In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 15-1043V
Filed: June 2, 2020
PUBLISHED

CRYSTAL DOWNING-POWERS and ZACHARY POWERS, on behalf of their deceased minor child, M.D.P.,

Petitioners,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Sudden Infant Death Syndrome (SIDS); Order to Show Cause; Dismissal; Insufficient Proof

Andrew Downing, Esq., Van Cott & Talamante, PLLC, Phoenix, AZ, for petitioners.
Julia Marter Collison, Esq., U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On September 17, 2015, petitioners, Crystal Downing-Powers and Zachary Powers, filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012)[2] on behalf of their minor child, M.D.P., alleging that several routine childhood vaccinations, including Haemophilus influenzae type B ("Hib"), pneumococcal conjugate ("PCV"), and Pediarix, a three-in-one of diphtheria-tetanus-acellular pertussis, hepatitis b, and inactivated polio vaccines, administered on October 7, 2013, caused or significantly contributed to his death, categorized as a case of Sudden Infant Death Syndrome ("SIDS"). (ECF No. 1; Ex. 13.) For the reasons set forth below I find that petitioners are not entitled to compensation.

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

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; 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. 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, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

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-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, 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 v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

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 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e*., evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). 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. Section 13(a)(1).

In what has become the predominant framing of this burden of proof, the Court of Appeals for the Federal Circuit described the "causation-in-fact" standard in *Althen v. Secretary of Health & Human Services*, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (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. If Althen satisfies this burden, she is "entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine."

Althen, 418 F.3d at 1278 (citations omitted). 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. The court also indicated that, in finding causation, a Program fact-finder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." *Id*. at 1280.

In their petition, petitioners characterized M.D.P.'s alleged injury as "severe adverse reaction and death from vaccinations." (ECF No. 1, p. 1.) Subsequently, petitioners filed an expert report more specifically characterizing M.D.P.'s death as Sudden Infant Death Syndrome or "SIDS." (Ex. 13.) Since SIDS is not listed on the Vaccine Injury Table, petitioners must satisfy the above-described *Althen* test for establishing causation-in-fact.

II. Prior SIDS Cases in This Program and Guidance from the Federal Circuit

SIDS is not an injury or disease in itself. (Ex. 13, p. 5.) Rather, it is a way of classifying deaths of otherwise undetermined cause. (Ex. 13, p. 5; Ex. A, p. 3.) Generally, all infant deaths that are sudden and unexpected are termed as such – "Sudden Unexplained Infant Death" or "SUID." The term SIDS is further applied to those SUID cases that remain unexplained following an autopsy, investigation, and clinical history review. (Hannah C. Kinney & Bradley T. Thach, *The Sudden Infant Death Syndrome*, 361 N. ENGLAND J. MED. 795, 797 (2009) (Ex. A, Tab 1, p. 2).) The first standard definitions of SIDS was advanced by the National Institute of Health in 1969. Under that definition SIDS represents the "sudden death of an infant or young child, which is unexpected by history, and in which a thorough post mortem examination fails to demonstrate an adequate cause of death." (Kinney & Thach, *supra*, at Ex. A, Tab 1, p. 1.) As of the mid-to-late 2000s, SIDS has an incidence rate in the United

States of 0.57 out of 1,000. (*Id*.) About 80% of SUID cases are classified as SIDS. (*Id*. at Ex. A, Tab 1, p. 2.)

More specifically, experts for both parties in this case have pointed to research by Dr. Hannah Kinney which revealed that the serotoninergic network in infants suffering SIDS is frequently defective. (Ex. 13, pp. 5-6; Ex. A, pp. 3-4.) The serotoninergic network is a network by which arcuate nuclei use serotonin (5-hyrdoxytryptamine or "5-HT") as a neurotransmitter in the regulation of respiratory effort, including recovering from apnea and hypercarbia. (Ex. 13, pp. 5-6.) Thus, if the infant's serotoninergic network is underdeveloped or defective and the infant experiences a normal episode of apnea or an event which causes hypercarbia during sleep, the defective network cannot trigger the neurons which stimulate arousal and increased breathing effort for recovery from that apnea or hypercarbia, and the infant dies. (*Id*. at 6.) Severe deficits in the number of 5-HT receptors exist in 70-90% of SIDS cases. (*Id*. at 5.)

This discovery helped give rise to the "Triple Risk Theory" (also referred to herein as the "Triple Risk Model"). (*Id*.) First proposed in 1994, the Triple Risk Model identifies three factors that combine to result in SIDS: (1) an underlying vulnerability; (2) a critical developmental period; and (3) an exogenous[3] stressor. (Kinney & Thach, *supra*, at Ex. A, Tab 1, p. 2.) This theory hypothesizes that in an infant with a serotoninergic network vulnerability as described above, in the appropriate critical developmental period, an acute "stressor" or combination of stressors can cause apnea or hypercarbia leading to death, because of the failure of the network to stimulate the normal arousal inducing response. (Ex. 13, p. 6.)

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-labeled death. Generally, such cases have been dismissed in the first instance by the presiding special masters for insufficient evidence that any vaccine played a causal role in the death. *See*, *e.g.*, *Olasvicky v. Sec'y of Health & Human Servs*., No. 17-1806V, 2019 WL 2881009 (Fed. Cl. Spec. Mstr. June 4, 2019); *Nunez v. Sec'y of Health & Human Servs*., No. 14-863V, 2019 WL 2462667 (Fed. Cl. Spec. Mstr. Mar. 29, 2019), *review denied* 144 Fed. Cl. 540 (2019); *Frady v. Sec'y of Health & Human Servs*., No. 16-148V, 2017 WL 5379391 (Fed. Cl. Spec. Mstr. Sept. 20, 2017); *Pelton v. Sec'y of Health & Human Servs.*, No. 14-674V, 2017 WL 1101767 (Fed. Cl. Spec. Mstr. Feb. 27, 2017); *Jewell v. Sec'y of Health & Human Servs.*, No. 11-138V, 2016 WL 5404165 (Fed. Cl. Spec. Mstr. Aug. 29, 2016); *Copenhaver v. Sec'y of Health & Human Servs.*, No. 13-1002V, 2016 WL 3456436 (Fed. Cl. Spec. Mstr. May 31, 2016), *review denied*, 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); *Cozart v. Sec'y of Health & Human Servs.*, No. 00-590V, 2015 WL 6746616 (Fed. Cl. Spec. Mstr. Oct. 15, 2015), *review denied*, 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), *review denied* 123 Fed. Cl. 564 (2015);

[3] "Exogenous" refers to something "developed or originating outside the organism." (*Dorland's Illustrated Medical Dictionary*, p. 652 (33rd ed., 2019.)

Sanchez v. Sec'y of Health & Human Servs., No. 11-651V, 2013 WL 4476750 (Fed. Cl. Spec. Mstr. Jul. 26, 2013); *Bigbee v. Sec'y of Health & Human Servs.*, No. 06-663V, 2012 WL 1237759 (Fed. Cl. Spec. Mstr. Mar. 22, 2012); *Nordwall v. Sec'y of Health & Human Servs.*, No. 05-123V, 2008 WL 857661 (Fed. Cl. Spec. Mstr. Feb. 19, 2008), *review denied* 83 Fed. Cl. 477 (2008); *Doe/11 v. Sec'y of Health & Human Servs.*, 2008 WL 649065 (Fed. Cl. Spec. Mstr. Jan. 31, 2008); *Heller v. Sec'y of Health & Human Servs*., No. 96-797V, 1998 WL 408612 (Fed. Cl. Spec. Mstr. June 22, 1998).

In some instances, the parties have litigated whether SIDS presents an alternative cause of 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)."). Significant to this case, 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 the framework of the Triple Risk Model of SIDS. In these prior decisions special masters 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; *Copenhaver,* 2016 WL 3456436; *Lord,* 2016 WL 806818; *Cozart,* 2015 WL 6746616.

In *Boatmon v. Secretary of Health & Human Services*, however, the special master concluded that a child's sudden, unexplained death was consistent with SIDS and that his death was caused, in part, by his vaccinations. No. 13-611V, 2017 WL 3432329 (Fed. Cl. Spec. Mstr. July 10, 2017). The *Boatmon* petitioners presented a theory through their expert, Dr. Douglas Miller (who also opines in this case), that vaccines produce a cytokine response that can act as an exogenous stressor on the 5-HT network to cause SIDS within the Triple Risk Model. The special master explained:

> I have concluded that petitioners have presented sufficient evidence and testimony to entitle them to compensation in the Vaccine Program. I have not concluded that vaccines present a substantial risk of SIDS. In fact, the evidence is to the contrary. The vast majority of vaccine recipients do not succumb to SIDS. Under the multi-factorial analysis of the Triple Risk Model, it is theorized that the ultimate fatal event may occur when multiple factors converge during this vulnerable period to cause death when one stressor acting alone may not have.

