In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 21-835V
Filed: February 11, 2025

JASON and TABITHA GASKIN, on behalf of their son, JASON GASKIN, JR., Deceased,

Petitioners,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Lisa A. Roquemore, Law Office of Lisa A. Roquemore, Rancho Santa Margarita, CA, for petitioners.
Zoe Wade, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On February 1, 2021, petitioners filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that their son, Jason, suffered fatal myocarditis as a result of influenza ("flu") and/or varicella vaccinations he received at his 15-month wellness check on February 7, 2019. (ECF No. 1.) 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 Jason's fatal myocarditis was caused by his vaccination(s). Thus, petitioners are *not* entitled to an award of compensation.

[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), 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, 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.

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

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. § 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 alleged that the vaccinations at issue caused myocarditis, which is not listed on the Vaccine Injury Table relative to any vaccine. Therefore, petitioners must meet the burden of proof for establishing causation-in-fact.

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, 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 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[.]" *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Ultimately, petitioner 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 all the relevant evidence of 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

Petitioners filed medical records and autopsy reports marked as Exhibits 1-7 in February of 2021 and later filed a Statement of Completion in June of 2021. (ECF Nos.

5, 8.) After the case was assigned to the undersigned in December of 2021, petitioners filed an expert report by pediatric cardiologist Anthony Chang, M.D. (ECF Nos. 13, 17-18; Exs. 8-18.) Dr. Chang opined that Jason died of acute myocarditis secondary to his vaccinations. (Ex. 8, p. 6.)

In July of 2022, respondent filed his Rule 4(c) Report, recommending against compensation in this case. (ECF No. 23.) Respondent asserted that Dr. Chang's medical theory implicating Jason's vaccinations in his myocarditis is unreliable and further contended that Jason's autopsy results are more consistent with an infectious lymphocytic myocarditis. (*Id*. at 3-6.) Respondent's report was accompanied by two expert reports by pediatric cardiologist Richard Ringel, M.D., and by immunologist Stephen Jameson, Ph.D. (ECF Nos. 21-22; Exs. A-D.) Respondent's experts pointed out that Dr. Chang relied on literature indicating that vaccine-related myocarditis results from a hypersensitivity response, which is evidenced by eosinophils. (Ex. A, pp. 4-6; Ex. C, pp. 4-5.) Due to the presence of lymphocytes and the absence of eosinophils in Jason's pathology, Dr. Ringel concluded that the medical examiner was correct to conclude that Jason's myocarditis was of "probable viral etiology." (Ex. A, p. 6.)

Petitioners filed a responsive expert report by Dr. Chang in October of 2022. (ECF No. 25; Exs. 19-26.) Dr. Chang challenged the notion that the absence of eosinophils is dispositive. (Ex. 19, p. 6.) I held a Rule 5 conference shortly thereafter. (ECF No. 26.) I explained why I felt that Dr. Chang's theory was vague and required clarification, but also noted that I did not agree that the literature cited by Dr. Chang supported the idea that eosinophils may be entirely absent in hypersensitivity myocarditis. (*Id*. at 2.) I also noted that the issues discussed by the experts had already been litigated in this program.[3] (*Id*.) Following the Rule 5 conference, petitioner filed a further report by Dr. Chang and respondent responded with a further report by Dr. Ringel. (ECF Nos. 28, 31; Exs. 27-31, Ex. E.)

Petitioners initially requested a hearing, but later agreed to proceed to briefing on the written record after being prompted to explain the need for an entitlement hearing. (ECF Nos. 32-34.) Petitioners filed a motion for a ruling on the written record on August 23, 2023, which was fully briefed. (ECF Nos. 35, 37, 40.) Thus, this matter is now ripe for resolution as to entitlement. 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. S*ee Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (citing *Simanski v. Sec'y of Health & Human*

[3] Specifically, I cited the following: *Yates v. Sec'y of Health & Human Servs*., No. 14-560V, 2020 WL 2313691, at *38 (Fed. Cl. Spec. Mstr. Apr. 16, 2020) (distinguishing eosinophilic myocarditis from lymphocytic myocarditis and noting that hypersensitivity myocarditis "is mainly characterized by eosinophils"), *mot. for rev. den'd*, 150 Fed. Cl. 575 (2020); *Matten v. Sec'y of Health & Human Servs*., No. 12-155V, 2021 WL 5768148, at *38 (Fed. Cl. Spec. Mstr. Nov. 2, 2021) (finding that the petitioner's experts offered a sound and reliable medical theory that the flu vaccine can cause eosinophilic and/or hypersensitivity myocarditis and death); *Bantugan v. Sec'y of Health & Human Servs*., No. 15-721V, 2019 WL 7602581, at *19-20 (Fed. Cl. Spec. Mstr. Dec. 20, 2019) (finding that petitioner failed to offer a reliable medical theory that the flu vaccine can cause hypersensitivity myocarditis and stating that "the relevant histology [of hypersensitivity myocarditis] is eosinophils, not lymphocytes").

Servs., 671 F.3d 1368, 1385 (Fed. Cir. 2012)); *see also* Vaccine Rule 8(d); Vaccine Rule 3(b)(2).

III. Factual History

Jason Gaskin, Jr., was born on September 14, 2017. (Ex. 1, p. 6.) He was generally a healthy baby with a regular heart beat and rhythm and no prior cardiac history. (ECF No. 1, pp. 1-2. (citing Ex. 2, pp. 9-16, 21-24, 27-34, 37-40, 46-49).) At his 15-month wellness checkup on February 7, 2019, he received an influenza vaccination as well as a varicella vaccination. (Ex. 2, pp. 46-48, 50-51.) No concerns were noted at this encounter. (*Id*. at 46-49.)

Two days later, on February 9, 2019, Jason was taken to the emergency department with a history of a one-day fever with a maximum temperature of 102.5ºF. (Ex. 1, p. 190, 193.) Jason's parents reported that he had received flu and varicella vaccinations two days prior and that he had recent contact with children at daycare who had been diagnosed with the flu. (*Id*. at 193.) His physical exam was "very reassuring," except for tachycardia. (*Id*. at 190, 193.) There were no signs of bacterial infection, and a flu test was negative. (*Id*. at 190.) Accordingly, it was suspected that Jason's fever was due to either teething or vaccination and he was discharged with instructions to use pediatric dosing of Tylenol/Ibuprofen as needed. (*Id*. at 193.) His discharge diagnoses were teething syndrome, postvaccination fever, and tachycardia, unspecified. (*Id*. at 190.)

EMS was dispatched to the Gaskin home in the early hours of the morning of February 13, 2019, regarding a report of a witnessed pediatric cardiac arrest. (Ex. 3, p. 3.) The report explains:

> Family on scene advise EMS that the patient has been sick for the past few days. They state that the patient has been eating and drinking less than normal, but has still been able to nurse at his regular times without difficulty. Mother states that she has been in contact with the patient[']s pediatrician about his illness over the past few days and had been following instructions from the pediatrician to maintain hydration and decrease vomiting. Mother states that the patient has had only [] 2-3 episodes of vomiting over the past few days but has been able to keep food down, although he has had a decreased appetite. Mother states that she noted that the patient appeared to be restless this evening and while she was nursing him, he became lethargic and stopped responding. Mother and father state that they contacted 911 at this time and followed instructions to perform CPR.

