

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 19-690V

Filed: August 8, 2025

NICHOLAS CATONE and MARJORIE
CATONE, parents of N.C., deceased,

Petitioners,

v.

SECRETARY OF HEALTH AND
HUMAN SERVICES,

Respondent.

Special Master Horner

Jessica Wallace, Siri & Glimstad, LLP, Aventura, FL, for petitioners.
Catherine Elizabeth Stolar, U.S. Department of Justice, Washington, DC, for respondent.

DECISION¹

On May 9, 2019, petitioners filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),² on behalf of their son, N.C. (ECF No. 1.) Petitioners allege that N.C.'s death on May 12, 2017, determined upon autopsy to be a sudden unexplained death, was caused by his diphtheria-tetanus-acellular-pertussis ("DTaP") vaccination administered on April 25, 2017. (*Id.*)

On November 30, 2023, petitioners filed a motion for a ruling on the written record finding them entitled to compensation for N.C.'s death. (ECF No. 85.) Petitioners contend that N.C.'s April 25, 2017 DTaP vaccine caused him to suffer from a witnessed seizure on May 7, 2017, and then an unwitnessed terminal seizure late on

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

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

May 11 or early on May 12, 2017. (ECF No. 85, pp. 25-26.) They theorize that N.C.'s vaccination "initiated a hyperimmune reaction that eventually led to a terminal seizure in N.C., a susceptible host." (*Id.* at 29.) Respondent opposes the motion, contending that the case should instead be dismissed for insufficient proof and stressing that N.C.'s death was appropriately categorized as a sudden unexplained death. (ECF No. 92.)

When sudden deaths remain unexplained after examination and investigation, such deaths are deemed either "SIDS" (Sudden Infant Death Syndrome) or "SUDC" (Sudden Unexplained Death in Childhood). (Ex. 13, p. 9.) The term SIDS is applied to infants under one year of age and the term SUDC is applied to children over the age of one, usually affecting children between ages one and three. (*Id.*) Some additional cases of sudden unexplained death are categorized as "SUDEP" (Sudden Unexpected Death in Epilepsy) when the decedent had a history of epilepsy. (Ex. 18, p. 21.) Because N.C. was over one year of age at the time of his death and with no prior history of epilepsy, his death would be termed SUDC based on his autopsy and the investigation into his death. (Ex. 18, pp. 8-9; Ex. 19, p. 10.) SIDS and SUDC are "diagnoses" of exclusion in that they by definition require that the death remain otherwise unexplained. (Ex. 13, p. 9.) Both phenomena usually occur during sleep and affect boys more often than girls. (*Id.*) Safe sleeping campaigns have been effective in reducing the rate of SIDS. (*Id.*) However, SIDS is the leading cause of infant mortality in the U.S., affecting 0.53 per 1,000 infants. (*Id.*) SUDC is rarer than SIDS, affecting 1.3 per 100,000 children (ages 1-4). However, it still accounts for 10% of unexpected deaths in childhood. (*Id.*)

Petitioners clearly suffered a profound loss, and I offer my sincerest condolences. However, for all of the reasons discussed below, there is not preponderant evidence that N.C.'s death was caused by his vaccination. Instead, the evidence preponderates in favor of a finding that N.C.'s death remains unexplained and therefore constitutes a SUDC. Thus, petitioners are *not* entitled to an award of compensation.

I. Applicable Statutory Scheme

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

In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to

the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A)-(B); § 300aa-11(c)(1)(C)(i); § 300aa-14(a). In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-11(c)(1)(C)(ii). In such a situation, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

In this case, petitioners have not alleged that the vaccination at issue caused any injury or condition listed on the Vaccine Injury Table. Therefore, petitioners must meet the burden of proof for establishing causation-in-fact. (*E.g.*, ECF No. 85, p. 56 (stating that "[p]etitioners are aware that they have a heavy burden to carry in order to prove an off-table claim").)

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

A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully

evaluated. § 300aa-13(b)(1). A petitioner may rely upon circumstantial evidence. See *Althen*, 418 F.3d at 1280. The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id.* at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019).

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

II. Procedural History

This case was initially assigned to another special master. (ECF No. 6.) Accompanying the petition, petitioners filed affidavits (Exs. 1-2) as well as N.C.'s pediatric care records (Ex. 3). Shortly thereafter, they filed N.C.'s death certificate. (ECF No. 8; Ex. 4.) In July of 2019 they filed autopsy and police reports (ECF No. 13; Exs. 5-6), and in August of 2019 they filed additional medical records, including the EMS report relating to N.C.'s death (ECF No. 15; Exs. 7-10) as well as scene and autopsy photographs related to the investigation of N.C.'s death (ECF Nos. 16-17; Exs. 11-12).

Petitioners were then given the opportunity to submit an expert opinion. In July of 2020 they filed a report and supporting literature by immunologist Omid Akbari, Ph.D. (ECF No. 28; Exs. 13-14.) In August of 2020 they filed a case review from the SUDCRRC (ECF No. 31; Ex. 15) and a nanodiagnostic report (ECF No. 31; Ex. 16). And, in September of 2020, they filed a report and supporting literature by pathologist Steven Rostad, M.D. (ECF Nos. 32-33; Exs. 17-18). Thereafter, respondent filed his

Rule 4 report recommending against compensation (ECF No. 41), along with responsive expert reports by pediatric immunologist Christine McCusker, M.D. (ECF No. 38; Exs. A-B), pediatric neurologist Gregory L. Holmes, M.D. (ECF No. 39; Exs. C-D), and pathologist Brent Harris, M.D., Ph.D. (ECF No. 40; Exs. E-F).

After the filing of respondent's report, the special master assigned to the case held a Rule 5 status conference to provide the parties preliminary guidance. (ECF No. 43.) The special master cautioned that, based on his review of the expert opinions, it would be difficult for petitioners to establish entitlement to compensation, especially in light of the *Boatmon v. Secretary of Health & Human Services*, 941 F.3d 1351 (Fed. Cir. 2019), a decision in which the Federal Circuit rejected the allegation that vaccination had contributed to a death categorized as SIDS. (ECF No. 43.) Nonetheless, he permitted petitioners an opportunity to have their experts respond to respondent's expert submissions. (*Id.* at 4.) Petitioners then filed supplemental expert reports by Drs. Akbari and Rostad in August of 2021. (ECF Nos. 45-46; Exs. 19-20.) In January of 2022, respondent filed further responsive reports from Drs. McCusker and Harris. (ECF Nos. 53-54; Exs. G-H.) Petitioner filed additional reports by Drs. Akbari and Rostad in February of 2022. (ECF No. 55; Exs. 21-22.)

The case was subsequently reassigned to the undersigned in March of 2022. (ECF Nos. 58-59.) Petitioners filed autopsy photos annotated by Dr. Rostad (ECF No. 60; Ex. 23) and requested a status conference, which was held in April of 2022. (ECF No. 62.) During the status conference, I advised that I felt this case may be appropriate for resolution on the written record; however, respondent was permitted an opportunity to determine if his expert(s) needed to respond to the annotated autopsy photos. (*Id.*) Respondent then filed an additional expert report by Dr. McCusker in September of 2022 (ECF No. 67; Ex. I), but no report responding to the photos.

During a follow up status conference in December of 2022, petitioners initially indicated they anticipated filing further expert reports (ECF No. 68); however, they instead advised in a status report filed February 27, 2023, that they wished to secure testimony from Dr. Ian Hood, the medical examiner that performed N.C.'s autopsy. (ECF No. 72.) Petitioners filed a motion seeking to stay or extend their deadlines so that they could pursue a deposition of Dr. Hood. (ECF No. 73.) During a follow up status conference, I granted petitioners' request in part and deferred in part. (ECF No. 74.) Specifically, I suspended petitioners' pending expert report deadline but required an offer of proof regarding the nature of Dr. Hood's opinion, and whether he intended to amend his official report. (*Id.*)

On June 12, 2023, petitioners filed an "addendum" to N.C.'s autopsy report (ECF No. 77; Ex. 24), and I directed the parties to file status reports indicating their respective positions regarding how to proceed (Non-PDF Scheduling Order, filed June 12, 2023). Petitioners argued that Dr. Hood's addendum rebutted points made by respondent's experts and that a deposition was therefore necessary for a fair and well-informed ruling. (ECF No. 79.) Respondent argued that a deposition was not reasonably necessary because Dr. Hood explicitly disclaimed having the knowledge necessary to

opine regarding petitioners' experts' theory of causation. (ECF No. 82.) I denied petitioner's request and instructed the parties to brief the case pursuant to Vaccine Rule 8(d). (ECF No. 83.) However, I allowed the parties to accompany their briefs with expert reports commenting on Dr. Hood's addendum to his report.³ (*Id.* at 3.)

Petitioner filed a motion for a ruling on the written record on November 30, 2023. (ECF No. 85.) Petitioners did not include any expert report with their motion; however, they subsequently filed a motion for leave to file newly published literature and a brief expert report. (ECF No. 87.) They filed a study regarding video analysis of sudden unexplained death in toddlers accompanied by a report by Dr. Rostad commenting on the study. (ECF No. 90; Ex. 25.) Petitioner's filing was permitted, and respondent was directed to file his response to petitioner's motion, accompanied by any expert response to petitioner's filing. (ECF No. 89.) Respondent filed his response to petitioner's motion on June 3, 2024 (ECF No. 92), along with a supplemental report by Dr. Holmes (ECF No. 91; Ex. J). Petitioners filed their reply brief on August 31, 2024. (ECF No. 96.)

In light of the above, I have concluded that the parties have had a full and fair opportunity to develop the record and that it is appropriate to resolve this case without an entitlement hearing.⁴ See *Kreizenbeck v. Sec'y of Health & Human Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020) (citing *Simanski v. Sec'y of Health & Human Servs.*, 671 F.3d 1368, 1385 (Fed. Cir. 2012)); see also Vaccine Rule 8(d); Vaccine Rule 3(b)(2). In total, the parties filed 314 exhibits, most of which are medical literature articles cited in the numerous expert reports submitted in this case. Although this decision discusses some but not all the literature in detail, the undersigned reviewed and considered all of the medical records and literature submitted in this matter. *Accord Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to

³ Specifically, I concluded that a deposition of Dr. Hood was not reasonably necessary for two reasons. First, I explained that "Dr. Hood's amendment to his report offers several specific statements with respect to N.C.'s autopsy findings. (ECF No. 83.) However, petitioner's status report cites these statements for the effect they contend the statements should have on interpreting *the experts'* reports rather than the autopsy report itself." (*Id.* at 2 (emphasis original) (internal citation omitted).) Second, "Dr. Hood further states that 'the most I can opine is that if [N.C.] had a terminal seizure his autopsy findings would be just as were present in this case. Whether his immunizations contributed in some way to causing such a postulated terminal seizure is beyond my area of expertise.' This strongly suggests that Dr. Hood has provided as much information as he believes he can." (*Id.* at 2-3 (quoting Ex. 24, p. 5).)

⁴ In this case, I advised the parties that I was "inclined to rule on the written record, potentially even over petitioners' objection, given that the issues presented are familiar within the program." (ECF No. 68, p. 1.) However, I instructed that "[t]o the extent that petitioners' object to proceeding with a ruling on the written record, petitioners may address such arguments in their brief. If I were to ultimately agree to hold a hearing in light of petitioners' arguments, then petitioners' brief would serve as a prehearing brief." (*Id.* at 2.) In their subsequent briefing, petitioners did not argue that they had been denied a full and fair opportunity to develop the record, request or argue in favor of an entitlement hearing, or object to proceeding on the written record pursuant to Vaccine Rule 8(d). (See ECF Nos. 85, 96.)

discuss every piece of evidence or testimony in [his or] her decision.” (citation omitted)), *aff’d*, 601 F. App’x 982 (Fed. Cir. 2015).

III. Factual History

N.C. was born on September 8, 2015, after an uncomplicated pregnancy. (Ex. 1, ¶ 3; Ex. 3, p. 7; Ex. 8, pp. 43-44.) His newborn screening labs were normal. (Ex. 7, p. 12.) N.C. initially had neonatal jaundice, abnormal weight loss, and umbilical granuloma; however, by the time of his one month well visit, he had a normal examination. (Ex. 3, pp. 7-10, 34-35.) Thereafter, N.C. had routine health encounters and received his routine vaccinations. (Ex. 10, pp. 17-63.) He also had some sick visits. (Ex. 10, p. 19 (November 16, 2015 encounter for rash); Ex. 3, pp. 17-19 (April 27, 2016 encounter for upper respiratory infection); Ex. 3, pp. 20-21 (June 11, 2016 encounter for upper respiratory infection); Ex. 3, pp. 23-24 (July 26, 2016 encounter for upper respiratory infection); Ex. 3, pp. 26-28 (January 3, 2017, encounter for bronchitis); Ex. 3, pp. 29-31 (February 3, 2017 encounter for bronchiolitis, upper respiratory infection, and left ear infection).) In her affidavit, Mrs. Catone, a registered nurse, recalled that

N.C. was a healthy baby but in the winter of 2016 when he was just over a year old, I noticed that N.C. always seemed to be sick with a cold and occasional fevers but these symptoms almost always came shortly after visiting the pediatrician for a well visit and vaccinations.

(Ex. 1, ¶ 7.) Mr. Catone echoed this observation.⁵ (Ex. 2, ¶ 5.)

On November 9, 2015, N.C. received a Pentacel vaccination, which includes DTaP as well as polio and Hib. (Ex. 3, pp. 36-37, 55.) One week later, on November 16, 2015, he presented to his pediatrician with a 3-to-5-day history of a rash over his face, back, chest, and abdomen, which was diagnosed as a “[r]ash and other nonspecific skin eruption.” (*Id.* at 11-12.) The parties disagree as to whether N.C.’s presentation was an adverse reaction to the Pentacel vaccination. (*Compare* ECF No. 85, p. 42 (petitioners citing Dr. Rostad as opining an adverse reaction occurred), *with* ECF No. 92, pp. 2-3, 26-27 (respondent citing the medical record as containing no suspicion of a vaccine reaction).) N.C. received the vaccination at issue in this case, which was his fourth dose of DTaP, on April 25, 2017, during a well exam occurring at about nineteen and a half months of age. (Ex. 3, pp. 54-56.) At that time there were no complaints and N.C.’s physical exam was normal. (*Id.*)

Seventeen days following his DTaP vaccination, on the morning of May 12, 2017, police and emergency medical services responded to the Catone home at 8:46 AM regarding a report of a “20 month old baby who was reportedly unconscious and not breathing.” (Ex. 6, p. 11; Ex. 9, p. 9.) Upon arrival, it was noted that N.C. was “not

⁵ Notably, this specific statement and several other observations are included in both affidavits verbatim. (See Exs. 1-2.) While this fact alone does not lead me to conclude that these statements are untruthful, it does significantly undercut any notion that that these affidavits present two independent recollections.

breathing, was cold to the touch, and was clearly deceased.” (Ex. 6, p. 11.) His skin was observed to be cyanotic. (Ex. 9, p. 9.) At that time, Mrs. Catone was holding N.C. in the living room; however, the police secured N.C.’s bedroom pending investigation. (*Id.*; Ex. 6, p. 11.) Police scene photographs show that N.C. slept in a crib. (Ex. 11.) Mr. and Mrs. Catone provided accounts indicating that N.C. had been placed in his crib at around 7:30 PM or 8:30 PM. (Ex. 6, p. 11; Ex. 9, p. 9.) Mr. Catone recalled hearing N.C. fussing (sneezing and coughing) between 11:00 PM and 11:30 PM, which was the last activity heard. (*Id.*) The next morning, Mrs. Catone discovered N.C. lying face down in his crib, not breathing, and cold to the touch, and immediately called 911. (Ex. 6, p. 11.) The family reported that N.C. “had a normal day the previous day” and that “they did not notice anything suspicious or out of the ordinary.” (Ex. 9, p. 9.) N.C. was officially pronounced dead as of 9:08 AM based on the medic’s consultation with Dr. Azubuine of Newark Beth Isreal Hospital. (*Id.*; Ex. 6, p. 11.)

Later in the day on May 12, Mrs. Catone spoke with N.C.’s pediatrician. (Ex. 10, p. 69.) The pediatrician recorded the following history as provided by Mrs. Catone:

She said that [N.C.] has been running fever with no other s/sx, since the weekend (May 5/6? around 102 temp- no ER visits/office visits or phone calls done) and she was about to bring him in last Monday (5/8) if it persists. Monday came and [N.C.] was doing well and was afebrile and so no visit was done . . . Today she discovered him in his crib around 8:30 not breathing, stiff and purplish (on prone).

(*Id.*)

However, in her affidavit prepared for this case in May of 2019, Mrs. Catone indicated that N.C. developed a rash and cold symptoms within 24 hours of his April 25, 2017 vaccination, noting it to be “probably the worst cold I had seen him have.” (Ex. 1, ¶ 8.) She recalled he was “coughing and had an on and off runny nose but there was a lot of nasal discharge.” (*Id.*) Mrs. Catone did not indicate when these symptoms resolved. (*See id.*) However, she further recalled, consistent with the contemporaneous account above, that on Sunday, May 7, 2017, N.C. was lethargic and ran a fever of up to 103.9 Fahrenheit. (*Id.* ¶ 9.) She indicated that “he had never before seemed this lethargic.” (*Id.*) While it is unclear from the affidavits alone whether N.C.’s symptoms occurring 24 hours post-vaccination and his symptoms occurring on May 7 were one continuous illness or two distinct events, the more contemporaneous account to N.C.’s pediatrician clearly indicates that N.C. had been sick “since” the weekend prior to his death and that his febrile illness did not include any other significant symptoms. (Ex. 10, p. 69.) Moreover, although Mrs. Catone did not specify when any of the earlier symptoms resolved, Mr. Catone did specifically recall that the rash lasted only “a day or so.” (Ex. 2, ¶ 7.) Thus, the record as a whole favors the conclusion that the symptoms N.C. reportedly experienced post-vaccination resolved and did not persist up to the time of his later May 7 illness.

Mrs. Catone recalled being out running errands on May 7, 2017. (Ex. 1, ¶ 10.) Accordingly, Mr. Catone was with N.C. that afternoon. Mr. Catone recalled in his May 2019 affidavit that

N.C. napped that day and when he woke up from his nap he was still feverish. After his nap I was playing quietly with him on the bed and reading him a book. As I was reading to him, N.C. rolled over, closed his eyes and fell asleep in an unusual manner.

(Ex. 2, ¶ 10.) Both Mr. and Mrs. Catone recalled that he called her, and they agreed it was not normal. (*Id.*; Ex. 1, ¶ 10.) Mrs. Catone told Mr. Catone to wake N.C. up (Ex. 1, ¶ 10), following which Mr. Catone recalled

I tried to wake N.C. I shook his arm for a few seconds calling his name trying to wake him and he wasn't waking up. I remember feeling like my heart stopped in my chest and then a few seconds later he finally opened his eyes. It really scared me.

(Ex. 2, ¶ 10.) Thereafter, Mrs. Catone recalled giving N.C. Tylenol when she returned home, which broke his fever, and both Mr. and Mrs. Catone recalled that he seemed "fine and well" by the next day, though Mrs. Catone additionally recalled that he threw up during the morning. (*Id.* ¶ 11; Ex. 1, ¶¶ 11-12.)

N.C.'s autopsy was performed by Dr. Ian C. Hood on May 12, 2017. (Ex. 5.) Dr. Hood's summary indicated that N.C. "had experienced 24 hours of a mild febrile illness a few days before his death, but had been in his vigorous normal state of health thereafter and was throughout the day he was last known alive." (*Id.* at 16.) He noted that N.C. was found "obviously deceased with the early onset of rigor mortis in his crib with his buttocks raised and his head turned to one side and no evidence of obstruction of the airway although two blankets and three stuffed toys were found in the crib with him." (*Id.*) Dr. Hood explained:

Autopsy disclosed a robust and apparently well nourished and well cared for male child of at least 50th percentile development in all his growth categories; no significant injuries, skin lesions or anomalies; visceral congestion and mild pulmonary edema; a mildly swollen and somewhat "dusky" brain, but no intracranial hemorrhage. No other significant trauma or pathology was noted in or about the body and there were no significant findings on toxicologic, histologic, metabolic or microbiologic studies and death is attributed to sudden unexplained death, presumably from some mechanism such as a sudden cardiac dysrhythmia on the basis of a congenital conduction defect or "channelopathy" as is responsible for long QT syndrome.