Id. at *42.

At the time the special master's decision in *Boatmon* was issued, petitioners in this case submitted a Notice of Additional Authority attaching the *Boatmon* decision. Petitioners suggested that the special master in that case had "fully analyzed the current state of the medical literature, as well as whether vaccination, under the right

circumstances, can serve as an extrinsic risk factor leading to a death characterized as SIDS." (ECF No. 45.) However, respondent successfully moved for review of the special master's decision in *Boatmon*. On July 3, 2018, the Court of Federal Claims reversed and vacated the special master's decision and dismissed the petition. *Boatmon v. Sec'y of Health & Human Servs.*, 138 Fed. Cl. 566 (2018).

Citing *Jewell*, *Copenhaver*, *Lord*, and *Cozart*, *supra*, the Court was critical of the special master for disregarding prior decisions by other special masters that uniformly found the same or similar theories unpersuasive. *Boatmon*, 138 Fed. Cl. at 571. More significantly, however, the Court found that the special master had erred by impermissibly lowering petitioners' burden of proof. *Id*. at 571-72. Specifically, the Court explained:

> This departure from the conclusions of other Special Masters can only be explained by improper application of the standard of proof required in vaccine cases. While scientific certainty is not required to establish causation under the Vaccine Act, the theory must be supported by a "sound and reliable" medical or scientific explanation. *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). In *Moberly*, 592 F.3d at 1322, the Federal Circuit noted that a Petitioner must provide a "reputable medical or scientific explanation" for causation, and that this standard requires more than mere "plausibility," which is "not the statutory standard." In the case at bar, the theory embraced by the Special Master has not been accepted by any other experts in the field of SIDS research.

Id.

The *Boatmon* petitioners appealed to the Court of Appeals for the Federal Circuit and on November 7, 2019, the Federal Circuit affirmed the Court of Federal Claims. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351 (Fed. Cir. 2019). Although the Federal Circuit noted that special masters are not obligated to rely on a *Daubert* analysis and are not obligated to distinguish the decisions of other special masters, the Court agreed with the Court of Federal Claims that the special master had impermissibly lowered petitioners' burden of proof. *Id*. at 1358-59.

Pertinent to this case, the Federal Circuit explained that, while petitioners are not required to demonstrate causation with scientific certainty, to satisfy *Althen* prong one petitioners must present expert opinion that provides a scientific explanation that is "sound and reliable." *Id*. The Federal Circuit stressed that theories that are merely "plausible" do not meet that standard. *Id.* at 1359. With regard to the Triple Risk Model of SIDS, the Federal Circuit held that petitioners' expert's theory that vaccination could act as the exogenous stressor pursuant to that theory was not sound and reliable. *Id*. at 1361.

First, the Federal Circuit explained that "outside of Vaccine Act litigation, vaccinations have not been identified as an exogenous stressor for SIDS." *Id*. The

Federal Circuit noted that petitioners' "extension of the Triple Risk Model to include vaccination-induced cytokine activity in the list of exogenous stressors" was based on "nothing more than the assertion of [petitioners' expert] Dr. Miller." *Id*. at 1360-61. Accordingly, the Federal Circuit concluded that "[t]he Special Master erred in adopting an unsound and unreliable theory that constitutes a significant extension of the Triple Risk Model in the absence of any indicia of reliability." *Id*. at 1362.

Second, the Federal Circuit determined that petitioners had "failed to show by a preponderance of the evidence that vaccinations cause cytokines to provoke an abnormal brainstem serotonin response or otherwise cause or contribute to a SIDS death." *Id*. The Federal Circuit discussed three studies (Frøen, Stoltenberg, and Brambilla) presented by petitioners as supportive of Dr. Miller's theory; however, the Federal Circuit concluded that "these studies do not provide support for Dr. Miller's proposed theory because they do not show that that cytokine activity is capable of impacting the brain's 5–HT system in the manner Dr. Miller claims or that vaccinations are capable of producing such cytokine activity in the brain." *Id*. at 1360-62.

With regard to the remaining *Althen* prongs, the Federal Circuit's guidance in *Boatmon* is limited to a discussion, relative to *Althen* prong two, of a brain stem abnormality alleged in that case to constitute an underlying vulnerability consistent with the Triple Risk Model. 941 F.3d at 1362-63. In that case, there was no physical evidence establishing the presence of a brain stem abnormality and the conclusion that the *Boatmon* child was in a vulnerable state was based in large part on statistical evidence that such an abnormality is present in 50-70% of SIDS cases. *Id*. The Federal Circuit found that conclusion to be error. *Id*.

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. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998).

In contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), ***aff'd*** 104 F. 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). Accordingly, 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.*, --- Fed. Appx. ----, 2020 WL 1685554 (Fed. Cir. April 7, 2020, at *7 (citing back to a prior Federal Circuit holding in *Paluck v. Secretary of Health & Human Services*, and noting with regard to the two Leigh Syndrome cases 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*.").

Petitioners suggest that the Federal Circuit decision in *Boatmon* "does not serve as a wholesale rejection of SIDS cases in the Vaccine Program" and is distinguishable from the instant case. (ECF No. 81, pp. 1-2.) Respondent characterizes the Federal Circuit decision in *Boatmon* as having a "binding nature" and argues this case "is not sufficiently distinguishable from *Boatmon* to avoid dismissal." (ECF No. 82, pp. 1-2.)

III. Procedural History

Petitioners filed the instant petition along with a statement from Crystal Downing-Powers on September 17, 2015. (ECF No. 1.) On September 21, 2015, the case was assigned to Special Master Laura Millman, who has since retired. (ECF No. 4.)

A status conference was held on December 17, 2015. Special Master Millman discussed the records filed in the case and noted an inconsistency in the record concerning whether M.D.P was fussy after receiving his routine vaccinations. (ECF No. 12.) She ordered petitioners to file the VAERS report and the coroner's records. (*Id*.) On January 14th, 2016, petitioners filed a status report addressing the inconsistency in petitioners' records. (ECF No. 14.) According to petitioners, the records were not inconsistent, and both parents reported that M.D.P had been fussy, but went to sleep without issue. (*Id*.) A subsequent status conference was held on February 11, 2016. (ECF No. 17.) Petitioners requested authority to issue a subpoena to the San Bernardino's coroner's office which was granted that same day. (ECF Nos. 17-18.)

On September 26, 2016, petitioners filed an expert report from Dr. Douglas Miller, the same expert who supported petitioners' claim in the *Boatmon* case. (ECF No. 28; Ex. 13.) A further status conference was held on November 8, 2016. (ECF No. 32.) Special Master Millman expressed her concerns regarding Dr. Miller's expert report, and requested a supplemental report from him addressing her concerns. (*Id*.) Petitioners were ordered to file an additional police report from Deputy Rivas, who was at the hospital on the day of M.D.P.'s death. (*Id*.)

On January 4, 2017, petitioners filed additional records from the San Bernardino Sherriff's Department's investigation of M.D.P.'s death. (ECF No. 38.) Special Master Millman noted that some of the details revealed in the investigation may have conflicted with petitioners' expert's report and ordered the expert to file a supplemental expert report responding to the possible conflicts. (ECF No. 39.) Petitioners filed the requested expert report on January 26, 2017. (ECF No. 40.) A status conference was held on March 21, 2017. Special Master Millman discussed the first supplemental expert report and requested a second supplemental expert report, which was filed on March 30th, 2017. (ECF Nos. 41-42.) On April 20, 2017, an additional status conference was held. (ECF No. 43.) Special Master Millman discussed the second supplemental expert report and ordered petitioner to file an additional expert report from a neuroimmunologist, which was filed on July 20, 2017. (ECF Nos. 43-44.)

On August 8, 2017, a status conference was held and respondent was ordered to file his Rule 4(c) report and expert reports. (ECF No. 49.) Respondent filed his report recommending that the claim be dismissed, along with expert reports from Drs. McCusker[4] and Harris on November 8, 2017. (ECF Nos. 51-52.) The next month, a status conference was held, and the parties discussed their intent to have a two-day entitlement hearing. (ECF No. 53.) Special Master Millman indicated that the hearing date would be scheduled when the case was reassigned following her retirement. (ECF No. 64.)