(*Id*.) Jason was unresponsive when EMS arrived. (*Id*.) EMS attempted CPR for 45 minutes. (Ex. 1, p. 287.) There was a further attempt at resuscitation at the hospital; however, Jason remained in asystole and was pronounced dead in the pediatric intensive care unit at 2:22 A.M. (*Id.*)

An autopsy with microscopic examination was performed on February 14, 2019. (Ex. 4, pp. 1, 3-4.) The autopsy concluded that the cause of death was "myocarditis, of probable viral etiology, not otherwise specified" with additional diagnoses of early pericarditis and left pleural effusion. (*Id*. at 1.) Pertinent to the analysis that follows, myocardium samples showed "extensive multifocal interstitial mononuclear inflammatory infiltrate, largely comprised of lymphocytes, with associated myocyte necrosis, early interstitial edema, and early pericardial involvement; no microorganisms identified." (*Id*. at 4.) Respiratory findings were "pulmonary parenchyma with congestion, patchy alveolar edema, focally increased alveolar macrophages, focal minimal peri-bronchial chronic inflammatory infiltrate, and focal alveolar expansion." (*Id*.) The medical examiner concluded:

> The cause of death of Jason Nathaniel Gaskin, Jr., an 18-month-old[4] boy, is attributed to likely fatal irregularity of the heart beat occurring in the setting of significant inflammation involving the heart muscle in association with probable recent viral infection.
>
> Postmortem examination additionally revealed no other contributor natural disease or physical injury; and toxicologic analysis of the blood detected no alcohol or drugs.

(*Id*.)

IV. Summary of Expert Opinions

a. Petitioners' Pediatric Cardiologist, Anthony Chang, M.D.[5]

Dr. Chang explains that myocarditis is defined as inflammation of the heart, which can be fatal. (Ex. 8, p. 4.) Clinical signs can include chest pain, dyspnea, palpitations, hemodynamic instability, and malignant tachydysrhythmias. (*Id*.)

[4] In fact, Jason was about 17 months of age.

[5] Dr. Chang received his bachelor's degree in molecular biology from Johns Hopkins University in 1980 and his medical degree from Georgetown University in 1984. (Ex. 9, p. 1.) He completed his residency in pediatrics at Children's Hospital National Medical Center in Washington, DC in 1987. (*Id.*) After his residency, Dr. Chang completed a fellowship in pediatric cardiology at the Children's Hospital of Philadelphia in 1990. (*Id.*) He then went on to earn additional graduate degrees, including a M.P.H. from the University of California at Los Angeles in 2008. (*Id.*) Dr. Chang has held numerous academic appointments, with professorships in pediatrics at Harvard University School of Medicine, the University of Southern California School of Medicine, and Baylor College of Medicine. (*Id.* at 2.) He served as the Director of the Pediatric Cardiac Intensive Care Service at Miami Children's Hospital from 1995 to 2000, and the Director of the Heart Institute at Children's Hospital of Orange County from 2006 to 2015. (*Id.*) Since 2015, Dr. Chang has served as the Director of the Heart Failure Program at Children's Hospital of Orange County. (*Id.*) Dr. Chang is board certified in pediatrics and pediatric cardiology, and he maintains his medical license in California. (*Id.* at 1.) Dr. Chang has been publishing manuscripts on myocarditis since 1992. (Ex. 8, p. 6.) At the time of publishing his initial report, Dr. Chang had published 115 scholarly articles as well as numerous book chapters and textbooks, primarily focusing on pediatric cardiology and pediatric critical care medicine. (Ex. 9, pp. 20-34.)

Myocarditis can be acute, chronic (active or persistent), or fulminant. (*Id.*) Fulminant myocarditis is an acute deterioration of cardiac function in a matter of hours to days. (*Id*.) Myocarditis can be further subdivided by histopathology, including classification as either lymphocytic or eosinophilic. (*Id*.) Viral illness is the most common cause of myocarditis. (*Id.*) Viruses can cause myocarditis either directly via cytotoxic proteins or indirectly by inducing inflammation. (*Id*.) Both the influenza and varicella viral infections have been described in medical literature as causes of myocarditis.[6] (*Id*.)

Dr. Chang opines:

> The influenza vaccine Flulaval Quadrivalent is an inactivated influenza virus vaccine from virus that was propagated in hens' eggs and is for the prevention of disease caused by influenza A subtype viruses and type B viruses. The antigenicity of this virus element can lead to inflammatory processes such as pericarditis or myocarditis. In other words, the influenza vaccine, with its viral antigens, can induce an inflammatory response of the host that involved the inflammatory cascade resulting in immune regulatory imbalance and subsequent pericarditis or myocarditis. The varicella vaccine is also a live-attenuated type of vaccine and therefore can have the same type of inflammatory response as the influenza vaccine. Both of these attenuated vaccines therefore have the potential to cause myocarditis as a result of a dysregulated immune system of the body due to its reaction to the viral antigen in the vaccines in the attenuated form.

(*Id*.)

Dr. Chang stresses that no other antecedent infection or other medical condition was present, leaving the vaccinations as the only significant event prior to Jason's sudden cardiac arrest. (Ex. 8, p. 5.) He opines that the symptoms of decreased appetite and vomiting that Jason experienced in the days leading up to his death were more likely symptoms of his myocarditis or part of a post-vaccination reaction rather than symptoms of a separate viral illness. (Ex. 19, p. 7.) He observes that Jason tested negative for influenza A and B and had no evidence of a bacterial infection at his emergency encounter on February 9, 2019. (*Id*.) He also notes that the autopsy did not identify a specific viral etiology for Jason's myocarditis. (Ex. 8, p. 5.) Thus, Dr. Chang considers respondent's reliance on a "hypothetical viral illness" to be "entirely groundless." (Ex. 19, p. 7.) In any event, even if one concluded Jason did have an infection, one should consider that the varicella vaccine, as a live virus vaccine, is

[6] Citing Paola Dolader et al., *Influenza Myocarditis in Paediatric Patients*, 32 CARDIOLOGY IN YOUNG 1188 (2021) (EX. 13); Nischit Baral et al., *Influenza Myocarditis: A Literature Review*, 12 CUREUS e12007 (2020) (Ex. 14); Zarha Raisi Estabragh & Mamas A. Mamas, *The Cardiovascular Manifestations of Influenza: A Systematic Review*, 167 INT'L J. CARDIOLOGY 2397 (2013) (Ex. 15); Kiran P. Sawardekar, *Fatal Varicella Myocarditis in a Child with Down Syndrome – A Case Report*, 62 J. TROPICAL PEDIATRICS 250 (2016) (Ex. 16); Emine Azak & Ibrahim I. Cetin, *Acute Myocarditis Following Varicella Zoster Infection in an Immunocompromised Adolescent: An Uncommon Complication*, 118 ARCHIVOS ARG. PEDIATRIA e12007 (2020) (Ex. 17).

capable of acting as a mild infection. (*Id.*) Therefore, the varicella vaccine could have caused myocarditis via the same kinetics as a wild-type viral infection.[7] (*Id*.)

Further, Dr. Chang contends that the autopsy otherwise shows an acute inflammatory process consistent with vaccine-causation. (Ex. 8, p. 5.) He opines that "[t]he relationship of the vaccines to the cardiac compromise is both plausible and nonspurious (there is no third variable involved) leading to the conclusion that the cause of the myocarditis is, more likely, the vaccine(s) itself." (*Id.*) He indicates that a vaccine would cause myocarditis in a matter of days to "maybe" weeks. (*Id*.) He opines that the timing of events in this case is consistent with the time over which fulminant myocarditis occurs. (*Id*.)