(*Id.*) The official cause of death was listed as "sudden unexplained death" and the manner of death "natural." (*Id.* at 16, 36.) A Sudden Unexplained Infant Death

Investigation (“SUIDI”) Reporting Form was completed. (*Id.* at 26-33.) It indicates that Mrs. Catone noted that in the 72 hours prior to death, N.C. had a fever but marked “no” for “lethargy or sleeping more than usual.” (*Id.* at 29.)

The autopsy originally resulted in the creation of three glass slides of stained tissue in May of 2017. (Ex. 18, p. 2.) However, an additional 19 slides were later created in April of 2019. (*Id.*) Further, four additional paraffin blocks of brain tissue were preserved. (*Id.*) Five cassettes of brain tissue fixed in formalin were also created. (*Id.*) Both parties’ pathology experts have confirmed that they reviewed all of this material.⁶ (*Id.*; Ex. E, p. 2.)

N.C. was enrolled in a study by the SUDC Registry and Research Collaborative (“SUDCRRC”).⁷ (Ex. 5, pp. 85-88; Ex. 15.) As part of the SUDCRRC study, whole exome sequencing determined that N.C. did not have any genetic variants clinically significant to SUDC. (Ex. 5, p. 85.) Additionally, a family interview was conducted on October 12, 2017. (Ex. 15, p. 3.) In that interview, Mrs. Catone explained that in the “two weeks to 72 hours” prior to death, N.C.

experienced a fever, cold symptoms, and took medications, which included one dose of Tylenol and one dose of Motrin. [N.C.] reportedly had no symptoms in his last 72 hours. [Mrs. Catone] described [N.C.’s] overall health and demeanor during his last 48 hours as: “He was fine with not one problem. Happy, healthy, and eating well. Normal self. Playful in the nice weather at the boardwalk.”

(*Id.*)

In September of 2019, the Catones commissioned an analysis of N.C.’s postmortem tissue samples by “Nanodiagnosics srl,” a consulting firm. (Ex. 16.) A report was authored by Dr. Antonietta M. Gatti and Dr. Stefano Montanari. (*Id.* at 1.) Nanodiagnosics srl indicates that “[i]ts main activity is detecting inorganic micro- and nano-particles in any medium (biological tissues, food, drugs, cosmetics, environmental samples, garments, etc.), through an innovative system of environmental scanning-electron microscopy.” (*Id.* at 3.) Neither the authors’ credentials, nor the literature cited

⁶ However, the parties’ experts have raised a question as to how many slides the SUDCRRC, discussed below, had reviewed. (*E.g.*, Ex. E, p. 4; Ex. 19, p. 3.) Additionally, petitioners’ pathology expert raised an issue as to the failure of SUDCRRC and respondent’s expert, Dr. Harris, to review gross autopsy photos. (Ex. 19, p. 3.) However, Dr. Harris asserted that gross autopsy photos would not have changed his opinion. (Ex. 15, p. 5; Ex. H, p. 2.)

⁷ SUDCRRC analyzes cases of sudden unexpected death in children to identify and understand risk factors and causes, and to develop preventative measures. (Ex. 15, p. 1.) SUDCRRC reviews available records, conducts a family interview, reviews the death investigation, and performs genetic analyses and special studies. (*Id.*) The purpose of SUDCRRC is “to increase the understanding of the characteristics, circumstances, medical histories, and pathologies of children who have died suddenly and unexpectedly, and in some instances, without explanation.” (*Id.*)

in the report, have been introduced into the record of this case.⁸ The Catones provided Nanodiagnosics srl three tissue samples preserved in paraffin by the medical examiner, though only two were examined, one sample of brain tissue and one sample of brain tissue with pancreatic tissue. (*Id.* at 4, 82, 86.)

After examining the tissue samples using an electron microscope, Nanodiagnosics srl concluded that the tissue

revealed the presence of numerous foreign bodies of obvious exogenous origin. A few of them had a micrometric size, others a sub-micrometric (in the size range of nanoparticles). Some debris were trapped in a reaction biological tissue that contains only Carbon, Oxygen Sodium, Phosphorus and Sulfur. Other elements do not belong to the human organism and are, therefore, exogenous.

(Ex. 16, p. 86.) They further remarked that “[w]hat was rather unexpected was finding such a great quantity of foreign bodies in the brain and in the pancreas.”⁹ (*Id.* at 87.)

⁸ The report includes a reference list of 49 publications. (Ex. 16, pp. 90-94.) A single publication from that bibliography by Gatti and Montanari was filed in connection with Dr. Rostad’s first supplemental report. (Antonietta M. Gatti & Stefano Montanari, *New Quality-Control Investigations on Vaccines: Micro- and Nanocontamination*, 4 INT’L J. VACCINES & VACCINATION 72 (2017) (Ex. 19, Tab J).) However, as was noted by another special master in a prior case, this particular paper “just provided a hypothesis with no evidentiary support.” *C.N. ex rel. Dinh v. Sec’y of Health & Human Servs.*, No. 16-171V, 2022 WL 730258, at *4, *8 (Fed. Cl. Spec. Mstr. Feb. 14, 2022).

⁹ It should also be noted that the report authors do purport to reach the conclusion that N.C.’s death was caused by his vaccination. The authors indicate that their finding “needs an explanation.” (Ex. 16, p. 88.) Although inhalation and absorption through the lungs would be the “usual” explanation for such findings, the authors hypothesize without any supporting citation that injection is a more likely explanation given N.C.’s age and, further, that N.C. had only had vaccine injections throughout his life. (*Id.* at 87-88.) The authors then suggest that vaccines are known to contain contaminants, though, again, the accompanying citations have not been submitted into evidence. (*Id.* at 88.) Citing statistics relative to payments made by the UK Vaccine Damage Payment Act of 1979 as well as following the introduction of the DPT vaccine to Guinea-Bissau in 1981, the authors infer an increased risk of death following these vaccinations. (*Id.* at 89.) Accordingly, they conclude that “[t]he pieces of evidence described in this pathological case are a proof of a brain contamination and that that is incompatible with life and that the phenomenon is not rare.” (*Id.*) This explanation is not persuasive on its face and, especially given the lack of evidence regarding the authors’ credentials and the unavailability of the supporting citations, I do not have sufficient evidence to conclude that the scientific/medical opinion rendered in this report was sound and reliable. Of particular note, this opinion cites the safety profile of the earlier whole cell pertussis vaccine (“DPT”) as evidence regarding the later acellular pertussis vaccine (“DTaP”), which is what is at issue in this case. In the *Althen* prong one analysis below, I explain that this is not sound and reliable. After respondent’s expert (Dr. McCusker) criticized the nanodiagnosics srl report, petitioner’s expert (Dr. Rostad) conceded that “I am limited in my responses to Dr. McCusker’s concerns, since I have no special expertise in this area, nor the research performed by Nanodiagnosics.” (Ex. 19, p. 24.) Dr. Rostad stated that “I cannot begin to completely defend the analysis” of the nanodiagnosics report and that “[w]ith regards to potential damage of these proposed contaminants to the CNS in N.C., no one knows this answer.” (*Id.*) Accordingly, the nanodiagnosics srl report need not be discussed further.

On June 1, 2020, SUDCRRC produced a case review report for N.C. (Ex. 15.) It indicated that:

Based on their review of the available information, the forensic pathologists consider the significant findings in [N.C.'s] investigation to be: 1) No significant anatomic abnormalities, 2) history of recent illness[,] 3) pattern of facial pallor suggestive of face-down position, and 4) presence of only alpha-hemolytic *Streptococcus pneumoniae* in both blood and lung cultures (not mixed flora). While these could both be postmortem contaminants, a possible underlying bacterial infection (*S. pneumoniae*) is possible.

(*Id.* at 6.) Thus, they indicate that “[i]t is the opinion of the reviewers that these findings – inclusive of the genetic analysis performed by SUDCRRC, the complete case information, and medical records made available to the reviewers – do not identify a specific cause of death with a high level of probability.” (*Id.*)

Much later, in a May 20, 2023 “supplement” to his original report, Dr. Hood added the following:

Since this autopsy nearly 6 years ago the case has been reviewed as a possible vaccine related injury and several expert reports have been provided to me as well as neuropathology review of the brain histology slides and the further information that the child may have had an “absence” attack or minor seizure in the presence of his father a few days before his death. All lung lobes were histologically sampled with no pneumonitis identified and staphylococcus is a common postmortem contaminant. Neuropathological examination found no encephalitis but some histological changes in the hippocampus of not yet established significance and they may have made the child’s brain more susceptible to exhibiting seizure activity. The brain was swollen and “dusky” in appearance which is not a postmortem phenomenon and would be in keeping with a terminal seizure but can result from almost any form of terminal hypoxia and the most I can opine is that if [N.C.] had a terminal seizure his autopsy findings would be just as were present in this case. Whether his immunizations contributed in some way to causing such a postulated terminal seizure is beyond my area of expertise.

(Ex. 24, p. 5.)

There are reports of a family history of febrile seizures, experienced by N.C.’s older sister. (Ex. 3, p. 1; Ex. 15, p. 3; Ex. 19, p. 11, n.1.)

IV. Summary of Expert Opinions

a. Petitioners' experts

i. Pathologist Steven Rostad, M.D.¹⁰

Dr. Rostad submitted four reports on petitioners' behalf along with a set of annotated autopsy photos.¹¹ (Exs. 18, 19, 22-23, 25.) In his first report, Dr. Rostad indicated he is satisfied that N.C.'s autopsy was of "adequate quality" and after review of the investigative materials, autopsy slides, and test results, he agreed with the medical examiner's conclusion that the best classification for N.C.'s death would be "sudden unexplained death," specifically SUDC. (Ex. 18, p. 8.) However, he does raise some differences from Dr. Hood in his own interpretation of the evidence.¹² (*Id.* at 8-13.)

¹⁰ Dr. Rostad earned his medical degree from the University of Washington in Seattle in 1983. (Ex. 17, p. 1.) He completed his internship in pathology at the University of Colorado in Denver in 1984. (*Id.*) After the conclusion of his internship, Dr. Rostad returned to the University of Washington in Seattle, where he completed a fellowship in neuropathology in 1986. (*Id.*) Thereafter, Dr. Rostad completed a residency in anatomic pathology and served a term as chief resident from 1987 through 1988. (*Id.*) Dr. Rostad became board-certified in anatomic pathology and neuropathology in 1988 and maintained his license to practice medicine in Washington. (*Id.* at 3.) From 1988 to 2017, Dr. Rostad was employed as a staff pathologist at CellNetix Pathology PLLC and as an attending pathologist and neuropathologist at Swedish Medical Center in Seattle. (*Id.* at 1.) In addition, Dr. Rostad served as a clinical associate professor of pathology at the University of Washington School of Medicine from 1990 to 2015. (*Id.*) During his thirty years of practice, Dr. Rostad reviewed more than 70,000 cases microscopically representing surgical, autopsy, cytologic, forensic, medical-legal, and pediatric specialties. (Ex. 18, p. 1.) He has published over thirty peer-reviewed journal articles, including articles on topics relating to adult epilepsy. (*Id.*; Ex. 17, pp. 3-6.)

¹¹ Dr. Rostad's reports are very long, including lengthy rebuttal to respondent's experts. His first report was 36 pages. (See Ex. 18.) His second report was 43 pages. (See Ex. 19.) And his third report was 27 pages. (See Ex. 22.) Because the length of these reports leaves a complete summary impractical, this summary emphasizes Dr. Rostad's primary contentions in preference to his rebuttal, though I have reviewed and considered each report in full. Exhibit 23 additionally includes five photographs of N.C.'s brain, both within the skull and after being sectioned. Although I have reviewed the annotated photos, it is not practical to attempt to describe the photographs and the many notations added by Dr. Rostad. (Ex. 23.) Suffice it to say that Dr. Rostad views the photographs as supportive of his interpretation of the pathology as otherwise explained in his reports.

¹² Additionally, Dr. Rostad was critical of the SUDCRRC report. (Ex. 18, p. 14; Ex. 19, p. 4; Ex. 22.) These critiques make up a significant portion of Dr. Rostad's reports. In discussing the SUDCRRC findings, Dr. Rostad raised several questions with respect to what evidence was reviewed before concluding that "the SUDCRRC pathology panel review was incomplete and therefore irrelevant for consideration in this matter. I suspect that upon review of these materials, as well as more detailed analysis of the information provided above, their conclusions would have been more coincident with mine." (Ex. 18, p. 14.) However, the SUDCRRC's ultimate conclusion was that N.C.'s death was an unexplained sudden death, and that the manner of death was undetermined. (Ex. 15, p. 6.) Even after accounting for Dr. Rostad's specific criticisms, it is difficult to see how he can reasonably fault the SUDCRRC report for reaching the same ultimate view that he did. As noted above, Dr. Rostad stated that "[a]fter review of the investigative scene report, scene photos and the autopsy slides supplementary tests, I agree with Dr. Hood that the best initial classification of N.C.'s death would be sudden unexplained death." (Ex. 18, p. 8 (citing Ex. 5, p. 37).) Moreover, while Dr. Rostad also proposed a

In summary, Dr. Rostad listed twelve anatomic diagnoses based on his review:

1. Cerebral edema with tonsillar and uncal herniation.
2. Hippocampal formation abnormalities (dentate gyrus (“DG”) malformations) including focal bilamination of DG, ectopic DG cells, irregular thickening, hyperconvolution of DG[.]
3. Heavy lungs with diffuse pulmonary edema and vascular congestion.
 - a. [P]atchy mild infiltrates of lymphocytes and histiocytes involving interstitium and terminal bronchioles.
4. Splenomegaly.
5. Hepatomegaly.
6. Diffuse vascular congestion, most organs, including brain and spinal cord[.]
7. Focal mild dropout of Purkinje cells, cerebellum.
8. Foci of acute hemorrhage, dura.
9. Increased weight of thymus.
10. Focal lymphohistiocytic infiltrates of prostate.
11. Petechiae on the inferior surface of thymus.
12. Toxicology, metabolic screen, microbiologic cultures, whole exome sequencing for 200 genes associated with sudden and unexpected death show no significant findings.

(*Id.* at 12-13.)

Based on these findings, Dr. Rostad opined that the cause of N.C.’s death was cerebral edema with tonsillar and uncal herniation resulting from a suspected seizure, with a hippocampal malformation also significantly contributing. (Ex. 18, p. 13.) In his second and third reports, Dr. Rostad addressed at length his disagreements with Dr.

specific cause of death, it is apparent that he does not view his own explanation of N.C.’s death as being mutually exclusive of the SUDC categorization. In fact, Dr. Rostad stated that “N.C.’s death overlaps with the description of sudden unexpected death in epilepsy (SUDEP), except N.C. had no history of epilepsy-like symptoms and no evidence of genetic abnormalities related to epilepsy.” (*Id.* at 21.) Accordingly, while all competing evidence must be weighed, Dr. Rostad is not persuasive in contending that the SUDCRRRC report is either suspect or irrelevant. Dr. Rostad has no personal knowledge regarding the details of the SUDCRRRC investigation and his assertion that, had the SUDCRRRC conducted a different investigation, they would have reached a conclusion similar to his, is speculative. *Accord Anklam v. Sec’y of Health & Human Servs.*, No.17-2061V, 2023 WL 8890662, at *17 (Fed. Cl. Spec. Mstr. Nov. 29, 2023) (petitioner’s pathology expert acknowledging that he cannot explain how the SUDCRRRC reached their findings without knowing the neuropathologist and what he reviewed.) Indeed, some of Dr. Rostad’s specific criticisms fail to raise any reasonable point of disagreement. For example, in his third report, Dr. Rostad criticized the SUDCRRRC pathologist for relying on Dr. Hood’s autopsy report for the conclusion that there was facial pallor suggestive of a face-down position, regarding this to be “poor practice of pathology.” (Ex. 22, p. 14 (citing Ex. 15, p. 6).) Yet in the same report, Dr. Rostad acknowledged that the SUDCRRRC report reasonably explained that the “sheriff’s office refused the release of their death scene investigations report and photos for our review.” (*Id.* at 14-15 (quoting Ex. 15, p. 5).) But, in any event, despite stressing this limitation, Dr. Rostad never disputed the finding that N.C. had been in a face-down position.

Harris, respondent's pathology expert, regarding the presence of cerebral edema and herniation. (Ex. 19, pp. 6-9; Ex. 22, pp. 7-12.) In his autopsy photo annotations, he further drew attention to his findings relating to the flattening of the gyri, compression of cerebral sulci due to edema, and herniation of the cerebellar tonsils and unci. (Ex. 23.)

Dr. Rostad explained:

The swelling and herniation of N.C.'s brain would be expected to contribute to dysfunction of the heart and lungs with resultant cardiopulmonary arrest. The documentation for these pathologic processes is challenging given the absence of a witness and N.C.'s inability to express detailed clinical symptoms. Several studies have raised whether febrile seizures may be associated with some cases of SUDC and SIDS. A recent report of a 20-month-old with prior febrile status epilepticus who died 2 days after her seizure with diffuse cerebral suppressions and severe bradycardia, suggest a mechanism of death like SUDEP. Another child with a history of febrile seizures, died suddenly and unexpectedly after a suspected seizure. As for N.C., although one could implicate the [hippocampal formation] malformation or febrile seizure as the cause of a fatal seizure (and death), as has been suggested by other studies, I argue that DTaP vaccination N.C. received on April 25, 2017, initiated the chain of events that led directly and inevitably to his untimely death.

(Ex. 18, p. 21 (internal citations omitted) (citing Hannah C. Kinney et al., *Sudden Death, Febrile Seizures, and Hippocampal and Temporal Lobe Maldevelopment in Toddlers: A New Entity*, 12 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 455 (2009) (Ex. 18, Tab DDD); Hannah C. Kinney et al., *Witnessed Sleep-Related Seizure and Sudden Unexpected Death in Infancy: A Case Report*, 9 FORENSIC SCI., MED., & PATHOLOGY 418 (2013) (Ex. 18, Tab EEE); Kenneth A. Myers et al., *Sudden Death After Febrile Seizure Case Report: Cerebral Suppression Precedes Severe Bradycardia*, 140 PEDIATRICS 1 (2017) (Ex. 18, Tab FFF); Brian J. Dlouhy et al., *Unexpected Death of a Child with Complex Febrile Seizures – Pathophysiology Similar to Sudden Unexpected Death in Epilepsy?*, 8 FRONTIERS NEUROLOGY 1 (2017) (Ex. 18, Tab GGG)).)

Dr. Rostad opined that the hippocampal maldevelopment he identified would have lowered N.C.'s seizure threshold. (Ex. 18, pp. 24-25.) Citing studies by Kinney et al. and Hefti et al., he opined that

[t]hese studies suggested that [hippocampal formation] malformation represented a marker of an impaired forebrain/limbic network that increases the risk of sudden death due to potential brainstem cardiorespiratory related circuits nuclei, or to a subclinical seizure in an infant predisposed to epilepsy. The association of epilepsy-related pathology (such as [hippocampal formation] maldevelopment) with SIDS, SUDEP and SUDC leads to possible epilepsy-related mechanisms in sudden death.

