitis and was positive for use of cannabis during the pregnancy.[4] Id.; R Report at 2, n. 2; P Ex 1; P Ex 2 at 4, 8, 14-16, 18. Kaleaf was shown to be hypoglycemic, was treated for jaundice and given antibiotics pending negative blood cultures. P Ex 11 at 2; P Ex 2-20. He was released from the infant ICU after 19 days[5] and was below the normal limits for weight, length and head circumference. Id.; P Ex 10 at 1. The discharge summary indicated he had apnea and bradycardia due to prematurity and was jaundiced but not septic. P Ex 11 at 2; P Ex 10 at 1; P Ex 2 at 6-7. Kaleaf also received his first hepatitis B vaccination on June 13, 2005, which was uneventful. P Ex 2 at 7. A follow-up exam on June 20, 2005, was also uneventful. P Ex 2 at 18; P Ex 10 at 1; P Ex 11 at 2.

On September 19, 2005, Kaleaf was seen for his three-month check-up. According to the appointment check-in ledger and his mother's testimony, they arrived to the appointment at 10:04 a.m. P Ex 25; Hr'g Tr. 9:21-10:8. During the exam, it was noted that he was having some trouble breathing, possibly due to congestion. P Ex 3 at 1; November 11-12, 2010 Hr'g Tr. 9:13-17, filed Dec. 6, 2010.[6] The record reads, "trouble breathing, sniffs/noisy sounds from the nose on and off." P Ex 3 at 1. The exam showed "nares patent" with "scant rhinorrhea."[7] Otherwise, the exam and his development were normal. Id. He received five vaccinations: hepatitis B ("Hep B"), hemophilus influenza type B ("HIB"), diphtheria-tetanus-acellular pertussis ("DTaP"), inactivated poliovirus ("IPV"), and pneumococcal ("PCV"). Id. As acknowledged by petitioners' expert, there is no indication whether or not Kaleaf had a fever before or after his vaccinations or up to the time of his death, on September 19, 2005. See, e.g., Hr'g Tr.49:23-50:12.

After returning home from the appointment, according to Kaleaf's mother, she fed him formula and put him down for a nap. P Affidavit of Bridgette Bigbee, filed Sept. 21, 2006; Hr'g Tr. 12:4-13:9; P Ex 5 at 4. It was approximated that Kaleaf was put down for his nap around

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Sudden infant death syndrome, SIDS, is "[t]he sudden, unexpected death of an infant that is unexplained by a thorough postmortem examination . . . ." NELSON TEXTBOOK OF PEDIATRICS 1421 (19th ed. 2011).

[3] Receipt of less prenatal care and initiating prenatal care later in a pregnancy is also a risk factor for SIDS. NELSON TEXTBOOK OF PEDIATRICS 1424 (19th ed. 2011). See also Hr'g Tr. 220:11-221:11 (noting a relevant lists risk factors for SIDS in the present case to be "smoking and drug use during pregnancy, ethnicity. It's higher in the Afro-American population. . . . Lack of breast feeding, lack of prenatal care . . . position of the baby in bed, and the type of bed . . . .").

[4] Maternal prenatal drug use is also a risk factor for SIDS. NELSON TEXTBOOK OF PEDIATRICS 1424 (19th ed. 2011).

[5] Intensive neonatal care requirement is a risk factor for SIDS. R Ex H-1 at 5, Enid Gilbert-Barness & Diane E Debich-Spicer, HANDBOOK OF PEDIATRIC AUTOPSY PATHOLOGY (Humana Press, 1st ed. 2004).

[6] Tachypnea is defined as "excessive rapidity of breathing." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1891 (31st ed. 2007). Dr. Byers described tachypnea as "rapid breathing and deep breathing," "more of a gasping." Hr'g Tr. 35:22-36:6.

[7] Nares are one's nostrils; Patent means "open, unobstructed, or not closed;" Rhinorrhea is "the free discharge of a thin nasal mucus" or a runny nose. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1251, 1416, 1665 (31st ed. 2007).

11:00 am. Hr'g Tr. 14:6-19.[8] It was reported that Kaleaf was placed on his parents' bed after being fed and "was placed on his back with [his] upper torso propped up with a pillow."[9] P Ex 5 at 4. Kaleaf was checked on once after being put down for his nap and no problems were observed at that time. Hr'g Tr. 15:1-8. Kaleaf's mother checked on him a second time, just prior to placing the 911 call at 12:45 pm, and found Kaleaf unresponsive. Hr'g Tr. 15:8-12; P Ex 7 at 1-5; P Ex 4 at 1. She reported finding him "on his stomach face down on the pillow and unresponsive."[10] P Ex 5 at 4. Kaleaf's mother screamed and his father entered the room. P Ex 5 at 4. It was noted that there was a light, bloody discharge from his nose. P Ex 5 at 4. Emergency personnel arrived at petitioners' home at 12:49 pm and found Kaleaf unconscious, with no pulse or breathing and showing signs of cyanosis. P Ex 7 at 5. Kaleaf was transported to the hospital, CPR and infusions were performed en route, and he was declared dead upon arrival. P Ex 8 at 2; P Ex 4.[11]

The cause of death was noted by the medical examiner to be Sudden Infant Death Syndrome ("SIDS").[12] Id.; P Ex 5. A majority of the autopsy findings were normal with the exception that many organ weights were greater than average, the significance of which is a major point of contention between the parties.[13] The autopsy diagnosis noted hyperplasia of the thymus,[14] generalized visceral congestion and no evidence of injury or congenital anomaly. P Ex 8 at 6. In an interview with the deputy coroner, Kaleaf's father noted Kaleaf had been "suffering from a rapid breathing problem that had been reported to the hospital . . . ." P Ex 5 at 4. Also in an interview with the deputy coroner, Kaleaf's mother noted she was told he "was suffering from some type of congestion." P Ex 5 at 4. Petitioners' expert referenced the "rapid breathing problem" in her report and later characterized this as tachypnea in hearing testimony. E.g., R Ex 17 at 2 (referring to the condition as a "rapid breathing problem"; Hr'g Tr. 34:15-23.

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[8] There was some discrepancy in the medical records relating to the time Kaleaf was put down for his nap; however, testimony from Kaleaf's mother during the November 2010 Hearing clarified the events and these facts are not in dispute.

[9] Soft sleep surfaces or bedding, such as a pillow, are risk factors for SIDS. NELSON TEXTBOOK OF PEDIATRICS 1424, 1425 (19th ed. 2011).

[10] A prone, face down, sleep position is important risk factor for SIDS. NELSON TEXTBOOK OF PEDIATRICS 1424, 1428 (19th ed. 2011).

[11] There was an admission from Kaleaf's mother that marijuana was smoked in the home in Kaleaf's presence on the day of his death. P Ex 8 at 20; P Ex 4 at 3.

[12] Sudden infant death syndrome, SIDS, is "[t]he sudden, unexpected death of an infant that is unexplained by a thorough postmortem examination . . . ." NELSON TEXTBOOK OF PEDIATRICS 1421 (19th ed. 2011).

[13] P Ex 5 at 6-8; P Ex 5 at 6 (noting Kaleaf was between the 95th and 99th percentile in weight for his age); P Ex 5 at 6 (noting brain weight of 593 grams and a mean for age of 567 ± 81 grams); P Ex 5 at 7 (noting heart weight of 48.1 grams and a mean for age of 30 ± 7 grams); P Ex 5 a6 7 (noting combined lung weight of 139.9 grams and a mean for age of 89 ± 23 grams); P Ex 5 at 7 (noting liver weight of 268.3 grams and a mean for age of 179 ± 41 grams); P Ex 5 at 7 (noting spleen weight of 22.8 grams and a mean for age of 16 ± 5 grams); P Ex 5 at 8 (noting thymus weight of 52.2 grams and a mean for age of 10 ± 5 grams); P Ex 5 at 8 (noting a combined kidney weight of 64 grams and a mean for age of 45 ± 10 grams).

[14] The thymus is "a bilaterally symmetric lymphoid organ . . . situated in the anterior superior mediastinum. . . . The thymus is the site of production of T lymphocytes." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1950 (31st ed. 2007).

EXPERTS OPINIONS AND TESTIMONY

Dr. Alan Levin and Dr. Vera Byers testified for petitioners. Dr. Levin was accepted as an expert in pathology; respondent objected to qualifying Dr. Levin as a pediatric pathologist during the Hearing. Dr. Byers was accepted as an expert in immunology. At Hearing, respondent objected to Dr. Byers' qualification as an expert in pediatric immunology. The undersigned acknowledged respondent's objections and noted this could affect the weight given to their testimony.

In summary, and as will be discussed in depth below, petitioners' experts opine that Kaleaf suffered from a lung infection, pneumonia or pneumonitis, prior to receiving his vaccinations. According to petitioners' experts, a vaccine or a vaccine component that Kaleaf received triggered an excessive release of cytokines that eventually inflamed his already-burdened lungs, causing increased circulatory resistance, ultimately causing respiratory distress, right heart failure and death. At different times, petitioners' experts refer to this as a vaccine-induced cytokine storm and, at other times, a vaccine-induced cytokine burst. What was clear was that petitioners' experts were opining to an exaggerated or aberrant cytokine response.

Respondent's experts disagreed with the opinions of Drs. Levin and Byers. Dr. Enid Gilbert-Barness testified for the respondent and was accepted as an expert in pediatric pathology, without objection. Dr. Christine McCusker testified for respondent and was accepted as an expert in pediatrics and pediatric immunology, without objection. Drs. Gilbert-Barness and McCusker found no evidence that Kaleaf suffered from a preexisting lung infection or a cytokine storm. Moreover, they contest petitioners' theory of causation concerning the cytokine storm or cytokine burst.

Regarding the findings of credibility and persuasiveness of expert witnesses, the Federal Circuit has noted:

> [T]his court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act. . . . In Moberly, we reiterated that a special master may not cloak the application of an erroneous legal standard in the guise of a credibility determination to shield it from appellate review. [Moberly v. Sec'y of the Dept. of Health & Human Servs.,] 592 F.3d [1315,] 1325 [Fed. Cir. 2010](discussing Andreu[v. Sec'y of the Dept. of Health & Human Servs.], 569 F.3d [1367,] 1379[Fed. Cir. 2009]). We went on to clarify that this does not mean that "a special master, as the finder of fact in a Vaccine Act case, is prohibited from making credibility determinations regarding expert testimony." Id. We indicated that "[a]ssessments as to the reliability of expert testimony often turn on credibility determinations" and "[f]inders 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." Id. at 1326.

Porter v. Sec'y of the Dept. of Health & Human Servs., 663 F.3d 1242, 1250-51 (Fed. Cir. 2011)(nonprecedential opinion).

The undersigned found the testimony from respondent's experts to be supported by evidence in the medical records and in the provided medical literature. The impressive academic and clinical experience of both of these experts, especially with a focus on pediatrics and infants, was evident and strengthened the persuasiveness of their testimony. Petitioners' experts' lack of recent clinical experience and their lack of a pediatric focus, when compared to that of respondent's experts, was palpable. Further, examining the medical literature filed by both parties demonstrated that the opinions given by petitioners' experts were, at best, speculative on certain critical issues and, at worst, unreliable. On the whole, after considering the entirety of the evidence, the undersigned found respondent's experts better qualified and vastly more persuasive; and thus, the undersigned relied heavily upon their information in resolving this case.

PETITIONERS' EXPERTS

Dr. Alan S. Levin, MD, JD

Dr. Levin's *curriculum vitae* is filed as petitioners' Exhibit 30. P Ex 30, Levin CV, filed February 9, 2011. Dr. Levin has a master's degree in biochemistry and received his medical degree from the University of Illinois in 1964. P Ex 30 at 1. Dr. Levin also received a juris doctor from Golden Gate University in San Francisco in August 1995 and currently practices law. P Ex 30 at 1. He is board certified in allergy immunology, clinical pathology, and emergency medicine. Id.; Hr'g Tr. 116.[15] Dr. Levin estimates that when he was practicing medicine, he performed fifty to sixty autopsies on children, half of which were "probably" infants, with the last infant autopsy being performed in the "1980's." Hr'g Tr. 120:14-121:3. His CV lists numerous publications and states these publications are primarily in the subjects of immunology, immunopathology, cancer biology and treatments. See P Ex 30 at 4-5. Dr. Levin testified that this was an abbreviated form of his bibliography and noted that article number 6 involved pathology in the death of a six year old child. Hr'g Tr. 126:11-129:19. He last saw a patient as a treating doctor in 1993 and has only worked as a physician consult since that time. Hr'g Tr. 130:130:12-17. At present, Dr. Levin approximates that 99% of his time is spent practicing law, specifically toxic torts. Hr'g Tr. 129:23-130:9.

Petitioners offered Dr. Levin as an expert in pathology. Hr'g Tr.119:3-4. The respondent engaged Dr. Levin in a significant *voir dire* examination; at the conclusion of which, respondent accepted Dr. Levin as an expert in general pathology but noted an objection if he was offered as an expert in pediatric pathology. Hr'g Tr. 119:6-131:5. The undersigned acknowledged respondent's point, stating this could affect the weight of his testimony. Hr'g Tr. 131:6-7.

[15] Respondent noted that Dr. Levin is not board-certified in pediatrics or pediatric pathology, but attested to being board-eligible in pediatrics. R Post-Hearing Brief at 19, n. 20. Respondent also noted that Dr. Levin testified that "guessed" that he has testified as a pathologist in "virtually all of the vaccine cases" in which he has testified. Id. However, respondent noted that in the two cases alluded to by Dr. Levin, he testified in the area of immunology. Id. In her citations, respondent also listed two cases in which Dr. Levin's opinions were called into question. Id. (citing Colon v. Sec'y of the Dept. of Health & Human Servs., No. 04-44V, 2007 WL 268781 (Fed. Cl. Spec. Mstr. 2007)(noting concern that Dr. Levin's conclusion was contrary to findings by the forensic and pediatric pathologists); Durden v. Sec'y of the Dept. of Health & Human Servs., No. 06-163V, 2007 WL 4962000 (Fed. Cl. Spec. Mstr. 2007)(finding Dr. Levin's testimony on the subject of immunology "confusing at best, and disingenuous at worst" and "involve[ing] circular logic."). See also Cohen v. Sec'y of the Dept. of Health & Human Servs., No. 94-0353V, 1998 WL 408784 (Fed. Cl. Spec. Mstr. 1998)(finding Dr. Levin's testimony unimpressive and noting that he relies on literature that does not support his opinion); but see Bragg v. Sec'y of the Dept. of Health & Human Servs., No. 08-477V, 2012 WL 404773 (Fed. Cl. Spec. Mstr. 2012)(crediting Dr. Levin's immunology opinion and finding petitioner entitled to compensation).

Dr. Levin's opinions are presented in petitioners' Exhibits 10 and 16, as well as in testimony from the November 2010 Hearing. P Ex 10, filed Sept. 9, 2009; P Ex 16, filed Sept. 23, 2010; Hr'g Tr. 114-210. In his first report, Dr. Levin briefly reviewed Kaleaf's medical history. He noted that there is no indication that the autopsy slides were reviewed by a medical examiner and that the diagnosis of SIDS was made at the autopsy. P Ex 10 at 1. He indicated that Kaleaf's measurements showed he was above the 95th percentile in size for his age; indicating that he "had attained normal length and weight by the time of his death" following his birth. Id. Dr. Levin found the autopsy was "remarkable for the heavy lungs, heavy viscera and heart, congested red pulp of the spleen, and thymic hyperplasia" and noted the organ weights. Id. at 1-2. He found that there is no indication of aspiration pneumonia. Id. at 2. He estimated the fatal event occurred approximately 2 ¾ hours after vaccination. Id. at 2. Dr. Levin found that the autopsy findings and his review of the pathology slides are inconsistent with aspiration pneumonia and SIDS, but did not explain how they are inconsistent or provide support. Id. at 3.

Regarding the autopsy slides, Dr. Levin stated they are "notable for cerebral gliosis, cerebral edema, and neuronal swelling in the brain." P Ex 10 at 2. He noted "**profound and massive**[] congest[ion of the] lungs, liver, spleen, and kidneys." Id. (emphasis added). Later in his report, Dr. Levin stated these findings are "consistent with right heart congestive failure." Id. at 3. Further, "[t]here were symptoms of marked reduced cardiac output, which would result in hypotension and congestion of the visceral organs. All these findings are characteristic of **overwhelming inflammation**[, which] occurred rapidly after the vaccination." Id. at 2 (emphasis added). Due to the gross brain weight being within normal limits, "the immediate cause of death was more cardiopulmonary than cerebral." Id. at 3. He opined that Kaleaf suffered "from a low grade viral infection for several weeks prior to vaccination," supported by the notation in the medical records of two weeks of 'sniffles' and normal breath sounds in the well baby exam" on the day of his death and his interpretation of the lung slides showing thickened alveoli and lymphocytic infiltrate. Id. According to Dr. Levin, "[t]he hypertrophic thymus and spleen are consistent with marked activation of T cells in the infant."

Dr. Levin ultimately opined that the case is "inconsistent with aspiration pneumonia and SIDS and [is] consistent with hypoxic death caused by right heart failure which in turn was caused by cytokine storm." P Ex 10 at 3. "The most probable chain of events [is] that the child was suffering from a mild chronic viral pneumonitis which became activated by his vaccination which, in turn, caused massive thickening of the alveolar membranes, increased pulmonary vascular resistance, right heart failure, hypoxia and death." Id.[16] In his testimony, Dr. Levin stated that the inflammation found on the slides of Kaleaf's lungs were actually patches of massive inflammation. Hr'g Tr. 160:18-161:15. An estimate of 10-15% involvement of inflammation in the lung was given by Dr. Levin. Hr'g Tr. 168:20-169:2. Dr. Levin's report included black and white pictures from the pathology slides, which he claimed evidence lymphocytic infiltrate on multiple slides, thickened alveolar walls and congestion in the lungs, and edema in the brain and lungs. P Ex 10 at 4-5.

[16] The terms hypoxia, anoxia and asphyxiation were used somewhat interchangeably during this case. Asphyxia is defined as "pathological changes caused by lack of oxygen in respired air, resulting in hypoxia and hypercapnia[, which is an excess of carbon dioxide in the blood.]" DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 167, 899 (31st ed. 2007). Anoxia is "a total lack of oxygen; often used interchangeably with hypoxia to mean a reduced supply of oxygen to the tissues." Id. at 97. Hypoxia is "reduction of the oxygen supply to tissue below physiological levels despite adequate perfusion of the tissue by blood." Id. 921.

In his supplemental report, Dr. Levin disagreed with respondent's expert's histopathology assessment, particularly focusing on the reading of the autopsy slides. P Ex 16. Specifically, he disagreed with respondent's expert in that he found the slides showed lymphocytic infiltrates and massive alveolar thickening in lung slides. P Ex 16 at 1-3. He also states that thymic hyperplasia "is seen 'often in the presence of respiratory distress or peripheral blood lymphocytosis, or both.'" Id. at 3 (citing P Ex 22).[17] Ultimately, Dr. Levin disputed Dr. Gilbert-Barness's finding that the child died of SIDS or simple asphyxia. P Ex 16 at 3.

Dr. Levin's Hearing Testimony

Expanding upon his reports, Dr. Levin's testimony was offered at the November 11-12, 2011 Hearing. In his testimony, Dr. Levin noted that the brain was of a normal weight. Hr'g Tr. 132:21-22. He also noted that Kaleaf's heart was large but otherwise normal. Hr'g Tr. 132:22-133:1. He characterized Kaleaf's lungs as "outrageously heavy" and "congested with blood." Hr'g Tr. 133:6-9. Dr. Levin described the thymus as large and also congested with blood. Hr'g Tr. 133:23-24.

Moving on to the autopsy slides, Dr. Levin pointed out an area of what he finds to be lymphocytic infiltrate, which he said is unequivocal evidence that Kaleaf suffered from "a chronic inflammatory process in the upper lung." Hr'g Tr.139:4-140:7. He found lymphocytes also in the slide of the lower lung, as well as thickened alveolar cells. Hr'g Tr. 140:10-141:4. He described viral pneumonias as "patchy," explaining that is why one portion of the lung slide looks normal while another portion shows lymphocytic infiltrate and thickened alveoli. Hr'g Tr. 143:20-23; Hr'g Tr. 147:19-25.[18] When the pneumonia or thickened alveolar walls reach a certain level, pumping blood through the lungs to be oxygenated becomes more difficult due to more resistance on the right ventricle of the heart. Hr'g Tr. 148:19-150:7. The right side of the heart "failed, and then the left heart was still pumping blood, so the blood was going to the kidney or liver, the spleen and the lungs, but it can't come out because the right heart isn't working;" to Dr. Levin, this explained why the autopsy showed heavy organ weights. Hr'g Tr. 150:3-13.

[17] C. Ricci, et al., True Thymic Hyperplasia: A Clinicopathological Study, 47 THE ANNALS OF THORACIC SURGERY 741 (1989). Once cited, exhibits of medical literature are referenced simply by the party ("P" or "R") and the exhibit number (e.g. "Ex 22").

[18] During the portion of Dr. Levn's testimony regarding the slides of the lungs, Dr. Levin intimated that a magnified slide image prepared by Dr. Gilbert-Barness was highly selective in cutting out portions of the slides that showed inflammation and infiltrates. Hr'g Tr. 134-135; Hr'g Tr. 143. In petitioners' Post Hearing Brief, petitioners accuse Dr. Gilbert-Barness of altering slides used in evidence. P Post Hearing Memorandum at 10-11, filed Apr. 8, 2011; Hr'g Tr. 253. Petitioners' counsel questioned Dr. Gilbert-Barness regarding this alleged alteration. Hr'g Tr. 253-54. From what the undersigned understood, each pathologist selected portions of slides they thought were relevant to magnify; it seems that petitioners accuse Dr. Gilbert-Barness of choosing or cropping a magnified image to exclude inflammation. The undersigned was confident from her testimony that what was done was standard procedure and constituted what Dr. Gilbert-Barness found relevant on the slide. Regardless of that explanation though, respondent's counsel took Dr. Gilbert-Barness through the other magnified portion of the lung slides relied upon by petitioners and she "demonstrated that neither slide showed abnormal levels of lymphocytic infiltration or alveolar thickening," which was opined by Dr. Levin. R Post Hearing Memorandum at 21-22, n. 26, filed May 9, 2011 (citing Hr'g Tr. 235-36; Hr'g Tr. 254-55). The undersigned finds petitioners' accusations baseless.