On June 5, 2019, this case was reassigned to my docket. (ECF No. 70.) Later that month, I ordered petitioners to file a joint status report on behalf of the parties confirming whether the case remained ripe for a two-day entitlement hearing. (ECF No. 72.) On July 15, 2019, petitioners filed the status report confirming that the case was ripe for hearing, and on July 25, 2019, I issued a prehearing order scheduling a two-day entitlement hearing to commence on January 23, 2020. (ECF No. 73-74.)

Subsequently, on November 7, 2019, the Federal Circuit issued the above-discussed decision affirming the Court of Federal Claims reversal of the special master's decision in *Boatmon*. I ordered petitioners to file a joint status report indicating how the parties wished to proceed in light of the decision in *Boatmon*. The parties confirmed jointly that a hearing would not be necessary in this case and requested that the Prehearing Order be vacated and all prehearing deadlines terminated. (ECF Nos. 79-80.) They differed, however, on how to resolve this case. Petitioners requested a briefing schedule and adjudication on the written record. (ECF No. 79.) Respondent's position was that the petitioners had lost reasonable basis to proceed with their claim following the *Boatmon* decision and that the case should be dismissed without further expenditure of Program resources. (*Id*.)

In response to the joint status report, I issued an order for petitioners to show cause why the case should not be dismissed for insufficient proof in light of the *Boatmon* decision and cancelled the previously scheduled entitlement hearing.[5] Petitioners filed their response to the order to show cause on February 3, 2020. (ECF No. 81.) They argued that the current case does not possess the same evidentiary shortcomings present in *Boatmon*. (ECF No. 81, p. 5.) In response, on February 26, 2020, respondent argued that petitioner's case is not sufficiently distinguishable from *Boatmon* to avoid dismissal. (ECF No. 82.)

[4] Dr. McCusker's report was stricken; however, an amended/corrected expert report from Dr. McCusker was filed later. (ECF No. 55.)

[5] Although the hearing in this case was cancelled with the agreement of the parties (ECF No. 79), I note that I have also separately determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve this case without a hearing. *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Secretary of Health & Human Services*, 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record.").

This case is now ripe for a ruling on the record.

IV. Factual History

a. Medical Records

M.D.P. was born to Crystal Downing-Powers at 37 weeks on April 26, 2013, weighing 6 pounds, 13 ounces, and was discharged from Valley View Medical Center the next day. (Ex. 5, pp. 5-6.) Ms. Downing-Powers' pregnancy was largely unremarkable. M.D.P. was her fourth child. (*See generally* Ex. 9.) On May 1, 2013, M.D.P. saw Dr. Naila Tariq for a newborn exam. (Ex. 4, pp. 12-13.) The visit was unremarkable and two-month follow-up was recommended. (*Id*.)

On May 8, 2013, M.D.P. was seen at Children's Medical Center presenting with upper respiratory symptoms. (Ex. 4, p. 10.) He was diagnosed with an upper respiratory tract infection and gastroenteritis. (*Id*. at 11.) On May 10, 2013, he went to Children's Medical Center for an upper respiratory infection follow-up. (Ex. 4, p. 8.)

On July 2, 2013, M.D.P. saw Dr. Tariq for a two-month well baby examination. (Ex. 4, p. 6.) The parents did not report any active major problems, but noted that at the time M.D.P was not breast feeding, and had been taking Enfamil Gentle-Ease. (*Id*.)

On August 2, 2013, M.D.P. presented to Children's Medical Center with two weeks of rash. (Ex. 4, p. 4.) He was diagnosed with dermatitis and a follow-up in one to two weeks was recommended. (Ex. 4, p. 5.)

On October 7, 2013, M.D.P. visited Tri-State Community Healthcare for a comprehensive physical, where he received his second dose of routine vaccinations. (Ex. 2, pp. 1-3.) Two days later, he was brought to the Colorado River Medical Center emergency room unresponsive. (Ex. 7, p. 2.) The clinical impression was "Sudden Death." (*Id*. at 3.) Mr. Powers noted that they put M.D.P. down to sleep at 7:00pm and he slept through the night. (*Id*. at 8.) He checked on M.D.P. at 7:00am and the infant was fine. (*Id*.) However, M.D.P. was later found cold and blue. (*Id*.) M.D.P. died on October 9, 2013. (Ex. 6, p. 1.)

The County of San Bernardino Sherriff's Department conducted a Death Investigation after M.D.P.'s death. (Ex. 42.) Officer Timothy Preston noted that M.D.P. slept in a "pack-n-play" playpen with one pillow, two blankets, and an 8-ounce bottle of formula milk. (Ex. 42, p. 7.) Mr. Powers was asked to create a reenactment of the incident using a doll, and he placed the doll on its back, covered the doll with a blanket, and placed a bottle near the doll's mouth. (*Id*. at 8.) The case status was listed as "unknown cause of death" and the case was forwarded to the Coroner's Division. (*Id*. at 5, 9.)

Dr. Chanikarn Changsri conducted an autopsy on October 10, 2013. (Ex. 11, pp. 10-13.) Dr. Changsri noted faint lividity on the chest and prominent lividity on the

posterior of the body, but no fatal trauma or abnormalities. (*Id*. at 13.) Dr. Changsri noted "petechial hemorrhages in the epicardium of the heart, visceral pleura of the lungs, and thymus." (*Id*.) The cause and manner of death were undetermined. (*Id*.)

b. Affidavit

Along with their petition, on September 17, 2015, petitioners filed an affidavit to support their claim. (Ex. 1, p. 1.) The affidavit was written by M.D.P.'s mother, Crystal Downing-Powers. She affirmed that M.D.P. was born on April 26, 2013 and was 5 ½ months of age at the time he allegedly suffered an adverse reaction to the vaccinations received on October 7, 2013. (*Id*.)

On October 7, 2013, M.D.P. went for his four-month check-up and petitioners were advised that he was late on his vaccination schedule. (*Id*.) At that visit, M.D.P. received multiple vaccinations. (*Id*.) His mother reported that after his vaccinations, M.D.P. was "fussy and he was crying" which she believed was a normal reaction to vaccinations, and since he did not have a fever, she did not give him any medication. (*Id*.) Ms. Downing-Powers further noted that he was lethargic, and not acting like himself, so she continued to check on him every hour. (*Id*. at 1-2.)

According to Ms. Downing-Powers, M.D.P. continued to be irritable and refused to eat the next day. (*Id*. at 2.) On October 9, 2013, when she went to check on M.D.P. he was not breathing. She and M.D.P.'s father attempted CPR but were unable to revive him, and when 911 took too long to respond, they took M.D.P. to the hospital two blocks away where hospital staff also attempted CPR. (*Id*.) M.D.P.'s body was transferred to the coroner's office in San Bernardino, and after a month and a half, the parents were advised that M.D.P.'s body was not in a condition they would want to remember him in, and he was cremated. (*Id*.)

V. Expert Reports

a. Petitioners' Experts

i. Douglas Miller, M.D., Ph.D.

Dr. Miller is a neuropathologist, who earned both his medical degree and his Ph.D. in physiology and biophysics at the University of Miami. (Ex. 13, pp. 1-2.) Dr. Miller has served as a neuropathy consultant for medical examiners for much of his medical career, including serving as a neuropathy consultant for the Office of the Chief Medical Examiner of the City of New York. (*Id*. at 2.) He is also board certified in anatomic pathology and neuropathology. Currently, Dr. Miller serves as a clinical professor of pathology at the University of Missouri School of Medicine and has served in that capacity for thirteen years. (*Id*.)

Upon review of M.D.P.'s medical history, the coroner's report, and paraffin block cuts of M.D.P.'s medulla, Dr. Miller opined that M.D.P.'s case "fits the standard

definition of SIDS" and, in fact, "is a case of SIDS." (Ex. 13, p. 4.) Consistent with the Triple Risk Model, Dr. Miller explained that SIDS has been found to be related to an underlying central nervous system abnormality, causing a number of factors which, when combined, cause sudden death during sleep. (*Id*. at 5.) This combination of factors is believed to occur only in infants 2-6 months of age with a particular abnormality of the neuronal systems in the medulla. (*Id*. at 5, 8.) These neuronal systems are responsible for mediating "arousal from apnea (which commonly occurs in sleep and from which normal infants readily recover), from hypercarbia (an excess of carbon dioxide in the blood) by gasping respirations and arousal, and indeed the sensory mechanism for detecting carbon dioxide levels above normal in the blood." (*Id*. at 5.)