(*Id.* at 25 (citing Hannah C. Kinney et al., *Hippocampal Formation Maldevelopment and Sudden Unexpected Death Across the Pediatric Age Spectrum*, 75 J. NEUROPATHOLOGY & EXPERIMENTAL NEUROLOGY 981 (2016) (Ex. 18, Tab XXX); Marco M. Hefti et al., *Hippocampal Malformation Associated with Sudden Death in Early Childhood: A Neuropathologic Study*, 12 FORENSIC SCI., MED. & PATHOLOGY 14 (2016) (Ex. 13, Tab S) (see also Ex. C, Tab 2)).) Further to this, Dr. Rostad opined that N.C. was in a critical period of development wherein the maturing brain is more vulnerable to seizures. (*Id.* at 25-26 (citing Sanjay N. Rakhade & Frances E. Jensen, *Epileptogenesis in the Immature Brain: Emerging Mechanisms*, 5 NATURE REVIEWS NEUROLOGY 380 (2009) (Ex. 18, Tab V)).) Significantly, however, Dr. Rostad acknowledged that “only a subset of SIDS and SUDC show these pathologic changes [of hippocampal malformation], so that not all individuals would be affected. In other words, these population groups are not homogeneous. In addition, individuals who are outside these groups, including ‘controls’ also may show these hippocampal changes.” (Ex. 19, p. 31.) Thus, while positing that these findings counsel “less distinction between SIDS and SUDC, and even SUDEP,” he agreed that present data does not allow for “adoption of a unified hypothesis.” (*Id.*)

Notably, however, Dr. Rostad confirmed that he agrees that N.C. did not suffer SUDEP as he did not have a history of epilepsy, even if he did have a witnessed seizure. (Ex. 19, p. 10.) Although respondent’s experts stressed the absence of evidence that a terminal seizure occurred, Dr. Rostad opined that specific signs of a seizure may be absent or nonspecific. (*Id.* at 11.) In his final report, Dr. Rostad further opined regarding a study in the journal *Neurology* by Gould et al. (Ex. 25 (discussing Laura Gould et al., *Video Analyses of Sudden Unexplained Death in Toddlers*, 102 NEUROLOGY e208038 (2024) (Ex. 25, Tab 1) (see also Ex. J, Tab 1)).) In this final report, Dr. Rostad firmly placed petitioners’ theory of causation within the SIDS/SUDC context. (*Id.*) Discussing the risk factors that have been identified in SIDS/SUDC, he explained that

a history of febrile seizures is noted in 30% of the children who suffered from an unexplained death, a significant increase over the controls in the studies. Another identified risk factor identified through these studies includes children who have malformations in the hippocampi in the brain. These risk factors can lower a child’s threshold for suffering a seizure. When a child has these risk factors and another internal or external stimulus is applied, a terminal seizure can result.

(*Id.* at 1.)

According to Dr. Rostad, the study by Gould et al. adds “strong evidence” that convulsive seizures are a cause of deaths in infants and toddlers that may otherwise be considered either unexplained or sleep-related deaths. (Ex. 25, p. 1.) The Gould et al. study reviewed crib camera footage that was available for seven out of 301 children that had been enrolled in the SUDCRR. (Ex. 25, p. 2 (discussing Gould et al., *supra*, at Ex. 25, Tab 1).) The study found that six cases had suggestive or definite convulsive

movements before death, suggesting that terminal seizures may be much more common than previously believed. (*Id.* at 2-3 (citing Gould et al., *supra*, at Ex. 25, Tab 1, p. 6).) He explained that “[t]his article is extremely relevant to the instant case as it is directly on point with Petitioner’s proposed medical theory that N.C. more likely than not suffered from a terminal seizure and therefore, his death is not unexplained. The question turns on whether the DTaP vaccine N.C. received caused or contributed to this terminal seizure.” (*Id.* at 3.)

Given the likelihood of contamination and a lack of a substantial acute inflammatory response as expected in pneumonia, Dr. Rostad opined that *S. pneumoniae* infection is “highly unlikely.” (Ex. 18, p. 14.) However, he did otherwise opine that N.C. was suffering a viral upper respiratory infection in the several days prior to his death, beginning on May 7, 2017. (*Id.* at 16-17.) Dr. Rostad opined that onset of an upper respiratory infection “amplified” N.C.’s ongoing cytokine response to his prior vaccination. (*Id.* at 16.) He proposed that the episode described by Mr. Catone occurring on May 7 likely represented an atonic seizure, which he attributed to either N.C.’s high fever or the cytokine elevation initially due to the DTaP vaccination.¹³ (*Id.* at 16-17.) Thus, he hypothesized that “[t]his same phenomenon of cytokine elevation and neuronal excitation is the most likely trigger for the final proposed seizure which likely occurred on May 12, 2017.” (*Id.* at 17.) Dr. Rostad cited a number of papers¹⁴ for the

¹³ Dr. Rostad acknowledged that febrile seizures are typically tonic-clonic but suggested that atonic seizures have also been reported. (Ex. 18, p. 16 (citing Alexander K.C. Leung et al., *Febrile Seizures: An Overview*, 7 DRUGS CONTEXT 1 (2018) (Ex. 18, Tab L)).)

¹⁴ Specifically: Nina Dupuis & Stéphane Auvin, *Inflammation and Epilepsy in the Developing Brain: Clinical and Experimental Evidence*, 21 CNS NEUROSCIENCE & THERAPEUTICS 141 (2015) (Ex. 18, Tab R); Luca Bartolini et al., *Viral Triggers and Inflammatory Mechanisms in Pediatric Epilepsy*, 56 MOLECULAR NEUROBIOLOGY 1897 (2019) (Ex. 18, Tab S); Karen S. Wilcox & Annamaria Vezzani, *Does Brain Inflammation Mediate Pathological Outcomes in Epilepsy?*, 813 ADVANCES EXPERIMENTAL MED. & BIOLOGY 169 (2014) (Ex. 18, Tab T); Amna Rana & Alberto E. Musto, *The Role of Inflammation in the Development of Epilepsy*, 15 J. NEUROINFLAMMATION 144 (2018) (Ex. 18, Tab U); Sanjay N. Rakhade & Frances E. Jensen, *Epileptogenesis in the Immature Brain: Emerging Mechanisms*, 5 NATURE REVIEWS NEUROLOGY 380 (2009) (Ex. 18, Tab V); Giovanna Vitaliti et al., *Molecular Mechanisms Involved in the Pathogenesis of Early Onset Epileptic Encephalopathy*, 12 FRONTIERS MOLECULAR NEUROSCIENCE 1 (2019) (Ex. 18, Tab W); Pavanish Kumar et al., *Proinflammatory IL-17 Pathways Dominate the Architecture of the Immunome in Pediatric Refractory Epilepsy*, 4 JCI INSIGHT 1 (2019) (Ex. 18, Tab Y); R.S. Rao et al., *Role of Different Cytokines and Seizure Susceptibility: A New Dimension Towards Epilepsy Research*, 47 INDIAN J. EXPERIMENTAL BIOLOGY 625 (2009) (Ex. 18, Tab Z); Fatma Mujgan Sonmez et al., *Blood Levels of Cytokines in Children with Idiopathic Partial and Generalized Epilepsy*, 22 SEIZURE 517 (2013) (Ex. 18, Tab AA); Kavitha Kothur et al., *Etiology is the Key Determinant of Neuroinflammation in Epilepsy: Elevation of Cerebrospinal Fluid Cytokines and Chemokines in Febrile Infection-Related Epilepsy Syndrome and Febrile Status Epilepticus*, 60 EPILEPSIA 1678 (2019) (Ex. 18, Tab BB); James G. Heida et al., *The Role of Interleukin-1 β in Febrile Seizures*, 31 BRAIN DEV. 388 (2009) (Ex. 18, Tab CC); James G. Heida & Quentin J. Pittman, *Causal Links Between Brain Cytokines and Experimental Febrile Convulsions in the Rat*, 46 EPILEPSIA 1906 (2005) (Ex. 18, Tab DD); Bo Feng et al., *Transient Increase of Interleukin-1 β After Prolonged Febrile Seizures Promotes Adult Epileptogenesis Through Long-Lasting Upregulating Endocannabinoid Signaling*, 6 SCI. REPS. 1 (2016) (Ex. 18, Tab EE); Adam L. Numis et al., *Early Changes in Pro-Inflammatory Cytokine Levels in Neonates with Encephalopathy are Associated with Remote Epilepsy*, 86 PEDIATRIC RSCH. 616 (2019) (Ex. 18, Tab FF); Aram Kwon et al., *Cytokine Levels in Febrile Seizure Patients: A Systematic Review and Meta-Analysis*, 59 SEIZURE 5 (2018) (Ex. 18, Tab GG); Hong-Mei Yu et al., *IL-1 β : An Important Cytokine Associated with*

proposition that inflammatory mediators, including pro-inflammatory cytokines, can incite seizures. (*Id.* at 17-20.) He also includes several citations for the proposition that vaccines, and in particular whole cell pertussis vaccines, are known to cause seizures and encephalopathy.¹⁵ (*Id.* at 22.)

Dr. Rostad also, like Dr. Akbari (as discussed further below), cited cytokine storms as evidence that uncontrolled cytokine responses can occur and can lead to death. (Ex. 18, pp. 20-21.) He opined that the acute respiratory distress that significantly contributed to mortality from Covid-19 may have been due to cytokine storm and that “it is worth noting that the histopathologic findings are like those seen with N.C.” (*Id.* (citing Farinaz Safavi & Avindra Nath, *Silencing of Immune Activation with Methotrexate in Patients with COVID-19*, J. NEUROLOGICAL SCI. 116942 (2020) (Ex. 18, Tab SS); Yu-Ting Yen et al., *Modeling the Early Events of Severe Acute Respiratory Syndrome Coronavirus Infection In Vitro*, 80 J. VIROLOGY 2684 (2006) (Ex. 18, Tab CCC)).) Dr. Rostad stressed that, whereas Dr. Harris appeared unconcerned regarding findings relative to the thymus, liver, and spleen, Dr. Rostad felt the liver and spleen were heavy and congested, qualifying as hepatomegaly and splenomegaly, which he viewed as “a likely sign of a generalized reaction, perhaps to a seizure or inflammation.” (Ex. 19, pp. 2-3.) Dr. Rostad confirmed that he viewed increased weight of the liver, spleen, and thymus, as evidence of a generalized inflammatory reaction consistent with a cytokine storm. (Ex. 22, p. 2.)

Febrile Seizures?, 28 NEUROSCIENCE BULL. 301 (2012) (Ex. 18, Tab HH); Kyungmin Kim et al., *Analysis of Plasma Multiplex Cytokines and Increased Level of IL-10 and IL-1Ra Cytokines in Febrile Seizures*, 14 J. NEUROINFLAMMATION 1 (2017) (Ex. 18, Tab II); Anne A. Kan et al., *Prolonged Increase in Rat Hippocampal Chemokine Signalling After Status Epilepticus*, 245 J. NEUROIMMUNOLOGY 15 (2012) (Ex. 18, Tab JJ); Jieun Choi et al., *Increase Levels of HMGB1 and Pro-Inflammatory Cytokines in Children with Febrile Seizures*, 8 J. NEUROINFLAMMATION 1 (2011) (Ex. 18, Tab KK); Miia Virta et al., *Increased Plasma Levels of Pro- and Anti-inflammatory Cytokines in Patients with Febrile Seizures*, 43 EPILEPSIA 920 (2002) (Ex. 18, Tab LL); Nikola Šutulović et al., *Glial Cells, Blood Brain Barrier and Cytokines in Seizures: Implications for Therapeutic Modalities*, 69 MEDICINSKI PODMLADAK 33 (208) (Ex. 18, Tab MM); Bridgette D. Semple et al., *Immune Challenges and Seizures: How Do Early Life Insults Influence Epileptogenesis?*, 11 FRONTIERS PHARMACOLOGY 1 (2020) (Ex. 18, Tab NN); Narges Karimi et al., *Frequent Convulsive Seizures in an Adult Patient with COVID-19: A Case Report*, 22 IRANIAN RED CRESCENT MED. J. 1 (2020) (Ex. 18, Tab OO); Dominik Kobylarek et al., *Advances in the Potential Biomarkers of Epilepsy*, 10 FRONTIERS NEUROLOGY 1 (2019) (Ex. 18, Tab PP).

¹⁵ Specifically: Jennifer Tam et al., *Review of Pediatric Encephalitis and Encephalopathy Cases Following Immunization Reported to the Canadian Immunization Monitoring Program Active (IMPACT) from 1992 to 2012*, 38 VACCINE 4457 (2020) (Ex. 18, Tab JJJ); Marie R. Griffin et al., *Risk of Seizure and Encephalopathy After Immunization with the Diphtheria-Tetanus-Pertussis Vaccine*, 263 JAMA 1641 (1990) (Ex. 18, Tab KKK); CTRS. FOR DISEASE CONTROL & PREVENTION, *Prevention of Pertussis, Tetanus, and Diphtheria with Vaccines in the United States: Recommendations of the Advisory Committee on Immunization Practices (ACIP)*, 67 MORBIDITY & MORTALITY WEEKLY REP. 1 (2018) (Ex. 18, Tab LLL) [hereinafter CDC Report]; Yuelian Sun et al., *Risk of Febrile Seizures and Epilepsy After Vaccination with Diphtheria, Tetanus, Acellular Pertussis, Inactivated Poliovirus, and Haemophilus Influenzae Type B*, 307 JAMA 823 (2012) (Ex. 18, Tab MMM) (see also Ex. C, Tab 14); Pedro L. Moro et al., *Safety Surveillance of Diphtheria and Tetanus Toxoids and Acellular Pertussis (DTaP) Vaccines*, 142 PEDIATRICS 1 (2018) (Ex. 18, Tab NNN); Prescribing Information, DAPTACEL (Diphtheria and Tetanus Toxoids and Acellular Pertussis Vaccine Adsorbed): Suspension for Intramuscular Injection [DAPTACEL Package Insert] (Ex. 18, Tab OOO).

Dr. Rostad also invoked the immune stimulant effect of aluminum containing adjuvants. (Ex. 18, pp. 23-24.) He further discussed the nanodiagnostics srl findings of multiple metals, including aluminum and silicon, as strengthening “[t]he linkage of aluminum to N.C.’s potential seizure and ultimate death.” (*Id.* at 24.) However, he ultimately concluded that any potential role for aluminum in the hypothesized seizures “is not clear.” (*Id.*) Dr. Rostad purported to rebut Dr. McCusker’s opinion (discussed below) as well as her criticisms of the nanodiagnostics srl report; however, he acknowledged that “I am limited in my responses to Dr. McCusker’s concerns, since I have no special expertise in this area, nor the research performed by Nanodiagnostics.” (Ex. 19, p. 24.) Dr. Rostad stated that “I cannot begin to completely defend the analysis” of the nanodiagnostics report and that “[w]ith regards to potential damage of these proposed contaminants to the CNS in N.C., no one knows this answer.” (*Id.*)

ii. Immunologist Omid Akbari, Ph.D.¹⁶

Dr. Akbari presented three reports in this case. (Ex. 13¹⁷, Ex. 20, Ex. 21.) He observed that cytokines are “key regulators of the inflammatory and immune responses,” explaining that “the immune cells involved in the body’s immune response need to communicate with each other. They do this by releasing a set of proteins that serve as chemical messengers. These proteins, called cytokines, tell immune cells what to do and also tell the body to produce more if needed.” (Ex. 13, p. 7.) Cytokines can be blamed for triggering symptoms associated with inflammation, such as fever, aches, or runny nose. (*Id.*) In some “severe cases,” the cytokine response can “grow out of control.” (*Id.*) In such instances, chemokines, a subset of cytokines that recruit immune cells to sites of inflammation, create a positive feedback loop called a “cytokine storm.” (*Id.*) Cytokine storms begin locally but spread throughout the body, causing

¹⁶ Dr. Akbari earned his Ph.D. in cellular and molecular immunology in 1998 from the National Institute for Medical Research in London. (ECF No. 28-48, p. 1.) Thereafter, he completed a postdoctoral fellowship at Stanford University in 2001. (*Id.*) Currently, Dr. Akbari serves as a Professor of Medicine and Professor of Allergy and Immunology at the University of Southern California Keck School of Medicine. (*Id.* at 2.) Additionally, he holds adjunct professorships at the University of California, Los Angeles David Geffen School of Medicine’s Department of Pediatrics and Chiba University’s Department of Immunology in Japan. (*Id.*; Ex. 13, p. 1.) Dr. Akbari’s research focuses on the role that immune cells play in inducing autoimmune and allergic diseases. (Ex. 13, p. 1.) He serves as an associate editor and as a reviewer on various journals. (*Id.* at 2.) He has authored or co-authored over eighty publications. (ECF No. 28-48, pp. 9-15.)

¹⁷ Petitioners filed a medical journal article marked Exhibit 13, Tab O with Dr. Akbari’s initial report at Exhibit 13. (ECF No. 28.) Petitioners’ exhibit list and Dr. Akbari’s bibliography identified the exhibit as the following article: K. Helweg-Larsen & E. Garde, *Sudden Natural Death in Childhood. A Review of Forensic Autopsy Protocols in Cases of Sudden Death Between the Ages of One and Five Years, 1982-1991, with a Special View to Sudden Unexplained Death*, 82 ACTA PAEDIATRICA 975 (1993). (See Ex. 13, p. 16; ECF No. 98-2, p. 3.) However, the medical literature actually filed by petitioners as Ex. 13, Tab O is the following article: Ali Riza Tümer et al., *Sudden Unexpected Child Deaths: Forensic Autopsy Results in Cases of Sudden Deaths During a 5-Year Period*, 51 J. TROPICAL PEDIATRICS 131 (2005). (See Ex. 13, Tab O.) Within the copy of this article by Tümer et al., petitioners highlighted the text relevant to Dr. Akbari’s report. (See *id.*) Accordingly, use of the Tümer article does not appear to have been inadvertent and all citations to Exhibit 13, Tab O within this decision refer to the article by Tümer et al.

redness, swelling, edema, heat, pain, and loss of function. (*Id.*) Cytokine storms can lead to organ damage, especially affecting the lungs and central nervous system, and can be fatal. (*Id.*)

Cytokines can be activated by either infection or vaccination. (Ex. 13, p. 7.) Citing a study by Kashiwagi et al., Dr. Akbari indicated that vaccination has been shown to increase the following cytokines: IL-1 β , IL-6, G-CSF, and TNF- α . (*Id.* at 12 (citing Yasuyo Kashiwagi et al., *Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influenzae Type b (Hib), and 7-Valent Pneumococcal (PCV7) Vaccines*, 10 HUM. VACCINES & IMMUNOTHERAPEUTICS 677 (2014) (Ex. 13, Tab OO) (see also Ex. A, Tab 10)).) However, he indicated that “the presence of antigen stimulation alone is not sufficient to trigger cytokine storm.” (*Id.*) Instead, “activation of the innate immune system by adjuvants or microbial products is necessary to break self-tolerance and cause reactions including autoimmunity.” (*Id.* (citing Chiaki Fujimoto et al., *Pertussis Toxin is Superior to TLR Ligands in Enhancing Pathogenic Autoimmunity, Targeted at a Neo-Self Antigen, by Triggering Robust Expansion of Th1 Cells and their Cytokine Production*, 177 J. IMMUNOLOGY 6896 (2006) (Ex. 13, Tab PP); Hanspeter Waldner et al., *Activation of Antigen-Presenting Cells by Microbial Products Breaks Self Tolerance and Induces Autoimmune Disease*, 113 J. CLINICAL INVESTIGATION 990 (2004) (Ex. 13, Tab QQ)).) Dr. Akbari posits that the initial innate immune reaction led to the production of autoreactive T cells that “initiated an adverse reaction that ultimately led to his untimely death.” (*Id.* at 10; Ex. 20, pp. 4-5.) In particular, Dr. Akbari suggests that there is “sequence homology between components of the DTaP vaccine and key biological proteins involved in regulating the immune response” and that N.C.’s death resulted from an autoimmune reaction. (Ex. 20, pp. 4-5.) He also suggested that tetanus toxoid is known to bind to gangliosides and neuronal cells. (*Id.* at 4.) Dr. Akbari opined that

it is more likely than not as well as biologically possible that N.C.’s immune system produced autoreactive T cells or antibodies in response to the administration of the DTaP vaccine he received on April 25, 2017 and that these autoreactive immune cells initiated an adverse reaction that ultimately lead to his untimely death on May 12, 2017.

(*Id.* at 5; Ex. 13, p. 10.) However, he is no more specific than to endorse Dr. Rostad’s suggestion of a terminal seizure and state that cytokines trafficked across the blood brain barrier had “neuropathological effects.” (Ex. 20, p. 5.)

Yet, despite invoking an autoimmune process to explain the 17-day latency in this case, Dr. Akbari stressed that “[o]ne very important aspect of this case is the evidence which supports the presence of febrile seizure in N.C.” (Ex. 20, p. 5.) He explained that

[t]he role of cytokines in the induction of fever is very relevant to this case and it is important to point out that although, in many cases innate cytokines

are able to cause fever and high temperature, recent data suggest that these cytokines play an important but not obligatory role in fever induction.

(Ex. 21, p. 6 (citing Katarina Alheim et al., *Hyperresponsive Febrile Reactions to Interleukin (IL) 1 α and IL-1 β , and Altered Brain Cytokine mRNA and Serum Cytokine Levels, in IL-1 β -Deficient Mice*, 94 PROCS. NAT'L ACAD. SCIS. 2681 (1997) (Ex. 21, Tab 10)).) In that regard, Dr. Akbari opined that N.C. had likely suffered a seizure on May 7, 2017. (*Id.* at 9.)