Dr. Levin also showed what he says is evidence of cytokine damage to Kaleaf's brain. Hr'g Tr.154:14-155:3 (noting a perineuronal halo in Exhibit 27, page 3, indicative of cerebral edema). Dr. Levin described what he finds to be swelling of the astroglial cells as they die, which he opined would not happen in a five to ten minute hypoxic event such as positional asphyxia. Hr'g Tr. 10-22. However, Dr. Levin did not explain the basis for this statement or state how long this process should take.

At this point in his testimony, Dr. Levin answered in the negative when asked if the heavy weight of Kaleaf's organs could be explained by a hypoxic death. Hr'g Tr.156:23-157:4; see also Hr'g Tr. 158:7-159:5. Dr. Levin's testimony on this point was confusing as he testified on the second day of the Hearing that, generally speaking, both parties were advancing a theory of an asphyxial death. See Hr'g Tr. 356:10-13.

A significant portion of the cross-examination time of Dr. Levin centered on his own reading of several slides that are in evidence and the support for his views on the slides and their significance. Hr'g Tr. 160:12-173:8. Much of this testimony is confusing. The undersigned ultimately relied upon Dr. Gilbert-Barness for the interpretation of these slides due to her vastly superior knowledge and experience in pediatric pathology.

Dr. Levin was also asked about the natural history of the thymus in infants. Hr'g Tr. 174:24-176:4. As a child ages, the thymus gets smaller; however, Dr. Levin stated that the thymus actually grows when an infant is under stress and cited petitioners' Exhibit 22, the Ricci article, for support. Hr'g Tr. 174:24-176:4 (citing P Ex 22). At Hearing, Dr. Levin also quoted from petitioners' Exhibit 18, the abstract of the Ricci article, and stated "[t]rue thymic hyperplasia is often present in respiratory distress."[19] Hr'g Tr. 177:19-20 (quoting P Ex 18).[20] The undersigned notes that this statement of correlation is not the same as saying respiratory problems cause thymic hyperplasia. Regarding Kaleaf's thymus, which was larger than normal, Dr. Levin opined that it is four times the normal size of a thymus due to infiltration of lymphocytes; this is evidence of an ongoing, chronic illness. Hr'g Tr. 182:12-190:4. He stated that the large thymus, or hyperplasia[21] of the thymus, is not an incidental finding. Hr'g Tr. 184:10-185:18. Again confusingly, though, he admitted the structure of Kaleaf's thymus, as shown in the pathology slides, was normal. Hr'g Tr. 184:25-189:7; Hr'g Tr. 193:19-24.

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[19] This quote supplied by Dr. Levin from the article abstract, petitioner's Exhibit 18, or the article itself was somewhat inaccurate. The article actually says, "[o]ur study of 4 patients and review of the literature indicate that true thymic hyperplasia has a well-defined clinicopathological profile: prevalence in children or young male patients, absence of associated autoimmune disease, and often presence of respiratory distress . . . ." P Ex 22 at 1; see also P Ex 18.

[20] C. Ricci, et al., True Thymic Hyperplasia: A Clinicopathological Study, 47 THE ANNALS OF THORACIC SURGERY 741 (1989). This is only the abstract of the Ricci article. The whole article is filed at petitioners' Exhibit 22.

[21] Hyperplasia is defined as "abnormal multiplication of increase in the number of normal cells in normal arrangement in a tissue." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 906 (31st ed. 2007). Petitioners' Exhibit 22 discussed "true" thymic hyperplasia. The authors refer to true thymic hyperplasia as a "controversial thymic lesion . . . only recently . . . described as a variable, often massive enlargement of the thymus . . . ." P Ex 22 at 1. "Pathological enlargement of the thymus is due in most instances either to neoplasms of epithelial and non-epithelial origin or to hyperplastic conditions." Id. "True thymic hyperplasia should be considered in the differential diagnosis of anterior mediastinal masses in children and young adolescents." Id.

However, the undersigned's review of Dr. Levin's cited article by Ricci, *et al.*, shows this article does not appear to support Dr. Levin's contention. See P Ex 22. This study focuses on four patients ranging from five years to sixteen years of age; the article further looks at seven cases of thymic hyperplasia reported in literature. P Ex 22 at 1-3 (noting the seven patients reported in literature ranged from seven months to fifteen years of age). The undersigned notes that Kaleaf's thymus weighed 52.2 grams. P Ex 5 at 8. In the Ricci article on thymic hyperplasia, the smallest thymic weight was 120 grams in a twelve year old. P Ex 22 at 2. Referencing the study patients that were closer to Kaleaf's age, the thymic weights were 224 grams in a seven month old and 420 grams in a twelve month old. Id. at 3. These weights appear well beyond what was reported for Kaleaf. Moreover, it appears that symptoms of respiratory problems in the study were caused by the thymic hyperplasia; not that the thymic hyperplasia was caused by the respiratory problems. P Ex 22. As noted by respondent in cross-examination, the study discussed children older than Kaleaf; the youngest children discussed were referenced from other literature and were seven and twelve months of age. P Ex 22 at 3. When this was pointed out, Dr. Levin shifted away from the Ricci article and attempted to find support elsewhere. Hr'g Tr. 176:20-180:5. Dr. McCusker addressed petitioners' Exhibit 22 in her testimony and confirmed the undersigned's reading of this article. Hr'g Tr. 470:4-472:2; see infra p. 30.

When he shifted away from the Ricci article, P Ex 22, Dr. Levin utilized respondent's Exhibit F, Tab 18 for support. R Ex F-18.[22] Dr. Levin cited to the first page, "[m]ean CT/T was significantly greater . . . in those babies with [respiratory distress syndrome.]" R Ex F-18 at 1.[23] Again, the undersigned notes this statement is not the same as saying respiratory problems, infection specifically, cause thymic hyperplasia. Also, the undersigned is not a medical expert; however, my review of this article indicates the authors are describing thymic size related to a specific disorder, respiratory distress syndrome,[24] which does not appear to be relevant to this

[22] Ira H. Gewolb, *et al.*, Thymus size and its relationship to the respiratory distress syndrome, 95-1 THE JOURNAL OF PEDIATRICS 108 (1979).

[23] Dr. McCusker selected this article to note the relatively large size of an infant's thymus in general. R Ex D at 6.

[24]

> Respiratory distress syndrome (RDS) is a breathing disorder that affects newborns. RDS rarely occurs in full-term infants. The disorder is more common in premature infants born about 6 weeks or more before their due dates. RDS is more common in premature infants because their lungs aren't able to make enough surfactant. Surfactant is a liquid that coats the inside of the lungs. It helps keep them open so that infants can breathe in air once they're born. Without surfactant, the lungs collapse and the infant has to work hard to breathe. He or she might not be able to breathe in enough oxygen to support the body's organs. The lack of oxygen may damage the infant's brain and other organs if proper treatment isn't given. Most infants who develop RDS show signs of breathing problems and a lack of oxygen at birth or within the first few hours that follow.

National Institutes of Health, National Heart Lung and Blood Institute, What is Respiratory Distress Syndrome? (Sept. 1, 2009), www.nhlbi.nih.gov/health/health-topics/topics/rds/ (last visited Jan. 5, 2012). The syndrome is associated with premature birth; Kaleaf was premature but the records note that he did not require respiratory support or intervention following birth when this syndrome normally presents. See P Ex 2 at 2-20.

case. Dr. Levin did not explain this syndrome's significance to Kaleaf's situation and the article was not fully investigated during Dr. Levin's testimony. However, Dr. McCusker discussed why the article does not support Dr. Levin's opinion during her testimony. See Hr'g Tr. 464:24-467:24 (noting that the study examined thymus size prior to onset of respiratory distress and thymus size being a predictor of potential respiratory distress syndrome); see infra p. 30.

Dr. Levin opined similarly regarding the other pathology slides. He testified that Kaleaf's spleen was heavy and thus infiltrated by lymphocytes due to the ongoing infection proffered by petitioners. Hr'g Tr. 193:25-199:24. Regarding several examples of pathology slides of brain tissue, Dr. Levin was unable to note edema due to his complaints regarding the resolution of the slides. Hr'g Tr. 199:25-203:13. Ultimately, Dr. Levin disagreed that the comparison slides provided by Dr. Gilbert-Barness as examples of the "normal" brain were actually normal. Hr'g Tr. 203:23-206:10.

Following testimony from respondent's pediatric pathologist, Dr. Gilbert-Barness, Dr. Levin offered further testimony and the following exchange occurred. Petitioners' counsel asked Dr. Levin, "The government is countering our case by saying that this was an asphyxial death. Are we claiming this was an asphyxial death?" Hr'g Tr. 356:10-12. Dr. Levin replied in the affirmative and explained that the cause of the asphyxiation is the difference between the parties' positions. Hr'g Tr. 356:13; but see Hr'g Tr. 158:7-159:7 (Dr. Levin testifying that heavy organs are not seen with an asphyxial death but still finding heavy organs supportive of petitioner's theory injury). Dr. Levin clarified that petitioners' hypothesis was that Kaleaf died of asphyxiation from right heart failure, which was secondary to the increased resistance in the lungs from the allegedly preexisting pneumonia and vaccine-induced cytokine burst. Hr'g Tr. 356:18-357:19. In the circumstances of petitioners' theory, Dr. Levin stated this was a process: the right heart failure caused a "bottleneck" of fluid, allowing the tissues to fill up and become congested and heavy. Id. Dr. Levin's opinion was that this "bottleneck" and congestion is inconsistent with the theory proffered by respondent that Kaleaf died of positional asphyxiation. Hr'g Tr. 357:6-358:15.

As will be discussed below, respondent's expert countered Dr. Levin's opinion of right heart failure on multiple grounds, but noted significantly the absence of specific cells, hemosiderin-laden histiocytes or macrophages. In response, Dr. Levin stated these cells would be present in chronic heart failure, not in the acute heart failure to which he alleged Kaleaf succumb. Hr'g Tr. 358:16-359:22. Following the hearing, Dr. Levin provided an untitled piece of medical literature which states, "[a]cute passive congestion without hemosiderin deposition or alveolar septal fibrosis" is a microscopic finding with severe, acute left heart failure. P Ex 28, filed Feb. 9, 2011.[25] However, when questioned again by respondent, Dr. Levin admitted that he

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[25] Title, author and page cite are omitted from this filing. A website address within the filing leads to an educational database that requires a login and password. The undersigned's office was able to track the excerpt by the listed ISBN number. The exhibit appears to be from PATHOLOGICAL BASIS OF DISEASE: INTERACTIVE CASE STUDY COMPANION (1999). The undersigned notes that the article discusses **left** heart failure when petitioners' experts discuss **right** heart failure. See supra pp. 7-8, Hr'g Tr. 148:19-150:13. In the absence of an explanation from petitioners, the undersigned could assume the results would be the same considering Dr. Levin's description of the hemosiderin-laden cells; however, this is unclear from the article or the evidence submitted by petitioners. Further, there is no discussion of whether this article is discussing a pediatric or adult patient and whether the statement applies to both pediatric and adult patients. Both sides' experts testified that children are not simply small adults medically speaking. See, e.g., Hr'g Tr. 367:10-16; Hr'g Tr. 383:2-9.

had limited experience with heart failure in premature infants. "I've seen it I think once before." Hr'g Tr.360:4-361:7. He further admitted that this one experience was decades prior. Id. As will be discussed below, the undersigned was not impressed with Dr. Levin's relevant medical experience and testimony in this case.

Dr. Vera S. Byers, MD, PhD

Dr. Byers CV is filed as petitioners' Exhibit 29. P Ex 29, Byers CV, filed Feb. 9, 2011. Dr. Byers has a master's degree in microbiology, a PhD in immunology and received her medical degree from University of California, San Francisco, in 1981. P Ex 29 at 1. Dr. Byers is board certified in internal medicine but practices allergy immunology and sees both pediatric and adult patients. Hr'g Tr. 25:5-14.[26] She did a fellowship in clinical immunology and practiced allergy immunology for approximately twenty years. Hr'g Tr. 18:9-15. Describing her pediatric experience, Dr. Byers saw pediatric patients up until 1998, then reviewed records of pediatric patients primarily with Still's disease, a form of juvenile rheumatoid arthritis, for three years, and then returned to practice for another couple years thereafter. Hr'g Tr. 25:17-28:13. The frequency of pediatric cases she saw at those times was not discussed. Like Dr. Levin, Dr. Byers has numerous publications to her credit. P Ex 29 at 6-14.

Dr. Byers was accepted as an expert in immunology at the November 2010 Hearing. However, respondent offered an objection to qualifying Dr. Byers as an expert in pediatric immunology. Hr'g Tr. 30:20-32:21. In response, Dr. Byers did note that at the time she completed her residency in clinical immunology, pediatric patients were the primary class of patients she treated in the three year residency. Hr'g Tr.21:11-21. Again, the undersigned acknowledged respondent's point and stated this could affect the weight of her testimony. Hr'g Tr.31:21-32:2.

Dr. Byer's opinions are presented in petitioner's Exhibits 11 and 17, as well as in testimony from the November 2010 Hearing. P Ex 11, filed Sept. 9, 2009; P Ex 17, filed Sept. 23, 2010; Hr'g Tr. 17-112; 482-89. In her first report, Dr. Byers recited Kaleaf's medical history. P Ex 11 at 1-2. She noted that her review of the autopsy slides shows "chronic inflammation in the lungs, probably 2-4 weeks old, with alveolar thickening, and with marked lymphocytic infiltrates, and very congested lungs, right heart failure with congestion of other organs, together with cerebral and cerebellar edema and perineuronal halos and swollen

[26] Respondent notes that Dr. Byers is not a pediatrician and last examined an infant *circa* 1984. R Post-Hearing Brief at 12, n. 7. Respondent also notes a case in which Dr. Byers' opinions have been subject to criticism. Id. (citing Snyder v. Sec'y of the Dept. of Health & Human Servs., No. 01-162V, 2009 WL 332044, at *15, 52 (Fed. Cl. Spec. Mstr. 2009), aff'd, 88 Fed. Cl. 706 (2009)); see also Shepperson v. Sec'y of the Dept. of Health & Human Servs., No. 05-1064V, 2008 WL 2156748 (Fed. Cl. Spec. Mstr. 2008)(finding Dr. Byers engaged in unsupported leaps of logic and improper analogies from other diseases in reaching her opinion); Rego v. Sec'y of the Dept. of Health & Human Servs., No. 04-997V, 2008 WL 1990844 (Fed. Cl. Spec. Mstr. 2008)(finding Dr. Byer's testimony "confusing, speculative, and frankly suspect as it [was] not supported by the record in this case or other reliable sources."); Torsches v. Sec'y of the Dept. of Health & Human Servs., No. 06-192V, 2008 WL 440285 (Fed. Cl. Spec. Mstr. 2008)(finding Dr. Byers' testimony less credible than testimony provided by respondent's expert considering the experts' comparative experience and supporting literature); Lawson v. Sec'y of the Dept. of Health & Human Servs., No. 90-2455V, 2000 WL 246234 (Fed. Cl. Spec. Mstr. 2000)(criticizing Dr. Byers' opinion and noting her unfamiliarity with pediatric neurological diseases, the area in question).

astrocytes." P Ex 11 at 3.[27] Further, Dr. Byers remarked that the unusually heavy weight of the well-formed thymus is due to "marked lymphocytic infiltrate. A similar picture is noted in the spleen." To Dr. Byers, these characteristics were signs of overwhelming inflammation, occurring rapidly after vaccination. Id.

The systemic inflammatory event that caused Kaleaf's death, per Dr. Byers' first report, was due to cytokine[28] storm,[29] a "massive and unregulated cytokine release." P Ex 11 at 3. Although she notes that a combination of vaccinations Kaleaf received on the day of his death may be responsible, the single most likely cause was the hepatitis B vaccination. Id. She notes that the Institute of Medicine ("IOM") hypothesized the hepatitis B vaccination might act as a superantigen, activating a much larger T-cell response than normal. Id.[30] Whether or not the IOM espoused this theory is later discussed. To offer an example of the effects of a cytokine storm, Dr. Byers referenced petitioners' Exhibit 12, which is also respondent's Exhibit F-9. This article discusses a study wherein researchers were testing a treatment to protect against autoimmune disease; however, the drug had an unintended and unexpected result and inadvertently triggered a cytokine storm in test patients. Id. at 3-4 (citing P Ex 12).[31] This aberrant instigation of the patients' immune system resulted in a systemic inflammatory reaction, a cytokine storm, which eventually caused respiratory failure where death would have occurred had the patients not received rapid medical treatment. Id. at 4. Later in her report, the doctor also discussed bystander activation, also mentioned in the IOM report, P Ex 13 and R Ex K, as a possible means for the process that resulted in Kaleaf's death. P Ex 11 at 4.

Pertaining specifically to Kaleaf, Dr. Byers stated, "[t]he clear sounding lungs on the day the hepatitis B vaccine was given, compared with the pink exudates in the child's mouth at the time he was found near dead, pulmonary infiltrates, and cerebral edema resulting in his death within hours after the hepatitis B vaccination is consistent with death caused allegedly by cytokine storm following the hepatitis B vaccination." P Ex 11 at 4. She hypothesized that his mild illness prior to vaccination was a mild, viral pneumonia, resulting in "low grade activation of the pulmonary macrophages." Id. This feature overlapping with the "hepatitis B vaccination and subsequent release of additional cytokines and chemoattractants, more probably than not, caused a massive overstimulation of both the adaptive and the innate immune system causing

[27] The undersigned notes that Dr. Byer's expertise in interpreting pathology slides was not discussed. It does not appear she has such expertise. Presumably, for her opinion in this case, she relied upon the interpretation provided by Dr. Levin.

[28] Cytokine is a "generic term for nonantibody proteins released by one cell population . . . on contact with specific antigen, which act as intercellular mediators, as in the generation of an immune response." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 473 (31st ed. 2007). See also infra pp. 23, 25-27.

[29] Dr. McCusker described a cytokine storm as "systemic inflammatory response syndrome," meaning that relatively unregulated inflammation is occurring systemically. Hr'g Tr. 420-421.

[30] P Ex 13, Institute of Medicine, Kathleen Stratton, et al., Immunization Safety Review, Hepatitis B Vaccine and Demyelinating Neurological Disorders (2002). Petitioners' Exhibit 13 is only two pages of this IOM report. The entire report, which is relied upon by Dr. McCusker to refute Dr. Byers' use of it as proof of a theory of causation, is found at respondent's Exhibit K, filed on November 4, 2010.

[31] P Ex 12 and R Ex F-9, Ganesh Suntharalingam, *et al.*, Cytokine Storm in a Phase 1 Trial of the Anti-CD28 Monoclonal Antibody TGN1412, N ENGL J MED 355:1018-28 (2006). This was sometimes referred to as the "Suntharalingam article" or the "New England Journal of Medicine article" during the Hearing.

cytokine storm." Id. Dr. Byers opined that Kaleaf's symptoms, along with his "thymic hyperplasia . . . are consistent with massive lymphocyte and macrophage activation, characteristic of either bystander activation or of a superantigen." Id. Dr. Byers found the medical examiner's cause of death, SIDS, "absolutely wrong," referencing Dr. Levin's report as support. She concluded that SIDS was diagnosed as the cause of death because it appears that the autopsy slides were not read by the medical examiner. Id.

In her supplemental report, P Ex 17, Dr. Byers refuted the assertion of respondent's expert that cytokine storm has not been reported with the vaccination or wild infection of hepatitis B. Quoting from medical literature, Dr. Byers noted that hepatitis B **infection** "provided the first clue that the disease is caused by an indirect mechanism rather than the virus itself . . . ." P Ex 17 at 1 (citing P Ex 19)(emphasis added to distinguish between the infection and vaccination).[32] Dr. Byers did not make efforts to discuss the comparative differences and similarities between the effects of the wild hepatitis B **infection** and possible effects of a hepatitis B **vaccination**. Nor did Dr. Byers provide separate support for the vaccine causing such damage. The undersigned also notes that the text section is titled, "Virus-Induced Immunopathology." Although cytokine storm is mentioned near the portion Dr. Byers quoted, it is not clear whether the authors are speaking of "cytokine storm" or "virus-induced immunopathology" when they reference "the disease" in that quote. The sentence quoted by Dr. Byers also continues on to discuss tissue damage in the presence of low levels of the hepatitis B virus over "a long time" period, which does not coincide with the short time period leading to Kaleaf's death.

Faced with Dr. McCusker's assertion that Kaleaf suffered from none of the hallmark signs of cytokine storm, Dr. Byers indicated that the phrase cytokine storm encompasses cytokine damage generally, which is highly variable. P Ex 17 at 2. She claimed Dr. McCusker "is incorrect to consider only three signs and symptoms as being diagnostic of cytokine storm." Id. Of note though, Dr. McCusker's testimony clarified that although there can be many eventual symptoms of cytokine storm, the three she looked for in this case are generally seen in all patients with a large cytokine release. See infra pp. 25-27; Hr'g Tr. 446-47. Curiously – injecting another potential cause in this case – Dr. Byers' report continues, "[t]he problems suffered by this child are more akin to the acute lung injury which is usually induced by an immune response to influenza, the cause of most of the damage." Id. Beyond this statement, Dr. Byers did not develop this point of her report.