Dr. Miller listed a number of risk factors and potential stressors implicated in SIDS including premature births, mothers who smoked, and infants who sleep prone rather than supine. (*Id*. at 6.) He further opined that M.D.P.'s death was caused in part by his vaccinations received on October 7, 2013, which acted as such a stressor and which included vaccinations for "Hib, Hepatitis A, DTP, Hepatitis B, IPV, PCV, and Pediarix." (*Id*. at 3.)

Dr. Miller pointed to evidence demonstrating that otherwise innocuous upper respiratory viral infections are associated with SIDS in vulnerable infants to support his opinion that infectious agents, and thus vaccinations, could be acute stressors potentially leading to SIDS. (*Id*. at 7.) He explained that cytokines are chemical messengers which facilitate important interaction between the immune system and the central nervous system to invoke the appropriate response to a given antigen. (*Id*.) For example, cytokines might signal the hypothalamus to elevate body fever. (*Id.*) According to Dr. Miller, these cytokines can cross the blood-brain barrier, and are also produced by vaccinations, which present the immune system with antigens to stimulate adaptive immunity. (*Id*.)

According to Dr. Miller, cytokines, specifically cytokines IL-1β and IL-2, have been "shown to suppress the activity of the medullary serotoninergic network when administered to animals, the same network which is responsible for regulating respiration, producing arousal from apnea, and increasing respiration react to hypercarbia." (*Id*. at 8.) Thus, Dr. Miller concludes that in an infant with a medulla which is not properly developed and with a defective serotoninergic network, an upper respiratory viral infection or the receipt of multiple vaccines would produce an increase of cytokines which may suppress the activity of the normal medullary system and cause SIDS. (*Id*.) This was especially important, Dr. Miller noted, because of the reports that M.D.P. was "fussy" and feeling ill after the receipt of his vaccinations.[6] (*Id*.)

[6] On January 25, 2017, Dr. Miller filed a supplemental report responding to specific questions posed by Special Master Millman. (ECF No. 40; Ex. 45.) First, he explained that the current science is insufficient to explain why M.D.P.'s first vaccinations did not cause SIDS. Then, he noted that M.D.P.'s seemingly normal state the morning of his death does not contradict the possibility of SIDS. (Ex. 45.) On March 30, 2017, Dr. Miller filed a second supplemental report explaining his conclusions in light of new information that M.D.P.'s parents reported to the Sherriff's department that M.D.P. had no immediate reaction to his vaccines and did not mention that he was fussy, lethargic, or lacked an appetite. (ECF No. 42; Ex 46.)

ii. Dr. Lawrence Steinman, M.D.

On July 20, 2017, petitioners additionally filed an expert report from Dr. Lawrence Steinman. (Ex. 47.) Dr. Steinman is a board-certified neurologist. (*Id*. at 2.) He previously served as professor of the Departments of Neurology and Neurological Sciences, Pediatrics and Genetics at Stanford University, and currently serves as G.A. Zimmermann Chair Professor of Neurological Sciences, Neurology, and Pediatrics. (*Id*.) In 2004, he won the John M. Dystel Prize for Outstanding Contributions in Multiple Sclerosis Research, National MS Society and the American Academy of Neurology. (*Id*.) In 2009, he was elected to the Institute of Medicine, renamed in 2015 as the National Academy of Medicine. (*Id*.) Dr. Steinman has 38 patents and is associate editor of the journal Neurobiology of Disease. (*Id*. at 3.)

Dr Steinman agreed in full with Dr. Miller's opinion and further opined that the ADP-ribosylation triggered by pertussis toxin in vaccines, the ability of Alum, an adjuvant used in the DTaP vaccine, to induce cytokine IL-1β secretion, and the ability of the pertussis toxin to cross the blood brain barrier, all helped to explain how vaccination contributed to M.D.P.'s death. (*Id*. at 5-6.) He further explained that ADP-ribosylation has been associated with seizures and neuronal death. (*Id*. at 5.) He concluded by opining "[t]o a high degree of medical certainty that the contents of the vaccines received on March 7, 2013 triggered the tragic outcome with SIDS."[7] (*Id*. at 7.)

b. Respondent's Experts

i. Christine McCusker, MSc, M.D., FRCP

Dr. McCusker holds a Master of Science degree in Molecular Biology and an M.D. (Ex. A, p. 1.) She is board certified in both pediatrics and allergy and clinical immunology. She currently serves as an associate professor of Pediatrics at McGill University and as Division Director of Pediatric Allergy, Immunology, and Dermatology at the Montreal Children's Hospital. (*Id*.)

Dr. McCusker did not challenge the validity of the Triple Risk Model of SIDS; however, she opined that there is no evidence connecting vaccination to SIDs and that vaccination is not currently considered an exogenous stressor by recognized experts in the study of SIDS. She also noted that given his family history of SIDS, M.D.P. could have had an underlying cardiac arrhythmia which led to his death. (Ex. A, p. 15.)

Dr. McCusker also opined that, although vaccines do produce a cytokine response, they do so in the periphery and at very low levels. (*Id*. at 10.) She disputed that the studies relied upon by Dr. Miller are reflective of what happens *in vivo* in

Dr. Miller maintained that the affidavit and the medical records were consistent, and that regardless, the immune system produces and circulates cytokines which interacted with his central nervous system in suppressing serotoninergic pathways in his medulla which are responsible for recovery from apnea or hypercarbia during sleep. (Ex 46, p. 2.)

[7] In fact, the vaccines at issue were administered in October of 2013, not March.

response to vaccination or that there is sufficient evidence that they can affect respiration or be pathologically implicated in SIDS. (*Id*. at 13-14.) Moreover, she explained that cytokines have a role in normal brain function and are involved in basic brain physiology. (*Id*. at 10-11; 14.) Accordingly, the mere finding of cytokines in the brain is not indicative of a vaccine-reaction. (*Id*. at 14.) Specifically, cytokines present in the 5-HT system among SIDS victims may be a result of the acute brain injury associated with their terminal event rather than the result of vaccine crossing the blood brain barrier. (*Id*.)

ii. Brent Harris, M.D., Ph.D.

Dr. Harris is a board-certified anatomic neuropathologist and the neuropathology consultant for a number of organizations, including the Washington, DC office of the Chief Medical Examiner, Howard University Hospital, and MedStar Hospital System. (Ex. C, p. 1.) Dr. Harris indicated that he largely agrees with the autopsy findings. He also agreed that "[t]he autopsy did show a possible structural change in the brainstem." (*Id*. at 5.) However, he further opined that the most appropriate designation for M.D.P.'s cause of death would be Sudden Unexplained Infant Death. (*Id.* at 6.) He noted that M.D.P.'s prone sleeping position could have caused death by asphyxiation. (*Id*. at 5.) He acknowledged that the Triple Risk Model is well-reasoned and possible but noted that it has not yet been proven and does not suggest a link with vaccinations. (*Id*.)

VI. Discussion

As explained above, petitioners' burden is to demonstrate by preponderant evidence each of the three *Althen* prongs for determining causation-in-fact (i.e. a medical theory, a logical sequence of cause and effect, and a proximate temporal relationship). *Althen*, 418 F.3d at 1278. In this case, the first *Althen* prong is dispositive. Petitioners' burden under the first *Althen* prong is to provide, by preponderant evidence, "a medical theory causally connecting the vaccination and the injury." *Id*. at 1278. Such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 549. Moreover, scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu v. Sec'y of Health & Human Servs*., 569 F.3d 1367, 1380 (Fed. Cir. 2009.) However, to satisfy this prong, petitioners' theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548; *Boatmon*, 941 F.3d at 1359. In this case, I find that Dr. Miller's and Dr. Steinman's opinions, considered separately or in combination, fail to set forth a sound and reliable medical theory that could causally link M.D.P.'s death to his vaccinations.