Among the case reports cited by Dr. Chang, a report by Thanjan et al. (the "Thanjan case report"), proposed that multiple vaccinations (specifically DTaP, meningococcal, and hepatitis A vaccines) resulted in a hypersensitivity myocarditis. (Ex. 8, p. 4 (citing Maria T. Thanjan et al., *Acute Myopericarditis After Multiple Vaccinations in an Adolescent: Case Report and Review of the Literature*, 119 PEDIATRICS e1400 (2007) (Ex. 10)).) In that regard, Dr. Chang disagrees with respondent's expert's contention that eosinophils would need to have been present for a hypersensitivity reaction to lead to a vaccine-related myocarditis. (Ex. 19, p. 6.) He asserts that, while eosinophils have a high positive predictive value for a hypersensitivity reaction, the absence of eosinophils does not rule out a hypersensitivity reaction.[8] (*Id.*) Dr. Chang also noted that the language of the autopsy report did not definitively preclude the presence of eosinophils, noting only that the infiltrate "largely" consisted of lymphocytes. (*Id*.) He explains that eosinophilic myocarditis does not necessarily involve eosinophils exclusively. (*Id*.) Furthermore, he opines that the presence of eosinophils would have dissipated in the seven days between Jason's vaccination(s) and the autopsy. (*Id*.)

Dr. Chang filed a third report (Ex. 27); however, that report did not add any new information.

[7] Citing Mark K. Slifka & Ian Amanna, *How Advances in Immunology Provide Insight into Improving Vaccine Efficacy*, 32 VACCINE 2948 (2014) (EX. 24).

[8] Citing A.P. Burke et al., *Hypersensitivity Myocarditis*, 115 ARCHIVES PATHOLOGY & LAB'Y MED. 764 (1991) (Exs. 20, 32); Nobutaka Nagano et al., *Hemodynamic Collapse After Influenza Vaccination: A Vaccine-Induced Fulminant Myocarditis*, 36 Can. J. Cardiology 1554.e5 (2020) (Exs. 21, 31).

b. Respondent's Immunologist, Stephen Jameson, Ph.D.[9]

Dr. Jameson stresses that "naturally occurring viral infections are the likely cause of most cases of infant myocarditis." (Ex. C, p. 3.) Noting that Dr. Chang purported to rule out a viral cause for Jason's myocarditis, he cautioned that this assertion is misleading because there was no testing for any viruses at the time of Jason's death. (*Id.*) Accordingly, excluding a natural infection as the cause of Jason's myocarditis is unsupported by any evidence. (*Id*.) To the extent Dr. Chang cited case reports of myocarditis following acquired infections with both influenza and varicella viruses, Dr. Jameson explains that, even if live virus vaccines, the attenuated viruses contained in vaccines do not result in an immune response comparable to the virulent forms of the virus. (*Id*. at 4.) He notes that Dr. Chang provided no case reports indicating that the vaccines at issue,[10] rather than natural infection, can cause myocarditis. Dr. Jameson disagrees that the case reports regarding natural influenza or varicella infection support vaccine causation. (*Id*.) Moreover, he notes that a single case report cited by Dr. Chang proposed a mechanism of hypersensitivity reaction for myocardial injury post-vaccination. In that case the biopsy indicated the presence of "prominent" eosinophils. (*Id*. at 4 (discussing Thanjan et al., *supra*, at Ex. 10).) However, most viral infections lead to a "Type-1" immune response, which is characterized by infiltrating lymphocytes that may cause local tissue damage in the process of eliminating a viral antigen. (*Id.*) Hypersensitivity reactions are "Type-2" immune reactions in which eosinophils are the most prominent cell type. (*Id*.) Thus, the fact that Jason's autopsy showed that his myocardium had lymphocytes, but no mention of eosinophils, supports a viral, rather than post-vaccination, etiology for Jason's myocarditis. (*Id*. at 4-5.)

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[9] Dr. Jameson received his bachelor's degree in cellular pathology from the University of Bristol in England in 1984, and his Ph.D. in immunology from Cambridge University in 1988. (Ex. D, p. 1.) He completed postdoctoral training at Scripps Clinic and Research Foundation in La Jolla, California from 1988 to 1990. (*Id.*) From 1990 to 1995, Dr. Jameson worked as a senior fellow in the Department of Immunology at the University of Washington in Seattle. (*Id.*) In 1995, Dr. Jameson established his own research lab at the University of Minnesota. (Ex. C, p. 1.) Since joining the University of Minnesota in 1995, Dr. Jameson has held various academic appointments, and he is currently a professor for the Center for Immunology, Masonic Cancer Center and Department of Laboratory Medicine and Pathology. (*Id.*; Ex. D, p. 1.) His research primarily focuses on the use of animal models to study cellular immune responses against pathogens and vaccines. (Ex. C, p. 1.) Dr. Jameson has published over 125 scholarly articles and over 50 review articles, nearly all focusing on aspects of immunology and the immune response to pathogens. (*Id.*; Ex. D, pp. 13-28.)

[10] Dr. Chang did later file case reports regarding myocarditis following flu vaccination in connection with his second report. (Nagano et al., *supra*, at Exs. 21, 31; Youn-Jung Kim et al., *Acute Fulminant Myocarditis Following Influenza Vaccination Requiring Extracorporeal Membrane Oxygenation*, 34 ACUTE & CRITICAL CARE 165 (2019) (Ex. 22); Ryo Nakamura et al., *Acute Lymphocyte Myocarditis Associated with Influenza Vaccination*, 61 INTERNAL MED. 2307 (2022) (Ex. 23)). However, Dr. Jameson never filed a second report. In his first report, Dr. Chang had cited only a case report of myocarditis following vaccination with DTaP, meningococcal, and hepatitis A vaccines. (Ex. 8, p. 4 (citing Thanjan et al., *supra*, at Ex. 10).)

a. Respondent's Pediatric Cardiologist, Richard Ringel, M.D.[11]

Dr. Ringel explains that "[v]iral myocarditis is a rare, but extensively documented complication of common viral infections seen in infants and children on a regular basis. Almost every viral pathogen has at some point been documented as a cause of myocarditis." (Ex. A, p. 4.) Viruses common to childhood illness have been found in cardiac biopsy specimens in a majority of patients with clinical and microscopic findings of myocarditis.[12] (*Id*.) In this case, the autopsy finding of "interstitial mononuclear inflammatory infiltrate, largely comprised of lymphocytes, with associated myocyte necrosis" is typical of viral myocarditis. (*Id*. (quoting Ex. 4, p. 4.) Dr. Ringel further opines that Jason's symptoms of decreased appetite and vomiting in the days prior to his death are consistent with a viral illness. (*Id*. at 5.) More likely than not, the myocardial inflammation demonstrated by Jason's autopsy was the result of a viral infection of the heart. (*Id.*) Dr. Ringel contends that Dr. Chang's assertion that no viral etiology is evident is misleading insofar as no viral testing was undertaken apart from the emergency department testing for influenza A and B. (*Id*. at 6; Ex. E, p. 9.) On average, young children have seven to eight viral infections each year.[13] (Ex. E, p. 9.) He concurs with the conclusion of the medical examiner that Jason's myocarditis was of "probable viral etiology." (Ex. A, p. 6.)