Dr. Byers also countered Dr. McCusker's conclusion that cardiopulmonary resuscitation ("CPR") could cause the changes seen in Kaleaf. She argued that Kaleaf was already deceased at the time CPR was initiated and therefore cytokines would not be released after his death. Id. Dr. McCusker countered this by noting that circulation was still present, evidenced by a strong radial pulse. R Ex I at 2, 7; Hr'g Tr.; P Ex 4 at 2. Dr. Byers further contested other assertions made by Dr. McCusker. She claimed that Dr. McCusker's references for the fact that the thymus shrinks in the presence of infection are outdated. Also, like Dr. Levin, she cited to the Ricci article, P Ex 22, as proof that an infant's thymus grows when suffering a chronic infection. Dr. Byers also discussed the role of fever in cytokine storm, saying it would be highly unlikely that Kaleaf did not have a fever. P Ex 17 at 2. Regardless, Dr. Byers noted a fever is not necessary to cytokine activation. Id. Also at this point, Dr. Byers arguably broadened her theory of causation in this case to include cytokine release due to the other vaccines Kaleaf received and

[32] SHERRIS MEDICAL MICROBIOLOGY at 152-53 (Kenneth J. Ryan, MD, & C. George Ray, MD, eds., 5th ed. 2010).

the aluminum contained in those vaccines. P Ex 17 at 3. In conclusion, Dr. Byers discussed hepatitis B infection and the body's immune-mediated damage in association with it to again refute Dr. McCusker's statement that this infection does not cause systemic cytokine storm. P Ex 17 at 3-4. Although Dr. Byers appears to be correct in citing literature that some tissue damage associated with the infection is caused by cytokines, she does not point to any information that says an acute, systemic cytokine response or a cytokine storm is responsible for the damage associated with the wild hepatitis B infection.

Dr. Byers' Hearing Testimony

Testimony was taken from Dr. Byers at the November 2010 Hearing. The doctor started by discussing Kaleaf's medical history leading up to his death. Hr'g Tr. 33:16-34:9. She described Kaleaf's prematurity, noting that "the premature birth should be considered because it gave him a relative immunodeficiency disorder which is well documented in all of the literature as a side effect of premature birth." Hr'g Tr. 33:23-34:2. Dr. Byers did not provide support from Kaleaf's medical records that he was immunodeficient and, in the next sentence, Dr. Byers stated that he had an "uneventful neonatal and pediatric follow up over approximately the next three months . . . ." Hr'g Tr. 34:3-4. Regarding Kaleaf in the two weeks prior to his death, Dr. Byers held the opinion that he suffered from a stuffy nose and tachypnea, which is rapid breathing, and an early sign of viral pneumonia. Hr'g Tr. 34:19-23; but see P Ex 3 at 1 (failing to note rapid breathing or lung congestion); Hr'g Tr.73:25-74:17 (admitting there was no evidence, other than the report following death, that Kaleaf was suffering from rapid breathing or tachypnea); Hr'g Tr. 74:25-75:15 (likening what, in her opinion, Kaleaf suffered to "walking pneumonia."); Hr'g Tr. 79:3-80:3 (opining that the child had a very mild pneumonia from the note of rapid breathing in the post-mortem interview and the treating physician's decision to give vaccinations on the day Kaleaf died).

At this point, Dr. Byers illustrated the behavior and function of cytokines and macrophages in a person's immune system, ultimately leading to her description of lymphocytic infiltrates due to ongoing infection and inflammation. Hr'g Tr. 37:25-41:24. The doctor discussed immune responses to vaccination generally. Hr'g Tr. 41:25-43:12. Specifically referencing petitioner's Exhibit 13, a report from the Institute of Medicine, Dr. Byers pointed to what are, in theory, aberrant responses to vaccination. Hr'g Tr. 43:13-45:17. Dr. Byers noted that these theoretical mechanisms are those associated with autoimmune diseases,[33] Hr'g Tr. 44:5-10, which Dr. Byers admitted are not involved in this case. Hr'g Tr. 485:19-486:7; see infra pp. 18, 37. As discussed with Dr. Byers, this case involves something akin to an infectious disease condition, or a robust or overactive immune response. Hr'g Tr. 485:19-486:7. The undersigned also notes there was no allegation that Kaleaf suffered a demyelinating or neurological injury.[34] Dr. Byers also singled out the discussion of the hepatitis B vaccination,

[33] Demyelination refers to the "destruction, removal, or loss of the myelin sheath of a nerve or nerves." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 496 (31st ed. 2007). An autoimmune disease is "a disorder caused by an immune response directed against" autoantigens, id. at 536, which are antigens "that despite being [normal tissue, are] the target of a humoral or cell-mediated immune response." Id. at 182. Guillain-Barré Syndrome and transverse myelitis are autoimmune, demyelinating disorders the undersigned has heard testimony on in many vaccine cases.

[34] A neurological injury is one that involves the nervous system. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY at 1285 (31st ed. 2007).

which Kaleaf received on the day of his death, and its potential association with the mechanisms discussed in the article relating to autoimmunity: molecular mimicry, bystander activation and superantigen. Hr'g Tr. 44:5-19. Specifically regarding the superantigen theory, Dr. Byers explained this theory regards an antigen that has the potential to trigger large immune response as opposed to antigens that typically trigger very specific, discrete responses. Hr'g Tr. 45:2-24 ("It is **conceivable** that antigen stimulation from vaccines generally and hepatitis B vaccine in particular could trigger any of these potentially damaging mechanisms.")(emphasis added).

Dr. Byers next moved on to give examples where immune system activation causes such an aberrant effect to the detriment of the individual. Hr'g Tr. 45:25-48:4 (discussing tampon use and toxic shock syndrome, SARS,[35] multiple sclerosis, and ulcerative colitis). This was in response to being asked for examples of what were "aberrant responses . . . from super-antigens?" Hr'g Tr. 46:1-2. However, Dr. Byers later gave testimony that somewhat contradicted use of these examples as aberrant responses to superantigens. Hr'g Tr. 103:3-104:1. When asked if these disorders required exposure to a superantigen, Dr. Byers responded, "[n]o, and in fact in the case of many of these autoimmune diseases we really don't know what the trigger was." Hr'g Tr. 103:22-24.

Concerning Kaleaf, Dr. Byers theorized that he had an aberrant response to his vaccinations. One important factor, which Dr. Byers stressed affected Kaleaf, was a compromised immune system that "can" be present in premature infants. Hr'g Tr. 68:11-72:5 (citing P Ex 26).[36] "This is especially true for premature babies who began with lower levels of maternal IGG and also reach immune competence later after birth." Hr'g Tr. 71 (citing P Ex 26). Although there is no mention of Kaleaf being immune-compromised in his medical records, Dr. Byers utilized this assumption in her theory of the case. Notably, Dr. Byers asserted Kaleaf suffered from a compromised immune system and due to this, he could not have "cleared" the pneumonia that she and Dr. Levin speculate. However, in contradiction to the assumption that Kaleaf was suffering an immune deficiency, the undersigned notes that petitioners' theory of a cytokine storm or burst actually involves a robust and detrimental immune response to the vaccinations he received. Petitioners appeared to rely upon a deficient immune system to support the portion of their theory regarding the low-grade, chronic lung infection, but then assert an overactive immune system to support their theory of the cytokine storm or burst. This apparent contradiction was highlighted critically in testimony from Dr. McCusker. See infra pp. 29-30.

During cross-examination, Dr. Byers was asked what the most important factor in the chain of events of her theory. Hr'g Tr. 67:12-13. She responded that the "most important factor in this case was the fact that the child had a low-grade viral pneumonia when he received the vaccination." Hr'g Tr. 67:24-68:1. She went on further to state that, "[i]f the child did not have a low-grade viral pneumonia, it would have much lessened the chance that he would have an adverse reaction to the vaccines." She conceded that the only evidence, besides Dr. Levin's review of the slides, of the viral pneumonia is a mention of a rapid breathing problem in the post-mortem report. Hr'g Tr. 82:9-83:9. She noted that "if we were to assume that there is no

[35] SARS stands for severe acute respiratory syndrome, "an infectious respiratory illness caused by a coronavirus and characterized by fever, dry cough, and breathing difficulties, often accompanied by headache and body aches." DORLAND'S ILLUSTRATED MEDICAL DICTIONARY, 1871 (31st ed. 2007).

[36] Charles Janeway, IMMUNOBIOLOGY at 526, "In some infants, this can lead to a period of heightened susceptibility to infection."). The undersigned is unclear which edition of the Janeway text petitioners submitted.

evidence Kaleaf was suffering from tachypnea," this would not affect her opinion very much as she is relying heavily on Dr. Levin's review of the pathology slides for evidence of a chronic lung infection. Hr'g Tr. 81:7-12. The critical point to note is that Dr. Byers testified she could not give a supportive opinion of causation if it was found that Kaleaf did not suffer from the low-grade viral pneumonia. Hr'g Tr. 67:12-68:1. As noted by respondent's counsel and admitted by Dr. Byers, the only note regarding rapid breathing in Kaleaf's medical records was in the post-mortem report. Hr'g Tr. 73:25-77:3. When her attention was drawn to the exam performed on the day Kaleaf passed away, Dr. Byers admitted that the notation indicates a normal chest exam but she also says there is no "box" for a physician to note rapid breathing or tachypnea. Hr'g Tr. 77:4-78:2. Although there is no indication of pneumonia or problems seen on the chest exam, Dr. Byers continued to assert Kaleaf had low-grade pneumonia but that the physician felt he was "well enough to get a vaccine." Hr'g Tr. 78:3-80:6.

Finally, Dr. Byers reviewed the evidence for indications of a local or systemic response to vaccinations: fever, malaise, and erythema. Hr'g Tr. 85:9-15. Admittedly, Dr. Byers noted that there was no evidence of these symptoms of a reaction. Hr'g Tr. 85:18-88:5.

On cross-examination, building from petitioners' theory that the vaccinations given in one's legs could trigger an overwhelming immune response targeting a distant organ, respondent's counsel presented a hypothetical question to Dr. Byers. "Assume that I'm walking on the beach and I step on a piece of glass, and I have an infection in my foot, and I already have a preexisting cold. . . . Would you expect more nasal running?" Hr'g Tr. 89:5-11. To this, Dr. Byers responded that she would not be surprised if that happened but ultimately admitted she was unsure. Hr'g Tr. 89:12-90:2. This hypothetical was discussed with respondent's expert and was utilized to show the relative compartmentalization that occurs with immune responses and why it is unlikely a vaccination in a child's thigh could cause an overwhelming response in the his lungs. See infra pp. 27, 38.

Petitioners' Exhibit 12, the Suntharalingam article, was reviewed by the expert; again, petitioners used this article to demonstrate the effects and timing of a cytokine storm in the setting of a clinical trial. Hr'g Tr. 48:5-49:17. This article was filed by both parties at petitioners' Exhibit 12 and respondent's Exhibit F-9. On cross, regarding the timing of the alleged injury and referencing this article, P Ex 12, Dr. Byers admitted that the article did not support lung involvement from a cytokine storm or burst in less than five hours. Hr'g Tr. 90:3-91:17. Further, she acknowledged that there is no indication between the time of vaccination and death that Kaleaf was suffering from a cytokine storm or burst. Hr'g Tr. 91:18-92:1. Regarding the theory that the hepatitis B vaccination is a superantigen, activating a detrimental immune response, Dr. Byers admitted that the article she relied upon ultimately concluded that "there is no evidence that the hepatitis B surface antigen is capable of bystander activation . . ., is a superantigen, is a molecular mimic . . ., or otherwise induces nonspecific polyclonal activation." Hr'g Tr. 92:10-95:18 (quoting R Ex K at 44.). See also Hr'g Tr. 96:2-97:14 (discussing with the expert R Ex L at 4, which provides a table of organisms that are associated with confirmed or putative superantigen production, which does not list the hepatitis B vaccination or any of its components).

During her testimony at the November 11-12, 2010 Hearing, Dr. Byers backed away from or broadened her previous use of the phrase "cytokine storm" and characterized the event resulting from vaccination as a "cytokine burst;" she explained this was a difference in scale.

Hr'g Tr. 104:19-108:14. "[I]t's really a whole range from the very extreme example shown here[, in the NEJM article, P Ex 12,] to just a burst of cytokines which you get with vaccinations." Hr'g Tr. 48:19-49:5. She summed up the sequence of events in petitioner's theory of causation as follows:

> The child had a low-grade viral pneumonia. The child received vaccinations. The vaccinations then activated both the cells of the innate immune system as well as the T-cells that had been primed by the prior vaccinations, producing a flood of cytokines throughout the body[. This] then activated lymphocytes causing them to start moving, ultimately caus[ing] them to target the already inflamed lungs, which then produced an increased lymphocytic infiltrate as well as increased macrophages, but primarily lymphocytes, then produced cytokines, additional cytokines then affecting the vasculature and resulting in hypotension[. But this] also resulted in an increase in the pneumonia, producing an increased peripheral resistance so that the right heart failed and there was just a massive organ failure because of both the right heart failure, which then resulted in congested organs behind it[,] as well as hypotension and basically a loss of consciousness and ultimately death.

Hr'g Tr. 66:18-67:11. As stated previously, Dr. Byers testified she could not give an opinion supportive of causation if it was found that Kaleaf did not suffer from the low-grade viral pneumonia. Hr'g Tr.109:16-109:23; Hr'g Tr. 67:12-68:1 (noting that her opinion relied heavily upon Dr. Levin's testimony).

Petitioners' counsel called Dr. Byers to testify again following testimony from respondent's experts. Hr'g Tr. 482:13-488:6. Dr. Byers explained that macrophages, cells of the innate immune system, circulate through the body systemically and in Kaleaf's case were drawn to the alleged tissue damage and viral infection in his lungs. Petitioners' counsel and Dr. Byers used autoimmune, demyelinating diseases[37] as an example and how these conditions affect parts of the body that are distant to where the initial insult occurred. Hr'g Tr. 483:6-485:12. Of note though, petitioners' expert again did not discuss how one could liken the target of autoimmune, demyelinating disease, which is not at issue here, to the target of an infectious or inflammatory process. Again, Dr. Byers admitted this case does not deal with an autoimmune disease. Hr'g Tr. 485:19-486:7. To support her theory that a distant vaccination in the thigh could trigger an immune response in the lungs, Dr. Byers referenced petitioners' Exhibit 12, the Suntharalingam article, which showed the cytokine storm in the study patients eventually affected their respiratory system. Hr'g Tr. 486:8-488:2. However, after cross-examination on this article, there appears to be little left in support of petitioners' case. Supra p. 17. Although Dr. Byers' opinions were somewhat less confusing and more coherent than Dr. Levin's, the undersigned did not find the opinions persuasive in light of testimony from Dr. McCusker and the submitted medical literature.

[37] Dr. Byers has testified before in many cases concerning autoimmune, demyelinating diseases. Specifically, she has testified to the time frame in which an autoimmune disease manifests clinical signs. She has stated that it takes multiple days for signs to manifest. See, e.g., Doe/64 v. Sec'y of the Dept. of Health & Human Servs., 2010 WL 1783539 (Fed. Cl. Spec. Mstr. 2010)(finding the appropriate time from vaccine to onset of the demyelinating disorder was 3-30 days after vaccination based upon testimony from Dr. Byers). Even if we were discussing an autoimmune, demyelinating disease, which we are not, the time frame between vaccination and Kaleaf's death, less than three hours, is far too short for such an occurrence.

RESPONDENT'S EXPERTS

Dr. Enid Gilbert-Barness, MD

Dr. Gilbert-Barness' CV is found at respondent's Exhibit B, filed February 25, 2010. Her CV includes numerous references to pediatric pathology and SIDS. During the Hearing, Dr. Gilbert-Barness testified that at the time of her testimony in another Vaccine Act case, she had written twenty-three papers on SIDS; that number has since expanded and does not include chapters in textbooks and her own books. Hr'g Tr. 213:19-214:15. She has also written an entire book on pediatric autopsy pathology. Hr'g Tr. 214:6-215:1. She is a Professor of Pathology and Cell Biology, and a Professor of Obstetrics and Gynecology at the University of South Florida. R Ex B at 1. She is also Professor Emeritus in Pathology and Laboratory Medicine and Distinguished Medical Alumni Professor Emeritus at the University of Wisconsin. Id. Dr. Gilbert-Barness received the equivalent of an MD at the University of Sidney, Australia. Id. She is board certified in pediatrics, clinical pathology, anatomic pathology, and pediatric pathology. Id. at 3-4. She served on the examining board for pediatric pathology for a number of years and was involved in instituting board certification for pediatric pathology. Hr'g Tr. 216:17-25.

Dr. Gilbert-Barness estimates that she has performed 10,000 pediatric autopsies; she has seen cases of SIDS in autopsy frequently and also estimates 10-15% of the pediatric autopsies she has performed involved pneumonia and/or pneumonitis. Hr'g Tr. 216:215:15-216:8. In her CV, Dr. Gilbert-Barness lists pediatric pathology and Sudden Infant Death among her major research interests. Id. at 2; Hr'g Tr. 215:2-8. Relevant to the case at hand, she has also received a citation by the Governor of Wisconsin for "outstanding contributions to Sudden Infant Death Syndrome in Wisconsin, 1982-85." Id. at 4. Dr. Gilbert-Barness testified in another vaccine case wherein it was discussed that she served on a National Institutes of Health panel tasked with studying SIDS. Doe/11 v. Sec'y of the Dept. of Health & Human Servs., 83 Fed. Cl. 157, 164 (Fed. Cl. 2008), aff'd 601 F.3d 1349 (Fed. Cir. 2010), cert. denied 131 S.Ct. 573 (2010). She was accepted as an expert in the area of pediatric pathology at the Hearing. Hr'g Tr. 217:16-22. Dr. Gilbert-Barness' experience is highly relevant to the issues at hand; also, the undersigned finds her experience and knowledge in her areas of expertise impressive. Dr. Gilbert-Barness' experience with pediatric pathology simply dwarfed and overwhelmed Dr. Levin's knowledge and experience with the relevant matters. See supra pp. 6-7. The substance of her testimony exhibited that professional dominance.

Dr. Gilbert-Barness' first report is respondent's Exhibit A. In it, she briefly discussed Kaleaf's history and autopsy. R Ex A at 1. She noted no abnormalities being found at autopsy except for congestion and hyperplasia of the thymus. Id. She also noted there was no indication of anaphylaxis. Id. Notably, she pointed out that Kaleaf's position, face down, when he was discovered unresponsive. Id. "The organ weights are slightly increased . . . ." Id. at 1-2 (referencing R Ex C, Enid Gilbert-Barness & Diane E Debich-Spicer, Handbook of Pediatric Autopsy Pathology at 57 (Humana Press, 1st ed. 2004)). Further, "[t]hese are values that would be seen and are consistent with congestion which is evident in the microscopic sections I examined and entirely consistent with asphyxiation in addition to hyperaeration of the lungs." R Ex A at 2. She found no evidence of preexisting viral pneumonitis. Id. Dr. Gilbert-Barness agreed to some extent with Dr. Levin's statement that the lungs, liver and spleen were heavy, which can be consistent with right heart failure; however, in contrast to Dr. Levin's opinion, she

noted this heaviness is "always seen IN ASPHYXIATION WITH POOLING OF BLOOD IN THE ORGANS." Id. (emphasis in original). Regarding the brain edema, Dr. Gilbert-Barness found it was minimal and consistent with respondent's theory of asphyxiation; "absence of inflammation in the brain excludes infection." Id.

Dr. Gilbert-Barness went on to explain that the term SIDS in coroner's reports is frequently used when there is asphyxiation and that the major risk factor for SIDS is sleeping in the prone position. R Ex A at 2. She commented that Dr. Levin's conclusion was purely speculative and noted that she "did not see massive thickening of the alveolar membranes causing pulmonary vascular resistance and right heart failure hypoxia death." Id. Dr. Gilbert-Barness held the opinion that Kaleaf suffered "an asphyxial death due to the prone position" he slept in and being further at risk due to his prematurity.

In her supplemental report, Dr. Gilbert-Barness confronted the criticisms Dr. Levin levied against her report. R Ex G, filed Oct. 29, 2010. She disagreed with Dr. Levin's contention regarding any evidence of acute infection or viral pneumonitis and describes her findings on the autopsy slides of the lungs, thymus, spleen, liver, and brain. Id. She found the "ischemic neuronal changes" to be minimal and only mild edema in the brain; evidence of an "acute global hypoxic encephalopathy" was not present. Id. Finally, Dr. Gilbert-Barness stated that there were no post-mortem findings that Kaleaf had a fever at the time of death and it is not possible to "speculate or conclude the presence of fever based on pathology." Id. This was reiterated during her testimony; when asked if one could ascertain if Kaleaf had a fever from the post-mortem findings, Dr. Gilbert-Barness responded in the negative. Hr'g Tr.242:19-243243:17.

Dr. Gilbert-Barness' Hearing Testimony

Consistent with her reports, Dr. Gilbert-Barness testified that Kaleaf's death was "a classical example of positional asphyxia, which in the past has frequently been referred to as SIDS." Hr'g Tr. 218:24-219:11. She opined that the process of asphyxiation could have occurred "within minutes or even less than that." Hr'g Tr. 221:21-222:2; see also Hr'g Tr. 244:5-21. Explaining further, Dr. Gilbert-Barness stated "an asphyxial death is a death that occurs due to obstruction of the airway, and that causes usually an immediate response and the baby or person may die from asphyxial death or lack of oxygen to the brain in particular." Hr'g Tr. 219:15-19. Regarding how this is associated to SIDS, Dr. Gilbert-Barness explained:

> Well, SIDS has been a term that's been used for many, many years now. It's a term that really is meaningless. Some years ago I was instrumental in supporting the Back to Sleep Campaign, which resulted in a reduction of the so-called SIDS deaths by 70 to 80 percent. SIDS is a term that I do not use anymore because most of them now have turned out to be an asphyxial death due to the position of the infant in the prone position, and now that we've changed that to the supine position the incidence of so-called SIDS has been reduced by, as I mentioned, 70 to 80 percent.