Petitioners argue that "[t]he aspect of *Boatmon* that was determined to be 'unreliable' by the Federal Circuit was how vaccination could serve as the extrinsic risk factor for SIDS. Petitioner has already produced evidence in this case that bridges that gap." (ECF No. 81, p. 1.) Petitioners further contend that "[t]he Federal Circuit gave several reasons why Petitioner failed to satisfy his burden of proof –none of which apply

here." (*Id*. at 2.) I disagree. Although petitioners are correct that the record of this case is not identical to the prior *Boatmon* case, several issues related to *Althen* prong one that were discussed by the Federal Circuit in *Boatmon* remain relevant based on the instant record. Moreover, contrary to their assertions, petitioners in this case have not remedied the short-comings that caused the Federal Circuit to reject the *Boatmon* theory of causation. That is, upon my own review of the complete record of this case, I am not persuaded that Dr. Miller's extension of the Triple Risk Model to include vaccines as an exogenous stressor is sound and reliable or that Dr. Steinman has otherwise provided a sound and reliable theory supporting causation.

a. Triple Risk Model

First, as described above, the Federal Circuit explained in *Boatmon* that "outside of Vaccine Act litigation, vaccinations have not been identified as an exogenous stressor for SIDS." *Boatmon*, 941 F.3d at 1360. The Federal Circuit noted that petitioners' "extension of the Triple Risk Model to include vaccination-induced cytokine activity in the list of exogenous stressors" was based on "nothing more than the assertion of [petitioner's expert] Dr. Miller." *Id*. at 1361-62. I have reviewed the complete record of this case, including each of Dr. Miller's reports and each study cited. Although the concept of the Triple Risk Model itself is not seriously debated in this case[8] and has resulted in a significant body of literature, none of the cited literature extends the Triple Risk Model of SIDS to vaccination.

Indeed, respondent's expert, Dr. McCusker, persuasively explained that the list of recognized extrinsic factors:

> Include[s] prone and side-sleeping positions, bedclothes, sleep on sofas, high ambient temperature in the sleeping environment, soft bedding, bed sharing and mild infections, including colds. Petitioners' expert, Dr. Miller[,] opines that vaccination should be on the list of extrinsic factors, yet large epidemiological studies do not reveal increased frequency of vaccination in SIDS[9] and recent published work from recognized experts in the field, including Dr. Kinney, do not include vaccination as an extrinsic factor.

[8] Respondent's neuropathology expert, Dr. Harris, did stress that the Triple Risk Model remains an unproven hypothesis; however he also agreed that it is well-reasoned and possible. (Ex. C, p. 5.) Respondent's immunology expert, Dr. McCusker, accepted the Triple Risk Model without challenge. (Ex. A, pp. 3-6.)

[9] Petitioners did submit a number of publications (attached both to Dr. Miller's and Dr. Steinman's reports) addressing whether epidemiological evidence of an association exists; however, that evidence does not preponderate in favor of such a relationship. Although petitioners cannot be required to come forward with epidemiological evidence, the special master may consider such evidence when filed. *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325–26 (Fed. Cir. 2006). In 1991 the Institute of Medicine ("IOM") examined whether there is any relationship between SIDS and the pertussis vaccine. (Christopher P. Howson, Cynthia J. Howe & Harvey V. Fineberg, eds., *Adverse Effects of Pertussis and Rubella Vaccines* (1991) (Ex. 27).) The resulting report noted that "[a]ll controlled studies that have compared immunized versus nonimmunized children have found either no association or a decreased risk of SIDS among immunized children." (*Id*. at 20 (internal citations

(Ex. A, p. 5 (citing Kinney & Thach, *supra*, at Ex. A, Tab 1, R.L. Haynes et al., *High Serum Serotonin in Sudden Infant Death Syndrome*, 114 PROC. NAT'L ACAD. SCIENCE USA 7695 (2017) (Ex. A, Tab 2); Richard D. Goldstein, Hannah C. Kinney & Marian Willinger, *Sudden Unexpected Death in Fetal Life Through Early Childhood*, 137 PEDIATRICS e20154661 (2016) (Ex. A, Tab 11)).) Respondent's neuropathology expert, Dr. Harris, likewise agreed that vaccination has not been identified as an exogenous stressor under the Triple Risk Model. (Ex. C, pp. 5-6.) Although petitioners have also submitted an expert report by neuro-immunologist Lawrence Steinman, Dr. Steinman did not invoke or separately discuss the Triple Risk Model of SIDS independent of his blanket acceptance of and reliance on Dr. Miller's opinion. (*See* Ex. 47, p. 7 (stating that "I fully agree with Dr. Miller's report.").) Accordingly, petitioners' theory that vaccination can be considered an exogenous stressor within the Triple Risk Model for SIDS remains both novel and based on Dr. Miller's *ipse dixit*.

---

omitted).) The IOM concluded that "[t]he evidence does not indicate a causal relation between DPT vaccine and SIDS. Studies showing a temporal relation between these events are consistent with the expected occurrence of SIDS over the age range in which DPT immunization typically occurs." (*Id*. at 21.) Dr. Miller did cite several more recent case reports originating in Germany related to a suspicion that certain hexavalent vaccines administered in Europe beginning in 2000 might be associated to sudden unexplained deaths. (B. Zinka et al., *Unexplained Cases of Sudden Infant Death Shortly After Hexavalent Vaccination*, 24 VACCINE 5779 (2006) (Ex. 24) (reporting six cases); Giulia Ottaviani, Anna Maria Lavezzi & Luigi Matturri, *Sudden Infant Death Syndrome (SIDS) Shortly After Hexavalent Vaccination: Another Pathology in Suspected SIDS?*, 448 VIRCHOWS ARCH 100 (2006) (Ex. 25) (single case report.) In 2005 von Kries et al. questioned whether unexplained deaths temporally associated with the hexavalent vaccines represented a "signal." (Rüdiger von Kries et al., *Sudden and Unexpected Deaths After the Administration of Hexavalent Vaccines (Diphtheria, Tetanus, Pertussis, Poliomyelitis, Hepatitis B, Haemophilius Influenzae Type B): Is There a Signal?*, 164 EUR J. PEDIATRIC 61 (2005) (Ex. 23).) Overall, they concluded that there is no causal relationship between sudden unexplained death and the vaccines studied; however, one of the vaccines identified as "Vaccine A" saw an unexpected temporal association to unexplained deaths following the second-year booster. This was based on three reported deaths. (*Id*. at 6-8.) They recommended enhanced surveillance. (*Id*. at 8.) A follow-up study was subsequently conducted in Italy, the second largest market for the hexavalent vaccines, over a six-year period (1999-2004), resulting in a study population of 604 unexplained deaths occurring between 31 to 729 days of age. (Giuseppe Traversa et al., *Sudden Unexpected Deaths and Vaccinations During the First Two Years of Life in Italy: A Case Series Study*, 6 PLOS ONE e.16363 (2011) (Ex. 26).) The Italian study did not confirm the "signal" found in the German study. (*Id*.) In 2012, the IOM looked at four further publications reporting SIDS after the administration of vaccines containing diphtheria toxoid, tetanus toxoid, and acellular pertussis antigens alone or in combination. (Kathleen Stratton et al., *Adverse Effects of Vaccines: Evidence and Causality* (2012) (Ex. 59, pp. 610-11).) The IOM concluded that "[t]he publications did not provide evidence beyond temporality" and that "the mechanistic evidence regarding an association between diphtheria toxoid–, tetanus toxoid–, or acellular pertussis–containing vaccine and SIDS is lacking." (*Id*. at 611.) Petitioners also filed the package insert for Pediarix, one of the vaccines administered to M.D.P. According to the manufacturer, Pediarix saw a rate of SIDS of 0.25/1,000 infants over the course of 14 clinical trials. (Ex. 54, p. 8.) By comparison, SIDS has an incidence rate in the United States of 0.57/1,000. (Kinney & Thach, *supra*, at Ex. A, Tab 1, p. 1.) Moreover, respondent's expert, Dr. McCusker, submitted several epidemiologic studies which she indicated further suggest the lack of a causal association between vaccination and SIDS. (Vennemann MM et al., *Sudden Infant Death Syndrome: No Increased Risk After Immunisation*, 25 VACCINE 336 (2007) (Ex. A, Tab 20); Jonville-Bera AP et al., *Sudden Unexpected Death in Infants Under 3 Months of Age and Vaccination Status – A Case-Control Study*, 51 BRITISH J. CLIN. PHARMACOLOGY 271 (2001) (Ex. A, Tab 21); Toro K et al., *Change in Immunisation Schedule and Sudden Infant Death Syndrome in Hungary*, 42 FEMS IMMUNO. & MED. MICROBIOLOGY 119 (2004) (Ex. A, Tab 22); Moro PL et al., *Deaths Reported to the Vaccine Adverse Event Reporting System (VAERS), United States, 1997-2013* (2015) (Ex. A, Tab 23).)