If a hypersensitivity reaction to vaccination were implicated, as hypothesized in the Thanjan case report cited by Dr. Chang, then one would expect to detect eosinophils during examination of Jason's heart muscle; however, this was not found. (Ex. A, pp. 4-5.) Toxic or allergic myocarditis typically includes findings of macrophages and/or eosinophils.[14] (*Id*. at 5.) Dr. Ringel acknowledges that no test is 100% sensitive and that in rare circumstances it is possible a biopsy or microscopic examination could

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[11] Dr. Ringel received his bachelor's degree from State University of New York at Stony Brook in 1974 and his medical degree from Albert Einstein College of Medicine in New York in 1977. (Ex. B, p. 1.) He completed his internship and residency in pediatrics at the University of Maryland Hospital in Baltimore in 1980. (*Id.*) After finishing his residency, Dr. Ringel held a fellowship position in pediatric cardiology at the University of Maryland Hospital. (*Id.*) Dr. Ringel has held a number of academic appointments, with professorships in pediatrics and pediatric critical care medicine at the University of Maryland School of Medicine, George Washington University School of Medicine, and Johns Hopkins School of Medicine. Currently, Dr. Ringel serves as an emeritus professor of pediatrics and a senior consultant in pediatric cardiology at Johns Hopkins School of Medicine. (Ex. A, p. 2.) He is board certified in pediatrics, pediatric cardiology, and pediatric critical care medicine. (Ex. B, p. 7.) Dr. Ringel is a board diplomate of the American Board of Pediatrics and the National Board of Medical Examiners. (*Id.*) He maintains a license to practice medicine in Maryland. (*Id.*) Dr. Ringel has published approximately 30 peer-reviewed articles and case reports primarily focusing on pediatric cardiology. (*Id.* at 1-3.)

[12] Citing Othman A. Aljohani et al., *Spectrum of Viral Pathogens Identified in Children with Clinical Myocarditis (Pre-Coronavirus Disease-2019, 2010-2018): Etiologic Agent Versus Innocent Bystander*, 242 J. PEDIATRICS 18 (2022) (Ex. A Tab 1).

[13] Citing KAREN J. MARCDANTE ET AL., NELSON ESSENTIALS OF PEDIATRICS 408-09 (9th ed. 2023) (Ex. E Tab 4).

[14] Citing Ornella Leone et al., *The Spectrum of Myocarditis: From Pathology to the Clinics*, 475 VIRCHOWS ARCHIV 279 (2019) (Ex. A Tab 2).

fail to detect eosinophils in a patent with hypersensitivity myocarditis; however, in those circumstances, other diagnostic clues would guide interpretation if the heart muscle biopsy was not diagnostic. (Ex. E, p. 7.) In this case, those clues still point to a viral etiology with no other clinical evidence of a hypersensitivity reaction. (*Id*.) Dr. Ringel contends that Dr. Chang misinterprets the paper by Burke, et al. (*Id.* at 8.) Dr. Ringel explains that the cases examined by Burke were already screened using diagnostic criteria that required the presence of eosinophils to identify hypersensitivity myocarditis. (*Id.*) In any event, Jason's autopsy results were also significant for an absence of histiocytes, which are also characteristic of hypersensitivity myocarditis. (*Id*.)

Dr. Ringel acknowledges that myocarditis has rarely been reported following administration of the Smallpox vaccine; however, he stresses that the Smallpox vaccine is a live vaccine. (Ex. A, p. 6.) Therefore, he opines that reports relating to the Smallpox vaccine cannot be extrapolated to the inactivated (flu) and attenuated (varicella) viral products contained in the vaccinations at issue in this case. (*Id.*) Although Dr. Chang cited a case report by Nagano, et al., proposing that a flu vaccination led to fulminant myocarditis, Dr. Ringel stresses that the authors acknowledge that this case report does not evidence causation. (Ex. E, p. 7.) He further stresses that the title of the case report is itself couched merely as a question – "A Vaccine-Induced Fulminant Myocarditis?" (*Id*.) Dr. Ringel asserts that the case reports cited by Dr. Chang generally involve adolescents and adults and therefore cannot be extrapolated to an infant. (*Id.* at 7-8.) He asserts that this "fails to account for the well-recognized differences in immune system development between these populations." (*Id*. at 8.)

With respect to timing, Dr. Ringel disagrees with Dr. Chang's assessment that the timing is appropriate to infer Jason's vaccination(s) as the cause of his myocarditis. (Ex. E, pp. 9-10.) Because vaccinations have not been shown to cause myocarditis in toddlers, there is no basis for proposing an appropriate temporal relationship. (*Id*.) The proximity of two events does not in itself prove causality. (*Id*. at 10.)

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).

Prior cases in this program have come to differing conclusions as to whether vaccinations can cause eosinophilic myocarditis. *Compare Matten v. Sec'y of Health & Human Servs*., No. 12-155V, 2021 WL 5768148, at *38 (Fed. Cl. Spec. Mstr. Nov. 2, 2021) (finding that the petitioner's experts offered a sound and reliable medical theory that the flu vaccine can cause eosinophilic and/or hypersensitivity myocarditis and death), *with Bantugan v. Sec'y of Health & Human Servs*., No. 15-721V, 2019 WL 7602581, at *19-20 (Fed. Cl. Spec. Mstr. Dec. 20, 2019) (finding that petitioner failed to offer a reliable medical theory that the flu vaccine can cause hypersensitivity myocarditis). However, prior cases are consistent in stressing that eosinophilic (or hypersensitive) myocarditis is distinct from lymphocytic myocarditis and that there has not been a showing sufficient to implicate vaccinations as a cause of lymphocytic myocarditis. *Yates v. Sec'y of Health & Human Servs*., No. 14-560V, 2020 WL 2313691, at *38 (Fed. Cl. Spec. Mstr. Apr. 16, 2020) (distinguishing eosinophilic myocarditis from lymphocytic myocarditis and noting that hypersensitivity myocarditis "is mainly characterized by eosinophils"), *mot. for rev. den'd*, 150 Fed. Cl. 575 (2020); *Bantugan*, 2019 7602581, at *19 (stating that "the relevant histology [of hypersensitivity myocarditis] is eosinophils, not lymphocytes").

Petitioners' brief engages in extensive discussion of these prior cases while their counsel provides very little by way of direct explanation of their own theory and what evidence on this record supports it. As a result, the brief is difficult to follow. However, based on my review, petitioners raise five separate assertions as significant to *Althen* prong one: (1) a hypersensitivity reaction, comparable to what was proposed by the Thanjan case report, is a viable mechanism of causation regardless of whether eosinophils are evidenced (ECF No. 35, p. 39); (2) the inflammatory effects of vaccination are sufficient to explain a cascade of inflammation affecting the heart muscle (*Id*. at 48); (3) the record of this case also otherwise includes case reports sufficient to implicate the flu vaccine as a cause of myocarditis (*Id*. at 32); (4) as a live virus vaccine, the varicella vaccine can mimic the immune response to infection to cause myocarditis in the same manner as a natural infection. (*Id*.; ECF No. 40, p. 5); and (5) the varicella vaccine is capable of causing a varicella infection that, in turn, can cause myocarditis. (ECF No. 35, pp. 52-53.) Collectively, these points present three distinct theories – that vaccinations can cause hypersensitivity responses leading to (typically eosinophilic) myocarditis; that the inflammatory response to an inactivated or attenuated vaccination can mimic the immune response to infection, albeit in a weaker form, thereby potentially resulting in myocarditis; and, finally, that a varicella vaccine can result in an uncontrolled and disseminated varicella infection that, in turn, can cause a viral myocarditis.