Hr'g Tr. 219:20-220:10.

In contrast to the opinion of Dr. Levin, Dr. Gilbert-Barness found "no evidence whatsoever in the sections of the lung that would indicate that this baby had interstitial

pneumonitis." Hr'g Tr. 222:9-14. Dr. Gilbert-Barness opined in the negative when asked if she found evidence that the baby was suffering from a low-grade infection. Hr'g Tr. 222:15-223:6. "The baby was described as having a stuffy nose, and there might have been what we usually call a cold, a mild upper respiratory infection . . . ." Hr'g Tr. 222:19-22. Dr. Gilbert-Barness noted that this was significant in that babies are obligate nasal breathers in the first six months of life; "In other words, the nose passages must be patent for the baby to be able to breathe." Hr'g Tr. 222:22-25. "In the prone position where the face is buried either in a soft pillow or the bed, and there is stuffiness of the nose, the baby cannot breathe normally, and I think that in this case it might have been a factor that certainly promoted or aggravated the situation which lead to an asphyxial dealth." Hr'g Tr. 223:1-6.

Dr. Gilbert-Barness also discussed how Kaleaf's prematurity may have been a risk factor for SIDS but noted that he had reached normal for his age. Regarding Kaleaf's organ weights, Dr. Gilbert-Barness noted they were slightly increased, "a little on the high side," which was due to congestion of pooled blood in her opinion. Hr'g Tr. 223:24-224:12. In contrast to Dr. Levin's opinion, Dr. Gilbert-Barness noted that this finding is "absolutely consistent" with an asphyxial death. Hr'g Tr. 224:5-12. Dr. Gilbert-Barness disagrees with Dr. Levin that the organs were massively heavy due to lymphocytic infiltrates. Hr'g Tr. 224:13-225:11.[38]

Regarding Kaleaf's thymus, Dr. Gilbert-Barness pointed out that it was more than moderately increased in weight. Hr'g Tr. 225:13-25. She did note that variation in thymus weight is great and commented that the organ "should be several times the average weight" to consider the organ to fall under the classification of thymic hyperplasia. Hr'g Tr. 225:16-25. Dr. Gilbert-Barness found Kaleaf's thymus, which weighed 52 grams, to be greater than average but of no real significance. Hr'g Tr. 226:5-8. Further, when asked what happens to thymus size when an infant is under stress, Dr. Gilbert-Barness stated "[t]he thymus decreases in size, and that is absolutely unequivocal." Hr'g Tr. 226:9-14 (citing R Ex H).[39] This was in direct conflict with the opinions of Dr. Levin and Dr. Byers who claim the infant thymus increases when under stress from infection. See supra pp. 9-11, 13-14. Later on in her testimony, Dr. Gilbert-Barness commented on the slides of Kaleaf's thymus and, beyond its organ weight, she stated she found no other abnormality. Hr'g Tr.237:2-238:8.

Dr. Gilbert-Barness walked the hearing participants through her interpretation of the pathology slides. Notably, Dr. Gilbert-Barness found Kaleaf's upper lung slides to be normal; however, she observed congestion in the lower lung slide, which she said is indicative of an asphyxial death. Hr'g Tr. 229:2-232:20. "I think the bottom line is that the lungs are essentially really showing normal pattern except in the lower lung that does show some congestion, and it certainly doesn't look like interstitial pneumonitis . . . ." Hr'g Tr.232:25-233:4. Dr. Gilbert-Barness also strongly disagreed with Dr. Levin's opinion that there was lymphocytic infiltrate in the lower lung; she explained that you would see white cells with congestion, since they are part

[38] There was a great deal of discussion at the hearing regarding how pathology tables determine what normal organ weight is and how abnormal weight is determined if the normal weights have great variance. See, e.g., Hr'g Tr. 255:16-257:15; Hr'g Tr. 354:19-356:4; Hr'g Tr. 361:12-367:2; Hr'g Tr. 375:23-382:25 Ultimately, in the opinion of Dr. Gilbert-Barness, the weights in this case were not so far outside the range of normal to be considered abnormal. Hr'g Tr. 255:16-257:15. "Double the weight" found to be normal in pathology tables is what Dr. Gilbert-Barness would find to be abnormal, which was not the case herein. Hr'g Tr. 382:13-25.

[39] Enid Gilbert-Barness, HANDBOOK OF PEDIATRIC AUTOPSY PATHOLOGY (2005).

of the blood, but that there were very few shown. Hr'g Tr.233:15-234:5; see also Hr'g Tr. 253:6-17. In responding to a question regarding whether she saw thickened alveolar walls, Dr. Gilbert-Barness noted some slight thickening but it was minimal and that it "certainly is not a cause of death . . . ." Hr'g Tr.235:1-236:1; see also Hr'g Tr. 252:6-253:3. On re-direct, Dr. Gilbert-Barness testified that the few instances of thickening that she saw were not indicative of massive inflammation. Hr'g Tr. 254:17-255:12.

Dr. Gilbert-Barness acknowledged the slides of the spleen were congested, consistent with asphyxiation, but otherwise unremarkable; she did note that the spleen was a lymph organ and thus full of lymphocytes, which is an insignificant finding. Hr'g Tr. 238:13-240:12. Dr. Gilbert-Barness also stated she had not heard of the phrase "density of lymphocytes" utilized when speaking of the spleen or the thymus, which was used by Dr. Levin. Hr'g Tr. 240:6-12.

Regarding the slides of Kaleaf's brain, Dr. Gilbert-Barness observed very minimal edema and that it was "probably insignificant" in this case. Hr'g Tr. 240:24-241:8. Bolstering her observation, Dr. Gilbert-Barness testified to what edema in an encephalopathic brain would look like, citing R Ex G-1, page 5, figure 4c, and estimated that she likely sees one encephalopathic infant brain per week. Hr'g Tr. 241:9-242:4.

On cross-examination, petitioners' counsel questioned Dr. Gilbert-Barness regarding the congestion and weight of Kaleaf's organs post-mortem. Hr'g Tr. 244:22-249:17. Dr. Gilbert-Barness explained that the weights were only slightly more than what one would expect but that this is consistent with an asphyxial death. Id. Further, regarding the mild brain edema, Dr. Gilbert-Barness stated this is expected in a SIDS death. Hr'g Tr. 250:9-18 (citing Valdes-Dapena, et al., Histopathology Atlas for the Sudden Infant Death Syndrome (1993)).[40]

When recalled to continue her testimony, Dr. Gilbert-Barness addressed what signs one would see with right heart failure, petitioners' theory. "First of all, the right side of the heart would be significantly dilated. The lungs would be congested, as in part they were, but much more so in right heart failure, and the liver would be congested, as it was but the overall appearance was not one of right heart failure." Hr'g Tr. 370:25-371:5. "There's just no mistaking right heart failure. There would be edema in the lungs, which was very minimal, and it just was not right heart failure." Hr'g Tr. 371:6-9. Further, "the heart [weight] may be increased, but significantly the right ventricle would be dilated." Hr'g Tr. 371:10-17. Dr. Gilbert-Barness testified that there was no evidence of this from the post-mortem findings. Hr'g Tr. 371:16-372:2.

Regarding the weight increases in many of Kaleaf's organs, Dr. Gilbert-Barness further testified that the fluids administered during the attempted resuscitation efforts would have contributed to the increased organ weights. Hr'g Tr. 372:3-12. Also, Kaleaf's size, in the 97th percentile for his age at his time of death, would also correlate with organ weight. Hr'g Tr. 372:13-23. With the findings in an asphyxial death, "the intensity of the condition is much greater than what one would normally see in any other sort of death. That I think in this case is exemplified by the increase of weight of several of the organs, and that I can document from my

[40] Although a page of this article is filed as R Ex O-2, this does not appear to be the page Dr. Gilbert-Barness was referring to regarding brain edema. Following the Hearing, additional pages from the Valdes-Dapena article were filed but these do not appear to discuss brain edema in SIDS cases. See R Ex O, filed Dec. 23, 2010.

own text, the Handbook of Pediatric Autopsy." Hr'g Tr. 374:19-25 (discussing R Ex H).[41] "On page 485, there is a description of asphyxia, and very clearly it's stated that there is intense venous congestion of the organs, which certainly would account for the mild increase of the weights of the organs." Hr'g Tr. 375:1-6.

Dr. Christine McCusker, MD

Dr. McCusker's CV is found at respondent's Exhibit E, filed February 25, 2010. She received her MD from McMaster University Medical School and is certified by the Royal College of Physicians and Surgeons in Canada[42] in general pediatrics and allergy and immunology; she is also board certified by the American Board of Pediatrics. R Ex E at 2-3; Hr'g Tr. 388:9-389:22. Dr. McCusker also is an examiner for the Royal College board for allergy and clinical immunology. Hr'g Tr. 389:20-22. She is presently the Director of the Clinical Immunology Laboratory at Montreal's Children's Hospital, as well as a Staff Physician in the Emergency Department there. Id. at 2-3. Dr. McCusker is also an associate professor in the Division of Allergy and Immunology at McGill University and Montreal Children's Hospital and Research Director of Meakins-Christie Laboratories at McGill University. Id. at 3-4.

Dr. McCusker estimated her time is split, 50/50, between clinical practice and research. Hr'g Tr. 389:23-390:2. Regarding her clinical practice with children, she sees children in her allergy clinic and in the immunodeficiency clinic at the hospital; she also sees children under allergy and immunology that are admitted while she is on service, primarily children with immune deficiencies; additionally, she works as a pediatrician at a local, semi-urgent clinic with walk-in patients and two to three times a month in the emergency department at Montreal's Children's Hospital. Hr'g Tr.390:9-23. Her primary research interest is in "understanding the development of the immune system from infancy through to obviously young adulthood." Hr'g Tr. 390:24-392:2. Dr. McCusker was accepted as an expert in pediatrics and pediatric immunology for the purposes of this case. Hr'g Tr. 392:24-394:6.

Dr. McCusker's initial report in this case is respondent's Exhibit D1.[43] Following a review of the case, Dr. McCusker explained the role of cytokines in an immune response, as well as the cytokine storm. R Ex D1 at 2-4. Cytokines, tightly regulated proteins responsible for one's immune response, may become imbalanced under specific circumstances, leading to uncontrolled inflammation and tissue damage: the cytokine storm. Id. at 2-3. She explained that infection from hepatitis viruses are not associated with systemic cytokine storm and that cytokine storm causing brain edema has only been reported when there is direct infection in the brain or surrounding tissue. Id. at 3 (citing R Ex F1[44]; R Ex F7[45]). Further, Dr. McCusker discussed the

[41] Enid Gilbert-Barness and Diane E. Debich-Spicer, HANDBOOK OF PEDIATRIC AUTOPSY PATHOLOGY (2004). Page 485 of Dr. Gilbert-Barness' textbook was submitted at respondent Exhibit O at page 7 ("The postmortem appearance of an individual who has died from asphyxia typically shows intense venous congestion . . . .").

[42] This is analogous to board certification in the United States. Hr'g Tr. 389:17-22.

[43] The report was initially filed as respondent's Ex D on February 25, 2010. It was filed again as Ex D1 on March 22, 2010, due to a typographical error in the original.

[44] Mathis Heydtmann, et al., Cytokines and chemokines in the immune response to hepatitis C infection, 14 CURR OPIN INFECT DIS 279-87 (2001).

"template for the symptoms of cytokine storm" being: "fever, headache, skin erythema followed by lung and other organ involvement." Id. at 4. She noted that these symptoms, or their observable signs, were not found in Kaleaf. Id.

Given the cardiopulmonary resuscitation attempts with Kaleaf, Dr. McCusker also opined that CPR subsequent to an acute life threatening event is "sufficient to induce cytokine activation, pulmonary edema and inflammation." R Ex D1 at 5 (citing R Ex F-10; [46] R Ex F-11).[47] Regarding the petitioners' theory that the hepatitis B vaccination is capable of producing a harmful immune response in the form of cytokine storm or cerebral edema, Dr. McCusker was unable to find any evidence of this relationship or association. Id. She also explained that the thymus is a large organ in infants, one that actually reduces in size due to the stress of infection, immune activation or surgery. Id. at 6 (citing R Ex F-19).[48] From this observation, she stated that Kaleaf's thymus would be expected to be smaller than average if he was suffering from a chronic pneumonitis as suggested by petitioners' experts. Id. Dr. McCusker cited a study that hypothesizes the "substantially greater thymic size" of SIDS infants "'is more representative of the living population than weights of infants dying of other causes who may have been exposed to various thymolitic factors.'" Id. (citing R Ex F-20).[49] Also regarding the thymus and petitioners' experts' opinions, she indicated that "it is impossible to conclude that there was a *de novo* or acute thymic lymphocytic infiltration" since the thymus is already an important location for development and maturation of T cells as well as other cells of the immune system . . ." without use of special staining of the autopsy slides. Id.

In her supplemental report, R Ex I, filed October 29, 2010, Dr. McCusker responded to the supplemental reports of Drs. Levin and Byers.[50] Specifically regarding the facts of this case, Dr. McCusker noted that the first responders noted "a strong radial pulse, and indication of adequate CPR, and that his lung sounds were clear bilaterally." R Ex I at 2, 7 (citing P Ex 4 at 2). This responded to Dr. Byers' assertion that CPR could not account for a cytokine response and inflammation. Dr. Byers had argued CPR would not induce systemic inflammatory response in a deceased child. Dr. McCusker noted a study wherein the authors demonstrated cytokine elevation in children following [SIDS] and notes the EMT reported a strong radial pulse Kaleaf, showing that circulation and blood pressure were potentially sufficient to permit pulmonary edema. Id. at 6-7.

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[45] Angela J. Frodsham, Host genetics and the outcome of hepatitis B viral infection, 14 TRANSPLANT IMMUNOLOGY 183-86 (2005).

[46] Shigekiyo Matsumoto, et al., Sivelestat treatment for acute respiratory distress syndrome in an infant, 23 J ANESTH 288 (2009).

[47] C. Caroline Blackwell, et al., Cytokine responses and sudden infant death syndrome: genetic, developmental, and environmental risk factors, 78 JOURNAL OF LEUKOCYTE BIOLOGY 1242 (2005).

[48] Helge Stalsbert, The Thymus in Infants Dead from Acute Disorders, 79A ACTA PATHOLOGICA MICROBIOLOGICA SCANDINAVICA SECTION A PATHOLOGY 37 (1971)(online publication 2009).

[49] JMD Thomspon, et al., Previous breastfeeding does not alter thymic size in infants dying of sudden infant death syndrome, 89 ACTA PAEDIATRICA 112 (2000).

[50] Dr. McCusker utilizes the text IMMUNOBIOLOGY by Charles Janeway, et al., 6th edition (2005), for support unless otherwise noted in her report. R Ex I at 1.

Regarding Dr. Byers' assertions that Kaleaf was suffering from a low grade viral pneumonia, likely due to influenza virus, Dr. McCusker differentiated Kaleaf's illness, as described in the medical records, from what would be seen in a child with pneumonia or an influenza infection. R Ex I at 2-3. Clear sounding lungs on the day of Kaleaf's vaccination are inconsistent with signs of pneumonia. Id. Regarding a possible influenza infection, Dr. McCusker noted that while there was scant rhinorrhea, there was no evidence of lower airway involvement by the pediatrician during the morning office visit or by the EMT during CPR. Id. at 3. She indicated that other signs of influenza, including fever, headache, and cough, were not present. Id. "Finally, influenza infection is self-limited, lasting 3-7 days, and thus would not be the cause of the 2 weeks of congestion noted in the coroner's report." Id.

In response to petitioners' theory of cytokine storm, Dr. McCusker again described the difference in scale between a normal cytokine release and a cytokine storm. R Ex I at 3-4. Citing medical literature, she noted that the symptoms suffered during a cytokine storm are not subtle, with fever, malaise and headache occurring within ninety minutes and respiratory distress occurring five hours after onset of symptoms. Id. at 5 (citing P Ex 12). In cytokine storm patients who developed the condition after infection from "SARS, fever malaise and cough developed 2-10 days after exposure to the virus, while signs of worsening respiratory distress developed later." Id. (citing R Ex J-10).[51] In the case *sub judice*, the infant received vaccinations between 10:00 a.m. and 10:30 a.m. and was pronounced dead at 1:10 p.m., showing no symptoms of a massive cytokine release. Id. at 6.

Finally, regarding Dr. Byers' assertion that hepatitis B virus infected individuals can sustain immune-mediated tissue damage, Dr. McCusker asserted this is a normal-functioning immune response while the body attempts to clear a chronic infection. R Ex I at 6. She stated, "[i]mmune-mediated end organ damage is one effect of chronic infection, as the immune system continues to attempt to eliminate infection. Thus cytokines can be etiologically associated with **local** organ damage at the site of infection, such as seen in chronic hepatitis infection . . . ." Id. (emphasis added). In Dr. McCusker and Dr. Gilbert-Barness' opinions, there was no evidence of chronic infection in Kaleaf. "In the case of Kaleaf . . . Dr. Byers postulates that massive cytokine release at the site of vaccination led to end-organ damage at a site **distant** from the local inflammation, leading to pulmonary failure alone with no other signs of systemic cytokine release." Id. (emphasis added). Further, Dr. McCusker addressed petitioners' claims regarding their theory and the alum adjuvant used in the vaccines. Again, Dr. McCusker noted that the effects of alum are local and noted that "it is unclear how [alum] would connect to an event at a distal site." Id.

Dr. McCusker's Hearing Testimony

Dr. McCusker testified consistently with her written reports that her opinion is that Kaleaf's death was due "to an acute life threatening event, probably due to positional asphyxia as described by Dr. Gilbert-Barness, and not related to a problem or an issue related to the immune response and the vaccine." Hr'g Tr. 395:1-5. The undersigned was impressed with the candor and clarity with which Dr. McCusker testified. It is clear from a reading of the Hearing

[51] D S C Hui, et al., Severe acute respiratory syndrome (SARS): epidemiology and clinical features, 80 POSTGRAD MED J 373-81 (2004).

Transcipt that Dr. McCusker brought much-needed clarity to many issues concerning the immune system and infant patients.[52]

Dr. McCusker then provided a very clear and useful explanation of the immune system. Hr'g Tr. 395:15-417. The immune system has two levels: the innate system, which is a person's first line of defense and includes barriers, such as skin or mucous membranes; and the adaptive system, which creates a memory of immunity and acts when the innate system is not enough to deal with an insult. Id. The innate immune system is comprised of cells, such as cells in your skin, that have "buttons on them, little button that we call toll receptors." Hr'g Tr. 397:7-398:7. Toll receptors are found throughout the body. Hr'g Tr. 398:23-10. When confronted with an insult, such as a vaccine, "these buttons are pushed or a series of buttons are pushed then that sends a signal. The cell is telling the body that something that has touched down on the cell is dangerous, and that's the first sign that something is going on." Hr'g Tr. 398:7-11. From this triggering, a cell may release cytokines. Hr'g Tr. 398:18-21. "Cytokines are actually designed to inform the body that something is going on . . .." Hr'g Tr. 413:5-9.

When discussing the innate immune system, Dr. McCusker discussed cytokine release when an individual receives a vaccination. "[W]hen you talk about cytokine release, the cytokine release is a little bit – you know, it's happening at the site, so at the site of the vaccination there would be signals going out to say 'there is something here we need to deal with.'" Hr'g Tr. 402:7-11. "And so the way it works is it's a gradient. So the cells that are releasing cytokines[,] obviously at the site of cytokine release is where you have the highest amount of cytokine. Then it radiates out in a gradient a little bit like a drop in a pond." Hr'g Tr. 402:13-17. "As you get further and further away from the signal, the amount of cytokine that is released or that is sent is less, so if there's not a lot of cytokine signal then it's only going to call in cells from a certain area and keep the problem somewhat localized." Hr'g Tr. 402: 18-23. This is the local response to vaccination from the innate immune system. Hr'g Tr. 402:24-403:6. It is from the release of cytokines that a person may feel a systemic response, such as fever, malaise, and muscle aches. Hr'g Tr. 415:3-6. Dr. McCusker opined that the systemic response, such as a fever, may be felt in two to four hours after vaccination; that this response was not instantaneous. Hr'g Tr. 415:11-20. Dr. McCusker was clear that in that normally, there is no systemic response felt when one is vaccinated; and even when there is a systemic response, such as fever or malaise, this does not constitute a cytokine storm. Hr'g Tr. 415:21-416:13.

During this local response, as described by the expert, the body's purpose is to contain and shut down the insult. Hr'g Tr. 403:12-19. In the case of vaccination, the particular vaccines received by Kaleaf were all killed vaccinations and were not infectious; however, the body does not realize they are not infectious. Hr'g Tr. 403:20-25. Initial signs of an innate immune response range from no symptoms at all, to a local reaction of tenderness, pain, swelling and redness at the vaccine site; this usually occurs in a couple hours but can happen faster in some individuals. Hr'g Tr. 406:4-407:6. Ultimately, a person gets systemic symptoms of an immune response through release of cytokines of the innate immune system. Hr'g Tr. 412:9-15. While the body is trying to contain and shut down the insult, macrophages and antigen presenting cells are gathering pieces of the vaccine and transporting them to lymph nodes, Hr'g Tr. 404:1-9, which initiates the adaptive immune response. Hr'g Tr. 404:22-405:3.

[52] This was in contrast to the testimony of Drs. Levin and Byers, which consistently obfuscated rather than illuminated issues.