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), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. Appx. 999 (Fed. Cir. 2013). In that regard, the fact that petitioners' theory ultimately rests upon Dr. Miller's *ipse dixit* extrapolation of the Triple Risk Model is especially significant because the evidence underlying his opinion that vaccines can act on the serotoninergic network is also weak. This relates to the second relevant point raised by the Federal Circuit in *Boatmon*. As in that case, petitioners have not satisfactorily demonstrated that vaccines produce cytokines that act on the 5-HT receptors in a manner consistent with the Triple Risk Model of SIDS.

This aspect of Dr. Miller's opinion is still largely based on animal-model studies, three of which were explicitly rejected by the Federal Circuit in *Boatmon*. Specifically, as in *Boatmon*, Dr. Miller relies in this case on two pig studies, by Stoltenberg et al., and Frøen et al., respectively, and one rat study by Brambilla et al.[10] He further cites an additional piglet study by Tang et al., and an additional rat study by Li et al.[11] Upon my own review, and further considering Dr. McCusker's competing opinion regarding the value of these studies (Ex. A, pp. 12-14), I find that they are insufficient to show that a cytokine response would affect the 5-HT receptors *in vivo*. In particular, Dr. McCusker stressed that the cytokine levels that would be achieved by vaccination would be "orders of magnitude less" than those examined in the above-discussed studies. (Ex. A, p. 13.) In that regard, both parties filed a study by Kashiwagi et al., that examined cytokine response to vaccination *in vivo* by drawing serum samples from 79 human vaccinees, 61 of whom experienced a febrile response. (Yasuyo Kashiwagi et al., *Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influenza Type B (Hib), and 7-Valent Pneumococcal (PCV7) Vaccines*, 10 HUMAN VACCINE & IMMUNOTHERAPEUTICS 677 (2014) (Ex. 31) (Ex. A, Tab 30).) While Dr. Miller cited this study for the proposition that vaccines did increase cytokine response, Dr. McCusker explained that the study found cytokine levels present within 48 hours of vaccination that are "very low" and in particular that IL-1β, the subject of the Stoltenberg, Frøen, and Brambilla studies, is not elevated at 48 hours post-vaccination. (Ex. A, pp. 9-10 (citing Kashiwagi et al, *supra*, at Ex. A, Tab 30).) In her

[10] L. Stoltenberg et al., *Changes in Apnea and Autoresuscitation in Piglets After Intravenous and Intrathecal Interleukin-1β Injection*, 22 J. PERINAT MED. 421 (1994) (Ex. 33); J.F. Frøen et al., *Adverse Effects of Nicotine and Interleukin-1β on Autoresuscitation After Apnea in Piglets: Implications for Sudden Infant Death Syndrome*, 105 PEDIATRICS E52 (2000) (Ex. 34); D. Brambilla et al., *Interleukin-1 Inhibits Firing od Serotonergic Neurons in the Dorsal Raphe Nucleus and Enhances GABAergic Inhibitory Post-Synaptic Potentials*, 26 EUROPEAN J. NEUROSCIENCE 1862 (2007) (Ex. 35).

[11] Samantha Tang, Rita Machaalani & Karen A. Waters, *Brain-Derived Neurotrophic (BDNF) and TrkB in the Piglet Brainstem After Post-Natal Nicotine and Intermittent Hypercapnic Hypoxia*, 1232 BRAIN RESEARCH 195 (2008) (Ex. 36); Qingqing Li, *Neonatal Vaccination with Bacillus Calmette-Guerin and Hepatitis B Vaccines Modulates Hippocampal Synaptic Plasticity in Rats*, 208 J. NEUROIMMUNOLOGY 1 (2015) (Ex. 40).

opinion, "there is no evidence to suggest that the levels are sufficient to influence the development of cytokine-mediated changes in respiration as hypothesized by petitioners' expert."[12] (Ex. A, p. 10.)

Dr. Steinman further added that the Pediarix vaccine (among those at issue in this case) contains alum, which is known to induce IL-1β secretion, as well as pertussis toxin, which is known to open the blood brain barrier. (Ex. 47, pp. 5-6.) However, as presented these points are neither specific enough nor well enough substantiated to add significantly to the above-described evidence of record more directly addressing the alleged relationship between vaccination, cytokines, and 5-HT receptors.[13] As above, on these points Dr. Steinman relies on *in vitro* and animal model studies. (Stephanie C. Eisenbarth et al., *Crucial Roe for the Nalp3 Inflammasome in the Immunostimulatory Properties of Aluminum Adjuvants*, 453 NATURE 1122 (2008) (Ex. 64); Kerstin E. Brückener et al., *Permeabilization in a Cerebral Endothelial Barrier Model by Pertussis Toxin Involves the PKC Effector Pathway and is Abolished by Elevated Levels of cAMP*, 116 J. CELL SCIENCE 1837 (2003) (Ex. 67).) Notably, however, among the vaccines studied in humans, Kashiwagi et al., examined an alum-containing acellular pertussis vaccine administered in Japan (referenced in the study as "DPT") and concluded that *in vivo* it did not necessarily produce higher levels of IL-1β compared to the other vaccines and vaccine combinations and, significantly, concluded that IL-1β production did not depend on the amount of aluminum adjuvant in the vaccine.[14] (Kashiwagi et al., *supra*,

[12] Prior decisions have also found that Kashiwagi et al., is inadequate to support the idea that cytokines produced in response to vaccination could negatively impact the brain. *See, e.g., Dean v. Sec'y of Health & Human Servs*., No. 13-808V, 2017 WL 2926605, at *17 (Fed. Cl. Spec. Mstr. June 9, 2017); *Copenhaver, supra,* at *9-14*; Cozart, supra,* at *6-7.

[13] Moreover, Dr. Steinman appears to overstate the evidence. For example, he states without qualification that "[o]f particular interest the pertussis toxin in Pediarix vaccine is critical for opening the blood brain barrier itself." (Ex. 47, p. 6.) Examining his source material, however, only one of the cited studies directly examines pertussis toxin and its authors are far more equivocal than Dr. Steinman. (Brückener et al., *supra*, at Ex. 67.) The study authors note with regard to pertussis toxin that its "role in the onset of systemic disease is still not completely understood. Especially, whether [pertussis toxin] might be instrumental in the development of neurological complications that are occasionally observed as a sequelae of pertussis disease *has not been elucidated*. In the pathogenesis of pertussis-related neurologic disorders an important step *might affect* the integrity of cerebral barriers represented either by the Plexus chorioideum epithelium or the cerebral capillary endothelium." (*Id*. at 1 (emphasis added).) In contrast to Dr. Steinman's phrasing, the study authors explain that "this study *implies a potential mechanism* for the onset of neurological disorders associated with pertussis disease due to the effect of [pertussis toxin] on the integrity of the blood-brain-barrier." (*Id*. at 2.) Moreover, the study was conducted on cell lines isolated from pig brains and utilized pertussis toxin itself, not an acellular pertussis vaccine formulation. With regard to vaccinations the authors note of a prior study that "it has been reported that whole-cell but not acellular pertussis vaccine induced convulsive activity in mice." (*Id*. at 7.)

[14] Specifically, the authors explained:

> The IL-1β levels were significantly higher in response to PCV7 than to DPT and this difference depended on the antigen-aluminum formulation. IL -1β levels with the simultaneous stimulation with DPT and Hib were the same as those induced by PCV alone, but were higher with the concurrent stimulation including of PCV7. IL -1β production did not depend on the amount of aluminum adjuvant. DPT and PCV7 contain aluminum

at Ex. 31, p. 5.) Additionally, Dr. McCusker explained that "there are currently 5 known pathways by which peripheral cytokine activation may influence cytokine expression in the [central nervous system], each with active regulation of the location in the [central nervous system] and the amount of cytokine expression." (Ex. A, p. 12.) Consistent with the above, she further explained that "[l]ocal cytokine production at the site of vaccination will stimulate cytokine release which can result in sickness behaviors but there is no evidence that these low concentrations of peripheral cytokines result in the respiratory arrest and SIDS as proposed by petitioners' experts." (*Id*.) Again, with regard to the above-discussed animal studies cited by Dr. Miller, she stressed that these studies "required cytokine levels over 1,000 times greater than that produced by vaccination" . . . "significantly limiting the relevance of these findings to the clinical setting." (*Id*.)