In addition to noting the tetanus toxoid molecules to be “antigenically complex and capable of cross-reactivity,” Dr. Akbari implicated the aluminum adjuvant included in the DTaP vaccine. (Ex. 13, pp. 10-12.) “Aluminum stimulates the innate immune system at the site of injection by directly recruiting naïve immune cells to the site and indirectly by activating the tissue resident macrophages to rapidly produce cytokines and chemokines, also attracting other immune cells of the innate system to the area.” (*Id.* at 11.) Additionally, he contended that aluminum adjuvants induce dendritic cells to secrete IL-1 β , which he asserted to be a “potent inflammatory cytokine” associated with acute and chronic inflammatory disorders. (*Id.* (citing Mirjam Kool et al., *Cutting Edge: Alum Adjuvant Stimulates Inflammatory Dendritic Cells Through Activation of the NALP3 Inflammasome*, 181 J. IMMUNOLOGY 3755 (2008) (Ex. 13, Tab JJ)).)

Regarding the details of this case, Dr. Akbari opined as follows:

When N.C. received the DTaP vaccination on April 25, 2017, the vaccine components, including the aluminum adjuvant, stimulated his immune system and a local inflammatory immune reaction ensued. This is evidenced by statements in the mother’s and father’s affidavits that “[w]ithin 24 hours of receiving this vaccine, N.C. developed a rash and symptoms of another cold. This was a very bad cold, probably the worst cold I had seen him have. N.C. was coughing and had an on and off runny nose but there was a lot of nasal discharge” and “[w]ithin 24-48 hours after receiving the vaccine, N.C. developed a rash that lasted for a day or so. He also developed symptoms of a very bad cold. He was coughing and had an on and off runny nose with lots of nasal discharge.” Initially a nonspecific innate response would have been employed. This nonspecific response has only recently become recognized for its role in initiation of inflammation. Only recently has understanding of immune response by adjuvant been advanced and there is no consensus regarding how aluminum-containing adjuvants potentiate the immune system. However, recent research shows that peripherally injected aluminum adjuvant nanoparticles engulfed by macrophages actively spread throughout the body eventually crossing the blood-brain-barrier (BBB) and blood-CSF barrier. Once in the CNS, aluminum adjuvant nanoparticles incite deleterious inflammatory responses, resulting in a range of neuropathological effects. It should be noted that aluminum on its own can alter the properties of BBB, making the brain more accessible to inflammatory and immune mediators. Aluminum

also increases endothelial adhesion of activated monocytes, which, in the case of aluminum penetration in the CNS can likewise facilitate the entry of immune-competent cells into the CNS and lead to adverse manifestations.

(Ex. 13, p. 11 (internal citations omitted) (citing Sunita Awate et al., *Mechanisms of Action of Adjuvants*, 4 *Frontiers Immunology* 1 (2013) (Ex. 13, Tab CC); Zakir Khan et al., *Slow CCL2-Dependent Translocation of Biopersistent Particles from Muscle to Brain*, 11 *BMC MED.* 1 (2013) (Ex. 13, Tab DD); Michael S. Petrik et al., *Aluminum Adjuvant Linked to Gulf War Illness Induces Motor Neuron Death in Mice*, 9 *NEUROMOLECULAR MED.* 83 (2007) (Ex. 13, Tab EE); Christopher A. Shaw & Michael S. Petrik, *Aluminum Hydroxide Injections Lead to Motor Deficits and Motor Neuron Degeneration*, 103 *J. INORGANIC BIOCHEMISTRY* 1 (2009) (Ex. 13, Tab FF); William A. Banks & Abba J. Kastin, *Aluminum-Induced Neurotoxicity: Alterations in Membrane Function at the Blood-Brain Barrier*, 13 *NEUROSCIENCE & BIOBEHAVIORAL REVS.* 47 (1989) (Ex.13, Tab GG); Robert A. Yokel, *Blood-Brain Barrier Flux of Aluminum Manganese, Iron and Other Metals Suspected to Contribute to Metal-Induced Neurodegeneration*, 10 *J. ALZHEIMER'S DISEASE* 223 (2006) (Ex. 13, Tab HH); Elizabeth Oesterling et al., *Alumina Nanoparticles Induce Expression of Endothelial Cell Adhesion Molecules*, 178 *TOXICOLOGY LETTERS* 160 (2008) (Ex. 13, Tab II).)

Dr. Abkari suggested that generally “cytokine storm after initial immunization reaches to the highest levels in 1-3 days.” (Ex. 13, p. 13.) However, he asserted that “large quantities of cytokine[s]” sufficient to cause death in individuals susceptible to febrile seizure, can be detected “for many weeks” after a booster vaccination. (*Id.*) In particular, Dr. Akbari indicated that pro-inflammatory cytokines, specifically IFN- γ and IL-13, are “strongly upregulated” for as much as two weeks following a DTaP booster vaccination. (*Id.* (citing Saskia van der Lee et al., *Robust Humoral & Cellular Immune Responses to Pertussis in Adults After a First Acellular Booster Vaccination*, 9 *FRONTIERS IMMUNOLOGY* 1 (2018) (Ex. 13, Tab RR).) Moreover, he asserted that a mouse model study by Bairwa et al. demonstrated that proinflammatory cytokines, including IL-6 and TNF- α , can cross the blood brain barrier to affect central nervous system function. (Ex. 21, pp. 3-4 (discussing S.C. Bairwa et al., *Cytokines Profile in Neonatal and Adult Wild-Type Mice Post-Injection of U.S. Pediatric Vaccination Schedule*, 15 *BRAIN, BEHAV., & IMMUNITY - HEALTH* 1 (2021) (Ex. 21, Tab 2)).)

In his initial conclusion, Dr. Akbari described three potential explanations for N.C.'s death, all three of which are cytokine-mediated: (1) “cytokines stimulated by mild infection and subsequent immunization accompanied with abnormal development of [the] ventral medulla fits well with occurrence of SUDC in minor N.C.”; or (2) in a susceptible person, such as N.C., having “received several doses of aluminum based adjuvants, immune responses are more likely triggered to produce higher amounts of cytokines capable of causing cytokine storm and eventually caused death”; or (3) “cytokines as [a] result of immunization, in a susceptible host such as N.C. with abnormal development of the ventral medulla and family history of febrile seizures, may directly suppress the medullary system, leading to death.” (Ex. 13, p. 14.) However, after Dr. McCusker challenged Dr. Akbari regarding the nature of cytokine storms, he

disclaimed *per se* reliance on the concept in his second report. (Ex. 20, pp. 2-3.) He suggested that Dr. McCusker's view of cytokine storms is "myopic" and indicated that

I am not suggesting that N.C. succumbed to a cytokine storm following a massive and systemic infection, rather, I am positing that N.C. suffered from an initial reaction to the DTaP immunization wherein cytokines were induced . . . The cytokines circulated throughout N.C.'s body, damaging local tissue structures. Over the next two weeks, active and passive regulatory systems in N.C.'s body worked to try and heal his body from the stimulation of these cytokines, however, due to his dysregulated immune system he was not able to overcome the damage to his tissues and organs.

(*Id.* at 3.)

b. Respondent's experts

i. Pathologist Brent Harris, M.D., Ph.D.¹⁸

Dr. Harris submitted two reports on respondent's behalf. (Exs. E, H.) He concluded that "I mostly concur with [the] medical examiner and SUDCRRRC neuropathologist reviewer that found no significant structural abnormalities in the brain. My opinion would differ only [in] that I recognize some mild, subtle changes in the hippocampus, changes that have been reported in the SUDC literature." (Ex. E, p. 5 (citing Marco M. Hefti et al., *Sudden Unexpected Death in Early Childhood: General Observations in a Series of 151 Cases*, 12 FORENSIC SCI., MED. & PATHOLOGY 4 (2016) (Ex. E, Tab 2) (see also Ex. 18, Tab F)).) However, Dr. Harris stressed that this literature indicates that about half of SUDC cases remain unexplained even after taking hippocampal malformation into account. (*Id.* at 6.) Thus, he characterized the evidence regarding presumed seizures to be "limited" and the issue in need of further investigation. (*Id.* at 5-6.)

Although he acknowledged a heavy brain weight, Dr. Harris observed that there were "[n]o hemorrhages within the brainstem or neuronal hypoxia-ischemia change to the neurons was seen, so I do not believe the swelling was enough to cause a lethal

¹⁸ Dr. Harris earned his Ph.D. in pharmacology and his medical degree from Georgetown University in 1995. (Ex. F, p. 1.) He completed his internship and residency in pathology at Stanford University in 1996 and 1998 respectively. (*Id.*) After finishing his residency, Dr. Harris went on to complete a fellowship in neuropathology at Stanford University in 1999. (*Id.*) From 1999 to 2002, he completed a postdoctoral fellowship at Stanford University Department of Neurobiology. (*Id.* at 1-2.) He is board-certified in anatomic pathology and neuropathology. (*Id.* at 3.) Dr. Harris has held various academic appointments, including professorships at Stanford University Medical center, Dartmouth Medical School, Howard University Medical School, and Georgetown University School of Medicine. (*Id.* at 1.) Currently, Dr. Harris serves as an Associate Professor for the Departments of Neurology and Pathology, staff neuropathologist, Director of Neuropathology, and Director of the Brain Bank at Georgetown University. (*Id.* at 1-2.) He has reviewed over 10,000 surgical pathology and autopsy cases throughout the course of his career. (Ex. E, p. 1.) Dr. Harris has published over 200 peer-review articles, book chapters, and meeting abstracts. (*Id.*; Ex. F, pp. 4-18.)

herniation.”¹⁹ (Ex. E, p. 6.) He further concluded that “[t]he neuropathological examination was not sufficient to rule in or out a seizure related incident,” additionally noting that no astro- or microgliosis was seen, which would be expected with multiple prior seizures. (*Id.* at 5-6.) Dr. Harris observed that the lungs were congested, but with no evidence of pneumonia. (*Id.* at 5.) However, due to the limitations of the tissue samples, and given culture findings of streptococcus in the blood and lung tissue, a bacterial infection remains possible. (*Id.*)

Dr. Harris characterized his observations regarding the autopsy as being “very similar” to those of Dr. Rostad, stressing, in particular, that both experts agree with the medical examiner’s classification of N.C.’s death as a natural death and as a sudden unexplained death. (Ex. E, p. 6.) However, he does not agree that the medical literature supports the causal link to vaccination that Drs. Rostad and Akbari seek to draw. (*Id.*) Further, he is “not aware of medical literature describing this type of delay between receiving a medication such as vaccination and seizure related side effects.” (*Id.*)

Dr. Harris agreed that the thymus, liver, and spleen had increased weight; however, he stressed that neither he, nor Dr. Rostad, nor Dr. Hood, observed significant inflammation in these organs on slide review. (Ex. H, p. 2.) Instead, he explained that these are non-specific findings often seen in autopsies as a result of ante- or perimortem congestion and edema. (*Id.*) Dr. Harris acknowledged that Dr. Hood had noted a few petechiae on the inner surface of the thymus but noted that this does not change his overall opinion. (*Id.* at 1.) Dr. Harris noted that much of Dr. Rostad’s rebuttal was devoted to Dr. Rostad’s strong disagreement with Dr. Harris’s observation that there was not enough swelling to result in herniation. (*Id.* at 2.) In that regard, Dr. Harris stressed that N.C. was not in any distress when he was put down to sleep prior to his death and that this “is not the behavior you would see with an emerging herniation.” (*Id.*)

¹⁹ Additionally, Dr. Harris noted that “Hefti and colleagues found no excess brain weight in their series of cases from children with explained, seizure-related, undetermined, or SUDC groups. (Ex. E, p. 6 (citing Hefti et al., *supra*, at Ex. E, Tab 2).) However, Drs. Rostad and Harris disagree as to the correct interpretation of the data from this study. (Ex. 19, pp. 5-6; Ex. H, p. 2; Ex. 22, p. 7.)

ii. Pediatric Neurologist Gregory Holmes, M.D.²⁰

Dr. Holmes presented two reports in this case. (Exs. C, J.) Dr. Holmes agreed with the aspect of Dr. Rostad's opinion that classified N.C.'s death as a SUDC. (Ex. C, p. 7.) He disagreed with Dr. Rostad's further opinion that N.C. experienced a DTaP vaccine-related seizure that resulted in cerebral edema and fatal herniation. (*Id.* at 7-9.)

Dr. Holmes indicated that tonsillar and uncal herniation is very rare in children under two and that, while cerebral herniation has rarely been reported following infection, "Dr. Rostad provides no supporting evidence that cerebral edema following a seizure results in herniation and death in a 15 months-old^[21] child." (Ex. C, p. 7.) Dr. Holmes acknowledged that edema can occur with status epilepticus, but explained that status epilepticus involves either a single epileptic seizure of greater than 30 minutes or a series of epileptic seizures from which function is not regained between seizures. (*Id.* (citing Eugen Trinka et al., *A Definition and Classification of Status Epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus*, 56 *EPILEPSIA* 1515 (2015) (Ex. C, Tab 22) (see also Ex. J, Tab 4)).) Dr. Holmes explained:

Cerebral edema is associated with vascular congestion, swelling and degenerative features in astrocytes, distention of perivascular spaces and pallor of myelin staining, with eosinophilic, PAS-positive exudate, none of which were reported in the pathological report. Dr. Hood's report showed no histological evidence of either cytotoxic or vasogenic edema. Dr. Rostad states "No definite ischemic neuronal injury is seen, particularly in the CA1 region of both hippocampi although there appears to be shrinkage of the neurons and increased basophilia of the nuclei in these areas. I believe these hippocampal abnormalities are identical to those described by Hefti and other researchers and associated with SUDC." While Dr. Rostad agrees with the diagnosis of SUDC and cites Hefti et al., these authors do not describe cerebral edema in the patients with SUDC. Hefti et al. also described hyper-eosinophilic neurons in the cerebral cortex and/or other vulnerable regions, consistent with agonal hypoxia–ischemia. It is not clear whether these are the cells Dr. Rostad refers to in his report. While there

²⁰ Dr. Holmes earned his medical degree from the University of Virginia School of Medicine in 1974. (Ex. D, p. 1.) He completed his internship and residency in pediatrics at Yale University School of Medicine in 1976. (*Id.*) Thereafter, Dr. Holmes returned to the University of Virginia School of Medicine where he completed a second residency in pediatric neurology in 1979. (*Id.*) He is board-certified in pediatrics, psychiatry and neurology with special qualification in child neurology, and clinical neurophysiology. (*Id.*) Dr. Holmes has held various academic appointments, including professorships at the University of Connecticut Health Center, the Medical College of Georgia, Harvard Medical School, Dartmouth Medical School, and the University of Vermont College of Medicine. (*Id.* at 2.) Currently, Dr. Holmes serves as a Professor of Neurological Sciences and Pediatrics and the Chair of the Department of Neurological Sciences at the University of Vermont College of Medicine. (*Id.*; Ex. C, p. 1.) Dr. Holmes serves as a reviewer and on the editorial board for various journals and has published over 200 articles. (Ex. D, pp. 6-7, 38-62.) Dr. Holmes has published and lectured on a wide range of topics in pediatric neurology with an emphasis on pediatric epilepsy. (Ex. C, p. 1.)

²¹ In fact, N.C. was 20 months old.

may have been some mild cerebral edema, there is lack of histological evidence for either cytotoxic or vasogenic edema.

(*Id.* at 8 (internal citations omitted) (citing Hefti et al., *supra*, at Ex. C, Tab 2; Herbert J. Manz, *The Pathology of Cerebral Edema*, 5 HUM. PATHOLOGY 291 (1974) (Ex. C, Tab 28)).)

Dr. Holmes further stressed that the timing of N.C.'s death is not compatible with Dr. Rostad's theory, because even where children have been shown to suffer cerebral edema following status epilepticus, the development of the cerebral edema is not instantaneous. (Ex. C, pp. 8-9 (citing Shih-Yun Lan et al., *Analysis of Fulminant Cerebral Edema in Acute Pediatric Encephalitis*, 57 PEDIATRICS & NEONATOLOGY 402 (2016) (Ex. C, Tab 32); Kenneth A. Myers et al., *Fatal Cerebral Edema with Status Epilepticus in Children with Dravet Syndrome: Report of 5 Cases*, 139 PEDIATRICS e1 (2017) (Ex. C, Tab 33); Setsuri Yokoi et al., *Hippocampal Diffusion After Febrile Status Epilepticus is Related to Subsequent Epilepsy*, 60 EPILEPSIA 1306 (2019) (Ex. C, Tab 30)).) In contrast, "a heavy brain may be the result of simple brain swelling due to blood congestion in the terminal phase." (*Id.* at 7 (citing Johanna Marie Lundesgaard Eidahl et al., *Postmortem Evaluation of Brain Edema: An Attempt with Measurements of Water Content and Brain-Weight-to-Inner-Skull-Circumference Ratio*, 64 J. FORENSIC & LEGAL MED. 1 (2019) (Ex. C, Tab 20)).) In that regard, Dr. Holmes noted that Dr. Hood observed only "mild bilateral uncal impressions and a modest cerebellar 'cone,' but no other herniation of any of its parts." (*Id.* (quoting Ex. 5, p. 19).)

Dr. Holmes agreed that the Gould et al. study cited by Dr. Rostad in his final report "provides some limited support for the idea that seizures may be involved in sudden, unexpected death in children." (Ex. J, p. 1 (discussing Gould et al., *supra*, at Ex. J, Tab 1).) However, he disagreed that the study supports the specific argument advanced in this case that the DTaP vaccine contributed to N.C.'s death. (*Id.*) In particular, Dr. Holmes explained that the study found the children experienced convulsive seizures of between 8-50 seconds whereas "[m]ost convulsive seizures, whether occurring during the awake or sleep states, febrile or afebrile, last longer than five minutes, and rarely, if ever, cause death." (*Id.* (internal citations omitted) (citing Dale C. Hesdorffer et al., *Distribution of Febrile Seizure Duration and Associations with Development*, 70 ANNALS NEUROLOGY 93 (2011) (Ex. J, Tab 3); Trinka et al., *supra*, at Ex. J, Tab 4; Shlomo Shinnar et al., *How Long Do New-Onset Seizures in Children Last?*, 49 ANNALS NEUROLOGY 659 (2001) (Ex. J, Tab 5); Manoj Chungath & Simon Shorvon, *The Mortality and Morbidity of Febrile Seizures*, 4 NATURE CLINICAL PRAC. NEUROLOGY 610 (2008) (Ex. J, Tab 6); Victoria Nesbitt et al., *Risk and Causes of Death in Children with a Seizure Disorder*, 54 DEVELOPMENTAL MED. & CHILD NEUROLOGY 612 (2012) (Ex. J, Tab 7)).)

Dr. Holmes noted that "the [Gould et al.] authors do not conjecture on how such short seizures could cause death nor whether the seizures could have been secondary to cerebral hypoxia-ischemia complicating a cardiorespiratory disorder of unknown etiology." (Ex. J, p. 1 (citing Gould et al., *supra*, at Ex. J, Tab 1).) Moreover, while Dr.