Regarding the adaptive immune response, Dr. McCusker testified that although the adaptive immune system is triggered right away, you would not detect the outcome of the adaptive immune response for seven to ten days, depending on type of adaptive immune cells you are examining. Hr'g Tr. 407:12-408:11. The adaptive immune system takes over if the innate immune system cannot clear the initial insult; it also has a recall response when the person encounters the same insult after that first encounter; and with time, the adaptive immune response to the insult becomes more finely tuned and more powerful, which requires tight regulation so it does not cause damage to the person. Hr'g Tr. 408:12-411:16.

Seemingly in response to the questions asked of Dr. Byers regarding a symptom of immune response – a runny nose – when one gets a cut on the foot, Dr. McCusker testified that the body's goal is to contain and compartmentalize an insult and the body's reaction to the insult. See supra p. 17. "It's no good for you to get symptoms or to have a sore finger when the problem is in your thigh [from a vaccination]. You need to keep it focused so that you don't waste time and energy trying to go madly off in all directions." Hr'g Tr. 404:14-21. Later in her testimony, Dr. McCusker noted that the immune system "compartmentalizes, and while cytokines can stimulate cells relatively nonspecifically, . . . generally these responses are maintained locally." Hr'g Tr. 444:19-445:24. "The immune system compartmentalizes, and for good reason. Otherwise we would be sick all the time." Hr'g Tr. 480:14-15. This discussion is relevant because, in Dr. McCusker's opinion, petitioners' theory that an immune challenge by a vaccination administered in the leg could have devastating effects on a distant location, Kaleaf's lungs, is untenable.

When asked at the close of the Hearing whether her opinion would change if the undersigned found there was the preexisting pneumonitis, Dr. McCusker opined that "there would still be problems [with petitioners' theory] connecting the dots, connecting the vaccine in the leg to the pneumonitis in the lung. . . . There isn't literature that I could find where immune response in the leg can turn up an immune response somewhere else." Hr'g Tr. 480:21-481:8. Even assuming the vaccination was a strong enough stimulus to trigger a distant response in Kaleaf's lungs, Dr. McCusker opined that one would expect to see signs such as swelling, pain, tenderness at that injection site, or irritability or inability to feed; these signs were not witnessed with Kaleaf. Hr'g Tr. 445:25-447:6. "I don't see evidence of active cytokine or cytokine activation to any significant degree in this child." Hr'g Tr. 447:10-12. Dr. McCusker also noted that there has been no evidence that a cytokine storm can happen without symptoms. Hr'g Tr. 447:7-12.

Dr. McCusker summed up her understanding of petitioners' theory of the case as Kaleaf having a "cytokine storm mediated inflammation of the lungs resulting in increase of the vascular resistance on the right heart and right heart failure, . . . that the vaccine in the leg stimulated an immune response in the lungs because [petitioners] felt that there was already evidence of lung infection." Hr'g Tr. 418:7-18. Her understanding of a second theory was that the heart failure was due to "the hepatitis B [vaccine] acting as a super antigen and up-regulating all of the immune responses and was related to the formation of T-cells on the initial [hep B] vaccine that [Kaleaf] received soon after birth." Hr'g Tr. 418:20-24. A third theory part of the theory perceived by Dr. McCusker was that Kaleaf's prematurity made him immunocompromised, which prevented him from clearing the lung infection, allowing the other events to occur. Hr'g Tr. 418:25-419:5.

Regarding petitioners' cytokine storm or burst mechanism, Dr. McCusker testified that she was not aware of the idea of a "cytokine burst." Hr'g Tr. 419:11-17. Dr. McCusker discussed the idea of a cytokine storm, noting that it is called a systemic inflammatory response syndrome with relatively uncontrolled inflammation throughout the body. Hr'g Tr. 420:11-19. The first symptoms of such a response are typically headache, chills and rigors. Hr'g Tr. 420:20-421:1. Thereafter, patients with cytokine storm go on to have vomiting, diarrhea, skin changes, redness of the skin, and fevers. Hr'g Tr. 421:2-4. Dr. McCusker noted that most, if not all, of the patients discussed in the Suntharalingam article, P Ex 12 and R Ex F9, developed most or all of these symptoms. Hr'g Tr. 421:4-7. Thereafter, various end organ problems developed, including pulmonary distress. Hr'g Tr. 421:8-12.

In the present case, Dr. McCusker found no evidence that a cytokine storm occurred in Kaleaf. Hr'g Tr. 422:1-5. Due to a fever and headache, one would expect Kaleaf to be irritable and in distress, possibly shaking – "a very, very, very unhappy child, extremely irritable." Hr'g Tr. 422:7-16. Dr. McCusker said it was unimaginable that a child undergoing such an ordeal would feed; the baby would be "miserable, unhappy, crying and really inconsolable." Hr'g Tr. 422:17-21. Dr. McCusker saw Kaleaf's feeding after returning from the doctor's office to be an important marker that there was not an aberrant immune response occurring. Hr'g Tr. 422:22-423:12. Regarding the Suntharalingam article, Dr. McCusker explained that the antigen used in that study, which triggered the patients' cytokine storm, was something that did not exist naturally and could be described as a "super-super-super antigen," stimulating around 60 percent of the person's T-cells. Hr'g Tr. 437:2-438:4. In comparison, an ordinary superantigen stimulates 10 to 20 percent. Hr'g Tr. 437:19-21. In the study, the patients had this extreme superantigen injected directly into their vein, which then activated the T-cells to release "all the cytokines that they could and activated every cell that they could and still with that there was an hour to the first symptoms and then two hours to [the] next set of symptoms and five hours minimum, in one patient, to symptoms in the lungs." Hr'g Tr. 437:22-438:4. Dr. McCusker describes the cytokine storm effect in this study as an example of the worst case scenario, Hr'g Tr. 440:4-7, and even in this worst case scenario, respiratory failure with tachypnea started in 300 minutes and longer. Hr'g Tr. 440:5-441:16.

Dr. McCusker also offered her opinion on whether vaccination could cause cytokine storm. Following her review of medical literature, Dr. McCusker could not find any evidence that vaccination could cause hyper activation or hyper release of cytokines, the cytokine storm. Hr'g Tr. 444:7-18.

Regarding whether Kaleaf had a preexisting lung infection, pneumonitis, Dr. McCusker's opinion was that the baby had a runny nose and a normal chest exam, as evidenced by the pediatrician's note on the day the vaccines were given. Hr'g Tr. 423:20-426:17. In response to Dr. Byers' assertion that there was no place for the pediatrician to denote rapid breathing or tachypnea, Dr. McCusker's opinion was that it would be malpractice if a pediatrician did not find a place to make such a note. Hr'g Tr. 426:18-25. Dr. McCusker found nothing in the pediatrician's notes that would indicate the child was suffering from pneumonia or pneumonitis. Hr'g Tr. 427:12-431:17. Responding to a supposition made by Dr. Byers that Kaleaf was suffering from an influenza infection, Dr. McCusker notes that there was nothing in the medical records to indicate Kaleaf was suffering from an influenza infection. Hr'g Tr. 431:19-432:23 (noting the symptoms of influenza infection to be fever, cough, malaise, chills, rigors, headache, and myalgias). Further, Dr. McCusker indicates, in her interpretation of the pediatrician's notes

and Kaleaf's weight gain, that he was feeding well leading up to that last visit; she testified that a refusal or inability to eat is common, observable sign of illness in infants. Hr'g Tr. 427:1-11; Hr'g Tr. 434.

Dr. McCusker testified that tachypnea would be taken very seriously when found in an infant, "because it's generally a sign that something is going on." Hr'g Tr. 433:19-434:1. This could be due to an infection, a cardiac problem or a very blocked, stuffy nose. Hr'g Tr. 433:2-23. "[P]art of the reason why it would be taken quite seriously is a baby had tachypnea would be that the baby can't feed." Dr. McCusker explained, "in order to feed properly you have to be able to generate suction, and in order to generate suction you actually have to be able to hold your breath for a bit, then you suck, then you breathe through your nose, then you hold your breath, you suck, you breathe through your nose." Hr'g Tr. 434:8-13. "[A]ll of that has to occur, and babies with tachypnea often end up in our emergency room because they can't feed. And that's a sign the parents note . . . ." Hr'g Tr. 434:14-18. Dr. McCusker found no evidence that Kaleaf suffered from tachypnea in the weeks leading to his death and further noted that he had continued to gain weight. Hr'g Tr. 434:19-23. In fact, Dr. McCusker noted that Kaleaf had risen from the 5th and 10th percentile in size, to the 95th percentile in just three months and twenty-four days. Hr'g Tr. 435:3-436:12.

Dr. McCusker responded to Drs. Levin and Byers' opinions regarding Kaleaf's immune system status considering his prematurity. First, Dr. McCusker argues that adjusting Kaleaf's age at the time of his death, he was functionally equivalent to a six week old baby and not a premature baby at the time of his death. Hr'g Tr. 447:25-449:1. Regarding premature infants' adaptive immune responses, Dr. McCusker discussed and cited literature showing the adaptive immune system was changed but that its function was still very good; thus, demonstrating her point further, the recommendation of vaccine schedule does not need to be changed for premature infants. Hr'g Tr. 450:24-452:17. Regarding Kaleaf's supposed lung infection, his adaptive immune would have been adequate to clear an infection in seven to ten days if his innate immune response was not able to do so. Hr'g Tr. 452:23-454:4.

Dr. McCusker reviewed literature regarding a premature infants' innate immune system. "[I]n fact the area where premature babies tend not to have as robust an immune system is in the innate area." Hr'g Tr. 454:17-19. Referencing literature, Dr. McCusker pointed out that premature babies could potentially have defects in their innate immune system, specifically in their ability to signal through toll receptions. Hr'g Tr. 454:20-455:1 (citing R Ex N).[53] She explained that the cited paper, R Ex N, examined "the innate immunity patterns, the patterns of toll receptors, how well they could signal, how well they could secrete cytokines and how well they recognized – how well their toll receptors worked and found that in the premature infant their toll receptor signaling was impaired." Hr'g Tr. 455:11-19. This impairment caused poor signaling within the cell, which affects cytokine secretion. Hr'g Tr. 455:20-456:8. "[T]herefore essentially, the problem with preterm newborns in terms of their ability to combat infection is they cannot ramp up their cytokine system to the extent of a normal child, let alone to the extent that would result in a cytokine burst . . .." Hr'g Tr. 456:24-457:4.

Ultimately, the undersigned reached the conclusion Dr. McCusker was making regarding immune problems with preterm infants:

[53] Sadeghi et al., Immaturity of Infection Control in Preterm and Term Newborns is Associated with Impaired Toll-like Receptor Signaling, 195 JOURNAL OF INFECTIOUS DISEASES 296 (Jan. 2007).

> THE COURT: . . . The fact that the child was premature actually works against [petitioners'] theory?
>
> THE WITNESS[, Dr. McCusker]: Well, if you examine the nature of the relative immunodeficiency in premature infants you cannot – I cannot – see how you can argue that theory.
>
> THE COURT: Both because the innate system would not ramp up the cytokines [for a cytokine storm] plus the adaptive system would have cleared out the viral pneumonitis?
>
> THE WITNESS: That's correct. Now, I mean, truthfully this is all hypothesis, but when you try and look at the literature and say what is a reasonable sequence of events, that what the literature is showing me.
>
> THE COURT: It's a hypothesis, but based on the literature –
>
> THE WITNESS: That's correct.

Hr'g Tr. 457:5-21. "[I]n truth there's no evidence that this child [was] in immunodeficiency. If the child did have an immunodeficiency, what the literature says to me is it would be in the innate system, in which case the child couldn't have had cytokine mediated burst or whatever." Hr'g Tr. 458:6-11.

Regarding the theory alleged by petitioners that the hepatitis B vaccination is a superantigen, Dr. McCusker clarified that the IOM report, relied upon by petitioners for this point, found no evidence that hepatitis B vaccine or its components could act as a superantigen; molecular mimicry, bystander activation and superantigen were three theories the IOM postulated and ruled out. Hr'g Tr. 458:23-460:15 (citing R Ex K, the full report from the IOM). Dr. McCusker further testified that she did her own search of medical literature and was unable to find any support for the superantigen hypothesis. Hr'g Tr. 460:16-23.

Dr. McCusker also testified, similar to the opinions given in her reports, regarding Kaleaf's thymus. Dr. McCusker explained the natural history of an infant's thymus. Hr'g Tr. 461:5-464:6. Dr. McCusker notes that the thymus has been studied for a long time and "one of the classical clinical characteristics of a child whose body is undergoing stress . . . because of acute infection – pulmonary infection, sepsis, any kind of infection – one of the classic features is that the thymus shrinks." Hr'g Tr. 463:22-464:6; Hr'g Tr. 468:13 (referencing R Ex F-19). Dr. McCusker would have expected the size of Kaleaf's thymus to have been smaller than usual if he was suffering from a viral pneumonitis, as alleged by petitioners. Hr'g Tr. 464:7-23. It was at this point that Dr. McCusker commented on Dr. Levin's reliance on respondent's Exhibit F-18 regarding thymus size and respiratory distress syndrome. Dr. Levin had quoted the first page of this article, "[m]ean CT/T was significantly greater . . . in those babies with [respiratory distress syndrome.]" See supra p. 10. Dr. McCusker clarified that the study examined the size of patient's thymus **prior** to onset of respiratory distress to decide if thymic size was a **predictor** of patients who may develop respiratory distress syndrome; so the larger than average thymus actually **predated** the respiratory problems. Hr'g Tr. 464:24-467:24. At the undersigned's questioning, Dr. McCusker explained that petitioners' reliance on the Ricci article, P Ex 22, was also misplaced in that the respiratory distress of two patients in the study was due to true thymic hyperplasia, where the enlarged thymus was actually pressing on the patients' airway. Hr'g Tr. 470:4-472:2; see supra pp. 9-10. In reviewing medical literature regarding SIDS, Dr. McCusker showed that a larger than normal thymus was a routine finding in SIDS cases where the children

were not under stress prior to their unexpected deaths. Hr'g Tr. 472:4-473:11 (referencing R Ex F-20 for support in the finding of larger than normal thymus size in children who were not suffering from preexisting stress such as infections, hospitalizations or surgery).

Dr. McCusker summed up her opinion of petitioners' case saying, "I feel very comfortable with my understanding of how infants work, the way infections work and the way the immune system works that the mechanisms that are postulated, there's just not any evidence to support that." Hr'g Tr. 479:20-24.

LEGAL STANDARD

In Vaccine Act cases, causation can be established either through the statutorily prescribed presumption of causation or by proving causation in-fact. For presumptive causation claims, the Vaccine Injury Table lists certain injuries and conditions, which create a rebuttable presumption that the vaccine caused the injury or condition if they are found to occur within a prescribed time period. §14(a); 42 C.F.R. § 100.3. Petitioners here argue the vaccinations in-fact caused their son's injury, a so-called "off-Table" case. See P Post Hearing Memorandum at 3, filed Apr. 8, 2011 ("In the instant case, petitioners have proceeded under a non-"table" theory").

According to §13(a)(1)(A), claimants must prove their case by a preponderance of the evidence.[54] To demonstrate entitlement to compensation in a causation in-fact case, petitioner must affirmatively demonstrate by a preponderance of the evidence that the vaccination in question more likely than not caused or significantly aggravated the injury alleged. See, e.g., Bunting v. Sec'y of Dept. of Health & Human Servs., 931 F.2d 867, 872 (Fed. Cir. 1991); Hines v. Sec'y of Dept. of Health & Human Servs., 940 F.2d 1518, 1525 (Fed. Cir. 1991); Grant v. Sec'y of Dept. of Health & Human Servs., 956 F.2d 1144, 1146, 1148 (Fed. Cir. 1992); see also §§11(c)(1)(C)(ii)(I) and (II). To prevail, petitioner must produce "preponderant evidence both that [the] vaccinations were a substantial factor in causing the illness, disability, injury or condition and that the harm would not have occurred in the absence of the vaccination." Pafford v. Sec'y of Health and Human Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006) (citing Shyface v. Sec'y of Health and Human Servs., 165 F.3d 1344, 1352 (Fed. Cir. 1999)). The vaccination "must be a 'substantial factor'" in bringing about the injury, but "it need not be the sole factor or even the predominant factor." Id. at 1357 (quoting Shyface, 165 F.3d at 1352-53).

In Althen v. Sec'y of Dept. of Health & Human Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005), the Court of Appeals for the Federal Circuit explained that petitioner's burden is to produce "preponderant evidence" demonstrating: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between the vaccination and injury."

[54] A preponderance of the evidence standard requires a trier of fact to "believe that the existence of a fact is more probable than its nonexistence before the [special master] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." In re Winship, 397 U.S. 358, 371-72 (1970)(Harlan, J. concurring)(quoting F. James, CIVIL PROCEDURE, 250-51 (1965)). Mere conjecture or speculation will not establish a probability. Snowbank Enter. v. United States, 6 Cl. Ct. 476, 486 (1984).

The evidence relating to these three prongs "must cumulatively show that the vaccination was a 'but-for' cause of the harm, rather than just an insubstantial contributor in, or one among several possible causes of, the harm." Pafford, 451 F.3d at 1355. Petitioner must provide a "reputable 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 Dept. of Health & Human Servs., 592 F.3d 1315, 1322 (Fed. Cir. 2005); Broekelschen v. Sec'y of the Dept. of Health & Human Servs., 618 F.3d 1339, 1350 (Fed. Cir. 2010), reh'g en banc denied (Dec. 8, 2010). Petitioners do not satisfy this burden by merely showing a proximate temporal association between the vaccination and the injury. Grant, 956 F.2d at 1148 (quoting Hasler v. United States, 718 F.2d 202, 205 (6th Cir. 1983), cert. denied, 469 U.S. 817 (1984) (stating "inoculation is not the cause of every event that occurs within the ten day period [following it]. . . . Without more, this proximate temporal relationship will not support a finding of causation")); Hodges v. Sec'y of the Dept. of Health & Human Servs., 9 F.3d 958, 960 (Fed. Cir. 1993). Also, petitioners do not demonstrate actual causation by solely eliminating other potential causes of the injury. Grant, 956 F.2d at 1149-50; Hodges, 9 F.3d at 960.

Petitioners must support their proposed causation theory with a "sound and reliable medical or scientific explanation." Knudsen v. Sec'y of the Dept. of Health & Human Servs., 35 F. 3d 543, 548 (Fed. Cir. 1994).[55] As the Federal Circuit reiterated:

[55] The general acceptance of a theory within the scientific community can have a bearing on the question of assessing reliability while a theory that has attracted only minimal support may be viewed with skepticism. Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 594 (1993). Although the Federal Rules of Evidence do not apply in Program proceedings, the United States Court of Federal Claims has held that "Daubert is useful in providing a framework for evaluating the reliability of scientific evidence." Terran v. Sec'y of Dept. of Health & Human Servs., 41 Fed. Cl. 330, 336 (1998), aff'd, 195 F.3d 1302, 1316 (Fed. Cir. 1999), cert. denied, Terran v. Shalala, 531 U.S. 812 (2000). See also Cedillo v. Sec'y of Dept. of Health & Human Servs., 617 F.3d 1328, 1338-39 (Fed. Cir. 2010)(approving the use of the Daubert factors in examining the reliability of expert testimony); Moberly v. Sec'y of Dept. of Health & Human Servs., 592 F.3d 1315, 1324 (Fed. Cir. 2010)(citing Daubert; approving of the use of the Daubert factors in determining expert reliability). In Daubert, the Supreme Court noted that scientific knowledge "connotes more than subjective belief or unsupported speculation." Daubert, 509 U.S. at 590. Rather, some application of the scientific method must have been employed to validate the expert's opinion. Id. In other words, the "testimony must be supported by appropriate validation – i.e., 'good grounds,' based on what is known." Id. Factors relevant to that determination may include, but are not limited to:

> Whether the theory or technique employed by the expert is generally accepted in the scientific community; whether it's been subjected to peer review and publication; whether it can be and has been tested; and whether the known potential rate of error is acceptable.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 43 F.3d 1311, 1316 (9th Cir. 1995)(Kozinski, J.), on remand from, 509 U.S. 579 (1993); see also Daubert, 509 U.S. at 592-94.

However, the court also cautioned about rejecting novel scientific theories that have not yet been subjected to peer review and/or publication. The court pointed out that the publication "does *not* necessarily correlate with reliability," because "in some instances well-grounded but innovative theories will not have been published." Daubert, 509 U.S. at 593. However, the Supreme Court has provided guidance to the lower courts in determining the reliability of a novel proposition:

> [S]ubmission to the scrutiny of the scientific community is a component of "good science," in part because it increases the likelihood that substantive flaws in methodology will be detected. (citation omitted). The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a particular technique or methodology on which an opinion is premised.

> Although Althen and Capizzano make clear that a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the special master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury. See Daubert, 509 U.S. at 593-97, 113 S.Ct. 2786 (noting that one factor in assessing the reliability of expert testimony is whether the theory espoused enjoys general acceptance within a relevant scientific community). . . . Althen makes clear that a claimant's theory of causation must be supported by a "reputable medical or scientific explanation." 418 F.3d at 1278.

Andreu v. Sec'y of Dept. of Health & Human Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); see also Grant, 956 F.2d at 1148 ("A reputable or scientific explanation must support this logical sequence of cause and effect."). The Federal Circuit further explained in Andreu:

> The assessment of whether a proffered theory of causation is "reputable" can involve assessment of the relevant scientific data. Medical literature and epidemiological evidence must be viewed, however, not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard . . .

Andreu, 569 F.3d at 1380 (citing Bunting, 931 F.2d 867, 873 (Fed. Cir. 1991)). Proving causation in-fact by proving the Althen standards requires preponderant proof of each of the three prongs. de Bazan v. Sec'y of the Dept. of Health & Human Servs., 539 F.3d 1347, 1351-52 (Fed. Cir. 2008); Moberly, 592 F.3d at 1315, 1322; Caves v. Sec'y of the Dept. of Health & Human Servs., 100 Fed. Cl. 119, 132 (Fed. Cl. 2011) aff'd per curiam, No. 2011-5108, slip op. (Fed. Cir. Feb. 14, 2012).

A finding that petitioners established their *prima facie* burden does not end the inquiry. The Act provides that a petitioner may not receive compensation "if the court finds by a preponderance of the evidence on the record as a whole 'that the illness, disability, injury, condition, or death described in the petition is due to **factors unrelated to the administration of the vaccine** described in the petition.'" Knudsen, 35 F.3d at 547 (citing §13(a)(1)(B))(emphasis in original); Walther v. Sec'y of the Dept. of Health and Human Servs., 485 F.3d 1146, 1150 (Fed. Cir. 2007)("[W]e conclude that the Vaccine Act does not require petitioner to bear the burden of eliminating alternative causes when the other evidence on causation is sufficient to establish a prima facie case."). Since the undersigned finds that petitioners have not provided preponderant evidence on vaccine causation, a factor unrelated analysis is unnecessary. However, a brief discussion is had at the conclusion of the decision regarding petitioners' and respondent's evidence regarding the cause of death noted in Kaleaf's autopsy, Sudden Infant Death Syndrome. Although not considered as a factor unrelated, the medical records and respondent's evidence provide persuasive rebuttal evidence. See Doe/11 v. Sec'y of the Dept. of

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Id. at 593-94; see Althen v. Sec'y of Dept. of Health & Human Servs., 418 F.3d 1274,1280 (Fed. Cir. 2005)("the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body."); see also, Gall v. Sec'y of Dept. of Health & Human Servs., No. 91-1642V, 1999 WL 1179611, at *8 (Fed. Cl. Spec. Mstr. Oct. 31, 1999).

Health & Human Servs., 601 F.3d 1349, 1358 (Fed. Cir. 2010)(holding the special master did not err in considering evidence of an idiopathic condition, SIDS, as rebuttal evidence).

DISCUSSION

Where "medical evidence [is] not definitive," the special master may rely heavily on expert medical testimony. Broekelschen v. Sec'y of the Dept. of Health & Human Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010). Expert medical testimony is particularly important in off-Table injury cases because "[t]he special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories." Id. (citing Lampe v. Sec'y of the Dept. of Health & Human Servs., 219 F.3d 1357, 1361 (Fed. Cir. 2000)). "Weighing the persuasiveness of particular evidence often requires a finder of fact to assess the reliability of testimony, including expert testimony, and we have made clear that the special masters have that responsibility in Vaccine Act cases." Moberly, 592 F.3d at 1325-26 (holding special masters "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 the evidence"). However, in weighing the expert testimony, the special master may not "cloak the application of an erroneous legal standard in the guise of a credibility determination, and thereby shield it from appellate review." Andreu v. Sec'y of the Dept. of Health & Human Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009). This case is not the about the credibility of the experts, but it is a case of reliability and persuasiveness. On that score, respondent prevailed by the widest of margins.

In this case, the undersigned relies heavily on the testimony of Dr. Gilbert-Barness and Dr. McCusker to resolve the underlying key issue of whether Kaleaf's vaccinations were the cause in-fact of his tragic death. As discussed in detail below, petitioners were unable to provide a reliable medical theory connecting Kaleaf's vaccines to his death or to petitioners' alleged injury leading to his death. Furthermore, the respondent's experts persuasively rebutted petitioners' theory and the evidence presented by their experts on vaccine causation. The undersigned finds it was respondent's experts who convincingly explained why it was exceedingly unlikely that the vaccines were the cause of Kaleaf's death.

Both of respondent's experts were far superior to petitioners' experts in their background, experience and in the quality of the information proffered in this case. Petitioners' experts' thin experience – especially in clinical medicine and specifically with infants and young children – and their often confusing, contradictory testimony, which seemed at times to meander through the medical information, was very unhelpful and unpersuasive.

On virtually every critical point in this case, petitioners' proposed analysis of the medical information was met with a resounding rebuttal from respondent's experts. Petitioners' experts simply did not have the comparative experience and their generalized testimony was exposed for what it was – conclusory, speculative, unsupported and unreliable. Dr. Levin's testimony in particular was extremely unhelpful – as would be expected from someone who practices law 99% of the time and thus medicine 1% and has not see a patient since 1993. Further, as he testified here in the capacity of a pathologist, Dr. Levin has not performed an autopsy on a child since the 1980's and the number of autopsies performed on children, fifty or sixty, is dwarfed by the experience of Dr. Gilbert-Barness, who estimates she has performed approximately 10,000 pediatric autopsies. See supra p. 6, 19. With the change in his career focus from medicine to

law, it is a reasonable question to ask whether Dr. Levin should be accepted to provide a medical opinion at this point in his career. Dr. Byers has better experience in the medical area in which she is opining, but consistent with the undersigned's and my colleagues' experience with Dr. Byers as an expert, her testimony tends to be highly generalized and missing a strong connection to the facts and medical information in the specific case. As such, Dr. Byers' opinions tend to falter when examined by the lay person and are exposed as unreliable when examined by a competent expert.

The differences between the experts were detailed above, and the critical factors for decision will be discussed below. However, significant disputes are highlighted here to exemplify the disparity between the experts and the evidence. Some, but not all, of the issues are summarized as follows:

- Drs. Levin and Byers proffered opinions that an infant's thymus grows when faced with stress, such as chronic infection, in contrast to the supported and clear testimony of Drs. Gilbert-Barness and McCusker that the thymus under stress actually shrinks.

- Dr. Levin testified that the heavy, congested organs in this case were significant in that they were evidence of right-sided heart failure and were not explained by respondent's theory of an asphyxial death. This was contradicted directly by Dr. Gilbert-Barness and respondent's literature that showed heavy organs were not unique to petitioners' theory.

- Dr. Levin opined that the lung slides showed evidence of chronic infection and inflammation, a keystone of petitioners' theory of causation, while Dr. Gilbert-Barness, who estimates she has performed 10,000 pediatric autopsies and estimates 10-15% of those involved pneumonia or pneumonitis, found no such evidence.

- As part of petitioners' theory, Dr. Levin opines Kaleaf died of asphyxiation via right-sided heart failure but was unable to show, nor did the medical examiner find, dilation in the heart that Dr. Gilbert-Barness states would be found with right-sided heart failure.

- Drs. Levin and Byers speculate Kaleaf's prematurity caused him to be immune deficient and opined this factored into the timing of the alleged vaccine-related death; however, they were unable to show evidence that Kaleaf was actually immune compromised. Dr. McCusker testified that there was no evidence Kaleaf was immune deficient and, even if he was, this conflicts with petitioners' theory of the case.

- Dr. McCusker, who presently has a very active pediatric practice in comparison to petitioners' experts, utilized the medical records to show no signs of pneumonia or chronic lung infection leading up to Kaleaf's death – he had clear sounding lungs at his last exam, no note of lung infection or problems by EMTs or the medical examiner, there were no notes of problems with weight gain or feeding, and there was nothing in the record to support the post-mortem report of tachypnea – while Dr. Byers speculated the existence of the lung infection based on the one post-mortem report of tachypnea and Dr. Levin's interpretation of the pathology slides.

Additionally, in conflict with Dr. Levin's reading of the pathology slides, Dr. Gilbert-Barness found no evidence of a chronic lung infection in the pathology slides.

- Dr. Byers presented a theory of cytokine storm, or a theory of an aberrant immune response, that overwhelmed Kaleaf's lungs and pulmonary system despite the fact that Dr. McCusker opined that a cytokine storm cannot occur without symptoms. She also pointed out that there is no support for an immune response in the leg producing a response in a distant part of the body. If that had occurred, she said one would expect to see other systemic signs of such an immune response, which were not found here.

There was very little agreement between the representative experts as to the interpretation of the medical information in this case. Ultimately, the undersigned had to determine which experts were more reliable, and thus more persuasive. See, e.g., Moberly, 592 F.3d at 1325-26. This was not a close call; respondent's experts were vastly more persuasive.

I. ALTHEN PRONG I – THEORY CONNECTING VACCINE AND THE INJURY

The first prong of Althen requires preponderant evidence of a "medical theory causally connecting the vaccination and the injury." This requirement has been referred to as the "can cause" prong: can the vaccine cause the alleged injury. Pafford v. Sec'y of the Dept. of Health & Human Servs., No. 01-165V, 2004 WL 1717359 (Fed. Cl. Spec. Mstr. 2004), aff'd 451 F.3d 1352, 1355-56 (Fed. Cir. 2006). The undersigned views the first prong of Althen as inquiring whether the vaccines received could cause the alleged injury, akin to general causation. Pafford v. Sec'y of the Dept. of Health & Human Servs., 451 F.3d 1352, 1354-56 (Fed. Cir. 2006)(noting the special master applied the tests of Althen and Shyface correctly and discussing the "can cause" aspect of the first prong and the "did cause" aspect of the second prong); see also Veryzer v. Sec'y of the Dept. of Health & Human Servs., 100 Fed. Cl. 344, 352-53 (Fed. Cl. 2011)(explaining the first two prongs of Althen in terms of general causation, prong one, and specific causation, prong two). To meet the first prong of Althen, petitioner "must provide a reputable 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, 592 F.3d at 1322 (citing Knudsen, 35 F.3d 543, 548-49 (Fed. Cir. 1994)); see also Broekelschen, 618 F.3d 1339, 1345 (Fed. Cir. 2010). Petitioner's proof cannot merely establish a "plausible" or "possible" causal link between the vaccine and the injury; the proof must meet the statutory standard of preponderance. Moberly, 592 F.3d at 1322; see also Caves, 100 Fed. Cl. at 132, 144, aff'd per curiam No. 2011-5108, slip op. (Fed. Cir. Feb. 14, 2012). Here, the question is whether the vaccines Kaleaf received can cause the injury alleged by petitioners that caused Kaleaf's death.

Dr. Byers provided the primary opinion regarding petitioners' theory of causation in this case. In her original report, P Ex 11, Dr. Byers opines that Kaleaf's death was "caused by a cytokine storm following the Hepatitis B vaccination." P Ex 11 at 4. She explained that the alleged "slight illness" in the two weeks before vaccination caused low-grade activation of macrophages in the lungs. Id. Then, "[t]he overlay of the Hepatitis B vaccination and subsequent release of additional cytokines and chemoattractants, more probably than not, caused a massive overstimulation of both the adaptive and the innate immune system causing cytokine storm." Id. She opined that, due to Kaleaf's symptoms and the finding of thymic hyperplasia,

this process was caused through bystander activation or a superantigen.[56] With this report, petitioners submitted as support their Exhibit 12, the New England Journal of Medicine article discussing an inadvertent cytokine storm induced in study subjects, and Exhibit 13,[57] an Institute of Medicine ["IOM"] report discussing the hepatitis B vaccine and demyelinating neurological disorders. As stated previously, respondent also filed the New England Journal Article. R Ex F-9. Respondent also filed the IOM report in its entirety. R Ex K.[58]

Petitioners' Exhibit 12 does not address a medical theory for how the hepatitis B vaccine, or any of the vaccines or vaccine components, could cause injury to Kaleaf. This piece of medical literature was used to describe the respiratory effects of a cytokine storm and its timing. Supra pp. 13, 17. Although it shows the process petitioners' allege, it fails to evidence a **medical theory causally connecting the vaccinations to the alleged injury**. Althen, 418 F.3d at 1278. Petitioners' Exhibit 13, though, appears to have been proffered to show a causation theory; particularly the superantigen or bystander activation theories.

First and foremost, the undersigned notes that this IOM report deals with the hepatitis B vaccine and a speculated link to **demyelinating neurological** injuries. P Ex 13; R Ex K. This was the evidence Dr. Byers relied upon to opine the hepatitis B vaccine, and presumably the other vaccines Kaleaf received, could have been superantigens or elicited bystander activation to cause the aberrant cytokine response. Dr. Byers characterized the report as "looking at autoimmune disease," and how the hepatitis B vaccine could be "associated with autoimmune disease, and specifically demyelinating autoimmune disease." Hr'g Tr. 44. At issue in this case is not a demyelinating injury; nor is a neurological condition at issue. Dr. Byers, herself, testified this was not an autoimmune case, albeit while trying to equivocate. Hr'g Tr. 485-86 ("I agree with you though. This is more of an infectious disease . . . ."). There was no evidence offered that Kaleaf suffered a demyelinating condition, let alone evidence that he suffered an autoimmune condition. Further, other than petitioners' experts' bare assertions, there is no evidence in this record that the theories discussed in this report relate to non-demyelinating injuries. On that basis alone, the article is not relevant or persuasive in establishing petitioners' theory of causation.

Second, as discussed by Dr. McCusker, Hr'g Tr. 458, "I think it's very clear when you read the full IOM that there is no evidence in the extant literature[,] in the clinical epidemiological literature[,] or in animal models that hepatitis B or any of the components of the

[56] It is noted that petitioners pepper the record with references of bystander activation but fail to develop this theory. Dr. McCusker did respond to the suggestion though and thoughtfully testified that there would be other evidence of a systemic reaction in the record if any of the vaccines Kaleaf received were potent enough to cause bystander activation. Ultimately, petitioners failed to develop this theory.

[57] Kathleen Stratton, et al., Institute of Medicine, Immunization Safety Review, Hepatitis B Vaccine and Demyelinating Neurological Disorders 8-9 (National Academy of Sciences 2002).

[58] Dr. Levin testified on the second day of the Hearing that petitioners were not contesting Kaleaf suffered an asphyxial death; the disagreement regarded how the asphyxia occurred. Hr'g Tr. 355-59. Dr. Levin testified that the resistance caused by the inflammation attracted to the lung built up and resulted in the right side of the heart failing. Id. It appears that respondent alleges Kaleaf died of SIDS, often used in reference to positional asphyxia, while petitioners allege lung inflammation lead to right heart failure and asphyxiation. The question discussed was whether there was post-mortem evidence that Kaleaf suffered acute right heart failure. Because this evidence centered on Kaleaf's specific autopsy findings, the undersigned believes the most logical place to analyze this testimony is under the second prong of Althen. See infra pp. 46-47.

vaccine that Kaleaf received can act as a superantigen." Hr'g Tr. 459. Dr. McCusker points out that the IOM hypothesized the mechanisms of bystander activation and superantigen could possibly be associated with demyelinating injuries but concluded that they were not viable theories after review of medical literature. Hr'g Tr. 459-460. Dr. McCusker went even further than just reviewing the IOM report and could not, herself, find anything in the literature to suggest that the hepatitis B vaccine acts as a superantigen. Hr'g Tr. 460. "Although a Vaccine Act claimant is not required to present proof of causation to the level of scientific certainty, the special master is entitled to require some indicia of reliability to support the assertion of the expert witness." Moberly, 592 F.3d at 1324 (citing Terran v. Sec'y of the Dept. of Health & Human Servs., 195 F.3d 1302, 1316 (Fed. Cir. 1999); see also Dobrydnev v. Sec'y of the Dept. of Health & Human Servs., 98 Fed. Cl. 190, 207 (Fed. Cl. 2011)("Moberly stands for the proposition that, as the fact finder, a special master may consider the lack of support in medical literature as a factor in determining whether a petitioner has established a prima facie case of causation under a preponderance of evidence standard."). Petitioners presented these theories based on literature that simply is not supportive.

Third, regarding the superantigen and bystander activation theories, Dr. McCusker dismissed this theory when responding to the hypothetical question regarding whether a cut on one's foot could cause an immune response in a distant location, for example, a runny nose; she noted that this does not happen. Hr'g Tr. 444-46. "[G]enerally [immune] responses are maintained locally. And so you really need to have a stimulus that is extremely strong or extremely unusual . . . because really what you're talking about there is this bystander activation; that there's activation of an immune response far away from the initial immune response, and it doesn't occur." Hr'g Tr. 445. She explained you would need a "very strong stimulus" for a response in one part of the body to affect another, distant part of the body. Hr'g Tr. 446. Even if the vaccines were this very strong stimulant, "you would have expected to see signs, and in this situation the baby came home, there was not any swelling or pain or tenderness noted at the site. The baby was not irritable. He fed well. No evidence of fever in this child. There's no evidence that this child did anything other than go for his nap."[59]

Leaving the precise mechanisms of bystander activation and superantigen behind, Dr. McCusker disagreed more generally with Dr. Byers regarding the association between the hepatitis B vaccine, which is the alleged agent of injury in Dr. Byers' first report, and cytokine storm. Dr. Byers relied upon literature noting immune-mediated tissue damage in found in association with the hepatitis B **infection**. Supra p. 14. As noted previously though, Dr. Byers did not provide evidence that this infection leads to the acute damage she alleged occurred with Kaleaf or that the vaccination is capable of the same damage. Supra pp. 14, 23, 25. Dr. McCusker admitted that in specific conditions, the immune system may become imbalanced, leading to detrimental inflammation, the cytokine storm. R Ex D1 at 3. She listed situations in which cytokine storm has been observed: bacterial sepsis, infections with certain viruses such as SARS and influenza, severe burns and trauma, and acute pancreatitis. Id. Dr. McCusker stated, "[i]n contrast, viral infections caused by hepatitis viruses are not associated with systemic

[59] The undersigned observes that this testimony is most applicable under an Althen prong two analysis and is not attempting to conflate the evidence relevant to these two Althen prongs. However, this was Dr. McCusker's explanation for why bystander activation was not a viable theory of causation herein and petitioners offered no reliable support for their allegation that the vaccines Kaleaf received or their ingredients could actually instigate this bystander activation.

cytokine storm." Id. (citing R Ex F1[60] and R Ex F7).[61] In the responsive report, P Ex 17, Dr. Byers wrongly assumed Dr. McCusker's statement applied to any immune-mediated pathology caused by a hepatitis B infection, not just cytokine storm, and countered this by quoting a sentence from a textbook. P Ex 19.[62] The section of this exhibit discussed the immune responses to viruses actually causing some inflammation and pathology. P Ex 19 at 1. It stated, "[t]his could be true in viral infections in which a large number of cells are infected in an individual before the immune response is turned on and in which destruction of these infected cells by immune response may have severe or fatal pathologic outcomes." Id. The text provides a table of immune-mediated pathology, which includes the wild hepatitis B virus but not the vaccine. Id. at 2.

However, there was no attempt by petitioners to show that the hepatitis B vaccine could induce the same type of immune pathology as the wild type hepatitis B infection can. Also, the undersigned notes that the section of the text, R Ex 19, relied upon by petitioners does not state that cytokine storm has been reported with the hepatitis B infection, only that one's own immune system causes tissue damage in the presence of the wild infection. It states, "**[c]hronic** hepatitis B virus infection provided the first clue that the disease is caused by an indirect mechanism rather than the virus itself because a low level of virus can be present in **chronically** infected people without any damage to the target tissue (liver) for a **long time**." R Ex 19 (emphasis added). The section goes on to note that damage distant to the liver can occur, including specifically inflammation and tissue damage and possible renal damage. R Ex 19 at 1. The text discusses the pathology of the wild hepatitis B infection and the detrimental action of the host's immune system with this infection, which provided scientists with clues to immune-induced pathology. The article does not state the hepatitis B infection causes a cytokine storm or even a large, acute cytokine response; there is no description of the time frame wherein this immune-mediated damage occurs – does the damage occur over hours like a cytokine storm or does it occur over days, months or years?

Dr. Byers is correct, P Ex 17 at 3-4, that the Sherris text and respondent's exhibits, R Ex F-1 and R Ex F-7, discuss immune-caused damage in response to the wild hepatitis B infection. However, to reiterate the two flaws in this reliance, review of these articles show that they do not state that the wild infection causes cytokine storm or any acute event, nor do they show the *vaccine* induces the same immune-mediated pathology that the wild infection may cause. It appears that petitioners' expert is stretching the statements in these article and ultimately fails to offer evidence to connect the cytokine storm or other aberrant effect to the hepatitis B vaccine. See Poulos v. Sec'y of the Dept. of Health & Human Servs., No. 90-2315V, 1994 WL 470622 (Fed. Cl. Spc. Mstr. 1994)(finding there was not enough evidence to conclude the rubella vaccination could cause the same condition that could be caused by the wild rubella infection).

Ultimately, petitioners' case rests on Dr. Byers' unsupported theory that the hepatitis B vaccine or the other vaccines received can cause a cytokine storm or cytokine damage – damage enough to cause Kaleaf's death. Petitioners' experts fail to address the obvious gaps left between

[60] Mathis Heydtmann, *et al.*, Cytokines and chemokines in the immune response to hepatitis C infection, 14(3) CURR OPIN INFECT DIS 279 (2001).

[61] Angela J. Frodsham, Host genetics and the outcome of hepatitis B viral infection, 14 TRANSPLANT IMMUNOLOGY 183 (2005).

[62] SHERRIS MEDICAL MICROBIOLOGY 152 (5th ed. 2010).

their theories and the literature they claim support their opinions. Again, petitioners' experts rely on an IOM report that discusses hypothetical mechanisms for autoimmune, demyelinating injuries, which are not alleged here, and failed to relate autoimmune, demyelinating injuries to the injury they allege Kaleaf suffered. Moreover, the report also **first theorizes and then rejects** the superantigen and bystander activations theories petitioners proffer. Petitioners' experts also rely upon a textbook that discusses how immune-mediated damage occurs in a wild type, hepatitis B infection. As stated above, two problems are obvious with this reliance: first, the experts fail to explain whether the hepatitis B vaccine can instigate the same immune-mediated damage as the wild infection; second, they also make no effort to show that the damage arising from the wild hepatitis B infection rises to the acute type of damage that they allege caused Kaleaf's death. Without support from the medical literature they offer and the unpersuasive testimony of their experts, petitioners' theory fails. Between their flawed theory and Dr. McCusker's highly persuasive testimony, petitioners' case does not approach preponderant evidence.