Moreover, even assuming *arguendo* that cytokines can be shown to be present in the brain as alleged, the evidence still does not necessarily implicate vaccinations. This was likewise an issue directly addressed by the Federal Circuit. *Boatmon*, 941 F.3d at 1361 (noting that during oral argument petitioner was asked to distinguish between the presence of cytokines and the function of cytokines). Although Dr. Miller cited two studies that showed some evidence post-mortem of cytokines within the brain tissue of SIDS victims, Dr. McCusker explained that cytokines are "induced at times of increased neuronal activity and IL1 and IL 6 are involved in basic brain physiology." (Ex. A, p. 11.) Cytokines can be increased within the brain for a number of reasons, including stressors such as psychological stress, acute brain injury, or neurodegeneration. (*Id*.)

Consistent with Dr. McCusker's opinion, one of the studies cited by Dr. Miller found evidence of increased levels of the cytokine Interleukin-2 ("IL-2") among post-mortem brain tissue samples of eighteen infants experiencing SIDS as well as among ten controls that died of known, diverse causes, including "infectious, hemodynamic, metabolic, severe congenital, or other serious conditions." (Hazim Kadhim et al., *Interleukin-2 as a Neuromodulator Possibly Implicated in the Physiopathology of Sudden Infant Death Syndrome*, 480 NEUROSCIENCE LETTERS 122, 123 (2010) (Ex. 39, p. 2).) Although the study authors suggested that their results evidenced a "central neuro-cytokine connection" in SIDS, they could not distinguish from among the wide range of "various biological stressors" (including infectious/inflammatory, ischemic/anoxic, immune conditions, and metabolic disorders) that affected both the SIDS and non-SIDS study groups. (*Id*. at 4.)

A different study cited by petitioners examined the presence of a different cytokine – Interlueken-6 ("IL-6") – and found "abnormal IL-6 [receptor] expression in the arcuate nucleus in SIDS cases," but noted that 44% of those cases had mild infections prior to death. (Ingvar Jon Rognum et al., *Interleukin-6 and the Serotonergic System of*

> adjuvants and the concurrent stimulation with DPT and PCV7 induced higher IL-β levels, but lower than those induced by PCV7 plus Hib.

(Kashiwagi et al., *supra*, at Ex. 31, p. 5.)

the Medulla Oblongata in the Sudden Infant Death Syndrome, 118 ACTA NEUROPATHOLOGY 519, 529 (2009) (Ex. 38, p. 11).) The authors concluded that the deaths were "potentially induced by the combined effect of prone position and mild infection." (*Id*.) The study authors further postulated that "[t]he increased expression of the IL-6R in the arcuate nucleus may be a compensatory mechanism as defective arcuate neurons may require excessive IL-6 stimulation in order to respond to altered CO2 levels." (*Id*. at 10.) This would suggest that the presence of these cytokines evidences the biological process proposed by the Triple Risk Model itself rather than evidencing that a vaccine reaction triggered that process. In that regard, Dr. McCusker persuasively explained that "[b]reathing dysregulation, specifically resistive breathing and increased work of breathing have been shown to increase plasma cytokine levels through increased production of cytokines, including IL 1β and IL6 by the muscle of the diaphragm." (Ex. A, p. 14.) Dr. McCusker noted that this is especially significant given the relationship between SIDS and prone sleeping and further suggested that "[d]ifferences in IL6 and IL6R levels seen in SIDS children are likely a marker of the acute brain injury prior to death and the presence of cytokines in the brains of SIDS patients represent normal physiologic responses to the stressors associated with their terminal events." (*Id.*)

b. ADP-Ribosylation

Nonetheless, petitioners also stress that the addition of Dr. Steinman's opinion regarding ADP-ribosylase is a significant factor that resolves the deficiencies cited by the Federal Circuit in *Boatmon*. (ECF No. 81, pp. 3-4.) Dr. Steinman opined that "ADP-ribosylation plays a role in seizures and in neuronal death" and that "pertussis toxin, contained in the acellular component of the DTaP vaccine has ADP-ribosylase activity." (Ex. 47, p. 5.) Accordingly, he opined that "[p]ertussis toxin in the DTaP vaccine was highly likely to have contributed to SIDS in this case via the effects on ADP ribose moieties." (*Id*.) As discussed above, Dr. Steinman also ascribes significance to the ability of pertussis to cross the blood brain barrier and alum to induce IL-1β secretion. (*Id*. at 5-6.) Upon my review, I do not find that this theory by Dr. Steinman, either alone or in conjunction with Dr. Miller, presents a sound and reliable explanation connecting M.D.P.'s death to his vaccinations.[15]

[15] It is not entirely clear whether Dr. Steinman intended his theory of ADP-ribosylation as an explanation for how vaccines can act as exogenous stressors pursuant to the Triple Risk Model or whether he intended ADP-ribosylation as a separate explanation for how vaccinations could contribute to death independent of the Triple Risk Model. Although he cited approvingly to Dr. Miller's report generally and in specific regard to his discussion of alum opening the blood brain barrier, he never explicitly linked his discussion of ADP-ribosylation to the Triple Risk Model. (Ex. 47, pp. 5-7.) Instead, as noted above, he stated only that "[p]ertussis toxin in the DTaP vaccine was highly likely to have contributed to SIDS in this case via the effects on ADP ribose moieties." (*Id*. at 5.) In response to my show cause order, petitioners suggest on the one hand that Dr. Steinman's opinion explains "a conduit by which cytokines can gain access to the brainstem, including the area of the brainstem responsible for regulating respiration, producing arousal from apnea, and increasing respiration to hypercarbia. Obviously, if these functions are compromised in an infant under six months, a catastrophic consequence can occur." (ECF No. 81, p. 3 (internal citation omitted).) This suggests that petitioners believe Dr. Steinman's opinion supports the idea that vaccines contribute to the Triple Risk Model. On the other hand, petitioners also asserted regarding *Boatmon* that "[w]hile some of Dr. Miller's opinions in this case are still relevant to entitlement

First, although Dr. Steinman characterizes ADP ribosylation as "contribut[ing] to SIDS," his explanation does not directly relate his ADP-ribosylase theory to SIDS (or to any other specific manner of death), but instead suggests that excessive ADP ribosylation leads to seizures which in turn lead to neuronal death. (Ex. 47, p. 5.) In fact, Dr. Steinman has previously, and unsuccessfully, presented this theory in relation to seizure disorders. *Zumwalt v. Sec'y of Health & Human Servs*., No. 16-994V, 2019 WL 1953739 (Fed. Cl. Spec. Mstr. Mar. 21, 2019), *review denied* 146 Fed. Cl. 525 (2019). However, even accepting that seizures can sometimes be fatal, there is no seizure disorder alleged in this case. Nor is there any evidence that M.D.P. ever experienced a seizure at any point in his life. Accordingly, Dr. Steinman's theory, and much of the literature it is based on (several of the cited articles relate specifically to seizures[16]), is inapposite.

Nor, for that matter, is the theory persuasive even as applied to seizure disorders. Significantly, Dr. Steinman acknowledged in the prior *Zumwalt* case that much of the literature he relied upon linking pertussis to ADP-ribosylation was "outdated." 2019 WL 1953739, at *17. Similarly, in this case, several of the articles Dr. Steinman cites are studies from the 1980's relating to the development of a safer vaccine to replace whole cell pertussis vaccines. (W.J. Black et al., *ADP-Ribosyltransferase Activity of Pertussis Toxin and Immunomodulation by Bordetella Pertussis*, 240 SCIENCE 656 (1988) (Ex. 55); J.R. Okensenberg et al., *Multiple T and B Cell Epitopes in the S1 Subunit ("A"-Monomer) of the Pertussis Toxin Molecule*, 143 J. Immunology 4227 (1989) (Ex. 56); Jorge R. Okesenberg et al., *MHC-Restricted Recognition of Immunogenic T Cell Epitopes of Pertussis Toxin Reveals Determinants in Man Distinct from the ADP-Ribosylase Active Site*, 168 J. EXP. MED. 1855 (1988) (Ex. 57).) Moreover, a key piece of evidence in *Zumwalt*, also presented in this case as the sole citation relating specifically to modern DTaP vaccines, is Gomez et al., a study that examined ADP-ribosylation related to pertussis vaccination using histamine-sensitisation tests (an accepted method of testing pertussis vaccine for residual toxicity or reversion). (S.R. Gomez et al., *ADP-Ribosylation Activity in Pertussis Vaccines and its Relationship to the In Vivo Histamine-Sensitisation Test*, 25 VACCINE 3311 (2007) (Ex. 63).) Petitioners argue that Gomez et al. demonstrates the switch from DTP to DTaP vaccines to be irrelevant because the study shows "all versions of the DTaP vaccine currently on the market continue to demonstrate ADP-ribosylation." (ECF No. 81, p. 5 (citing Gomez et al, *supra*, at Ex. 63).) Notably, however, the Gomez study did

---

determination (after all, he is a neuropathologist with extensive expertise in SIDS), the primary difference here is that Petitioner has an explanation of the direct effect/impact on the brainstem from Dr. Steinman, and Petitioner is not relying on a supposed pre-existing defect in the medulla to satisfy *Althen*. Dr. Steinman provides an explanation as to how vaccination can open the Blood Brain Barrier, and more importantly, how the vaccination M.D.P. received became pathogenic." (*Id*. at 5-6.) This seems to suggest petitioners are advancing Dr. Steinman's opinion in preference to Dr. Miller's opinion and in preference to the Triple Risk Model. For these reasons I stress that I have considered Dr. Steinman's opinion both as further support for Dr. Miller's extension of the Triple Risk Model and also as a standalone theory of causation.