Rostad had hypothesized that N.C.'s death resulted from cerebral edema leading to herniation and death, he has not explained how such pathology could result from an 8-50 second seizure. (*Id.*) By contrast, Dr. Holmes explained that, while the DTaP vaccine is associated with a slight increased risk of febrile seizures within 1-2 days of immunization, the DTaP vaccine is not associated with afebrile seizures. (Ex. C, p. 7 (citing John Hansen et al., *Safety of DTaP-IPV/Hib Vaccine Administered Routinely to Infants and Toddlers*, 34 *VACCINE* 4172 (2016) (Ex. C, Tab 13); Sun et al., *supra*, at Ex. C, Tab 14; Jennifer C. Nelson et al., *Adapting Group Sequential Methods to Observational Postlicensure Vaccine Safety Surveillance: Results of a Pentavalent Combination DTaP-IPV-Hib Vaccine Safety Study*, 177 *Am. J. Epidemiology* 131 (2013) (Ex. C, Tab 15); INSTITUTE OF MED., *ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY* 539-44 (Kathleen Stratton eds., 2012) (Ex. C, Tab 16)).) Nor would a fever occurring 17 days post-vaccination be attributable to a DTaP vaccine.²² (*Id.* (citing Sun et al., *supra*, at Ex. C, Tab 14; Emmanuel B. Walter et al., *Fever After Influenza, Diphtheria-Tetanus-Acellular Pertussis, and Pneumococcal Vaccinations*, 145 *PEDIATRICS* 1 (2020) (Ex. C, Tab 17); Lisa A. Jackson et al., *Retrospective Population-Based Assessment of Medically Attended Injection Site Reactions, Seizures, Allergic Responses and Febrile Episodes After Acellular Pertussis Vaccine Combined with Diphtheria and Tetanus Toxoids*, 21 *PEDIATRIC INFECTIOUS DISEASE J.* 781 (2002) (Ex. C, Tab 18); Deborah G Hirtz et al., *Seizures Following Childhood Immunizations*, 102 *J. PEDIATRICS* 14 (1983) (Ex. C, Tab 19)).)

iii. Immunologist Christine McCusker, M.D.²³

Dr. McCusker presented three reports in this case. (Exs. A, G, I.) She opined that N.C. suffered a SUDC and that his prior vaccination was unrelated and non-contributory. (Ex. A, p. 14.) She agreed that vaccinations produce cytokines, often resulting in symptoms such as pain and redness at the injection site, fever, or malaise. (*Id.* at 4.) However, the effects are limited and resolve once the pathogen is

²² Dr. Holmes further opined that Dr. Akbari's initial report "adds little useful information to this case." (Ex. C, p. 9.) While Dr. Akbari addressed the role of cytokines and immune dysregulation broadly, he provided no explanation of a mechanism by which a DTaP vaccine would be related to N.C.'s death occurring 17 days later. (*Id.*)

²³ Dr. McCusker earned her medical degree in 1993 from McMaster University Medical School in Hamilton, Ontario, Canada. (Ex. B, p. 1.) In 1996, she completed her residency in pediatrics at McGill University, training at Montreal Children's Hospital. (*Id.* at 2.) Thereafter, Dr. McCusker completed a clinical fellowship in allergy and immunology in 1999 at McGill University in Montreal, Quebec, Canada. (*Id.*) She board-certified in pediatrics by the American Board of Pediatrics and is certified by the Royal College of Physicians and Surgeons of Canada and by the College des Medecins du Quebec in both pediatrics and allergy and clinical immunology. (*Id.*; Ex. A, p. 1.) Since completing her fellowship, Dr. McCusker has held various academic appointments at McGill University and clinical appointments at Montreal Children's Hospital. (Ex. B, p. 3.) Currently, Dr. McCusker serves as an Associate Professor of Pediatrics at McGill University and as Division Director of Pediatric Allergy, Immunology and Dermatology at Montreal Children's Hospital. (*Id.*) Additionally, Dr. McCusker is a research director at Meakins-Christie Laboratories of McGill University Health Centre Research Institute where her research focus is the regulation of the immune response. (Ex. A, p. 1; Ex. B, p. 3.) She has published over fifty peer-reviewed articles. (Ex. B, pp. 33-39.)

neutralized. (*Id.*) The release of cytokines happens immediately as part of the first phase of the immune response, intended to fight the pathogen locally while activating the adaptive immune response. (*Id.*) In the context of repeat exposure, “the presence of immunological memory, for example in the form of antibodies, results in rapid clearance of the pathogen from the system, with only limited activation of the innate, pro-inflammatory pathways.” (*Id.* at 4-5.) She opined that “[t]here is no evidence in the extant literature of prolonged subclinical inflammation post vaccination” as proposed by petitioners. (*Id.* at 5 (discussing Claire-Anne Siegrist, *Vaccine Immunology*, in PLOTKIN’S VACCINES 16 (2015) (Ex. 13, Tab A)).)

The studies cited by Dr. Akbari do not support the idea of a sustained or delayed cytokine storm. (Ex. A, pp. 5-6 (discussing Rui Huang et al., *Profiles of Acute Cytokine and Antibody Responses in Patients Infected with Avian Influenza A H7N9*, 9 PLoS ONE 1 (2014) (Ex. 13, Tab G); Lee et al., *supra*, at Ex. 13, Tab RR).) Instead, citing a clinical trial of a monoclonal antibody, Dr. McCusker explained the course of a cytokine storm as follows:

Within 60 minutes of the intravenous injection, the patients developed headache and myalgias followed by high fever. All patients had notable rash and rigors. Respiratory distress and nausea, vomiting and diarrhea also developed followed by low blood pressure. Over the course of the next few days patients developed multi-organ failure. Fortunately, due to the rapid response of the critical care teams, all patients recovered with resolution of most symptoms within 30 days. Serum cytokine levels were measured (Figure 3) and there were significant cytokine elevations within 4 hours of injection of the trial drug, including TNF α (levels between 4000-5000pg/ml), interferon- γ (5000pg/ml), IL6 (range 2000-5000pg/ml) and IL1 β (5000pg/ml). Of note, despite the extreme levels of peripheral cytokines released during this event, no patient developed clinical signs of significant brain edema. Neurologic sequelae included delirium associated with the high fever, partial amnesia, localized numbness, difficulty concentrating, and headaches (see Table 3).

(*Id.* at 6-7 (discussing Ganesh Suntharalingam et al., *Cytokine Storm in a Phase 1 Trial of the Anti-CD28 Monoclonal Antibody TGN1412*, 355 NEJM 1018 (2006) (Ex. A, Tab 8)).)

By contrast, this was not N.C.’s presentation and the study by Kashiwagi et al., (also cited by petitioners), found post-vaccination cytokines were produced at much lower levels. (Ex. A, p. 7 (Kashiwagi et al., *supra*, at Ex. A, Tab 10).) Dr. McCusker explained that “while peripheral cytokines are released by vaccination, the levels are extremely low, even in the case of booster vaccination.” (*Id.* at 13 (citing Kashiwagi et al., *supra*, at Ex. A, Tab 10).) According to Dr. McCusker, the cytokine levels demonstrated in the Kashiwagi et al. study are lower than would be expected to affect the seizure threshold as hypothesized by petitioners’ experts. (*Id.*; see also *Id.* at 14 (citing Wen-Hsuan W. Lin et al., *Plasma Cytokines and Chemokines in Zambian*

Children with Measles: Innate Responses and Association with HIV-1 Coinfection and In-Hospital Mortality, 215 J. INFECTIOUS DISEASES 830 (2017) (Ex. A, Tab 33)); Celine Dubé et al., *Interleukin-1 β Contributes to the Generation of Experimental Febrile Seizures*, 57 ANNALS NEUROLOGY 152 (2005) (Ex. A, Tab 26)).) Here, “there were no significant clinical signs or symptoms in N.C. consistent with chronic cytokine activation sufficient to cause [central nervous system] damage. Indeed, he was in a good state of health from May 8-11, 2017, when he was put to bed on the evening of his death.” (Ex. G, p. 3 (citing Ex. 1, p. 3).) Moreover, Talaat et al. shows that post-vaccination proinflammatory cytokines peak after 16-24 hours and return to baseline by 44 hours. (Ex. I, p. 3 (citing Kawsar R. Talaat et al., *Rapid Changes in Serum Cytokines and Chemokines in Response to Inactivated Influenza Vaccination*, 12 INFLUENZA & OTHER RESPIRATORY VIRUSES 202 (2018) (Ex. I, Tab 2)).) In mice, cytokine levels have been shown to peak at 48 hours post-vaccination and return to baseline within 7 days. (*Id.* at 3-4 (citing Tetsuo Nakayama et al., *Long-term Regulation of Local Cytokine Production Following Immunization in Mice*, 62 MICROBIOLOGY & IMMUNOLOGY 124 (2018) (Ex. I, Tab 3)).) Talaat et al. showed that patients experiencing systemic side effects after vaccination (specifically the flu vaccine) had elevated cytokine levels for only 1-2 days post-vaccination. (Ex. I, p. 4 (citing Talaat et al, *supra*, at Ex. I, Tab 2).)

Contrary to Dr. Rostad’s suggestion that the cytokine response to vaccination may have acted in concert with N.C.’s May 7 infection, Dr. McCusker explained that prior studies have shown that vaccinations do not worsen viral illness or inflammatory autoimmune disease. (Ex. A, pp. 7-8 (discussing Thomas G. Boyce et al., *Pertussis Vaccination and the Risk of Respiratory Syncytial Virus-Associated Hospitalization*, 23 PEDIATRIC INFECTIOUS DISEASE J. 897 (2004) (Ex. A, Tab 11); Johanna Westra et al., *Vaccination of Patients with Autoimmune Inflammatory Rheumatic Diseases*, 11 NATURE REVS. RHEUMATOLOGY 135 (2015) (Ex. A, Tab 12)).) After reviewing Dr. Akbari’s supporting citations, she further concluded that Dr. Akbari has not presented literature that would support the hypothesis that the vaccine components N.C. received “induced a catastrophic autoimmune response either T cell or cytokine mediated.” (*Id.* at 10.)

Regarding the relationship between cytokines and seizures, Dr. McCusker explained that certain cytokines, namely IL-1 β and IL-6, act as neurotransmitters and so it is not surprising that they would be increased in epilepsy or following a seizure. (Ex. A, p. 10.) Cytokine upregulation is a *consequence* of seizure activity rather than an etiologic factor. (*Id.* at 10-11 (citing Gang Li et al., *Cytokines and Epilepsy*, 20 SEIZURE 249 (2011) (Ex. A, Tab 22); A. Vezzani et al., *Powerful Anticonvulsant Action of IL-1 Receptor Antagonist on Intracerebral Injection and Astrocytic Overexpression in Mice*, 97 PROCS. NAT’L ACAD. SCIS. 11534 (2000) (Ex. A, Tab 23); Annamaria Vezzani & Tallie Z. Baram, *New Roles for Interleukin-1 Beta in the Mechanisms of Epilepsy*, 7 EPILEPSY CURRENTS 45 (2007) (Ex. A, Tab 24)).) A study by Li et al. examined the role of proinflammatory cytokines in causing seizures and determined based on an animal model that, while super-physiological levels of IL-1 β had epileptogenic potential, levels comparable to what occurs in vaccination appeared to have an anti-epileptic effect. (*Id.* (Li et al., *supra*, at Ex. A, Tab 22).) Moreover, IL-1 β has a very short half-life, with clearance occurring within 19 minutes of release. (*Id.* at 13 (citing Shoji Kudo et al.,

Clearance and Tissue Distribution of Recombinant Human Interleukin 1 β in Rats, 50 *CANCER RSCH.* 5751 (1990) (Ex. A, Tab 28)).) While cytokines can cross the blood brain barrier, the pathways for this to occur regulate both the location and amount of cytokines expressed. (*Id.* at 9.) “Local production of cytokines by the [central nervous system] parenchymal cells are more likely to mediate any neuroimmune communication compared with those produced in the periphery.” (*Id.* (citing Ning Quan, *In-Depth Conversation: Spectrum and Kinetics of Neuroimmune Afferent Pathways*, 40 *BRAIN, BEHAVIOR, & IMMUNITY* 1 (2014) (Ex. A, Tab 18)).) In fact, Dr. McCusker opined that “[t]here is no evidence submitted that low levels of peripheral cytokines released during vaccination result in pathological increases in [central nervous system] cytokines.” (*Id.* at 13.)

Dr. McCusker does not agree that the hippocampal changes discussed by Dr. Rostad have been clearly implicated as a cause of seizures or a contributor to SUDC. (Ex. I, p. 5 (citing Dominique F. Leitner et al., *Blinded Review of Hippocampal Neuropathology in Sudden Unexplained Death in Childhood Reveals Inconsistent Observations and Similarities to Explained Paediatric Deaths*, 48 *NEUROPATHOLOGY & APPLIED NEUROBIOLOGY* 1 (2021) (Ex. I, Tab 5); Declan McGuone et al., *Neuropathologic Changes in Sudden Unexplained Death in Childhood*, 79 *J. NEUROPATHOLOGY & EXPERIMENTAL NEUROLOGY* 336 (2020) (Ex. I, Tab 6)).) In any event, while Dr. Rostad cited work by Kinney et al. to support a role for seizures in SUDC, Dr. McCusker stressed that the cited study does not identify vaccination as a risk factor. (Ex. A, p. 12 (discussing Kinney et al., *supra*, at Ex. 18, Tab DDD).) By contrast, she cited several studies²⁴ that she indicated constitute evidence against a causal relationship between vaccines and SIDS. (Ex. G, pp. 8-9.) Moreover, while the Kinney et al. study hypothesized an association between febrile seizures and SUDC, the study results were not necessarily clear in distinguishing either hippocampal anomalies or family history of febrile seizures from controls. (Ex. A, p. 12 (citing Kinney et al., *supra*, at Ex. 18, Tab DDD).) Additional studies by Hesdorffer et al. and Hefti et al. did not find that a family history of febrile seizures was significantly associated with SUDC. (*Id.* (discussing Dale C. Hesdorffer et al., *Sudden Unexplained Death in Childhood: A Comparison of Cases with and Without a Febrile Seizure History*, 58 *EPILEPSIA* 1294 (2015) (Ex. A, Tab 25) (see also Ex. 13, Tab R); and Hefti et al., *supra*, at Ex. 13, Tab S).) In any event, Dr. McCusker cited a number of studies²⁵ for the proposition that the DTaP vaccine has not

²⁴ Specifically: Pedro L. Moro et al., *Deaths Reported to the Vaccine Adverse Event Reporting System (VAERS), United States, 1997-2013*, 61 *CLINICAL INFECTIOUS DISEASES* 980 (2015) (Ex. G, Tab 3) (see also Ex. 21, Tab 13); Richard D. Goldstein et al., *Overall Postneonatal Mortality and Rates of SIDS*, 137 *PEDIATRICS* 1 (2016) (Ex. G, Tab 4); Ronny Kuhnert et al., *Reanalyses of Case-Control Studies Examining the Temporal Association Between Sudden Infant Death Syndrome and Vaccination*, 30 *VACCINE* 2349 (2012) (Ex. G, Tab 5) (see also Ex. 21, Tab 14); Jacqueline Müller-Nordhorn et al., *State-Level Trends in Sudden Unexpected Infant Death and Immunization in the United States: An Ecological Study*, 21 *BMC PEDIATRICS* 1 (2021) (Ex. G, Tab 6) (see also Ex. 21, Tab 16)).

²⁵ Specifically: Sarah von Spiczak et al., *A Retrospective Population-Based Study on Seizures Related to Childhood Vaccination*, 52 *EPILEPSIA* 1506 (2011) (Ex. A, Tab 29); Nienke E. Verbeek et al., *Etiologies for Seizures Around the Time of Vaccination*, 134 *PEDIATRICS* 658 (2014) (Ex. A, Tab 30); Wan-Ting Huang et al., *Lack of Association Between Acellular Pertussis Vaccine and Seizures in Early Childhood*, 126 *PEDIATRICS* e263 (2010) (Ex. A, Tab 31); Jo M. Wilmshurst et al., *Summary of Recommendations for the*

been shown to be a cause of seizures. (*Id.* at 13-14.) She further suggested that the literature cited by petitioners' experts likewise do not support a causal relationship between the DTaP vaccine and seizures and further disputing that evidence related to the whole cell DPT vaccine is relevant. (Ex. A, p. 14 (citing at CDC Report, *supra*, at Ex. 18, Tab LLL; Sun et al., *supra*, at Ex. 18, Tab MMM; Paolo Bellavite, *Causality Assessment of Adverse Events Following Immunization: The Problem of Multifactorial Pathology*, 9 F1000 RSCH. 1 (2020) (Ex. 18, Tab VVV)); Ex. G, pp. 3-4 (citing Paddy Farrington et al., *A New Method for Active Surveillance of Adverse Events from Diphtheria/Tetanus/Pertussis and Measles/Mumps/Rubella Vaccines*, 345 LANCET 567 (1995) (Ex. 19, Tab N); Moro et al., *supra*, at Ex. 13, Tab NNN).)

V. Analysis

a. Medical theory of causation (*Althen* prong one)

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

There have been a significant number of prior cases in this Program that have addressed allegations that one or more childhood vaccines caused or contributed to a SIDS or SUDC-labeled death. However, given that these deaths have necessarily been deemed "unexplained," prior petitioners have, unsurprisingly, failed to come forward with sound and reliable evidence that suggests a more likely than not cause of the death. See, e.g., *Anklam v. Sec'y of Health & Human Servs.*, No. 17-2061V, 2023 WL 8890662 (Fed. Cl. Spec. Mstr. Nov. 29, 2023) (SUDC); *Whitesell v. Sec'y of Health & Human Servs.*, No. 17-1557V, 2022 WL 3081327 (Fed. Cl. Spec. Mstr. July 12, 2022) (SUDC); *Bohn v. Sec'y of Health & Human Servs.*, No. 16-0265V, 2021 WL 4302367 (Fed. Cl. Spec. Mstr. Aug. 23, 2021) (SIDS); *Wunderler v. Sec'y of Health & Human Servs.*, No. 19-1468V, 2020 WL 4346694 (Fed. Cl. Spec. Mstr. July 7, 2020) (sudden unexplained death ("SUDS")); *Martin v. Sec'y of Health & Human Servs.*, No. 15-789V,

Management of Infantile Seizures: Task Force Report for the ILAE Commission of Pediatrics, 56 EPILEPSIA 1185 (2015) (Ex. A, Tab 32)); see also Ex. G, pp. 3-4 (citing Dana Craiu et al., *Vaccination and Childhood Epilepsies*, 36 EUR. J. PAEDIATRIC NEUROLOGY 57 (2022) (Ex. G, Tab 1)).

2020 WL 4197748 (Fed. Cl. Spec. Mstr. May 8, 2020) (SUDC), *mot. rev. den'd*, 158 Fed. Cl. 459 (2020); *Olsavicky v. Sec'y of Health & Human Servs.*, No. 17-1806, 2019 WL 2881009 (Fed. Cl. Spec. Mstr. June 4, 2019) (SIDS); *Nunez v. Sec'y of Health & Human Servs.*, No. 14-863V, 2019 WL 2462667 (Fed. Cl. Spec. Mstr. Mar. 29, 2019) (SIDS), *mot. rev. den'd*, 144 Fed. Cl. 540 (2019), *aff'd*, 825 F. App'x 816 (Fed. Cir. 2020); *Fraday v. Sec'y of Health & Human Servs.*, No. 16-148V, 2017 WL 5379391 (Fed. Cl. Spec. Mstr. Sept. 20, 2017) (SIDS); *Pelton v. Sec'y of Health & Human Servs.*, No. 14-674V, 2017 WL 1101767 (Fed. Cl. Spec. Mstr. Feb. 27, 2017) (SIDS); *Jewell v. Sec'y of Health & Human Servs.*, No. 11-138V, 2016 WL 5404165 (Fed. Cl. Spec. Mstr. Aug. 29, 2016) (SIDS); *Copenhaver v. Sec'y of Health & Human Servs.*, No. 13-1002V, 2016 WL 3456436 (Fed. Cl. Spec. Mstr. May 31, 2016) (SIDS), *mot. rev. den'd*, 129 Fed. Cl. 176 (2016); *Lord v. Sec'y of Health & Human Servs.*, No. 12-255V, 2016 WL 806818 (Fed. Cl. Spec. Mstr. Feb. 9, 2016) (SIDS); *Cozart v. Sec'y of Health & Human Servs.*, No. 00-590V, 2015 WL 6746616 (Fed. Cl. Spec. Mstr. Oct. 15, 2015) (SIDS), *mot. rev. den'd*, 126 Fed. Cl. 488 (2016); *Waterman v. Sec'y of Health & Human Servs.*, No. 13-960V, 2015 WL 4481244 (Fed. Cl. Spec. Mstr. June 30, 2015) (SIDS), *mot. rev. den'd*, 123 Fed. Cl. 564 (2015); *Sanchez v. Sec'y of Health & Human Servs.*, No. 11-651V, 2013 WL 4476750 (Fed. Cl. Spec. Mstr. July 26, 2013) (SIDS); *Bigbee v. Sec'y of Health & Human Servs.*, No. 06-663V, 2012 WL 1237759 (Fed. Cl. Spec. Mstr. Mar. 22, 2012) (SIDS); *Heller v. Sec'y of Health & Human Servs.*, No. 96-0797V, 1998 WL 408612 (Fed. Cl. Spec. Mstr. June 22, 1998) (SIDS).²⁶

In some instances, the parties have litigated whether SIDS presents an alternative explanation to what petitioners otherwise alleged to have been a vaccine-caused death. See, e.g., *Doe 11 v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1351 (Fed. Cir. 2010) (holding that “the special master did not commit legal error in considering evidence of SIDS, an allegedly alternative cause. Nothing in the Vaccine Act prohibits the government from presenting evidence that the petitioner's injury was due to ‘factors unrelated’ to the vaccine (here, SIDS)”); *Bigbee*, 2012 WL 1237759, at *47-49. However, many of these prior cases have directly addressed at length allegations that one or more vaccines directly caused or contributed to a child's death within a framework of SIDS or SUDC itself. For example, it has been repeatedly found that attempts to establish vaccination as an exogenous stressor under the accepted Triple Risk Model of SIDS were unpersuasive. See, e.g., *Jewell*, 2016 WL 5404165, at *13; *Copenhaver*, 2016 WL 3456436, at *12-13; *Lord*, 2016 WL 806818, at *14; *Cozart*, 2015 WL 6746616, at *13. One such case, *Boatmon v. Secretary of Health and Human Services*, was reviewed by the Federal Circuit. 941 F.3d 1351 (Fed. Cir. 2019).²⁷

²⁶ Special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Human Servs.*, 76 Fed. Cl. 328, 338–39 (2007) (“[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim”). Nonetheless, special masters are not bound by the prior decisions of other special masters or of the Court of Federal Claims. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). Thus, these cases do not dictate the outcome in this case.