The undersigned notes that the theory proposed in this case, the aberrant cytokine response, is similar to the theory discussed in Doe/11. Doe/11 v. Sec'y of the Dept. of Health & Human Servs., No. 99-212, 2008 WL 4899356 (Fed. Cl. Spec. Mstr. 2008), aff'd, 87 Fed. Cl. 1 (Fed. Cl. 2009), aff'd, 601 F.3d 1349 (Fed. Cir. 2010). In that case, Dr. Levin testified on the immunology portion of the case and utilized some of the same literature presented herein.[63] The theory offered in Doe/11 was that an abnormal cytokine response caused inflammation, some of which was directed at the infant's brain, leading to the child's death. This theory in Doe/11 was strongly criticized. In the case at hand, the undersigned finds there is no persuasive evidence of a medical theory causally connecting the vaccinations received to the alleged injury and death of Kaleaf. See Althen, 418 F.3d at 1278. Petitioners fail to meet their burden under analysis of Althen prong one.

II. ALTHEN PRONG II – SEQUENCE OF CAUSE AND EFFECT SHOWING THE

Having found petitioners' unable to meet their burden regarding a legally plausible medical theory, the undersigned need not consider the evidence of the second and third prongs of Althen. See Althen, 418 F.3d at 1278 (requiring petitioner to show preponderant evidence of each of the three factors in the causation analysis); Broekelschen, No. 07-137V, 2009 WL 440624, *24 (Fed. Cl. Spec. Mstr. 2009)("[Petitioner] has failed to establish one of the three elements required by Althen[. . . and] is not entitled to compensation. Furthermore, because of the lack of proof on this element, discussing the remaining two prongs from Althen is not necessary."), aff'd, 89 Fed. Cl. 336, 346 (Fed. Cl. 2009)("Thus, petitioner failed to establish a prima facie case with respect to one of the three elements required by Althen, and the special master properly concluded that petitioner was therefore not entitled to compensation.")(citing Althen, 418 F.3d at 24-28), aff'd, 618 F.3d at 1345-46 ("causation is relative to the injury," and the theory must pertain specifically to the petitioner's case), reh'g en banc denied (Dec. 8, 2010); see also Lombardi v. Sec'y of the Dept. of Health & Human Servs., 656 F.3d 1343, 1353 (Fed. Cir. 2011)(affirming special master's finding that petitioner did not suffer a medically recognized "injury" and special master's decision to decline conducting the Althen analysis). Logically, if petitioners cannot show the vaccines could cause the alleged injury and Kaleaf's death, they would be unable to show that the vaccines actually did cause his death. However, five aspects of petitioners' case, one of which was admittedly critical, relied on findings

[63] Dr. Levin testified here on the pathology issues of the case.

specifically related to Kaleaf and are appropriately addressed under the second prong of Althen. The undersigned, for the sake of thoroughness, discusses these critical aspects of the case and finds petitioners' again could not meet their burden of proof under Althen prong two even assuming *arguendo* they could show their theory was legally probable under prong one.

The second prong of Althen requires preponderant evidence of a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." This prong is sometimes referred to as the "did cause" test: in petitioner's case, did the vaccine cause the alleged injury. Pafford v. Sec'y of the Dept. of Health & Human Servs., No. 01-165V, 2004 WL 1717359 (Fed. Cl. Spec. Mstr. 2004); see also Broekelschen v. Sec'y of Health & Human Servs., 618 F.3d 1339, 1345 (Fed. Cir. 2010)("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…"); Moberly, 592 F.3d at 1324-26. Petitioner must show that the vaccine was the "but for" cause, or in other words, "the vaccine was the 'reason for the injury.'" Pafford v. Sec'y of the Dept. of Health & Human Servs., 451 F.3d 1352, 1356 (Fed. Cir. 2006). This is akin to actual or specific causation.

First, petitioners' theory of causation hinged upon the finding that Kaleaf suffered from a preexisting lung infection, pneumonia or pneumonitis, that attracted the inflammation brought on by the vaccine and ultimately caused the pulmonary resistance that petitioners' allege caused Kaleaf's death. In fact, petitioners' expert noted that she could not offer a supportive opinion on causation if the undersigned failed to find this preexisting lung infection. Second, both of petitioners' experts stressed Kaleaf's status as a premature infant could have caused or did cause an immune-compromised state, which affected his ability to fight off the alleged lung infection, which in turn attracted and hastened the cytokine effect on his lungs. Third, assuming *arguendo* that petitioners' theory of general causation was found reliable, the undersigned would necessarily examine Kaleaf's case for evidence that the cytokine storm, or burst, actually occurred. See Moberly v. Sec'y of the Dept. of Health & Human Servs., 592 F.3d at 1324 ("Even setting aside the question whether the 'blood-brain barrier' theory was reliable, [the expert] conceded that there was no evidence in the record suggesting that the proposed mechanism was at work in [the vaccinee's] case."). Fourth, considering petitioners agree this was a hypoxic or asphyxial death, both sides' experts discussed petitioners' allegation of right heart failure. Finally, petitioners argue the increased organ weights found during the post-mortem exam are evidence of massive inflammation while respondent's experts claim this finding is consistent with the cause of death found by the medical examiner or positional asphyxia. As these are critical components to petitioners' case regarding whether Kaleaf's vaccines actually caused his death, the undersigned addresses these case-specific issues under the second prong of Althen. As discussed, petitioners fail to produce preponderant evidence on any of these critical issues.

a. Preexisting lung infection, pneumonia or pneumonitis, lymphocytic infiltration and evidence relating to the thymus

The evidence submitted by petitioners that Kaleaf was suffering from a lung infection prior to his vaccinations is scant. Dr. Byers' opinion on this subject relied upon Dr. Levin's interpretation of the pathology slides and the post-mortem reference of "rapid breathing." Hr'g Tr. 34:19-23; Hr'g Tr. 67:12-68:1; Hr'g Tr. 81:7-12; Hr'g Tr. 82:9-83:9. Beyond these two elements, Dr. Byers admitted there was no other evidence of a preexisting lung infection. Hr'g

Tr. 82:9-83:9. Dr. Byers asserted, with no apparent basis, that the pediatrician who examined Kaleaf on the day of his death likely omitted reference to any signs of a lung infection because there was no designated space for such a comment. Hr'g Tr. 77:4-78:2. To the undersigned, this base assertion strains credulity. Dr. McCusker averred that such an omission would be tantamount to malpractice. Hr'g Tr. 426:18-25. Dr. McCusker indicated that tachypnea or rapid breathing could signal a serious problem and would normally be investigated if found or reported. Hr'g Tr. 433. In fact, the record evidences a chest exam was performed at his final doctor visit and the indication was normal. P Ex 3. Although it appears that Kaleaf was suffering from some nasal congestion, "scant rhinorrhea," there is no indication that his pediatrician detected tachypnea or evidence of a lung infection. Moreover, as testified by Dr. McCusker, who treats infants and children on a regular basis, infants typically exhibit problems with fussiness, feeding and weight gain when they present with a chronic condition. Hr'g Tr. 427; Hr'g Tr. 434. Here, there is no evidence of such signs, the parents made no complaints that were recorded in the medical records, and Kaleaf had continued to gain weight. P Ex 2 at 18; P Ex 3; P Ex 5 at 4, 6. The undersigned finds many of Dr. Byers' assumptions either unsupported by the record or mere speculation. See Hr'g Tr. 74:25-75:15 (likening what, in her opinion, Kaleaf suffered to "walking pneumonia."); Hr'g Tr. 79:3-80:3 (opining that the child had a very mild pneumonia from the note of rapid breathing in the post-mortem interview and the treating physician's decision to give vaccinations on the day Kaleaf died). There simply is no persuasive basis for Dr. Byers' opinion that Kaleaf suffered from a lung infection.

Regarding Dr. Levin's opinion, he opines that Kaleaf suffered "from a low grade viral infection for several weeks prior to vaccination," supported by the notation regarding two weeks of 'sniffles' and normal breath sounds in the well baby exam" on the day of his death. P Ex 10 at 3. The large thymus and heavier-than-average spleen were also used to support of his opinion of a preexisting lung infection in that they showed "marked activation of T cells in the infant." P Ex 10 at 3; Hr'g Tr. 193:25-199:24. Further, Dr. Levin referenced "massive" patches of inflammation in 10-15% of Kaleaf's lungs, lymphocytic infiltrates and "massive" alveolar thickening as found by him on the pathology slides. P Ex 16 at 1-3; Hr'g Tr. 139:4-141:4; Hr'g Tr. 143:20-23; Hr'g Tr. 147:19-25; Hr'g Tr. 160:18-161:15; Hr'g Tr. 168:20-169:2.

In regard to the "rapid breathing" note in the post-mortem report relied upon by petitioners as evidence of the alleged lung infection, P Ex 5 at 4, the undersigned finds this is the only reference to a potential problem in Kaleaf's records. The post-mortem narrative discusses that this problem was "reported to the hospital two weeks" prior to the infant's death and the mother was told it was "some type of congestion." P Ex 5 at 4. Petitioners could show no place where such a problem was actually reported two weeks prior. What was reported, however, was evidence of a runny or stuffy nose at his vaccination appointment. P Ex 3 at 1; see also Hr'g Tr. 423:20-426:17. Further, the medical record from the day of his death actually indicates a normal chest exam. P Ex 3. The undersigned notes that pneumonia, lung infection or other lung abnormalities were not noted at autopsy. See P Ex. 5.

Again, in response to Dr. Byers' assertion that there was no place for the pediatrician to denote rapid breathing or tachypnea, Dr. McCusker's opinion was that it would be malpractice if a pediatrician did not find a place to make such a note. Hr'g Tr. 426:18-25. Further, Dr. McCusker indicates, in her interpretation of the pediatrician's notes and Kaleaf's weight gain, that he had been feeding well leading up to that last visit. Hr'g Tr. 427:1-11. Dr. McCusker found nothing in the pediatrician's notes that would indicate the child was suffering from a

chronic condition, pneumonia or pneumonitis. Hr'g Tr. 427:12-431:17. The medical examiner made no findings pertaining to this. Dr. McCusker, referencing the medical records, also showed there was no evidence of lower airway issues at the pediatrician's office or by the EMT during CPR. R Ex I at 3. In conclusion, based upon the medical records of Kaleaf's last doctor visit, the post-mortem examination, and the EMT report, there is simply no evidence that any of these three health professionals even suspected a lung infection. This is further supported by Dr. McCusker's interpretation of the records.

In reference to the pathology slides, Dr. Levin testified that there was no evidence that the pathology slides were examined. The undersigned notes the record is silent regarding whether the slides were or were not viewed by the medical examiner or a pathologist. Regarding the interpretation of those slides, Dr. Gilbert-Barness admitted that there was some small but insignificant evidence of infiltrates and alveolar thickening seen in some of the organs. However, her interpretation of these signs was that they were insignificant and, further, did not evidence a cause of death. Hr'g Tr.235:1-236:1; see also Hr'g Tr. 252:6-253:3; R Ex A at 2 (finding no evidence of preexisting pneumonitis). Dr. Gilbert-Barness noted there were lymphocytes present in the lungs, which she testified is normal, but strongly disagreed with Dr. Levin that the pathology slides showed lymphocytic infiltration and/or massive inflammation. R Ex A; Hr'g Tr. 224-45; Hr'g Tr. 254-55. Despite agreeing this is an asphyxial death generally, Dr. Levin cited the organ weights as evidence of petitioners' theory of massive inflammation and right-sided heart failure. Supra pp. 8-9, 11. Dr. Gilbert-Barness opined that the organ congestion would be even greater and the shape of the heart would be affected if Dr. Levin's theory of right heart failure were valid. Supra pp. 19-20, 22-23; infra pp. 46-47. Also, in rebuttal to petitioners' contention that congestion and raised organ weight is inconsistent with a SIDS death, Dr. Gilbert-Barness testified and provided medical literature support that this was consistent with SIDS, the medical examiner's cause of death. R Ex A; R Ex I; R Ex O1. The undersigned credits the testimony of Dr. Gilbert-Barness and finds the organ congestion and weights were not so abnormal as to identify the cause of death. Further, respondent's medical literature supported the testimony that the weight and congestion of the organs were not indicative of only inflammation and right heart failure, as asserted by petitioners.

Last, the experts disagreed about the significance of Kaleaf's heavier-than-average thymus. Petitioners' experts testified the thymus was larger than average and loaded with lymphocytes as evidence that he was suffering from a chronic infection. P Ex 10; P Ex 11; P Ex 17. Both of petitioners' experts pointed to evidence that they claimed supported the notion that an infant's thymus actually gets larger when faced with a stressor, such as a chronic lung infection. See supra pp. 9-11, 13-14. The medical literature cited by petitioners' experts for their assertion, as discussed, was inapt. See supra pp. 10-11. This was also in contrast to the respondent's experts and medical literature showing the thymus actually shrinks in the presence of stress, such as chronic infection. See supra pp. 21, 24, 30-31. Other than citing the Ricci and Gewolb articles that did not support their contention, petitioners' failed to offer reliable evidence that an infant's thymus actually increases in the presence of infection. Respondent's experts testified consistently and persuasively that the thymus would actually shrink in the presence of chronic infection and provided evidence, unrebutted by petitioners, to support this. In fact, respondent provided supportive medical literature showing that the weight of Kaleaf's thymus was likely consistent with the weight of infants dying unexpectedly and without preexisting stressors, such as infection or surgery, that affect thymic size. See supra 24 (noting Dr. McCusker cited a study that hypothesizes the "substantially greater thymic size" of SIDS infants

"'is more representative of the living population than weights of infants dying of other causes who may have been exposed to various thymolitic factors.'")(citing R Ex F-20). Again, petitioners are unable to show that the increased thymus size in this case is supportive of their theory that Kaleaf suffered from a preexisting lung infection that was the focus of the allegedly harmful immune response. The undersigned credits Drs. Gilbert-Barness and McCusker's convincing and supported testimony regarding the evolution of the thymus under stress.

This issue, whether or not there was a chronic infection and then acute inflammation in Kaleaf's lungs, evidences a true "battle of the experts." Lampe v. Sec'y of the Dept. of Health & Human Servs., 219 F.3d 1357 (Fed. Cir. 2000)("In actual causation cases such as this one, the ultimate decision often turns on the outcome of the 'battle of the experts' . . . ."). Both doctors are qualified to testify in the area of pathology, though respondent challenged Dr. Levin's qualifications to testify on pediatric pathology, and both experts examined the pathology slides. However, these experts strongly disagree in their impressions of these slides. When all of the testimony is considered, the undersigned finds Dr. Gilbert-Barness much more persuasive and reliable given her knowledge and vast experience with pediatric pathology. The differences between the pathologists experience in this area is enormous and cannot be gainsaid. See supra pp. 5-6, 12, 19, 23. After consideration of the totality of the evidence, the undersigned agrees with respondent's experts and finds that Kaleaf did not suffer from a preexisting lung infection. Neither the clinical information nor the pathology slides show evidence of massive inflammation and lymphocytic infiltrate to the degree necessary to support petitioners' experts' allegations. Necessarily, assuming petitioners' theory of causation had been accepted under Althen prong one, this finding defeats petitioners' case as their expert, Dr. Byers, stated she could not provide a supportive opinion on causation if Kaleaf did not suffer the preexisting lung infection.

b. Immune-compromised state due to premature birth

Most notably on the second day of the Hearing, petitioners' experts stressed Kaleaf's premature birth as a condition of their theory of the case. Both parties' experts admitted that prematurity may affect an infant's immune system. However, petitioners could not persuasively show Kaleaf was immune compromised. In rebuttal respondent's expert was more specific in describing how the medical community believes prematurity affects the infant's immune system, which does not comport with petitioners' theory of how the vaccines caused Kaleaf's death.

Petitioners believe Kaleaf's prematurity caused him to be immune-compromised. This alleged status fits into their theory of the case in two ways. First, being immune-compromised, Kaleaf was unable to clear the lung infection that petitioners' experts opine was present for approximately two weeks prior to the day of his death. Supra pp. 15-16. Second, the prematurity and immune-compromised states were also discussed regarding the shorter-than-expected time frame between vaccination and death, which would be relevant under Althen prongs two and three. Hr'g Tr. 90-91. However, as is characteristic of much of petitioners' experts' testimony, there is no evidence to support their contention of Kaleaf being immune-compromised.

Primarily, no evidence of actual immune compromise is found in Kaleaf's medical records, nor did Dr. Byers provide support from Kaleaf's medical records that he was immune deficient. In fact, Dr. Byers stated that he had an "uneventful neonatal and pediatric follow up over approximately the next three months . . . ." Hr'g Tr. 34:3-4. She also confirmed he

achieved the 98th percentile in weight gain according to medical records and autopsy measurements. Id. When reviewing the pathology slides for her first report, Dr. Byers also opined that "[t]he thymus is well formed, compatible with immune competence in the young child . . . ." P Ex 11 at 3. As pointed out by Dr. Levin, he "had attained normal length and weight by the time of his death" and was above the 95th percentile in size for his age. P Ex 10 at 1. These observations are in direct conflict with petitioners' assertion that Kaleaf was immune compromised and were not addressed by petitioners.

Respondent's expert noted the lack of evidence of immune deficiency in the medical record. Hr'g Tr. 454:5-7; 458:2-18. Dr. McCusker argued that adjusting Kaleaf's age at the time of his death, he was functionally equivalent to a six week old baby and not a premature baby at the time of his death. Hr'g Tr. 447:25-449:1. Kaleaf was found to be a well-baby at his two follow-up visits, with the exception of the runny nose at the last visit. P Ex 2 at 18; P Ex 3. Other than stuffiness and the unconfirmed post-mortem report of "rapid breathing," there is no other evidence of illness.

To reiterate, there is no evidence in the record showing Kaleaf was immunocompromised during the time leading up to his death. The allegations by petitioners' experts are not supported by the record and appear to be mere speculation.[64] There is simply nothing in this record to show that Kaleaf was immune compromised due to his prematurity.

c. Signs of a cytokine storm or aberrant cytokine release assuming *arguendo* that petitioners' theory of causation had been accepted

Regardless of whether the vaccine or vaccine components cause the alleged cytokine storm or unusual cytokine release, petitioners rely primarily on their Exhibit 12 regarding the effects of the cytokine storm on Kaleaf, particularly lung and respiratory involvement of the patients in the study. Petitioners' expert discussed other cytokine-related disorders but did not utilize those conditions to discuss similarities to the cytokine event allegedly suffered by Kaleaaf. Supra p. 16. However, as acknowledged by both immunologists, there is not one set of symptoms that necessarily manifest from a cytokine storm; they are somewhat variable. Dr. McCusker, though, provided testimony of general, systemic symptoms that accompany a large cytokine release and do not vary as much as the end-organ results of the different cytokine storm disorders. Supra pp. 23, 25-28.

In the New England Journal of Medicine article, P Ex 12, clinically observable symptoms of cytokine storm may include: headache, rigors, lumbar myalgias, fever, restlessness, nausea, vomiting, bowel urgency, diarrhea, and erythema. Dr. McCusker focused on the initial symptoms, which would manifest in infants as fussiness, crying, restlessness and refusal of food. Supra pp. 25-28.

Petitioners and their expert fail to cite to anything in the record to support their assertion that Kaleaf's death was actually caused by cytokine storm or an otherwise aberrant immune

[64] As discussed previously, the theory that Kaleaf was immune compromised due to prematurity actually harms petitioners' case in regards to Althen prong one. See supra pp. 29-30. As explained by Dr. McCusker, if he was immune compromised, his innate system would not have been able to respond to the point to induce an aberrant cytokine reaction and/or his adaptive system would have cleared any alleged chronic, low-grade infection since the adaptive system is altered but not hindered. Id.

response. It appears that, based on Dr. Levin's tenuous interpretation of the certain aspects of the pathology evidence and the simple fact that death occurred, Dr. Byers has assumed an abnormal immune response, cytokine storm or cytokine burst occurred without any evidence. See Moberly, 592 F.3d at 1324 ("Even setting aside the question of whether the [expert's] theory was reliable, [the expert] conceded that there was no evidence in the record suggesting that the proposed mechanism was at work in [the child's] case.").

Prior to finding Kaleaf unresponsive, there was no evidence that he was fussy, crying, uncomfortable, restless or otherwise *in extremis* following his vaccinations. In fact, Kaleaf took a bottle and was put down for a nap without consequence. If the vaccines or their components were hypothetically a strong enough stimulus to cause a cytokine storm or burst, Dr. McCusker testified that "you would have expected to see signs, and in this situation the baby came home, there was not any swelling or pain or tenderness noted at the site. The baby was not irritable. He fed well. No evidence of fever in this child. There's no evidence that this child did anything other than go for his nap." Hr'g Tr. 446:23-447:6. Dr. McCusker was clear: the "**clinical signs and symptoms associated with massive cytokine release are not subtle**." R Ex I at 5 (emphasis in original). In light of this lack of evidence, Dr. McCusker went on to explain that there is no evidence in medical literature that a "silent" cytokine storm, one occurring in an absence of symptoms, exists. Hr'g Tr. 447:7-12.