Regarding the first theory, petitioners offer very little to specifically support a causal relationship between vaccination and hypersensitivity/eosinophilic myocarditis in

particular, mainly the Thanjan case report. (Thanjan et al., *supra*, at Ex.10.) However, respondent's experts appear primarily to dispute the applicability of this theory to the instant case more so than arguing against its validity in a broader sense. (Ex. A, pp. 4-5; Ex. C, pp. 4-5; Ex. E, pp. 5, 8-9.) Petitioners similarly characterize the primary question presented as "whether a lymphocytic myocarditis indicates only an infectious process as opposed to a hypersensitivity response which is more consistent with [an] eosinophilic myocarditis." (ECF No. 35, p. 30.) Thus, as presented by the parties, this issue is better addressed under *Althen* prong two. Accordingly, it is not necessary to separately reach the question of whether vaccines can cause hypersensitivity reactions leading to eosinophilic myocarditis as a matter of general causation. The analysis under *Althen* prong two explains why this theory, even if accepted, would not support causation-in-fact in this particular case. Therefore, I will merely assume, but not decide, that the vaccinations at issue can cause eosinophilic myocarditis.

Setting aside hypersensitivity responses, Dr. Chang otherwise opines that "the influenza vaccine, with its viral antigens, can induce an inflammatory response of the host that involved the inflammatory cascade resulting in immune regulatory imbalance and subsequent pericarditis or myocarditis." (Ex. 8, p. 4.) He asserts that the varicella vaccine has "the same type of inflammatory response." (*Id*.) Therefore, "[b]oth of these attenuated vaccines [] have the potential to cause myocarditis as a result of a dysregulated immune system of the body due to it is reaction to the viral antigen in the vaccines in the attenuated form." (*Id*.) This is the second of petitioners' theories as described above.

However, even acknowledging that the vaccine can induce some inflammatory immune response, mere invocation of a vaccine's intended immune response is not in and of itself sufficient to carry petitioner's burden under *Althen* prong one. *See Elvira ex rel. D.E. v. Sec'y of Health & Human Servs.*, No. 17-531V, 2024 WL 4966035, at *20 (Fed. Cl. Spec. Mstr. Nov. 6, 2024); *Vanore v. Sec'y of Health & Human Servs.*, No. 21-0870V, 2024 WL 3200287, at *18 (Fed. Cl. Spec. Mstr. May 31, 2024); *Kalajdzic v. Sec'y of Health & Human Servs.*, No. 17-792V, 2022 WL 2678877, at *23 (Fed. Cl. Spec. Mstr. June 17, 2022), *mot. for rev. den'd*, No. 17-792V, 2024 WL 4524777 (Fed. Cl. Oct. 18, 2024), *aff'd*, No. 2023-1321, 2024 WL 3064398 (Fed. Cir. June 20, 2024); *Cordova v. Sec'y of Health & Human Servs.*, No. 17-1282V, 2021 WL 3285367, at *17 (Fed. Cl. Spec. Mstr. June 23, 2021). There must be some additional evidence linking the vaccine's immune response to the pathology of petitioner's actual condition. Here, however, Dr. Chang does not explain either the cascade or immune dysregulation he is positing. Nor did he provide any direct citation for this proposition or otherwise rely on any materials sufficient to explain how the immune response to vaccination could commence the immune response(s) underlying myocarditis. Dr. Chang summarily relied on the notion that "[t]he autopsy of the heart with microbiology showed an acute inflammatory process consistent with vaccination-related myocarditis" (Ex. 8, p. 5), but the presence of an acute inflammatory process does not, by itself, credibly point to the cause of the condition. Dr. Chang explained that myocarditis is, by definition, inflammation of the heart (Ex. 8, p. 4) and Dr. Ringel explained that viral illness leading

to viral infection and inflammation of the heart is a "recognized pathway" to myocarditis. (Ex. A, p. 5.)

Dr. Chang also cited several case reports. Apart from the Thanjan case report, which proposed a hypersensitivity response leading to myocarditis with "prominent" eosinophils (Ex. 10), petitioners filed three case reports of myocarditis occurring after a flu vaccination. (Exs. 22, 23, 31.) In 2019, Kim, et al., reported a case of fulminant myocarditis occurring three days after a flu vaccination. (Ex. 22, p. 1.) Like Dr. Chang, they offered the hypothesis that an inflammatory cascade may have played a role in the development of cardiovascular compromise, but acknowledged that this hypothetical mechanism cannot be proven. (*Id*. at 4.) Thus, the authors explained that a causal relationship to vaccination "is still controversial." (*Id*.) In 2020, Nagano, et al., reported a patient presenting with cardiopulmonary arrest and fulminant myocarditis 7 days after a flu vaccination. (Ex. 31, p. 1.) The authors observed that anaphylactic reaction leading to eosinophilic myocarditis has been reported post-vaccination but noted that their patient did not have eosinophils. (*Id.* at 3.) The authors nonetheless "speculate[d]" that their case was due to a vaccine reaction, but noted further evidence was needed to demonstrate causality. (*Id*.) Finally, in 2022, Nakamura, et al., reported a case of lymphocytic-predominant myocarditis in an elderly patient occurring two days after receiving the flu vaccination. (Ex. 23, p. 1.) The authors contrasted their patient with the prior Kim and Nagano subjects because he was elderly, had comorbidities, and had a mild and self-limited course of myocarditis in contrast to a fulminant myocarditis. (*Id*. at 5.) The authors noted that the mechanism underlying post-vaccination myocarditis remains unknown and further stressed that "[t]he causal relationship between influenza vaccination and acute myocarditis relies heavily on the temporal relationship between the timing of vaccination and the onset of acute heart failure (two days after vaccination in our case)." (*Id.* at 5-6.)

I have reviewed the case reports cited by Dr. Chang; however, without more, I do not find that they are sufficient to meet petitioners' burden of proof under *Althen* prong one. Petitioners in this program often highlight the usefulness of case reports in cases of rare diseases or unusual occurrences. *E.g.*, *Patton v. Sec'y of Health & Human Servs.*, 157 Fed. Cl. 159, 166-68 (2021). However, case reports "do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value," even though they are not entirely devoid of evidentiary value. *Paluck v. Sec'y of Health & Human Servs.*, 104 Fed. Cl. 457, 475 (2012) (quoting *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011)); *see also Crutchfield v. Sec'y of Health & Human Servs.*, No. 09-0039V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) ("[S]ingle case reports of Disease X occurring after Factor Y . . . do not offer strong evidence that the *temporal* relationship is a *causal* one—the temporal relationship could be pure random chance."), ***aff'd***, 125 Fed. Cl. 251 (2014). Without entirely discounting the value of case reports, respondent is persuasive in contending that the particular case reports filed in this case are equivocal in their conclusions. Based on my review of the case reports at issue, I conclude that they are entitled to little weight.