²⁷ In *Boatmon*, the special master initially found petitioners entitled to compensation before being overturned by the Court of Federal Claims. 941 F.3d. at 1353. The Court of Federal Claims was then

Although there are fewer prior SUDC cases, petitioners have not fared any better when seeking to assert vaccine causation within the research framework associated with SUDC. *E.g.*, *Anklam*, 2023 WL 8890662, at *40 (rejecting petitioners' theory in part because "the idea that hippocampal abnormalities cause children to be vulnerable and at risk of sudden death is likewise not sound and reliable"); *Martin*, 2020 WL 4197748, at *27 (distinguishing a case of SUDC from prior SIDS precedent, but explaining that "this claim, like the prior SIDS cases, relies on the theory that vaccine-induced cytokine interference with the brain in some way has pathologic, and ultimately fatal, outcomes under circumstances involving very young children that otherwise remain mysterious to medical science").

In *Boatmon*, the petitioners' expert (Dr. Miller) had theorized that post-vaccination cytokines "can inhibit the activity of 5-hydroxytryptamine ('5-HT' or serotonin) neurons in the medulla, causing prolonged apnea and interference with autoresuscitation." 941 F.3d at 1356. This was presented as an application of the so-called "Triple Risk Model" of SIDS by Kinney et al., which suggested that (1) a vulnerable infant, i.e. one having a defect of the medulla, may (2) experience an exogenous stressor, during (3) a critical developmental period, and thereby experience a sudden unexplained death. *Id.* at 1355-56. However, the Federal Circuit explained that "[i]t would be an extension of the Triple Risk Model to include vaccination-induced cytokine activity in the list of exogenous stressors as Dr. Miller proposes. Dr. Miller himself concedes that, outside of Vaccine Act litigation, vaccinations have not been identified as an exogenous stressor for SIDS." *Id.* at 1360. They further stressed that, petitioners were unable to move beyond asserting the presence of cytokines in the brain to explain how cytokine function in the brain supported their theory. *Id.* at 1361. Accordingly, the Circuit concluded that, whereas they were obligated to present "sound and reliable" scientific opinion, the petitioners had presented a merely "plausible" or "possible" theory that was insufficient to meet their burden of proof under *Althen* prong one.²⁸ *Id.* at 1361-62.

In this case, petitioners theorize that "the DTaP vaccine initiated a hyperimmune reaction that eventually led to a terminal seizure in N.C., a susceptible host." (ECF No.

affirmed by the Court of Appeals for the Federal Circuit, though with differences in reasoning. *Id.* The instant petition was filed between the time the Court of Federal Claims reversed the special master's ruling and the time when the Federal Circuit affirmed the Court of Federal Claims.

²⁸ Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014). However, the Federal Circuit has also stressed that "[c]ausation in fact under the Vaccine Act is ... based on the circumstances of the particular case." *Boatmon*, 941 F.3d at 1358-59 (quoting *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cr. 1994)). Thus, Federal Circuit precedents do not automatically control the outcome of subsequent cases even when they involve the same injury. *See, e.g., Sanchez v. Sec'y of Health & Human Servs.*, 809 Fed. Appx. 843, 851-52 (Fed. Cir. 2020) (citing back to a prior Federal Circuit holding in *Paluck v. Secretary of Health & Human Services* involving the same injury and noting that "while there are substantial parallels between this case and *Paluck*, the differences between the two cases are such that the outcome of this case is not dictated by *Paluck*").

85, p. 29.) This theory encompasses several underlying assertions. First, petitioners explain that the DTaP vaccine produces an innate immune response that includes the production of cytokines. (*Id.* at 30-31.) This is the only aspect of petitioner’s theory that is not controverted. Second, petitioners argue that immune dysregulation can result in a positive feedback loop that causes the cytokine response from vaccination to become a “cytokine storm,” a damaging and potentially fatal overproduction of cytokines. (*Id.* at 31.) Third, petitioners argue that, concomitantly, there are several pathways by which the DTaP vaccine can dysregulate the adaptive immune response to result in autoimmunity. (*Id.* at 32-33.) Most significantly, aluminum within the DTaP vaccine can damage the blood brain barrier, opening up the central nervous system to an inflammatory reaction. (*Id.*) Fourth, once the blood brain barrier has been made “more accessible,” the further inflammatory response from a subsequent upper respiratory infection can further inflame the brain. (*Id.* at 34.) And, fifth, where a subject is susceptible to seizures due to a hippocampal malformation, cytokine damage within the brain can decrease the seizure threshold and hyper-excite the neuronal network, resulting in a terminal seizure as evidenced in the SUDC literature. (*Id.* at 35-37.) Specifically, they assert that a seizure can result in cerebral edema and fatal brain herniation. (*Id.* at 38.)

Although petitioners’ theory invokes several different concepts and includes multiple steps, it is important to note that it remains firmly grounded in the idea that a vaccination can initiate a process that ultimately results in a terminal seizure consistent with what they assert is hypothesized as the cause of at least a subset of SUDC cases. Indeed, petitioners have filed some 32 publications, either introduced or discussed by their experts,²⁹ having the primary purpose of exploring the potential cause of SIDS, SUDC, and/or SUDEP, representing a body of literature current up to 2024, and their experts have affirmatively relied on concepts and data derived from this body of literature. Moreover, Dr. Rostad, petitioner’s pathology expert, agreed that N.C.’s autopsy was correct in initially classifying N.C.’s death as a sudden unexplained death. (Ex. 18, p. 8 (citing Ex. 5, p. 37).) Citing studies by Kinney et al. and Hefti et al., he opined that

[t]hese studies suggested that hippocampal formation] malformation represented a marker of an impaired forebrain/limbic network that increases the risk of sudden death due to potential brainstem cardiorespiratory related circuits nuclei, or to a subclinical seizure in an infant predisposed to epilepsy. The association of epilepsy-related pathology (such as HF maldevelopment) with SIDS, SUDEP and SUDC leads to possible epilepsy-related mechanisms in sudden death.

(*Id.* at 25 (citing Kinney et al., *supra*, at Ex. 18, Tab XXX; Hefti et al., *supra*, at Ex. 13, S).) Thus, despite the preceding steps of petitioners’ theory, this case still ultimately falls squarely within the prior line of cases, including *Boatmon*, in which petitioners have

²⁹ Some of these 32 filings are instances where petitioners’ experts resubmitted papers originally cited by respondent’s experts for purposes of highlighting counterpoints.

attempted to leverage research into the phenomena of SIDS and SUDC as evidence supporting a theory of vaccine causation within that framework. That is, rather than seeking to wholly supplant the SUDC concept, petitioners purport to have presented a theory that explains how the leading hypotheses of SUDC could actually support vaccination as a trigger. However, similar to what was explained by the Federal Circuit in *Boatmon*, this effort fundamentally lacks support in the relevant body of literature.

Petitioners' pathology expert has sought to distinguish this case from *Boatmon* based on differences between the leading hypotheses of SIDS and SUDC. (Ex. 19, pp. 30-32.) To that point, prior decisions have noted that SIDS and SUDC are not interchangeable. *Martin*, 2020 WL 4197748, at *27 (noting that "[a]ll of the experts testifying in this case agreed that SIDS and SUDC are not congruent, and that the 'triple risk' model employed in SIDS circumstances has no direct application here"). In particular, Dr. Rostad has explained that petitioners' theory of causation in this case is premised on a hippocampal abnormality leading to a terminal seizure rather than being premised on a medullary defect leading to arrested breathing as was at issue in *Boatmon*.³⁰ (Ex. 19, pp. 30-32.) Yet, Dr. Rostad conceded that "[d]espite the robust SUID/SIDS literature, research about the relationship of immunization and SUID/SIDS is scant. The papers evaluating vaccine as a risk factor in SUDC are even more rare." (Ex. 19, p. 37.) "Furthermore, there is a considerable gap in knowledge with regards to SUDC pathobiology." (*Id.*) Given these concessions, if it was unreliable to extend the hypotheses underlying SIDS to vaccination as was done in *Boatmon*, then it is *a fortiori* unreliable to do the same in the context of SUDC, which petitioners' experts explain to be even less well understood. *Accord Martin*, 2020 WL 4197748, at *27; *Anklam*, 2023 WL 8890662, at *39-40.

Indeed, of the 32 publications petitioners' experts have discussed regarding the potential cause(s) of SIDS, SUDC, and/or SUDEP, 20 include no mention of vaccinations whatsoever, even where other risk factors for these phenomenon are discussed.³¹ Of the remaining twelve, five (mostly case reports) reference a history of

³⁰ However, while Dr. Rostad explicitly disclaimed reliance on the Triple Risk Model, he nonetheless cited approvingly to a paper by Kinney, et al., for the proposition that "SIDS, SUDC, and SUDEP cases represent a spectrum of serotonopathy that affects the caudal and rostral 5-HT domains in the brainstem and their preferential targets, including the hippocampus." (Ex. 19, p. 32 (citing Hannah C. Kinney et al., *Sudden and Unexpected Death in Early Life: Proceedings of a Symposium in Honor of Dr. Henry F. Krous*, 8 FORENSIC SCI., MED. & PATHOLOGY 414 (2012) (Ex. 19, Tab R)); see also Kinney et al., *supra*, at Ex. 18, Tab XXX, p. 1 (suggesting hippocampal abnormalities may represent "a common vulnerability that defies the 1-year age cutoff between SIDS and SUDC").) Moreover, both of petitioners' experts explicitly rely in part on papers and data pertaining to SIDS. (*E.g.*, Ex. 13, pp. 13-14 (citing W.G. Guntheroth, *Interleukin-1 as Intermediary Causing Prolonged Sleep Apnea and SIDS During Respiratory Infections*, 28 MED. HYPOTHESES 121 (1989) (Ex. 13, Tab TT) (see also Ex. 20, Tab Q)); Ex. 20, p. 3 (citing Walker, *supra*, at Ex. 20, Tab C); Ex. 18, pp. 24-25 (citing Hannah C. Kinney et al., *Dentate Gyrus Abnormalities in Sudden Unexplained Death in Infants: Morphological Marker of Underlying Brain Vulnerability*, 129 ACTA NEUROPATHOLOGICA 65 (2015) (Ex. 18, Tab ZZZ).) In that regard, Dr. Akbari did initially invoke the idea that his theory was compatible with the idea that SUDC is partly explained by a medullary defect. (Ex. 13, p. 14.)

³¹ Specifically: Hannah C Kinney & Bradley T. Thach, *The Sudden Infant Death Syndrome*, 361 NEJM 795 (2009) (Ex. 13, Tab M); Felicia L. Trachtenberg et al., *Risk Factor Changes for Sudden Infant Death*

vaccination among subjects but without suggesting any causal role³² and five actually indicate that vaccines are exonerated as a cause of sudden unexplained deaths.³³ Importantly, among these 32 publications, eight specifically acknowledge concepts pertaining to petitioners' theory of causation – hippocampal abnormalities leading to seizure – as a mechanism of sudden death, but include no discussion of vaccination as a potential trigger.³⁴ By contrast, petitioners' experts have presented only two articles purporting to implicate vaccination as a cause of sudden death. (Ex. 20, p. 3 (citing to Alexander M. Walker et al., *Diphtheria-Tetanus-Pertussis Immunization and Sudden Infant Death Syndrome*, 77 AM. J. PUB. HEALTH 945 (1987) (Ex. 20, Tab C)³⁵; Neil Z.

Syndrome After Initiation of Back-to-Sleep Campaign, 129 Pediatrics 630 (2012) (Ex. 13, Tab N); Ali Riza Tümer et al., *Sudden Unexpected Child Deaths: Forensic Autopsy Results in Cases of Sudden Deaths During a 5-Year Period*, 51 J. TROPICAL PEDIATRICS 131 (2005) (Ex. 13, Tab O); Henry F. Krous et al., *Sudden Unexpected Death in Childhood: A Report of 50 Cases*, 8 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 307 (2005) (Ex. 13, Tab Q); Hefti et al., *supra*, at Ex. 13, Tab S; Guntheroth, *supra*, at Ex. 13, Tab TT; Hefti et al., *supra*, at Ex. 18, Tab F; James J. Filiano & Hannah C. Kinney, *Arcuate Nucleus Hypoplasia in the Sudden Infant Death Syndrome*, 51 J. NEUROPATHOLOGY & EXPERIMENTAL NEUROLOGY 394 (1992) (Ex. 18, Tab G); H.C. Kinney et al., *Subtle Developmental Abnormalities in the Inferior Olive: An Indicator of Prenatal Brainstem Injury in the Sudden Infant Death Syndrome*, 61 J. NEUROPATHOLOGY & EXPERIMENTAL NEUROLOGY 427 (2002) (Ex. 18, Tab H); Kinney et al., *supra*, at Ex. 18, Tab DDD; Myers et al., *supra*, at Ex. 18, Tab FFF; Dlouhy et al., *supra*, at Ex. 18, Tab GGG; Kinney et al., *supra*, at Ex. 18, Tab XXX; Hannah C. Kinney et al., *Abnormalities of the Hippocampus in Sudden and Unexpected Death in Early Life*, in SIDS – SUDDEN INFANT AND EARLY CHILDHOOD DEATH 661 (Jhodie R. Duncan & Roger W. Byard eds., 2018) (Ex. 18, Tab YYY); Kinney et al., *supra*, at Ex. 18, Tab ZZZ; Maxine Dibué et al., *Sudden Death in Epilepsy: There is Room for Intracranial Pressure*, 10 BRAIN & BEHAVIOR 1 (2020) (Ex. 19, Tab I); Laura Crandall & Orrin Devinsky, *Sudden Unexplained Death in Children*, 1 LANCET 8 (2017) (Ex. 19, Tab S); Matthew Halvorsen et al., *De Novo Mutations in Childhood Cases of Sudden Unexplained Death that Disrupt Intracellular Ca²⁺ Regulation*, 118 PROCS. NAT'L ACAD. SCIS. 1 (2021) (Ex. 21, Tab 12); Orrin Devinsky et al., *Underestimation of Sudden Deaths Among Patients with Seizures and Epilepsy*, 89 NEUROLOGY 886 (2017) (Ex. 22, Tab 9); Gould et al., *supra*, at Ex. 25, Tab 1.

³² Specifically: D.P. Southall et al., *Sudden and Unexpected Death Between 1 and 5 Years*, 62 J. ARCHIVES DISEASE CHILDHOOD 700 (1987) (Ex. 13, Tab P); Hesdorffer et al., *supra*, at Ex. 13, Tab R; Laura Gould Crandall et al., *Potential Role of Febrile Seizures and Other Risk Factors Associated with Sudden Death in Children*, 2 JAMA 1 (2019) (Ex. 13, Tab V); Kinney et al., *supra*, at Ex. 18, Tab EEE; Hannah C. Kinney et al., *Sudden Death in Toddlers Associated with Developmental Abnormalities of the Hippocampus: A Report of Five Cases*, 10 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 208 (2007) (Ex. 18, Tab WWW).

³³ Specifically: JHODIE R. DUNCAN & ROGER W. BYARD, SIDS SUDDEN INFANT AND EARLY CHILDHOOD DEATH: THE PAST, THE PRESENT AND THE FUTURE (2018) (Ex. 13, Tab J, pp. 46, 351, 479); Stephen M. Adams et al., *Sudden Infant Death Syndrome*, 79 AM. FAM. PHYSICIAN 870 (2009) (Ex. 13, Tab K, p. 3); Moro et al., *supra*, at Ex. 21, Tab 13, pp. 2, 5-6; Kuhnert et al., *supra*, at Ex. 21, Tab 14, pp. 7-8; Müller-Nordhorn et al., *supra*, Ex. 21, Tab 16, pp. 6-8.

³⁴ Specifically: Hefti et al., *supra*, at Ex. 13, Tab S; Crandall et al., *supra*, at Ex. 13, Tab V; Hefti et al., *supra*, at Ex. 18, Tab F; Kinney et al., *supra*, at Ex. 18, Tab DDD; Kinney et al., *supra*, at Ex. 18, Tab XXX; Kinney et al., *supra*, at Ex. 18, Tab YYY; Crandall et al., *supra*, at Ex. 19, Tab S; Devinsky et al., *supra*, at Ex. 22, Tab 9.

³⁵ In fact, although Dr. Akbari cites this paper by Walker et al., his report includes a quotation that he attributes to a presentation by William Torch. (Ex. 20, p. 3.) A paper by Dr. Torch is among the references listed in the Walker paper. (Walker et al., *supra*, at Ex. 20, Tab C, p. 6.) However, the quotation included within Dr. Akbari's report is not included in the paper and no paper by Dr. Torch has

Miller, *Vaccines and Sudden Infant Death: An Analysis of the VAERS Database 1990-2019 and Review of the Medical Literature*, 8 TOXICOLOGY REPS. 1324 (2021) (Ex. 26)).) However, these studies are unpersuasive.

Walker et al. is a study from 1987 that examined medical records for a population of 26,500 infants to determine immunizations status and mortality between 1972 and 1983. (Ex. 20, Tab C, p. 1.) The authors identified 29 instances of SIDS based on their definition (*i.e.* any death for which no cause could be discerned among infants of normal birth weight with no predisposing medical conditions). (*Id.*) Of that group, six were never immunized. (*Id.* at 2.) Of the remaining 23 subjects, there were increased incidences of death within three days of a DPT vaccine followed by decreased incidences between 4-29 days post-DPT vaccination. (*Id.* at 2, 4.) There are several reasons why this finding is not significant evidence in this case. First, despite the size of the population from which the study was drawn, the finding of elevated instances of SIDS in the three days post-vaccination was based on only four deaths, followed by a noted decrease in mortality wherein the confidence interval extended to into mortality deficit. (*Id.* at 2.) The authors suggest the subsequent decrease below typical mortality rates could be a “compensatory decline,” but also specifically caution that “[t]he relatively small number of SIDS cases in the present study also admits the possibility of substantial random error.” (*Id.* at 2, 4.) Second, based on the finding that six of the 29 SIDS subjects were not vaccinated, the authors found overall that the resulting mortality rate among the unvaccinated infants was 6.5 times higher than among vaccinated infants. (*Id.* at 1-2.) And third, this study predates the introduction of the acellular pertussis vaccine and assesses the earlier whole cell pertussis vaccine. Special masters have repeatedly found that the safety profile of the whole cell pertussis vaccine cannot be imputed to the acellular pertussis vaccine. See, e.g., *Bangerter v. Sec’y of Health & Human Servs.*, No. 15-1186V, 2022 WL 439535, at *21 (Fed. Cl. Spec. Mstr. Jan. 18, 2022); *Martin v. Sec’y of Health & Human Servs.*, No. 13-486V, 2020 WL 6865931, at *30 (Fed. Cl. Spec. Mstr. Oct. 27, 2020); *Kottenstette v. Sec’y of Health & Human Servs.*, No. 15-1016V, 2020 WL 4197301, at *8-9 (Fed. Cl. Spec. Mstr. June 2, 2020) (Horner), *rev’d on other grounds*, 861 F. App’x 433 (Fed. Cir. 2021); *Sharpe v. Sec’y of Health & Human Servs.*, No. 14-65V, 2018 WL 7625360, at *31-32 (Fed. Cl. Spec. Mstr. Nov. 5, 2018); *Taylor v. Sec’y of Health & Human Servs.*, No. 05-1133V, 2012 WL 4829293, at *30 (Fed. Cl. Spec. Mstr. Sept. 20, 2012); *Holmes v. Sec’y of Health & Human Servs.*, No. 08-185V, 2011 WL 2600612, at *20 (Fed. Cl. Spec. Mstr. Apr. 26, 2011); *Simon v. Sec’y of Health & Human Servs.*, No. 05-941V, 2007 WL 1772062, at *7 (Fed. Cl. Spec. Mstr. June 1, 2007); *Grace v. Sec’y of Health & Human Servs.*, No. 04-[redacted], 2006 WL 3499511, at *9 (Fed. Cl. Spec. Mstr. Nov. 30, 2006); *but see Kottenstette v. Sec’y of Health & Human Servs.*, 2017 WL 6601878 (Fed. Cl. Spec. Mstr. Dec. 12, 2017) (Millman), *aff’d* 861 F. App’x 433 (Fed. Cir. 2021). Indeed, Dr. Rostad effectively conceded this point in his final report. (Ex. 25, p. 3 (contending that the DTaP vaccine is not risk free for seizures but acknowledging the risk to at least be “dramatically” reduced).)

been filed by petitioners. However, Dr. McCusker nonetheless explained that the Torch presentation was a case series only, with no examination of background incidences of SIDS. (Ex. G, p. 8.)