[16] Welhal Ying et al., *Poly (ADP-Ribose Glycohydrolase Mediates Oxidative and Excitotoxic Neuronal Death*, 98 PNAS 12227 (2001) (Ex. 61); Chi, Wang & Li, *supra*, at 60; Wang et al., *supra*, at Ex. 62.

not find that these test results could be universally correlated to ADP-ribosylation, finding instead that "most formulations" of vaccine containing chemically detoxified pertussis showed no correlation between ADP-ribosylation and histamine-sensitisation tests. (Gomez et al. *supra*, at Ex. 63, p. 7.)

To the extent Dr. Steinman may intimate that this theory can be applied more broadly beyond the context of seizure disorders, he is extremely vague and unpersuasive. The literature cited by Dr. Steinman indicates that ADP-ribosylase is a "ubiquitous" enzyme involved in DNA repair and involved in many physiological processes, including gene expression and cell death. (Ling-yi Chi, Sheng-jun Wang & Xin-gang Li, *Poly (ADP-Ribose) Signal in Seizures-Induced Neuron Death*, 71 MEDICAL HYPOTHESES 283, 283-84 (2008) (Ex. 60, pp. 1-2); Sheng-jun Wang et al., *Poly (ADP-Ribose) Polymerase Inhibitor is Neuroprotective in Epileptic Rat Via Apoptosis-Inducting Factor and Akt Signaling*, 18 NEUROREPORT 1285, 1285 (2007) (Ex. 62, p. 1).) However, Dr. Steinman is never more specific than to assert that ADP ribosylases "are associated with neuronal death in a variety of neurodegenerative diseases." (Ex. 47, p. 5.) Apart from his endorsement of Dr. Miller's opinion based on the Triple Risk Model, Dr. Steinman never otherwise opined that any specific neurodegenerative condition or process could be explained by ADP ribosylation or be responsible for SIDS.[17] Moreover, Dr. Steinman himself cited a 2012 IOM report regarding adverse effects of vaccines, including an examination of SIDS following DTaP immunizations, as well as package insert material for the Pediarix vaccine administered to M.D.P., which likewise contained a discussion of incidences of post-vaccination SIDS. (Ex. 47, pp. 4-5.) Neither supports a causal relationship between DTaP and SIDS. (*See supra*, at n.9.)

c. Brainstem Abnormality

Lastly, I note that petitioners have stressed that, unlike the *Boatmon* case, there is evidence in this case that M.D.P. did in fact have a brainstem abnormality. Dr. Miller reviewed four sections of M.D.P.'s medulla preserved in paraffin block and opined that "[t]he section[s] of the medulla all show that there is only a thin ribbon of gray matter

[17] Notably, neither petitioners' nor respondent's experts in neuropathology identified a cause of death for M.D.P. While Dr. Harris characterized M.D.P.'s death as SUID potentially explained as asphyxia, Dr. Miller described M.D.P.'s death as SIDS. Accordingly, it is not clear on what basis Dr. Steinman could theorize broadly that M.D.P. experienced fatal, unspecified, vaccine-caused neurodegeneration. Indeed, Dr. Steinman confirmed that he agreed with Dr. Miller's report "in full." (Ex. 47, p. 7.) If instead Dr. Steinman associated ADP-ribosylation with neurodegenerative disease only as a means of demonstrating that it can be responsible for neuronal death in the most general sense and thereby potentially infer that the specific finding of hyperplastic nuclei in the medulla made by both neuropathologists could ultimately have been vaccine-caused, this would still leave a considerable unfilled gap in Dr. Steinman's theory. He has cited nothing to support – nor has he even explicitly asserted – the idea that his theory of ADP-ribosylation can result specifically in the type brainstem abnormality potentially present in this case. Dr. Steinman did separately discuss in the context of the unrelated condition of neuromyelitis optica that the area of the postrema in the medulla is "another conduit whereby cytokines can gain access to the brain stem." (*Id*. at 6.) However, he does not in any way link this discussion back to the neuronal death he discussed regarding ADP-ribosylation. (*Id*. at 5-6.) (Moreover, he cites this as relating to his opinion that vaccines containing pertussis toxin can open the blood brain barrier. (*Id*. at 6.) I addressed that point in the prior discussion section regarding the Triple Risk Model.)

representing the ventral arcuate nuclei on the anterior surface of the medullar pyramids. If this represents the maximum extent of these nuclei, they are hypoplastic." (Ex. 13, p. 4.) Respondent's expert, Dr. Harris also reviewed the four sections of the medulla. (Ex. C, p. 4.) He similarly opined that they "show normal appearing nuclei, with the exception of the arcuate nucleus that appears somewhat hypoplastic; a finding that has been associated with SIDS in some neuropathological studies (reviewed by Kinney, 2009). However, the full medulla is not represented, and the arcuate can appear hypoplastic in normal medulla at some levels." (*Id*.) Dr. Harris agreed that "[t]he autopsy did show a possible structural change in the brainstem." (*Id*. at 5.) However, he noted that "[s]erotonin receptor changes that have been extensively reported by Kinney and others (Kinney, 2009) are generally not available in forensic practice and were not done in this case." (*Id*.)

While petitioners are correct that this constitutes a significant difference between the two cases, the distinction has no bearing on the outcome of this case. The presence of a brainstem abnormality is an accepted part of the Triple Risk Model. Although the Federal Circuit in *Boatmon* found error in the special master's reliance on statistical evidence of the presence of such a brainstem abnormality in the *Boatmon* child, that holding was in regard to petitioner's burden of proof under *Althen* prong two, which requires a logical sequence of cause and effect showing that the vaccine at issue did cause the decedent's death. 941 F.3d at 1362-63. That is, the question of whether a brainstem abnormality actually exists in a given child relates to specific rather than general causation. Thus, the answer to that question neither defeated petitioner's *Althen* prong one theory in *Boatmon* nor supports petitioner's *Althen* prong one burden in this case. Here, I have concluded as a threshold matter that petitioners' failure to meet their burden under *Althen* prong one (general causation) is dispositive and I do not reach *Althen* prongs two (relating to specific causation) or three (temporal relationship) and therefore do not resolve the significance of Dr Miller's and Dr. Harris's opinions regarding the condition of M.D.P.'s brainstem.

VII. Conclusion

In light of all of the above and in consideration of the record as a whole, I find that petitioners have failed to meet their burden under *Althen* prong one. Neither Dr. Miller's nor Dr. Steinman's opinions, alone or in combination, provide a sound and reliable theory of causation that could link M.D.P.'s death to any of his vaccinations. M.D.P.'s unexplained death is tragic and a profound loss for his family. Mr. Powers and Ms. Downing-Powers have my deepest sympathy. I also appreciate their desire to know and understand the cause of their child's death. Unfortunately, upon my review of their claim, this Program cannot provide them the answer they seek. Petitioners have not established by preponderant evidence a medical theory causally connecting any of vaccines M.D.P. received to SIDS via the Triple Risk Model. Nor have their experts provided any alternate explanation for how vaccines could cause an otherwise unexplained death. **Accordingly, this petition is DISMISSED.** In the absence of a

motion for review filed pursuant to RCFC Appendix B, the clerk of the court is directed to enter judgment accordingly.[18]

IT IS SO ORDERED.

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

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