Petitioners also more specifically suggest that: (1) natural infection with influenza and varicella viruses can cause myocarditis (ECF No. 35, p. 48; Ex. 27, p. 4 (citing Exs. 13-17)); (2) the Institute of Medicine ("IOM")[15] recognizes that the ability of a wild-virus to cause a condition is some evidence to suggest that a vaccine can cause the same condition (Ex. 33); and (3) a paper by Slifka, et al., in particular shows that the varicella vaccine, as an attenuated live viral vaccine, can "act like an attenuated infection." (Slifka et al., *supra*, at Ex. 24;. ECF No. 40, p. 5.) While the first of these propositions is uncontroversial, respondent is persuasive in arguing that petitioners overstate the evidence pertaining to the second and third. (ECF No. 37, pp.16-17.)

While the IOM does state that the consequence of a wild-virus are *some* evidence regarding vaccine causation, they stress that "[e]vidence consisting only of parallels with the natural infections is never sufficient to merit a conclusion other than the evidence is inadequate to accept or reject a causal relationship." (Ex. 33, p. 42, n. 6.) In that regard, respondent stresses language from Slifka, et al., that "[m]ost live attenuated vaccines have been selected on the basis that they replicate less efficiently in their intended host, resulting in reduced pathogenicity and an improved safety profile." (ECF No. 37, p. 17 (quoting Ex. 24, p. 6).) Thus, Dr. Jameson persuasively explains, on respondent's behalf, that the consequences of live, virulent influenza or varicella infections are not indicative of the consequences of the immune response to the vaccinations for these same pathogens, which is comparatively mild. (Ex. C, p. 4.) The flu vaccine, in particular, cannot reproduce because it is inactivated. (*Id.*)

Finally, regarding the third theory, petitioners also contend that, more than simply mimicking the immune response to infection, the varicella vaccine can actually in some instances result in an active varicella infection. (ECF No. 35, pp. 52-53.) Thus, because infections generally, and varicella infections specifically, can cause myocarditis, this is a viable chain of events that could explain how a varicella vaccine could ultimately be the initiating cause of a myocarditis. (*Id*.) This contention is

[15] The Institute of Medicine (known as the National Academy of Medicine since 2015) is the medical arm of the National Academy of Sciences. The National Academy of Sciences ("NAS") was created by Congress in 1863 to be an advisor to the federal government on scientific and technical matters (*see* An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863)), and the Institute of Medicine is an offshoot of the NAS established in 1970 to provide advice concerning medical issues. When Congress enacted the Vaccine Act in 1986, it directed that the IOM conduct studies concerning potential causal relationships between vaccines and illnesses. *See* § 300aa-1 note. Special masters have previously observed that the IOM employs a standard for finding causation that is higher than what is required by petitioner's burden of proof. *E.g.*, *Raymo v. Sec'y of Health & Human Servs.*, No. 11-654V, 2014 WL 1092274, at *21 n.39 (Fed. Cl. Spec. Mstr. Feb. 24, 2014). Accordingly, IOM reports and findings are typically approached with caution and generally not treated as dispositive. *Porter v. Sec'y of Health & Human Servs.*, 663 F.3d 1242, 1252 (Fed. Cir. 2011) (noting the special master's comment that "IOM reports are favored, although not dispositive, in the Vaccine Act Program," then affirming special master's decision). However, numerous prior cases have demonstrated that special masters may account for IOM findings in reaching their decisions. In this case, petitioners have filed the IOM's complete 2012 report Adverse Effects of Vaccines: Evidence and Causality with their reply brief and requested judicial notice of the document. (ECF No. 40, p. 5, n.1; Kathleen Stratton et al., Committee to Review Adverse Effects of Vaccines, Institute of Medicine, eds., *Adverse Effects of Vaccines: Evidence and Causality,* Washington (DC): National Academies Press (2012) (Ex. 33).)

preponderantly supported. While petitioners overstate as a general matter the degree to which the intended effects of vaccinations mimic infections to potentially injurious effect, the IOM has specifically concluded that there is evidence that convincingly supports a causal relationship between varicella vaccination and disseminated vaccine-strain varicella infection, including infections with additional complications such as pneumonia, meningitis, and hepatitis. (Ex. 33, pp. 278, 285.) Importantly, however, while the committee concluded that disseminated viral infection could be caused by the varicella vaccination with or without demonstrated immunodeficiency, the causal conclusion with respect to disseminated infection leading to other organ involvement was limited to individuals with demonstrated immunodeficiency. (*Id*.) Although the IOM did not specifically examine myocarditis, it is not controversial on this record that the varicella virus can cause myocarditis. (Ex. A, p. 4 (Dr. Ringel noting that "[a]lmost every viral pathogen has at some point been documented as a cause of myocarditis."); Ex. A, Tab 1, p. 5 (literature filed by respondent identifying varicella among a list of viruses causative of myocarditis).)

Considering Dr. Chang's opinion and the arguments presented by petitioners, both singularly and collectively, *Althen* prong one is resolved as follows:

- It is assumed, but not decided, that vaccinations can cause hypersensitivity responses leading to eosinophilic myocarditis; and

- Petitioners have preponderantly established that, as a live attenuated vaccine, the varicella vaccine can sometimes result in uncontrolled, disseminated varicella infection. In immunocompromised individuals, such a post-vaccinal varicella infection can, in turn, lead to complications affecting other organs, likely including myocarditis; and

- Petitioners have not met their preponderant burden of proof under *Althen* prong one with respect to any other theory of causation.

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 v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 745 n.67 (2009) ("[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).

The certified cause of Jason's death is "myocarditis, of probable viral etiology, not otherwise specified." (Ex. 4, p. 1.) And, significantly, this assessment was rendered by a medical examiner with full awareness of Jason's history of vaccination, his post-vaccination fever, and his symptoms of decreased appetite and vomiting. (Ex. 5, p. 4.) Thus, the primary source medical opinion available in the medical records does not favor vaccine causation. 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. *Pelton v. Sec'y of Health & Human Servs.*, No. 14-674V, 2017 WL 1101767, at *13 (Fed. Cl. Spec. Mstr. Feb. 27, 2017); *Bohn ex rel. G.B. v. Sec'y of Health & Human Servs*., No. 16-265V, 2021 WL 4302367, at *11 (Fed. Cl. Spec. Mstr. Aug. 23, 2021). Of note, the medical examiner in this case does not appear to have been a medical doctor. (*See* Ex. 5, p. 1 (signed by Vincent J. Moylan, Jr., MS, PA (ASCP)).) However, the Vaccine Act specifies, without limitation, 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). Moreover, the medical examiner's conclusion is also ratified by respondent's expert, Dr. Ringel, who is a clinician and board diplomate of the American Board of Pediatrics (sub-boarded in both pediatric cardiology and pediatric critical care medicine) as well as the National Board of Medical Examiners. (Ex. A, pp. 5-6; Ex. B, p. 7.)