The Miller study similarly purported to find that instances of SIDS are clustered within three or seven days post-vaccination. (Miller, *supra*, at Ex. 26, p. 3.) That study examined reports of infant deaths to VAERS³⁶ between 1990 to 2019, identifying 2,605 reported deaths occurring within 60 days of vaccination, 1,048 of which were classified as SIDS. (*Id.*) Among the SIDS cases, 51% occurred within 3 days of vaccination and 75.5% occurred within 7 days of vaccination. (*Id.*) The author suggests that this finding accords with a cytokine-based theory of vaccination causation of SIDS (*Id.* at 9-10); however, as Dr. McCusker observed, this study had no control population or comparison to background rates (Ex. G, p. 8). Moreover, as the author himself notes, the fact that this data was drawn from a system for collecting *voluntary* reports of vaccine adverse events introduced an inherent selection bias. (Miller, *supra*, Ex. 26, p. 9.) He explains that “[t]he main weakness of this study is the potential for reporting bias. Doctors and parents may have been more likely to report a sudden death to VAERS when it occurred in close temporal proximity to vaccination than if it had happened several days or weeks later.” (*Id.* at 9.) By contrast, Dr. McCusker cited another study of VAERS data that concluded that “[n]o concerning pattern was noted among deaths submitted to VAERS during 1997-2013. The main causes of death were consistent with the most common causes of death in the U.S. population.” (Moro et al., *supra*, at Ex. G, Tab 3, p. 2 (see also Ex. 21, Tab 13, p. 1).)

Thus, as in *Boatmon*, petitioners’ experts are attempting to extend the leading hypotheses for SUDC to encompass a purported risk relative to vaccination based largely on their own *ipse dixit*. However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder v. Sec’y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); see also *Isaac v. Sec’y of Health & Human Servs.*, No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *aff’d*, 108 Fed. Cl. 743 (2013), *aff’d*, 540 F. App’x. 999 (Fed. Cir. 2013).

Moreover, petitioners’ theory of causation is also unreliable in its attempt to conflate SUDC and SUDEP. Respondent persuasively argues that the hippocampal abnormalities that petitioners’ experts incorporate into the causal chain they theorize are not actually predictive of SUDC. (ECF No. 92, p. 25.) In particular, respondent cites studies by Leitner et al. and McGuone et al. as undermining petitioners’ assertion that hippocampal malformation leaves an individual more susceptible to seizures. (*Id.* (citing

³⁶ The Vaccine Adverse Event Reporting System (VAERS) is a national safety surveillance program created by the National Childhood Vaccine Injury Act of 1986 that collects information about possible adverse reactions to vaccines that are administered in the U.S. (Miller, *supra*, at Ex. 26, pp. 1, 3.) VAERS collects and analyzes reports adverse events following the administration of a vaccination in the U.S., and each report includes information concerning the patient, the vaccine(s) administered, and the symptoms the patient experienced after vaccination. (*Id.* at 3.) Since the program’s creation in 1990, VAERS has received over 700,000 reports which describe adverse events ranging from mild sild events to serious life-threatening conditions, including death. (*Id.*) The VAERS database is available to and relied upon by both government and independent researchers. (*Id.*) VAERS allows for identification and detection of unusual patterns and important safety concerns regarding vaccines administered in the U.S. (*Id.*)

Leitner et al., *supra*, at Ex. I, Tab 5; McGuone et al., *supra*, at Ex. I, Tab 6; Dominique F. Leitner et al., *Proteomic Differences in Hippocampus and Cortex of Sudden Unexplained Death in Childhood*, 143 ACTA NEUROPATHOLOGICA 585 (2022) (Ex. I, Tab 7)).) Thus, Dr. McCusker opined that the data from these studies “shows that changes in the hippocampus are not predictive of cause of death and do not differentiate patients with known causes of death from those that fall into the SUDC group such as N.C.” (Ex. I, p. 6.) Although Dr. Rostad did cite papers hypothesizing that SIDS, SUDC, and SUDEP may exist as a spectrum (Ex. 19, p. 32 (citing Hannah C. Kinney et al., *Sudden and Unexpected Death in Early Life: Proceedings of a Symposium in Honor of Dr. Henry F. Krous*, 8 FORENSIC SCI., MED. & PATHOLOGY 414 (2012) (Ex. 19, Tab R)); Kinney et al., *supra*, at Ex. 18, Tab XXX)), he acknowledged that “only a subset of SIDS and SUDC show these pathologic changes [of hippocampal malformation], so that not all individuals would be affected. In other words, these population groups are not homogeneous. In addition, individuals who are outside these groups, including ‘controls’ also may show these hippocampal changes.” (*Id.* at 31.) Thus, while positing that these findings counsel “less distinction between SIDS and SUDC, and even SUDEP,” he agreed that present data does not allow for “adoption of a unified hypothesis.” (*Id.*) Prior petitioners have likewise been unpersuasive under *Althen* prong one where they “have extrapolated information taken from studies that relate to children with epilepsy (SUDEP) and applied it to children who do not have epilepsy or a history of prior seizures.” *Anklam*, 2023 WL 8890662, at *40. In this case, despite relying on the idea of “epilepsy-related mechanisms in sudden death” (Ex. 18, p. 25), Dr. Rostad conceded that N.C. did not suffer SUDEP as he did not have a history of epilepsy, even if he did have a witnessed seizure (Ex. 19, p. 10).

In his final report, Dr. Rostad introduced a study by Gould et al. for the proposition that terminal seizures may be an underrecognized cause of SUDC. (Ex. 25; Gould et al., *supra*, at Ex. 25, Tab 1.) He asserted that “[t]his article is extremely relevant to the instant case as it is directly on point with Petitioner’s proposed medical theory that N.C. more likely than not suffered from a terminal seizure and therefore, his death is not unexplained.” (Ex. 25, p. 3.) However, while that Gould et al. study observed convulsive events among seven cases otherwise determined to represent SUDC, the authors acknowledged that hippocampal findings did not correlate with febrile seizure history, that this was a small study that lacked physiologic monitoring to distinguish abnormal movements, and, importantly, that “[t]he cause of the convulsive events in our cases are unknown.” (Gould et al., *supra*, at Ex. 25, Tab 1, pp. 6-7.) Thus, Dr. Holmes observed of the Gould et al. study that “the authors do not conjecture on how such short seizures could cause death” and further noted that neither the Gould et al. study, nor Dr. Rostad, have explained how a short seizure of 8-50 seconds (as reported by Gould et al.) could result in cerebral edema, herniation, and death, especially given that convulsive seizures usually last much longer and yet still do not generally result in death. (Ex. J, p. 1.) The SIDS, SUDC, and SUDEP literature does explore the possibility that the mechanism of death in some sudden death cases may be a terminal seizure; however, contrary to what Dr. Rostad asserts, this literature does not suggest that such a hypothesis renders these deaths “explained” and Gould et al. is not to the contrary. As noted above, Gould et al. explicitly stated that the cause of the

convulsive events they observed remains unknown. (Gould et al., *supra*, at Ex. 25, Tab 1, pp. 6-7.)

Of further note, while the concept of a cytokine storm is indisputably a part of petitioners' theory, as repeatedly cited by their experts (e.g., Ex. 13, pp. 7-9, 12-14; Ex. 18, pp. 20-21; Ex. 19, p. 3; Ex. 20, pp. 2-3.) and as explained in their initial brief (e.g., ECF No. 85, p. 39 (heading titled "In summary, the combination of a DTaP booster vaccine, subsequent cytokine storm, aluminum adjuvant, subsequent infection, and host susceptibility creates a perfect storm for a catastrophic event"), petitioners have ultimately disclaimed any allegation that N.C. suffered a cytokine storm. (ECF No. 96, p. 2.) This issue is further discussed under *Althen* prong two. However, for purposes of *Althen* prong one, it is also important to note that petitioners in this program "must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Brokelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1345 (Fed. Cir. 2020). Given that petitioners' experts repeatedly invoked a cytokine storm to explain how a post-vaccination cytokine response -- which is itself otherwise an expected, and indeed intended, immune response -- could become fatally uncontrolled, petitioners' concession that N.C. did not suffer a cytokine storm dramatically undercuts the reliability of their experts' theory as it would apply in this case. These petitioners are not the first to seek unsuccessfully to use the "cytokine storm" concept in a SUDC case to bolster their cytokine-based theory despite its lack of applicability. E.g., *Martin*, 2020 WL 4197748, at *28 n.37 (noting in dismissal of a SUDC case that "[c]ircumstances might be different if the record supported the conclusion that a 'cytokine storm' had occurred, but Petitioner's experts plainly stated they did not believe this to have occurred herein"); *Whitesell*, 2022 WL 3081327, at *6 (rejecting cytokine storm theory to explain SUDC); see also *Bohn*, 2021 WL 4302367, at *21 (explaining in a SIDS case that, notwithstanding petitioner's assertions, her pathologist expert "agrees on petitioner's behalf that a cytokine storm is not a useful concept in this case. He notes that '[t]his is not a prototypical case of "cytokine storm," a term used to describe fatal or near-fatal events in the setting of severe infection with, usually, septicemia, which is clearly not the case here").

In addition to the above, respondent argues that petitioners' theory of causation fails for numerous other reasons. He contends that there is no evidence that the DTaP vaccine can cause a cytokine storm. (ECF No. 92, pp. 16-17.) Moreover, transient elevations in cytokine levels as would be seen post vaccination have not been shown to trigger seizures. (*Id.* at 17-18.) Petitioners have not demonstrated how peripheral cytokines would affect the central nervous system. (*Id.* at 19.) Dr. Akbari's invocation of autoreactive T cells is entirely unsupported. (*Id.* at 19-20.) Neither the fact that N.C.'s DTaP vaccination was a booster, nor the fact that it contained multiple antigens, indicates that it would be more likely to produce pathogenic levels of cytokines. (*Id.* at 20-21.) Petitioners have not put forward reliable or persuasive evidence regarding any pathologic role for aluminum adjuvant and have not demonstrated that nanoparticles play any obvious role in disease development. (*Id.* at 22-23.) And, finally, petitioners overstate the ability of the DTaP vaccine to cause any type of seizure. The evidence favors only a finding that there is an increased risk of febrile seizure within 1-2 days of

DTaP vaccination. (*Id.* at 23-24.) However, because petitioners' inability to meet their burden of proof is otherwise clear from the analysis herein, and especially the analysis under *Althen* prong two, I do not find it necessary to separately address each of these points as a matter of general causation. Nonetheless, all of these points raised by respondent are well taken and further demonstrate the shortcomings of petitioners' theory of causation.

In light of all of the above, and considering the record as a whole, petitioners have not met their burden of proof under *Althen* prong one.

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

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326-27; *Grant*, 956 F.2d at 1147-48. Medical records are generally viewed as particularly trustworthy evidence. *Cucuras*, 993 F.2d at 1528. However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. See § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder*, 88 Fed. Cl. at 745 n.67 ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."). A petitioner may support a cause-in-fact claim through either medical records or expert medical opinion. § 300aa-13(a). The special master is required to consider all the relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler*, 88 F.4th at 963 (citing *Hines*, 940 F.2d at 1528).

In this case, the available medical opinion *strongly* favors the conclusion that N.C.'s death is unexplained. N.C.'s case has undergone four different pathology reviews – by the medical examiner, the SUDCRRRC, and the two pathology experts opining in this case – and all four reviews resulted in the conclusion that no cause for N.C.'s death is evident. First, as part of the initial investigation, the medical examiner, Dr. Hood, concluded that N.C. died a natural death and concluded that the cause of death was "sudden unexplained death." (Ex. 6, p. 16.) Later, a secondary analysis was conducted by the SUDCRRRC. (Ex. 15.) Following an independent pathology review, as well as additional family interviews and genetic screening, the SUDCCRC also came to the conclusion that N.C.'s death was an unexplained sudden death. (*Id.* at 6.) Although Dr. Rostad criticized the SUDCCRC review, the SUDCCRC report simply characterized their review as "in agreement with the pathologist of record." (*Id.*) In that regard, Dr. Rostad acknowledged that Dr. Hood's autopsy was adequate and opined that he was correct in classifying N.C.'s death as SUDC. (Ex. 18, p. 8.) Additionally, despite seeking to otherwise explain N.C.'s death, Dr. Rostad at a minimum agreed that N.C.'s death "overlapped" with sudden unexplained death (*Id.* at 21 (characterizing N.C.'s death as similar to SUDEP, but without epilepsy)) and placed his findings within that context. Thus, even setting aside his explicit endorsement of Dr. Hood's conclusion, Dr. Rostad's own explanation for N.C.'s death is still firmly grounded in the SUDC literature

as explained under *Althen* prong one. (*Id.* at 24 (indicating that “[t]he autopsy findings, favor that N.C.’s seizure threshold was lowered by hippocampal formation (HF) maldevelopment. Recent evidence has emphasized the importance of the HF maldevelopment in unexplained deaths in the pediatric age group”).) As respondent’s pathology expert, Dr. Harris, summarized:

This tragic child’s death does not have a clear cause, an opinion shared by the multiple physicians evaluating it during the autopsy process and now in retrospect by our evaluations. I also have this opinion and believe this was correctly stated at autopsy and by the body of physicians of the SUDCRRC who evaluated this and many such cases.

(Ex. E, p. 7.) Moreover, after developing their theory of causation, petitioners sought to have the medical examiner revisit his initial conclusions. However, even after being apprised of petitioners’ theory, Dr. Hood did not change his conclusion as to the cause of N.C.’s death and explicitly declined to comment on whether N.C.’s vaccination contributed to his death.³⁷ (Ex. 24, pp. 1, 5.)

Although rebuttable, the “medical records and medical opinion testimony” of treating physicians can be “quite probative,” because “treating physicians are likely to be in the best position to determine whether ‘a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.’” *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1280); accord *Andreu*, 569 F.3d at 1376. This principle has also been applied in the context postmortem conclusions because, although not strictly speaking “treating” physicians, the opinions of coroners and medical examiners are based on their direct autopsy examinations and role in investigating the cause of death. *Nordwall ex rel. Tori v. Sec’y of Health & Human Servs.*, 83 Fed Cl. 477, 488 (2008) (explaining that “[a]n autopsy report by a medical examiner is without question a contemporaneous medical record” and that “[w]hile such records may not have been created in the context of diagnosis and treating a patient, they are contemporaneous records made by a health professional outside the context of litigation, and should be given the same probative weight as other medical records”); *Pelton v. Sec’y of Health & Human Servs.*, No. 14-674V, 2017 WL 1101767, at *13 (Fed. Cl. Spec. Mstr. Feb. 27, 2017) (indicating that “[a]lthough neither Dr. Eisenstat nor the coroner are treating physicians, it remains the case that they were better positioned than [petitioner’s expert] to evaluate the cause and effect based on in-person inspection

³⁷ Both parties raise competing arguments with respect to Dr. Hood’s report and addendum to his report. (ECF No. 92, pp. 30-32; ECF No. 96, pp. 6-10.) Of particular note, petitioner questions the reliability of Dr. Hood’s initial report because he presumed that N.C.’s SUDC was related to cardiac dysthymia related to long QT syndrome whereas the SUDCRRC report subsequently cast doubt on that presumption by ruling out long QT syndrome. (ECF No. 96, pp. 6-7, 9.) However, this is not very significant. Dr. Hood’s conclusion, in both his initial report and, by extension, his addendum, was that N.C.’s death was unexplained. The fact that Dr. Hood’s speculation regarding one potential mechanism of SUDC did not withstand further investigation does not undermine the conclusion that N.C.’s death remains unexplained or in any way suggest any other cause. In contrast, respondent argues that Dr. Hood’s addendum to his report should be given less weight given that it was not produced in the context of his initial evaluation. (ECF No. 92, pp. 31-32.) However, because I have not identified any manner in which Dr. Hood’s addendum meaningfully supports petitioners’ allegations, this issue is moot.

of the scene and physical examination of the body.”); *Bohn*, 2021 WL 4302367, at *11; *Gaskin v. Sec’y of Health & Human Servs.*, No. 21-835V, 2025 WL 786306, at *13 (Fed. Cl. Spec. Mstr. Feb. 11, 2025). Indeed, the Vaccine Act specifies that the special master “shall consider” any “autopsy or coroner’s report which is contained in the record” with respect to the nature or cause of a vaccinee’s death. § 300aa-13(b)(1)(A). Here, I have considered petitioners’ challenges to the medical examiner’s conclusion that N.C.’s death was a sudden unexplained death. However, despite having compiled an extensive record reviewing those findings, they have not presented any significant cause to doubt that conclusion. Accordingly, the medical opinion in this case preponderates in favor of a finding that N.C.’s death remains unexplained.

Dr. Rostad has not preponderantly established that N.C. suffered fatal cerebellar edema and brainstem herniation. Although Dr. Harris did not engage in great detail with Dr. Rostad’s observations, and in fact declined to respond to Dr. Rostad’s annotated autopsy photos, he was clear in asserting that there were “[n]o hemorrhages within the brainstem or neuronal hypoxia-ischemia change to the neurons was seen, so I do not believe the swelling was enough to cause a lethal herniation.” (Ex. E, p. 6.) This is consistent with Dr. Hood’s autopsy report. (Ex. 5, p. 19; Ex. 24, p. 4 (noting brain swelling “as evidenced by some effacement of sulci” and with “mild bilateral uncus impressions” and a modest cerebellar ‘cone,’ but no other herniation of any of its parts.”).) In that regard, Dr. Holmes further explained that a fatal cerebral edema is unlikely given N.C.’s own history. (Ex. C, pp. 7-9.) Whereas cerebral edema can be associated with status epilepticus, that is a more severe presentation than the two seizures alleged by petitioners. (Ex. C, p. 7 (citing *Trinka et al.*, *supra*, at Ex. C, Tab 22).) Moreover, Dr. Holmes opines that cerebral edema does not develop instantaneously following a seizure, meaning that Dr. Rostad’s implication that a terminal seizure resulted in a fatal degree of cerebral edema is unlikely. (Ex. C, pp. 8-9 (citing *Lan et al.*, *supra*, at Ex. C, Tab 32; *Myers et al.*, *supra*, at Ex. C, Tab 33).) In that regard, Dr. Harris noted that N.C.’s last known presentation was not consistent with a developing brain herniation. (Ex. H, p. 2.)