Despite the fact that Kaleaf's situation was fatal, petitioners are unable to show that Kaleaf actually suffered a cytokine storm that lead to his death.

d. Evidence of right heart failure

As discussed previously, supra p. 7-8, Dr. Levin testified that Kaleaf suffered right heart failure due to the circulatory resistance caused by inflammation in his lungs. On the first day of the Hearing, Dr. Levin noted red cells on the slides of Kaleaf's lower lungs, P Ex 27 at 2, which denotes congestion and right heart failure. Hr'g Tr. 149-50. In response, Dr. Gilbert-Barness stated one would expect to find changes in the heart and lungs – abilitation of the right ventricle and heart failure cells or hemosiderin-laden histriocytes – which were not present in Kaleaf. Hr'g Tr. 234. Dr. Gilbert-Barness also stated the right side of the heart would be "significantly dilated." Hr'g Tr. 370:25-371:1. She noted no dilation or abnormality of the right heart ventricle was found during the autopsy. Hr'g Tr. 371:10-18. Further, "[t]he lungs would be congested, as in part they were, but much more so in right heart failure, and the liver would be congested, as it was, but the overall appearance was not one of right heart failure." Hr'g Tr. 371:1-5. "There's just no mistaking right heart failure. There would be edema in the lungs, which was very minimal, and it was just not right heart failure." Hr'g Tr. 371:6-8. The undersigned notes that the medical examiner also did not note evidence of right heart failure. See P Ex 5. When asked if the medical examiner would note right heart failure or the indicative changes seen with it, Dr. Gilbert-Barness stated, "[y]es, you would have expected that. In fact, he mentions the thickness of the ventricles and they were within normal limits, and the heart essentially appeared to be normal from the gross description and from the autopsy report." Hr'g Tr. 371:19-372:2. With the exception of the slight increase in organ weight, petitioners made no assertion regarding a dilation or abnormality of the heart that would be seen with heart failure.

In response, Dr. Levin stated the cells, hemosiderin-laden histiocytes, would be present in **chronic** heart failure, not in the **acute** heart failure to which he alleged Kaleaf succumb. Hr'g

Tr. 358:16-359:22. Following the hearing, Dr. Levin provided an untitled excerpt of medical literature which states, "[a]cute passive congestion without hemosiderin deposition or alveolar septal fibrosis" is the microscopic finding with severe, acute **left** heart failure. P Ex 28, filed Feb. 9, 2011 (emphasis added). The undersigned notes that this piece has no context, as it is an excerpt, and discusses **left** heart failure. No testimony was given to confirm the same findings would be had with acute **right** heart failure. As noted before, when questioned by respondent, Dr. Levin admitted that he had limited experience with heart failure in premature infants. "I've seen it I think once before." Hr'g Tr.360:4-361:7. He further admitted that this one experience was decades prior. Id.

Reinforcing Dr. Gilbert-Barness' testimony, examination of the autopsy report shows the medical examiner noted the external surface of the heart was unremarkable. P Ex 5 at 7. The right ventricle was within normal limits. Id. No dilation or abnormality was noted. Id.; see also id. at 13 (lacking a note of any abnormality with the heart).

Assuming petitioners' Exhibit 28 supports Dr. Levin's assertion that absence of the hemosiderin-laden histiocytes is evidence of acute **right** heart failure, the undersigned still does not find petitioners' proposition of right heart failure persuasive. In light of the testimony from Dr. Gilbert-Barness, who has immense experience with pediatric pathology, and a complete absence of notations from the medical examiner of any heart abnormality, the undersigned does not find Dr. Levin's testimony persuasive regarding evidence of right heart failure.

e. Increased organ weight

Another portion of petitioners' case was that the weight of many of Kaleaf's organs, being higher than normal in several instances and congested to some extent, was evidence that he suffered the type of injury and death they allege due to massive inflammation.

Dr. Levin characterized Kaleaf's lungs as "outrageously heavy" and "congested with blood." Hr'g Tr. 133:6-9. Dr. Levin characterized the thymus as large and also congested with blood. Hr'g Tr. 133:23-24. Following the inflammation and pulmonary resistance in the lungs, in Dr. Levin's opinion, the right side of Kaleaf's heart "failed, and then the left heart was still pumping blood, so the blood was going to the kidney or liver, the spleen and the lungs, but it can't come out because the right heart isn't working;" to Dr. Levin, this explains why the autopsy showed heavy organ weights. Hr'g Tr. 150:3-13. At one point in his testimony, Dr. Levin answered in the negative when asked if the heavy weight of Kaleaf's organs could be explained by a hypoxic death and repeats this answer later in the Hearing. Hr'g Tr.156:23-157:4; Hr'g Tr. 158:7-159:5. As noted previously, this is slightly contradictory since Dr. Levin stated on the second day of the Hearing that, generally speaking, both parties were advancing a theory of an asphyxial death. Hr'g Tr. 356:10-13. In the circumstances of petitioners' theory, Dr. Levin states this was a process, the right heart failure, caused a "bottleneck" of fluid, allowing the tissues to fill up and become congested. Hr'g Tr. 356:18-357:19. Dr. Levin's opinion is that this "bottleneck" and congestion would not occur in the theory proffered by respondent that Kaleaf died of positional asphyxiation within a matter of minutes. Hr'g Tr. 357:6-358:15.

In contrast, Dr. Gilbert-Barness noted, "[t]he organ weights are slightly increased . . . ." Id. at 1-2. "These are values that would be seen and are consistent with congestion which is

evident in the microscopic sections I examined and entirely consistent with asphyxiation in addition to hyperaeration of the lungs." R Ex A at 2. Dr. Gilbert-Barness explained that this finding is "absolutely consistent" with an asphyxial death. Hr'g Tr. 224:5-12. Dr. Gilbert-Barness disagrees with Dr. Levin's characterization of the organs as massively heavy. Hr'g Tr. 224:13-225:11. Regarding the mild weight increases in many of Kaleaf's organs, Dr. Gilbert-Barness further testified that the fluids administered during the attempted resuscitation efforts would have contributed to the increased organ weights. Hr'g Tr. 372:3-12. Also, Kaleaf's size, in the 97th percentile for his age at his time of death, would also correlate with organ weight. Hr'g Tr. 372:13-23 ("There is a correlation between body weight and organ weight, and that is exemplified particularly in infants of diabetic mothers where the infant overall weight may be high and that corresponds to increased weight of organs."). Regarding the findings in an asphyxial death, "the intensity of the condition is much greater than what one would normally see in any other sort of death. That I think in this case is exemplified by the increase of weight of several of the organs, and that I can document from my own text, the Handbook of Pediatric Autopsy." Hr'g Tr. 374:19-25. "On page 485, there is a description of asphyxia, and very clearly it's stated that there is intense venous congestion of the organs, which certainly would account for the mild increase of the weights of the organs." Hr'g Tr. 375:1-6. Respondent further provided literature showing the heavy organs were, in fact, not inconsistent with a SIDS death. This was the opposite of what was hypothesized by Dr. Levin. See R Ex O-1 at 1 (noting organ weights are "significantly greater than published norms" of the thymus, liver, lungs, and brain in infants dying of SIDS and further discussing that organ weights more closely correlate with body weight than with age). R Ex O-1 at 1.

Reviewing petitioners' evidence regarding the heavy organ weights and congestion, the undersigned first sees clear contradiction in Dr. Levin's testimony. He testified that the organs were heavy and asphyxiation cannot explain heavy organs. However, confusingly, he states that both parties are alleging an asphyxial death. Dr. Levin's testimony on this issue simply made little sense. Regardless, respondent's supportive literature showed that increased organ congestion and weight are found in deaths attributed to SIDS and not just in asphyxiation by right heart failure, as alleged by petitioners. In the final analysis, as discussed above, respondent provided clear evidence and testimony that the organ weights found are not inconsistent with SIDS, which was the cause of death found by the medical examiner, and are consistent with Kaleaf's size at the time of his death.

For the numerous reasons discussed, petitioners fail to provide preponderant evidence of a logical sequence of cause and effect showing that the vaccines were the cause of Kaleaf's death.

III. ALTHEN PRONG III – MEDICALLY APPROPRIATE TIMEFRAME

Despite finding petitioners' case has not met their burden under prongs one and two of Althen, the undersigned will review the evidence regarding the appropriate temporal relationship for the sake of completeness. The third prong of Althen is "showing of a proximate temporal relationship between the vaccination and injury." In de Bazan v. Sec'y of the Dept. of Health & Human Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008), the Federal Circuit explained that "the proximate temporal relationship prong requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." Id. (citing Pafford, 451 F.3d 1352,

1358; Althen, 418 F.3d at 1281). The petitioner has the burden of proof. While petitioners were able to show a literal temporal association, the petitioners faltered in showing evidence of a medically-accepted temporal relationship between the vaccination and the alleged injury. Pafford, 451 F.3d at 1358; see also de Bazan, 539 F.3d at 1352.

In Kaleaf's case, he arrived at his doctor appointment on September 19, 2005, at 10:04 am. P Ex 25; Hr'g Tr. 9:21-10:8. Kaleaf's mother estimated he was seen by the doctor approximately five or six minutes later, Hr'g Tr. 10:17-10:21. The doctor performed an exam and conversed with the mother, then called in a nurse to administer the vaccinations. Hr'g Tr. 10:22-11:9. One could assume the entire appointment, since the exam was relatively normal, lasted ten to twenty minutes, ending around 10:25 am or 10:30 am; thus, Kaleaf likely received his vaccinations between 10:15 and 10:30 am that day.

Following the immunizations, Kaleaf's mother telephoned for a ride to return her and Kaleaf to their home; when the ride arrived, she estimates the car ride was approximately five or six minutes. Hr'g Tr. 11:10-12:3. The time they waited for the ride was not discussed so the undersigned estimates they arrived home around 10:40 am. Formula was prepared for Kaleaf, his father left for the store and his mother gave him his bottle. Hr'g Tr. 12:4-9. His mother testified that Kaleaf was already asleep when she fed him the bottle and she burped him while he was sleeping. Hr'g Tr. 12:9-12. Following the burping, she took Kaleaf upstairs. Hr'g Tr. 12:13-13:9. She estimated that from the time they arrived home until the time she took Kaleaf upstairs, fifteen or twenty minutes had passed. Hr'g Tr. 13:3-9. This would mean Kaleaf was put down for his nap around 11:00 am or shortly thereafter. This coincides with testimony given by his mother and information taken following Kaleaf's death. Hr'g Tr. 14:6-19. The emergency phone call was placed at 12:44 pm after Kaleaf was found unresponsive. P Ex 7 at 1-5. His mother noted that she checked on him once between the time he was put down and when he was found unresponsive, approximately thirty to forty-five minutes into his nap approximately 11:45 am. Hr'g Tr. 15:1-8. Kaleaf's mother noted that she checked on him, rubbed him and that he was okay at that time. Hr'g Tr. 15:3-7. She estimated the second check on Kaleaf, when he was found unresponsive, was approximately thirty to forty-five minutes thereafter. Hr'g Tr. 15:8-12.

Although these were approximations from a very traumatic day, they appear consistent with times recorded for the doctor appointment and the placement of the 911 call. Considering this sequence of events and times, the time between vaccination, approximately 10:15 am, and when Kaleaf was unexpectedly found unresponsive, shortly before the 12:44 pm 911 call, was approximately 150 minutes. Petitioners' expert, Dr. Byers, accepted the estimation of the time between vaccination and death as approximately two to two and a half hours. Hr'g Tr. 63:8-18; see also P Ex 11 at 3 (noting an approximate time lapse of 2 ¾ hours). Notably, nothing out of the ordinary was noted before finding Kaleaf unresponsive around 12:44 pm. In fact, everything seemed normal up to the point Kaleaf was first checked in on, approximately 11:30 or 11:45 am that day.

Discussion of what is a medically appropriate time for this injury and death was given by Dr. Byers and centered upon petitioners' Exhibit 12, the New England Journal of Medicine article titled that chronicled an inadvertent cytokine storm in study patients. Notably though, Dr. Byers acknowledged that the article did not support lung involvement, the substance of petitioners' theory of asphyxiation, in under five hours. Hr'g Tr. 90:3-91:17.

Examining this article, six test subjects received an infusion that caused the cytokine storm. P Ex 12 at 1-2. Regarding the study patients' initial response, the article notes that the onset of symptoms was a severe headache fifty to ninety minutes after infusion in five of the six patients. Id. at 2. These are also initial, general symptoms of a robust cytokine response as discussed by Dr. McCusker. This was followed closely by lumbar pain in all six patients between fifty-seven and ninety-five minutes after the infusion. "Subsequently, during this early phase, the patients were restless and had varying degrees of nausea, vomiting, bowel urgency, or diarrhea." Id. "Five subjects had short amnestic episodes associated with severe pyrexia,[65] restlessness, or both. All patients had systemic inflammatory response that included erythema and peripheral vasodilation (the timing of which was undocumented), with recorded rigors in four patients at a median of 59 minutes (range, 58 to 120) after infusion." Id. "Hypotension . . . developed in all patients a median of 240 minutes (range, 210 to 280)[, approximately 4 hours] after infusion, accompanied by tachycardia, with maximal heart rates of 110 to 145 beats per minute." Id. "At 300 minutes after infusion, Patient 1 had signs of respiratory failure, with tachypnea . . . ." Id. A timeline on page 5 of Exhibit 12 shows the onset of the symptoms in the study patients.

Dr. Byers testified the study patients suffered hypotension within ninety minutes. Hr'g Tr. 64:4-6. This appears to be in conflict with the study, which notes hypotension beginning around four hours after infusion, as discussed above. P Ex 12 at 2. Later in her testimony, Dr. Byers agreed that respiratory distress began approximately 300 minutes after infusion. Hr'g Tr. 90:13-18. When asked why it was reasonable that Kaleaf's respiratory problems arose in much less time, two and a half hours, Dr. Byers stated, "I do not have temporal support in the literature to account for a child that had a low-grade viral pneumonia because these were all healthy teenagers [in the study]." Hr'g Tr. 91:2-5.[66] Dr. Levin, without more specificity, noted that the changes he observed in the slide of Kaleaf's brain, allegedly due to cytokine release and subsequent inflammation, would take hours. Hr'g Tr. 153:25-155:22.

The undersigned notes that Dr. McCusker described the drug used in this study, which was directly infused into the patients' veins and the cause of the cytokine storm, to be beyond a typical superantigen; she characterized it as a "super-super-super antigen" that stimulated approximately 60% of the patients' T-cells while a typical super antigen stimulates only approximately 10-20%. See supra p. 28. Even with this extreme assault, it took almost an hour for an initial symptom of a headache to arise and almost five hours to affect the lungs. Hr'g Tr. 437:22-438:4.

As noted in the study and explained by Dr. McCusker, a most severe cytokine storm did not result in respiratory complications for four to five hours. Petitioners' expert, Dr. Byers, seems to account for the faster reaction in Kaleaf by speculating that the alleged chronic lung infection and, or, his prematurity affected the speed of the reaction. Hr'g Tr. 90-92. As discussed there is not persuasive evidence that Kaleaf suffered from a chronic lung infection or from immune compromise due to his premature birth. Further, even if Kaleaf was immune deficient, Dr. McCusker explained why such a condition would actually lessen the likelihood of

[65] Pyrexia is fever. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 1586 (31st ed. 2007).

[66] Respondent also notes that the study participants were not teenagers but were adults. R Post-Hearing Brief at 16 (citing P Ex 12 at 1019).

a robust and potentially harmful cytokine release due to the way in which premature infants are believed to be immune deficient. See supra pp. 29-30.

In the present case, petitioners have not shown that the vaccines or vaccine components Kaleaf received are traditional superantigens, let alone that they are the type of superantigens capable of the intense effect seen after several hours in the cited study, petitioners' Exhibit 12. Petitioners' own expert acknowledged that this article, upon which she relied for her opinion, did not support lung involvement in under five hours. Further, petitioners' attempts to explain the faster immune response by Kaleaf's prematurity were illogical and unpersuasive when considered alongside Dr. McCusker's supported testimony on how the alleged immune deficiency is thought to work in premature infants. Petitioners here fail to show that the time frame between vaccination and injury was medically appropriate and, thus, fail to provide "preponderant proof that [Kaleaf's death] occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352.

IV. FINDING A FACTOR UNRELATED, SIDS OR POSITIONAL ASPHYXIA, IS UNNECESSARY

Given the findings above that petitioners' evidence of causation was woefully short of preponderant evidence of causation, it is unnecessary to address the role of SIDS or positional asphyxia as either rebuttal evidence, Doe/11 v. Sec'y of the Dept. of Health & Human Servs., 601 F.3d 1349 (Fed. Cir. 2010), or as a factor unrelated. However, it is noted that evidence pertaining to SIDS and positional asphyxia was strong and persuasive. A very brief discussion follows.

Petitioners' experts disagree with the final diagnosis of SIDS found in the medical records. P Ex 10 at 3 ("These findings are inconsistent with aspiration pneumonia and SIDS . . . ."); P Ex 11 at 4 ("The SIDS diagnosis provide by the Medical Examiner was, absolutely wrong. The reasons are detailed in Dr. Levin's report."). Dr. Levin, in his conclusion, simply references his findings and states they are inconsistent with SIDS. P Ex 10 at 3. Dr. Byers states that death by SIDS is "histologically silent" and references the findings of Dr. Levin. P Ex 11 at 4. The testimony of respondent's experts and the submitted literature show that SIDS is not always and not necessarily "histologically silent." Review of one article on SIDS, R Ex H1,[67] shows petitioners' expert's assertion, that SIDS is histologically silent, is not true. R Ex H1 at 8-9 (noting, among other commonly encountered tissue findings, petechial hemorrhage on surface of thymus, pleural and epicardium; hyperinflated lungs with pulmonary congestion and edema; mild interstitial lymphocytic infiltrates and intraalveolar neutrophils; astrogliosis, delayed myelination, delayed maturation, megalencphaly, changes in the vagus nerve in the central nervous system; etc.).

Kaleaf's very tragic death in this case was attributed to SIDS by the medical examiner. P Ex 5.[68] Petitioners' experts actually introduced a discussion of SIDS in their original reports

[67] R Ex H1, Enid Gilbert-Barness and Diane E. Debich-Spicer, AUTOPSY PATHOLOGY (Humana Press 2004).

[68] Dr. Gilbert-Barness explains that the term SIDS in coroner's reports is frequently used when there is positional asphyxiation and that the major risk factor for SIDS is sleeping in the prone position. R Ex A at 2.

opining the vaccinations were causative; notably they both voiced their strong disagreement with that finding. P Ex 10 at 3; P Ex 11 at 4. Much like petitioners in Doe/11, petitioners' experts here attempted to distinguish Kaleaf's case from that of an infant dying of SIDS or positional asphyxia. Doe/11, 2008 WL 4899356, *19. It should be noted that no treating doctor blamed the vaccination for Kaleaf's death. See P Ex 5.

Throughout the evidence, however, respondent's experts opined consistently and persuasively that the evidence was consistent with a SIDS or positional asphyxiation. Respondent also provided medical literature supportive of this position. Dr. Gilbert-Barness' own textbook on pediatric pathology, R Ex H-1, confirms that Kaleaf faced several risk factors: found in a prone sleeping position, smoking and drug abuse, soft bedding, and his race. R Ex H1 at 5. Petitioners' case against SIDS mostly rests, in regards to the pathology opinion, on the increased weight of Kaleaf's organs at the time of his death and Dr. Levin's reading of the pathology slides. Petitioner's experts refer to tables of normal organ weights for the comparison. However, respondent's pathology expert testified that Kaleaf's organ weights were not so far outside the normal range to be of concern; she testified that she would consider an abnormal increase in weight to be twice the normal weight. See supra p. 21, n. 38. Further though, as testified by Dr. Gilbert-Barness and found in Respondent's Exhibit O1,[69] heavy organ weights are not unusual in SIDS. See supra p. 19; R Ex O1, filed Feb. 10, 2011. The article notes "significantly greater than published norms" regarding weight of the thymus, liver, lungs, and brain in infants dying of SIDS and further discusses that organ weights more closely correlate with body weight than with age. R Ex O1 at 1. This article also notes that organ weights more closely correlate with body weight than with age. R Ex O1 at 1. As discussed previously, Kaleaf was between the 95th and 99th percentile in size. P Ex 5. Table 1 on page 4 of the Exhibit notes average size and organ weights by age for infants who died in possible, probable and classic SIDS; review of that Table shows that Kaleaf's organ weights were found within or only slightly higher than the average found in the study for three and four month old infants.[70] Compare R Ex O1 at 4-5, Table 1; with P Ex 5. The organ weights found in Kaleaf were more similar to these suspected or classic SIDS cases than they were when compared to normal table of organ weights. The undersigned first reiterates that the medical examiner listed the cause of death as SIDS. P Ex 5.

The undersigned notes that, given her vast knowledge and experience regarding SIDS and positional asphyxia, Dr. Gilbert-Barness was better qualified to testify in this case and was more persuasive in comparison to petitioners' expert. Considering her testimony coupled with the evidence submitted regarding SIDS and positional asphyxia, the undersigned believes that there is no persuasive evidence in this case that is inconsistent with Kaleaf's cause of death being SIDS, as determined by the medical examiner. However, as it is unnecessary to this proceeding, the undersigned makes no finding regarding the cause of Kaleaf's death beyond that it was not related to the vaccinations he received on September 19, 2005.

CONCLUSION

[69] R Ex O-1, Joseph Siebert and Joel Haas, Organ Weights in Sudden Infant Death Syndrome, 14 PEDIATRIC PATHOLOGY 973 (1994).

[70] Kaleaf was three months and twenty-seven days old. See, e.g., P Ex 5 at 6.

This is a tragic case of an infant death. However, as detailed above, petitioners failed to produce preponderant evidence that the vaccines caused that death. For the foregoing reasons, petitioners' are denied compensation under the Act and the Petition is dismissed. The Clerk of the Court is directed to enter judgment accordingly.

IT IS SO ORDERED.[71]

s/ Gary J. Golkiewicz
Gary J. Golkiewicz
Special Master

[71] This document constitutes a final "decision" in this case pursuant to 42 U.S.C. § 300aa-12(d)(3)(A). Unless a motion for review of this decision is filed within 30 days, the Clerk of the Court shall enter judgment in accord with this decision.