The conclusion that Jason's myocarditis was of probable viral etiology is ultimately supported by three converging lines of evidence: (1) the autopsy finding of mononuclear inflammatory infiltrates, largely lymphocytes, rather than eosinophils (Ex. 4, pp. 3-4); (2) possible signs of viral illness, such as of loss of appetite and vomiting prior to death (Ex. 3, p. 3); and (3) the fact that both parties' experts agree that viral infection is the most common cause of myocarditis (Ex. 19, p. 4; Ex. A, p. 4; Ex. C, p. 3).[16] Although further testing that could have confirmed viral illness was not completed

[16] Further to these three points, respondent notes that Jason's autopsy found "pulmonary parenchyma with congestion, patchy alveolar edema, focally increased alveolar macrophages, focal minimal peri-bronchial chronic inflammatory infiltrate and focal alveolar expansion." (ECF No. 37, p. 2 (citing Ex. 4, p. 4).) According to Dr. Jameson's recitation of the relevant history, this is consistent with a recent lung infection. (Ex. C, p. 2.) However, Dr. Jameson is not a medical doctor and accordingly disclaimed offering any commentary on the clinical aspect of this case in his opinion. (*Id*.) Respondent's other expert, Dr. Ringel, did not address or rely upon the pulmonary findings in either of his reports. (Exs. A, E.) The autopsy report is silent with respect to whether this specific finding supported the ultimate conclusion that a viral etiology was probable. (Ex. 4, p. 4.) Due to the lack of any clinical opinion clearly supporting the significance of this finding, it is given little weight.

(*see* Ex. C, p. 3), these lines of evidence suggest viral illness as the cause of Jason's myocarditis, even if not definitively. *Accord Winkler*, 88 F.4th at 963 (explaining that "[e]specially given that lack of dispute regarding *C. jejuni* as a possible source of injury, evaluating the strength of Winkler's prima facie case did not require an explicit finding that Winkler actually suffered from a *C. jejuni* infection. The Special Master was free to consider evidence relating to whether or not Winkler suffered from a *C. jejuni* infection, as well as the likelihood that said infection triggered Winkler's GBS. Such contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law.") Petitioners are not persuasive in seeking to undermine these points. Moreover, in contrast to the above, there is no clinical evidence to suggest that Jason's myocarditis was related to his vaccinations.

Dr. Chang contends that Jason's loss of appetite and vomiting cannot necessarily be attributed to an unspecified viral illness when these same symptoms can otherwise be attributed either to a vaccine reaction or the myocarditis itself. (Ex. 19, p. 7; Ex. A, Tab 3, p. 4; *see also* Exs. 25 (Flulaval package insert), 26 (Varivax package insert).) Dr. Chang is persuasive in noting that Jason's loss of appetite and vomiting might be attributable to the myocarditis itself, meaning they would not separately evidence the presence of a viral infection. (Ex. 19, p. 7; Ex. A, Tab 3, p. 4.) However, this observation similarly undercuts any suspicion that these outward clinical symptoms are indicative of a vaccine reaction. Instead, it suggests these symptoms are simply uninformative as to the initial cause of Jason's myocarditis. Moreover, this observation does not overcome the additional points of evidence noted above, particularly the presence of predominantly lymphocytic infiltrate. Even if Jason's symptoms are typical prodromal symptoms of myocarditis itself, lymphocytic myocarditis is most often caused by viral infection. (Ex. A, p. 5.)

Petitioners further argue that the fact that Jason's autopsy showed primarily lymphocytic infiltration in the heart muscle does not exclude the possibility that it resulted from a hypersensitivity reaction rather than a viral infection. (Ex. 19, p. 6.) However, the predominance of lymphocytes in the heart muscle *favors* a viral etiology for Jason's myocarditis as explained by both Dr. Ringel and Dr. Jameson. (Ex. A, pp. 5-6; Ex. C, pp. 4-5; Ex. E, pp. 8-9.) In seeking to complicate that picture, petitioners ultimately offer only speculation. Dr. Chang is not persuasive in contending that, despite eosinophils being "strongly" suggestive of a hypersensitivity response, Jason's myocarditis can *reasonably* be attributed to a hypersensitivity reaction in the complete absence of eosinophils in the heart muscle. (Ex. 19, p. 6.)

First, Dr. Chang cites two sources for the proposition that, as a general matter, the absence of eosinophils is not necessarily meaningful as an indicator of a hypersensitivity myocarditis: a study by Burke, et al., and a case report by Nagano, et al. (Ex. 19, p. 6; *see also* Exs. 20-21, 31-32.) Burke, et al., examined 69 cases of hypersensitivity myocarditis, noting that only 20% involved primarily eosinophilic infiltrate. (Burke et al., supra, at Exs. 20, 32.) As noted by Dr. Ringel, however, the authors explained that all 69 cases qualified as hypersensitivity myocarditis because

they had already been screened for the presence of eosinophils. (Ex. 32, pp. 1-2; Ex. E, p. 8.) Thus, while Dr. Chang may be correct that Burke otherwise showed a spectrum of histopathology among hypersensitivity myocarditis patients, it is not accurate to conclude that Burke supports the proposition that eosinophils are diagnostically unimportant or may be entirely absent. Nor is the Nagano case report helpful on this point. (*See* Exs. 21, 31.) The Nagano patient was specifically diagnosed with lymphocytic myocarditis rather than hypersensitivity myocarditis. (Ex. 31, p. 2.) Although the case report noted that prior reports of post-vaccinal myocarditis hypothesized a hypersensitivity mechanism, nothing in the case reports purports to revisit the patient's lymphocytic myocarditis diagnosis in light of that observation. (*Id.* at 2-3.) Thus, even while Dr. Ringel acknowledges that no test is 100% sensitive (Ex. E, p. 7), Dr. Chang is not persuasive in seeking to minimize the importance of eosinophils in determining whether a patient can reasonably be diagnosed with a hypersensitivity-related myocarditis. (Ex. 19, p. 6.)

Second, Dr. Chang notes that the autopsy report in this particular case states only that the infiltrate was "largely," as opposed to exclusively, lymphocytic (Ex. 19, p. 6); however, the same medical examiner that wrote that finding also diagnosed myocarditis of probable viral etiology. (Ex. 4, p. 1.) Accordingly, while Dr. Chang is correct that the autopsy report is not definitively phrased, this is not strong evidence to suggest that a significant finding of eosinophils went unremarked upon. This is not ultimately a question of what degree of eosinophilic involvement can evidence a hypersensitivity reaction. There is a complete absence of any available clinical data on this record to support the invocation of a hypersensitivity response affecting the heart,[17] and petitioners have not substantiated that there is any reason to actually suspect the presence of undetected eosinophils in this case.

Alternatively, in an attempt to reconcile petitioners' allegation with the presence of lymphocytic infiltrate, Dr. Chang proposes that Jason's varicella vaccination may

[17] In their briefing, petitioners for the first time raise a question with respect to the fact that the autopsy report did find eosinophils in the gastrointestinal tract. (ECF No. 35, p. 30; ECF No. 40, p. 4.) Petitioners cite the prior *Matten* decision as support for the notion that this is a meaningful finding and stress that respondent's experts did not address this fact. (ECF No. 35, pp. 25, 46-47.) Apart from discussion of the *Matten* decision, which is not binding and was based on a different evidentiary record, petitioners never explicitly explain how eosinophils solely in the gastrointestinal tract support their claim; however, they assert that "Dr. Chang opined that Gaskin, Jr., suffered from a hypersensitivity myocarditis and that not all hypersensitivity cases involve eosinophils, although in this case, eosinophils did exist." (ECF No. 35, p. 47.) This is a misleading representation of the record evidence. In the course of three expert reports, Dr. Chang never relied on the presence of eosinophils in the gastrointestinal tract to support his opinion. In fact, his summary of the autopsy report did not even remark on that finding. (*E.g.* Ex. 8, p. 3.) Moreover, when Dr. Chang was challenged by respondent's experts regarding the lack of eosinophils, he opined only that "the absence of eosinophils in the heart muscle examination does not rule out a hypersensitivity." (Ex. 19, p. 6 (emphasis omitted).) He never asserted that eosinophils elsewhere in the body would be causally relevant. Accordingly, petitioners' assertion that eosinophils in the gastrointestinal tract may be causally significant to the etiology of Jason's myocarditis is not supported by any medical opinion. Accordingly, as with respondent's reliance on the pulmonary findings within the autopsy report (*see supra* note 16), the fact that the autopsy showed eosinophils in the gastrointestinal tract is given very little weight.