Despite this, Dr. Rostad effectively contends that he completed the most detailed pathology review and therefore his opinion should carry the most weight. (See, e.g., Ex. 19, pp. 3-8; Ex. 22, pp. 2-8 (arguing that the reviews completed by Dr. Harris and SUDCRRC were not complete or comprehensive, notably for their failure to review the scene and gross autopsy photographs).) However, this is not persuasive. Dr. Harris, Dr. Hood, and the SUDCRRC, each separately concluded that there were insufficient abnormalities to set aside the SUDC designation and Dr. Rostad at a minimum acknowledged Dr. Hood’s autopsy to be adequate. (Ex. 18, p. 8.) Criticism of Dr. Harris’s decision not to explicitly rebut Dr. Rostad’s annotated autopsy photos is misdirected given that Dr. Hood – the only pathologist to have completed an in person gross autopsy examination and who created the photographs at issue – was likewise explicit in noting the absence of any herniation despite also acknowledging some degree of swelling. And, as explained above, he did not revisit this finding even after being prompted by petitioners to address Dr. Rostad’s reports and provide a supplement to his original report. (Ex. 24, pp. 4-5.) Without entirely discounting Dr.

Rostad's competing medical judgment, preferring his review would effectively mean accepting that cerebral swelling significant enough to cause death could occur while leaving signs so subtle that only one out of four pathologists would note them and conclude they were significant. Even accounting for Dr. Rostad's criticisms of the completeness of the other reviews, and especially given the overall record, this seems less likely. To the extent this issue of clinical judgment may boil down to a difference of degrees, both Dr. Harris and Dr. Holmes confirmed that some degree of excessive brain weight and congestion remains consistent with a sudden unexplained death. (Ex. C, p. 7; Ex. E, p. 6.)

In any event, there is also not preponderant evidence that N.C. suffered a terminal seizure as has been proposed by petitioners. In his supplement to the autopsy report, Dr. Hood stated that N.C. had findings consistent with a terminal seizure, but he cautioned that the same findings "can result from almost any form of terminal hypoxia." (Ex. 24, p. 5.) Dr. Hood stated that "the most I can opine is that if [N.C.] had a terminal seizure his autopsy finding would be just as were present in this case." (*Id.*) This is not significant evidence supportive of petitioners' claim. Dr. Harris had similarly explained that "[t]he neuropathological examination was not sufficient to rule in or out a seizure related incident." (Ex. E, p. 6.) In response, Dr. Rostad stressed that "[f]requently there is scant evidence at autopsy to determine when a seizure has occurred and or that it was the cause of death in an unwitnessed case." (Ex. 19, p. 10.) He relied on the idea that specific features of a terminal seizure "may be absent or nonspecific." (*Id.* at 11.) Without more, the fact that the autopsy findings do not "rule out" a terminal seizure is not tantamount to preponderant evidence that such a seizure occurred.³⁸

Petitioners have suggested the undersigned's prior ruling in *Rodela v. Secretary of Health & Human Services*, No. 17-236V, 2024 WL 2034220 (Fed. Cl. Spec. Mstr. Apr. 5, 2024), supports the proposition that prior petitioners were "found to be entitled to compensation for an unwitnessed seizure in a toddler that resulted in the toddler's death following administration of multiple vaccinations." (ECF No. 96, p. 7.) However, the *Rodela* case is not informative, because it was not an unexplained death case. 2024 WL 20344220, at *28 (explaining that "I have concluded that this is *not* an instance of unexplained death consistent with any of the categorizations of sudden unexplained deaths in either infancy or childhood (i.e., SUDC, SUDEP, SIDS, or SUID).") Rather, the *Rodela* ruling addressed a claim of a Table Injury of encephalitis, which was entitled to a presumption of vaccine causation, and the seizures at issue in that case were attributable to the encephalitis. *Id.* at 24-25, 29. Here, however, the medical examiner has confirmed that "[neuropathological examination found no encephalitis." (Ex. 24, p. 5.) Absent the presence of an encephalitis, and more similar to this case, prior petitioners were unable to reliably hypothesize that an unwitnessed seizure explained how vaccination could result in SUDC. *Anklam*, 2023 WL 8890662, at *38-41.

³⁸ Additionally, to the extent respondent's experts acknowledged that the DTaP vaccine can cause febrile seizures (albeit only 1-2 days post-vaccination), it is also worth noting that there is no evidence that N.C. was suffering a fever around the time of his death. Instead, the contemporaneous accounts are explicit in indicating that he had seemingly recovered from his prior febrile illness days prior to his death. (See, e.g., Ex. 1, ¶ 12; Ex. 2, ¶ 11; Ex. 9, p. 9; Ex. 10, p. 69.)

Moreover, the specific finding in *Rodela* that a terminal seizure had likely occurred was based in significant part on affirmative autopsy findings consistent with seizure activity. 2024 WL 2034220, at *24. Specifically, the pathology evidence in *Rodela* included a finding of activated microglia in the hippocampus that respondent's expert agreed was consistent with seizure activity.³⁹ *Id.* Here, however, Dr. Harris has confirmed that this same finding is absent in this case. (Ex. E, p. 5 (explaining that “[n]o astro- or microgliosis is seen. Gliosis would be expected with multiple prior seizures”).)

If N.C. had experienced a recent witnessed seizure, as petitioners allege, that could potentially be viewed as some circumstantial evidence lending support to a theorized terminal seizure. However, there is not preponderant evidence that N.C. experienced a witnessed seizure on May 7, 2017. Without doubting petitioners' account of what happened on that date, it remains unclear that what N.C. experienced was a seizure. Mr. Catone does not describe any overt signs of a seizure but instead described N.C. as falling asleep while being read to and then being difficult to wake up “for a few seconds.” (Ex. 2, ¶ 10.) However, Mrs. Catone otherwise described N.C. as lethargic that day due to being ill and with a fever. (Ex. 1, ¶ 9.) This could reasonably explain what Mr. Catone described. While both Mr. and Mrs. Catone characterize this event as unusual and alarming in their hindsight recollections (*Id.* ¶ 10; Ex. 2, ¶ 10), this is not reflected in their more contemporaneous accounts. Multiple prior accounts of N.C.'s final days include no mention of this episode. (Ex. 9, p. 9; Ex. 10, p. 69; Ex. 15, p. 3; Ex. 5, pp. 26-30.) In particular, on the day N.C. was found deceased, Mrs. Catone specifically discussed his prior febrile illness of May 7 with his pediatrician without indicating N.C. had experienced any unusual or concerning event. (Ex. 10, p. 69.) While Dr. Rostad posited (and Dr. Akbari agreed) that what Mr. Catone described may have been a febrile seizure, he acknowledged that febrile seizures would more typically be tonic-clonic (involving violent muscle contractions) whereas what Mr. Catone described would represent an atonic seizure.⁴⁰ (Ex. 18, p. 16; Ex. 21, p. 9.)

Although there is no dispute that N.C. had the hippocampal malformation that Dr. Rostad seeks to place at issue, Dr. McCusker has cast significant doubt on any association between such a malformation and a lowered seizure threshold, as explained under *Althen* prong one. Indeed, in the addendum to his autopsy report, Dr. Hood noted that the changes he had observed in N.C.'s hippocampus are “of not yet established significance.” (Ex. 24, p. 6.) But in any event, even if a terminal seizure did

³⁹ But in any event, the finding of a terminal seizure was not dispositive. The ruling concluded that “[e]ven in the absence of a terminal seizure, the evidence of neuroinflammation and edema with intracranial pressure is sufficient to support Dr. Shuman's and Dr. Kinsbourne's conclusion that a fatal encephalitis occurred.” *Rodela*, 2024 WL 2034220, at *25.

⁴⁰ Petitioners also argue that Dr. Hood agreed that N.C. more likely than not experienced an atonic or absence seizure. (ECF No. 96, p. 3.) However, I do not agree with this interpretation of Dr. Hood's supplement to his autopsy report. Dr. Hood simply reported that he had been informed that N.C. may have experienced such an event. (Ex. 24, p. 6.) He stated: “Since this autopsy nearly 6 years ago the case has been reviewed as a possible vaccine related injury and several expert reports have been provided to me as well as neuropathology review of the brain histology slides and the further information that the child may have had an ‘absence’ attack or minor seizure in the presence of his father a few days before his death.” (*Id.*)

occur, Dr. Rostad's discussion of the hippocampal malformation leading to a terminal seizure was firmly grounded in the literature discussing SUDC and SUDEP. Thus, the occurrence of a terminal seizure in N.C.'s case standing alone would not actually disturb the conclusion that N.C.'s death is best categorized as SUDC and, as discussed under *Althen* prong one, petitioners have not established that vaccination can be implicated in SUDC. Thus, for example, Dr. Holmes explained that

[w]hile N.C. was at heightened risk for SUDC because of the family history of febrile seizures, there is no evidence that his death was in any way (and certainly not "more likely than not" related to vaccination . . . There is no support in the literature suggesting DTaP results in increased risk for SUDC.

(Ex. C, p. 6.)

Respondent's experts are also persuasive in opining that N.C.'s presentation prior to his death is not compatible with petitioners' theory. In their reply brief, petitioners attempt to disclaim reliance on a cytokine storm, stating that "[p]etitioners are not alleging a cytokine storm in the sense that it caused N.C. to suffer from critical and overwhelming systemic damage." (ECF No. 96, p. 2.) However, this is not credible. While it is fair to say that petitioners have not relied exclusively on a cytokine storm, Dr. Akbari was nonetheless explicit not only in invoking the concept of a cytokine storm as an explanation for N.C.'s death in particular, but also in describing a cytokine storm as widespread inflammation throughout the body that causes death through damage to organs, especially the lungs and central nervous system. (Ex. 13, pp. 7, 14.) He described cytokine storms as having hallmark features inclusive of redness, swelling, heat, pain, and loss of function. (*Id.* at 7.) In that regard, Dr. McCusker is persuasive in describing the expected signs and symptoms of a cytokine storm as being abrupt and severe. (Ex. A, pp. 6-7.) Prior decisions have likewise noted that those suffering a cytokine storm are "profoundly ill." *Bohn*, 2021 WL 4302367, at *20-22. In his second report, Dr. Akbari did disclaim a cytokine storm as one might see subsequent to infection; however, he still opined that N.C. experienced a two-week course of cytokine stimulation and that as a result "he was not able to overcome the damage to his tissues and organs." (Ex. 20, p. 3.)

Even setting aside the specific concept of a cytokine "storm," Dr. Akbari generally agrees that cytokine reactions cause systemic symptoms such as fever and characterized "redness, swelling, edema, and heat" as "hallmarks of acute inflammation." (Ex. 13, p. 7; Ex. 21, p. 5.) In N.C.'s case, however, he simply had a brief rash and cold-like symptoms following vaccination (Ex. 1, ¶ 8; Ex. 2, ¶ 7), followed, more than a week later, by a brief febrile illness (Ex. 1, ¶¶ 9-12; Ex. 2, ¶¶ 8-11; Ex. 10, p. 69). This cannot be readily accepted as consistent with Dr. Akbari's suggestion of two weeks of immune dysfunction and ultimately fatal organ damage. As Dr. Harris explained, while Dr. Rostad opined that N.C. suffered a fatal brain herniation, N.C.'s presentation when he was placed to bed on May 11 was not consistent with a developing herniation. (Ex. H, p. 2.) And, while Dr. Rostad felt N.C.'s heavy liver and spleen were "a likely sign of a generalized reaction, perhaps to a seizure or

inflammation” (Ex. 19, pp. 2-3), Dr. Harris stressed that neither he, nor Dr. Rostad, nor Dr. Hood, had appreciated any significant inflammation in these organs. (Ex. H, p. 2.) Petitioners cannot reasonably argue it both ways – invoking the concept of a cytokine storm to suggest that a cytokine reaction can be dangerous in rare and severe cases but backing away from that same concept when it comes to the expected signs and symptoms of such a severe reaction. *Accord Bohn*, 2021 WL 4302367, at *20-22.

Finally, petitioners suggest that respondent has not offered any credible alternative explanation for N.C.’s death, stressing, for example, that Dr. McCusker was equivocal in endorsing pneumonia as a contributory factor. (ECF No. 85, p. 41.) However, this observation is of no moment given that petitioners have not met their own prima facie burden of proof. While the elimination of other causes can be probative, it is not enough standing alone. *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149). As explained above, the evidence preponderates in favor of a finding that N.C.’s death is best categorized as SUDC, which does not implicate N.C.’s prior vaccination as causal regardless of whether any alternative contributing factor is identified. As noted at the outset, Dr. Akbari acknowledges that SUDC accounts for 10% of all unexpected deaths in childhood.⁴¹ (Ex. 13, p. 9.)

In light of all of the above, and considering the record as a whole, petitioners have not met their burden of proof under *Althen* prong two.

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

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

⁴¹ Also of note, the Federal Circuit has previously found that this type of concept can qualify in itself as a causal factor unrelated to vaccination. *See, e.g., Doe/11*, 601 F.3d at 1351 (holding that “the special master did not commit legal error in considering evidence of SIDS, an allegedly alternative cause. Nothing in the Vaccine Act prohibits the government from presenting evidence that the petitioner’s injury was due to “factors unrelated” to the vaccine (here, SIDS).”); *Winkler*, 88 F.4th at 963 (quoting *Stone v. Sec’y of Health & Human Servs.*, 676 F.3d 1373, 1379 (Fed. Cir. 2012) for the proposition that “evidence of other possible source of injury can be relevant . . . to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question”).

In their motion, petitioners' disclaim any obligation to show that N.C.'s vaccination could have caused a fatal seizure 17-days post vaccination. (ECF No. 85, p. 53.) Instead, they argue that N.C.'s DTaP vaccination was only the initiating factor in the development of his injury, amplified by his febrile illness and alleged seizure of May 7, 2017. (*Id.*) Thus, they suggest that they can satisfy *Althen* prong three by demonstrating that "significant levels of cytokine can persist for weeks after the DTaP vaccination" and that N.C. was "sustaining a heightened level of cytokines" at the time he suffered his upper respiratory infection," *i.e.* approximately 12 days post-vaccination. (*Id.*) However, this assertion is not preponderantly supported.⁴²

Dr. Akbari opined that in the context of infections, cytokines can remain in the blood in significant quantities for up to 100 days. (Ex. 13, p. 8 (citing Huang et al., *supra*, at Ex. 13, Tab G).) In the case of Covid-19, he asserted that a "delayed autoimmune cytokine storm" may occur. (*Id.* (citing Francesca Coperchini et al., *The Cytokine Storm in COVID-19: An Overview of the Involvement of the Chemokine/Chemokine-Receptor System*, 53 CYTOKINE & GROWTH FACTORS REVS. 25 (Ex. 13, Tab H); Shivraj Nile et al., *COVID-19: Pathogenesis, Cytokine Storm and Therapeutic Potential of Interferons*, 53 CYTOKINE & GROWTH FACTOR REVS. 66 (2020) (Ex. 13, Tab I).) Importantly, however, Dr. McCusker stressed that cytokines do not persist after the antigenic challenge is resolved. (Ex. A, pp. 4-5.) And, whereas infections include ongoing antigenic challenge, vaccination does not. (*Id.*) Thus, the cytokine response to vaccination does not persist in the manner of an infection. (*Id.*) In fact, both parties' experts (Drs. Akbari and McCusker) cited approvingly to a study by Kashiwagi et al. (Ex. 13, p. 12; Ex. A, p. 7; Ex. I, pp. 3-4), which showed experimentally that post-vaccination cytokines develop within six to 24 hours. (Kashiwagi et al., *supra*, at Ex. 13, Tab OO (see also Ex. A, Tab 7 & Ex. I, Tab 1).) Moreover, Dr. McCusker further cited Talaat et al., which showed in human subjects that post-vaccination cytokines peak after 16-24 hours and return to baseline by about 44 hours. (Ex. I, p. 3 (citing Talaat et al., *supra*, at Ex. I, Tab 2).) Although Dr. Akbari contends that the ultimate effects of a cytokine response may manifest over a longer course, he explained, consistent with Dr. McCusker's opinion, that "[c]ytokines are not meant to stay in circulation for a long time." (Ex. 20, p. 2.)

Dr. Akbari's opinion that DTaP booster vaccines produce "many weeks" of "large quantities of cytokines" is based primarily on a study by Lee et al. (Ex. 13, pp. 12-13 (citing Lee et al., *supra*, at Ex. 13, Tab RR).) He opines that the cytokines observed by Lee et al. two weeks post-vaccination are "capable of causing pathologies that may induce sudden death." (*Id.* at 13.) However, this conclusion is entirely unsupported.

⁴² Even if petitioners had preponderantly demonstrated that the 12 days between the time of N.C.'s vaccination and his febrile illness supported a causal inference, respondent also raises a separate argument with respect to Dr. Rostad's reliance on a terminal seizure resulting in fatal cerebral edema and herniation. (ECF No. 92, p. 39.) Dr. Holmes opines that, even if N.C. experienced a seizure prolonged enough to constitute status epilepticus, such a seizure would not result in fatal cerebral edema and herniation within the approximately nine hours between when N.C. was last heard in his crib and when he was found deceased the next morning. (Ex. C, pp. 8-9.) However, because petitioners are unpersuasive with respect to their own framing under *Althen* prong three, it is not necessary to resolve that additional question.

The Lee et al. study was intended to analyze the immunogenicity of acellular pertussis boosters in adults to assess whether the boosters have long-term protective effect. (Lee et al., *supra*, at Ex. 13-RR, p. 1.) Nothing in the study identifies the observed cytokines levels to be pathogenic and, in fact, the study does not even suggest that potential adverse outcomes were tracked. (*See id.*) Indeed, contrary to this assertion by Dr. Akbari, as discussed under *Althen* prong one, the only two studies that petitioners have presented that would potentially (if they had been credited) implicate vaccines as a cause of sudden unexplained death are very clear in asserting that the relevant risk period would be three, or perhaps up to seven, days post-vaccination. (Walker et al., *supra*, at Ex. 20, Tab C; Miller, *supra*, at Ex. 26.) Dr. Akbari himself opined that “cytokine storm after initial immunization reaches to the highest levels in 1-3 days.” (Ex. 13, p. 13.) Similarly, Dr. Holmes explained that elevated risk of fever, as well as any related febrile seizure, persists for only about 1-2 days post-vaccination. (Ex. C, p. 7 (citing Sun et al., *supra*, at Ex. C, Tab 14; Walter et al., *supra*, at Ex. C, Tab 17; Jackson et al., *supra*, at Ex. C, Tab 18; Hirtz et al., *supra*, at Ex. C, Tab 19).)

Nor does the evidence preponderate in favor of a finding that N.C. was symptomatic from the time of his vaccination through the time of his May 7 febrile illness. As discussed in the factual summary above, the evidence indicates that N.C. experienced a short-lived rash and symptoms of a cold within about 24 hours of his vaccination. Dr. Akbari reasonably suggests that this could be consistent with a cytokine response in that cytokines can produce redness and runny nose. (Ex. 13, p. 7.) The fact that there is little to no evidence to indicate that these symptoms persisted is consistent with the experts’ overall discussion regarding the short-lived nature of post-vaccination cytokine responses. There is not preponderant evidence that N.C.’s subsequent febrile illness of May 7, 2017, which the contemporaneous accounts indicate began the weekend before his death and lasted only about 24-hours (Ex. 10, p. 69; Ex. 5, p. 16), was any continuation of his prior symptoms or otherwise attributable to a cytokine reaction to a vaccination occurring 12 days prior.

With regard to *Althen* prong three, special masters should not set “a hard and fast deadline” for onset of an alleged vaccine injury. *Paluck v. Sec’y of Health & Human Servs.*, 786 F.3d 1373, 1384 (Fed. Cir. 2015). Thus, I stress that none of the above per se demonstrates that the timing of the events alleged by petitioners is *impossible*. However, it does indicate that, separate and apart from the shortcomings of their theory under *Althen* prong one, the timing of events leaves petitioners’ explanation *much less likely*. But in any event, even if they were persuasive in asserting the N.C.’s death occurred at a time appropriate to the theory they advanced, petitioners’ failure to substantiate their theory and to demonstrate a logical sequence of cause and effect would still be dispositive. *E.g., Hibbard v. Sec’y of Health & Human Servs.*, 698 F.3d 1355, 1364-65 (Fed. Cir. 2012) (holding the special master did not err in resolving the case pursuant to Prong Two when respondent conceded that petitioner met Prong Three).

In light of all of the above, and considering the record as a whole, petitioners have not met their burden of proof under *Althen* prong three.

VI. Conclusion

As noted at the outset, I offer my sincerest condolences to petitioners. Petitioners' loss is profound, and one can easily see how their grief may be compounded by being repeatedly told that N.C.'s death is unexplainable based on existing medical knowledge. However, for all of the reasons described above, there is not preponderant evidence that N.C.'s death was caused by his vaccination.

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

IT IS SO ORDERED.

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