have led to a mild varicella infection that in turn caused the myocarditis. (Ex. 19, p. 7.) However, here too, there is an absence of evidence to support the idea that Jason suffered a varicella infection in particular. As explained above, Jason's certified cause of death was myocarditis "of probable viral etiology, not otherwise specified." (Ex. 4, p. 1.) This is, in large part, because, as respondent stressed, postmortem testing to determine what virus may be at issue was not conducted. (Ex. C, p. 3.) Moreover, the varicella virus causes chicken pox, which has a characteristic presentation (Ex. 33, p. 268); however, respondent stresses that Jason had no specific signs of chicken pox. (ECF No. 37, p. 22.) Petitioners argue that Jason could have been sick with a varicella infection manifesting with only constitutional symptoms, such as fever, without yet having developed the characteristic rash. (ECF No. 40, p. 6.) In that regard, one of the case reports filed by petitioners does seem to confirm that cardiac complications due to varicella infection can pre-date the onset of the skin lesions associated with chicken pox. (Ex. 16, pp. 1-2 (onset of rash occurring just under 2 days after onset of cardiac complaints).) However, even if possible, this is still speculative insofar as these are non-specific symptoms and petitioners' own expert urges that Jason's pre-mortem symptoms may have been signs of his myocarditis regardless of its cause. (Ex. 19, p. 7.) Further still, post-vaccination disseminated varicella infection capable of resulting in other organ involvement has only been evidenced on this record among immunocompromised individuals. However, there is no evidence available to suggest that Jason was immunocompromised.

In any event, even setting aside all of the above, the time between Jason's varicella vaccination and his death casts doubt on petitioners' argument as it is likely too short for a post-vaccination infection to have taken hold. Jason was exposed to varicella via his vaccination on February 7 and died on February 13, leaving a maximum of six days for any infection to incubate. If one takes his prior symptoms of fever, loss of appetite, and vomiting as symptoms of a varicella infection, as petitioners seem to agree one should in this context, then the potential incubation period is even shorter. However, as cited by petitioners, the IOM explained that in general it takes 10-21 days from initial exposure for a natural varicella infection to incubate into illness. (ECF No. 40, p. 6; Ex. 33, p. 268.) Even though constitutional symptoms such as fever or malaise may precede the hallmark rash of chicken pox, this generally only occurs up to about two days prior to the rash, suggesting a minimum of about eight days for any symptoms of any kind to develop. (Ex. 33, p. 268.) Patients are not considered contagious until that time. (*Id*.) Further to that, the IOM examined direct viral infection resulting from the varicella vaccine and concluded that this process likewise takes eight or more days to result in a disseminated infection leading solely to a rash and ten or more days to result in a disseminated infection affecting other organs, including complications such as pneumonia, meningitis, or hepatitis. (*Id*. at 278, 285.) The case reports of myocarditis following varicella infection cited by petitioners are not to the contrary. In one, the date of viral exposure is not indicated. (Ex. 16.) In the other, the exposure to the varicella virus occurred "a few weeks" prior to onset of symptoms. (Ex. 17.)

Ultimately, Dr. Chang proposes that the vaccines are the more likely cause of Jason's myocarditis because the timing is reasonable and because it is "nonspurious" in

the sense that no other cause is evident, including a viral illness, in his opinion. (Ex. 19, p. 8.) However, this opinion of vaccine causation is lacking affirmative factual support. The relevant testing for viral illness is largely absent rather than negative. (*See* Ex. C, p. 3.) And, even if the absence of eosinophils is only probabilistic rather than definitive, the available findings – lymphocytes, but not eosinophils – still more strongly favor a viral rather than post-vaccinal etiology. However, having found a viral cause most likely, there is no evidence to suggest that the viral illness in question was varicella. Therefore, petitioners are unpersuasive in arguing that the burden of proof should have shifted to respondent to demonstrate the presence of a causal factor unrelated to vaccination. (ECF No. 35, p. 58; ECF No. 40, p. 2; *accord Winkler*, 88 F.4th at 963.) The Federal Circuit has explained that "[a]lthough probative, neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149).

Accordingly, petitioners have not preponderantly demonstrated under *Althen* prong two that there is a logical sequence of cause-and-effect supporting vaccine causation of Jason's myocarditis and death.

c. Proximate temporal relationship between vaccination and significant aggravation (*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).

The case reports filed by petitioners identify post-influenza vaccination myocarditis occurring between one to five days post-vaccination. (Ex. 22, p. 2; Ex. 23, p. 1; Ex. 31, p. 1.) Jason's death would be consistent with this timeframe in that he died six days post-vaccination and may have had symptoms of his myocarditis (loss of appetite and vomiting) in the days prior. (Ex. 1, p. 287; Ex. 3, p. 3.) However, for the reasons discussed under *Althen* prong one, petitioners were not persuasive in offering these case reports as evidence of any direct relationship between post-vaccination inflammation and myocarditis, as suggested by Dr. Chang. The case reports themselves are inadequate to evidence any other theory of causation. Thus, these

case reports do not help petitioners meet their burden of proof under *Althen* prong three.

The *Althen* prong one analysis above assumed, without deciding, that vaccines can cause hypersensitivity responses leading to eosinophilic myocarditis. In that regard, the subject of the Thanjan case report had onset of symptoms of such a presentation within one day of vaccination, suggesting a more rapid onset than was present in this case. (Ex. 10, p. 3.) Nonetheless, Dr. Chang explains that even acute fulminant myocarditis can include a decline spanning days. (Ex. 8, p. 5.) Accordingly, it may be possible that petitioners could have satisfied *Althen* prong three with respect to a hypersensitivity response leading to eosinophilic myocarditis. However, as explained under *Althen* prong two, there is not preponderant evidence that Jason suffered this form of myocarditis, rendering this potential temporal relationship moot. Accordingly, the Thanjan case report does not help petitioners meet their burden of proof under *Althen* prong three.

Finally, the *Althen* prong one analysis above also concluded that there is preponderant evidence that the varicella vaccine can cause a disseminated varicella infection and, further, that a varicella infection can, in turn, cause myocarditis. However, for the reasons discussed under *Althen* prong two, the timeframe between Jason's varicella vaccination and his death does not support an appropriate temporal relationship from which a causal inference can be made. Thus, petitioners cannot meet their burden of proof under *Althen* prong three relative to this theory based on the facts of this case. In any event, as additionally explained under *Althen* prong two, there is not preponderant evidence that Jason actually suffered a varicella infection.

Accordingly, petitioners have not met their burden under *Althen* prong three.

VI. Conclusion

As noted at the outset, I offer my sincerest condolences to petitioners on their loss. However, for all of the reasons described above, there is not preponderant evidence that Jason's fatal myocarditis was caused by his vaccination(s). Therefore, this case is dismissed.[18]

IT IS SO ORDERED.

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

[18] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.