In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 20-1455V
Filed: May 23, 2024

JULIA HUNT,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Amy A. Senerth, Muller Brazil, LLP, Dresher, PA, for petitioner.
Meghan Murphy, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On October 23, 2020, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2012),[2] alleging that she suffered dermatomyositis following an influenza ("flu") vaccination that she received on November 14, 2017. (ECF No. 1.) Petitioner alleged her injury was caused-in-fact by her vaccination. (*Id*. at 4.) For the reasons set forth below, I conclude that petitioner is *not* entitled to compensation and the petition is dismissed.

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

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

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), (B); § 300 aa-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-13(a)(1)(B); § 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, 1524-25 (Fed. Cir. 1991). Because petitioner's injury is not listed on the Vaccine Injury Table, petitioner must satisfy this burden of proof.

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; *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. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, 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. However, that expert's opinion must be based upon "sound and reliable" scientific explanation. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Knudsen ex rel. Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548-49 (Fed. Cir. 1994)). The *Althen* court also indicated that, in finding causation, a Program factfinder 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." 418 F.3d at 1280.

II. Procedural History

Initially petitioner filed medical records and an affidavit marked as Exhibits 1-12. (ECF Nos. 1, 16.) She filed her Statement of Completion in January of 2021. (ECF No. 17.) After completion of a Pre-Assignment Review, the case was assigned to the undersigned in February of 2021. (ECF No. 19.) Respondent filed his Rule 4 Report recommending against compensation in June of 2021. (ECF No. 24.) Respondent contended that the medical records were inadequate to support petitioner's burden of proof under the *Althen* test. (*Id*. at 11-14.)

Subsequently, petitioner filed an expert report by rheumatologist/immunologist M. Eric Gershwin, M.D., and accompanying literature. (ECF No. 25; Exs. 13-54.) Respondent filed responsive reports by rheumatologist Christopher Mecoli, M.D., and immunologist James Moy, M.D. (ECF Nos. 31-32; Exs. A-D.) Thereafter, the parties confirmed during a status conference held June 24, 2022, that the case is ripe for resolution on the written record. (ECF No. 34.)

Petitioner filed her motion for a ruling on the written record in December of 2022. (ECF No. 38.) Respondent filed his response in May of 2023 and petitioner filed her reply in July of 2023. (ECF Nos. 43-45.)

In light of the above, I have determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve entitlement on the

existing record. *See* Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *see also Kreizenbeck ex rel. C.J.K. v. Sec'y of Health & Human Servs*., 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"). Accordingly, this matter is now ripe for resolution.

III. Factual History

The details of petitioner's medical history are not disputed. Accordingly, only an abbreviated history is provided. However, I have reviewed the medical records in full.

Petitioner received the flu vaccination at issue during a routine exam on November 14, 2017. (Ex. 1, p. 2.) At that time she had no prior history of dermatomyositis, though she did have a history of thyroid nodules, vitamin D deficiency, prediabetes, and elevated cholesterol. (Ex. 4, pp. 74-75, 86-87.) Six days post-vaccination she presented to her endocrinologist for her thyroid nodules and reported no symptoms of joint or muscle pain or weakness. (Ex. 5, pp. 476-79.)

On December 5, 2017, she presented for care with joint and muscle pain, which she indicated had begun the day she received the flu vaccine. (Ex. 4, p. 59.) Petitioner did not have fever or chills at the time of this medical encounter. (*Id.*) She was diagnosed with muscle pain and prescribed naprosyn. (*Id*. at 60.)

On December 15, 2017, she presented to the emergency department with complaints of shortness of breath and swelling of her face and upper extremities, as well as malaise and dizziness. (Ex. 3, p. 12.) She indicated her symptoms had been present for three weeks. (*Id*.) She also stated that she "started having joint aches, fevers, chills, night sweats, facial swelling around her eyes, hand swelling and arm weakness, leg swelling and thigh weakness [two] days after [receiving her flu vaccination]. She said she thought it was a reaction to the flu shot, so when it didn't go away after [two] weeks, she went to go see her doctor. Her doctor prescribed her naproxen for a week, which did improve her symptoms except for thigh weakness." (*Id*. at 83.) Petitioner was admitted with a differential diagnosis of lupus flare versus polymyositis versus dermatomyositis and rheumatologist was consulted. (*Id*. at 14.)

Petitioner had elevated CK, AST, and ALT, which was concerning for inflammatory myositis. (Ex. 3, p. 16.) She was also positive for ANA and Anti-Jo-1 antibodies. (*Id*.) An MRI of petitioner's bilateral thighs showed diffuse myositis and a biopsy showed "[e]xtensive myofiber necrosis and regeneration, focal limited inflammation and a positive HLA Class 1 immunohistochemistry study, most consistent with an immune-mediated (inflammatory) myopathy." (Ex. 2, p. 30.) During hospitalization, IV fluids improved her symptoms and her CK trended downward. (Ex. 3, p.16.) At discharge she was instructed to continue on prednisone at 60 mg and to follow up with rheumatology. (*Id*.)

Petitioner then saw rheumatologist David Ozeri, M.D., on January 8, 2019. (Ex. 5, pp. 49-53.)[3] It was noted that petitioner had self-tapered her prednisone to 40 mg. (Ex. 5, p. 51.) Petitioner reported a history of symptoms arising post-vaccination and it was noted that "[s]he is suspicious that the flu vaccine triggered her polymyositis." (*Id*.) Petitioner was doing well with "no significant proximal muscle weakness" and Dr. Ozeri agreed to further taper prednisone to 30mg in light of petitioner's anxiety regarding insomnia from the prednisone. (*Id*. at 52-53.) Dr. Ozeri's impression was biopsy-proven myositis consistent with polymyositis. (*Id*. at 52.) He instructed petitioner to follow up with her primary care provider to rule out age-appropriate malignancies given high incidences of paraneoplastic immune mediated myopathy. (*Id*. at 52-53.)

Petitioner continued to follow up with Dr. Ozeri. (Ex. 5, pp. 83-88, 133-38, Ex. 12, pp. 4-8.) As of March 12, 2018, petitioner's lab work showed resolution of her myositis. (Ex. 5, p. 105.) Dr. Ozeri concluded that petitioner had been suffering Jo1 idiopathic inflammatory myopathy ("IIM") and remarked that it "may have been triggered by a flu vaccine suggesting possible autoimmune/autoinflammatory disease induced by adjuvants." (*Id*.) By June 4, 2018, petitioner had been tapered down to 4 mg of prednisone and was still doing well. (Ex. 12, p. 4.) Dr. Ozeri concluded she had "recovered fully." (*Id*. at 7.) He noted that "[m]y overall impression is that she has autoimmune syndrome induced by adjuvants . . . [and] she has been counseled to avoid flu vaccine in future." (*Id*.) His ultimate diagnoses were dermatomyositis and reaction to influenza immunization. (*Id*. at 7.)

Petitioner would subsequently see two different rheumatologists in management of her condition who diagnosed CPK with polymyositis (Ex. 5, p. 353) and inflammatory myositis (Ex. 2, p. 7). However, the initiating cause of her condition was not revisited.

IV. Expert Reports

a. Petitioner's rheumatology/immunology expert, M. Eric Gershwin, M.D.[4]

According to Dr. Gershwin, classification of inflammatory muscle diseases, including dermatomyositis, is "crude and enigmatic." (Ex. 13, p. 3.) "Causes leading to inflammation in idiopathic inflammatory myopathies (IIMs) are enigmatic, but autoimmune mechanisms are strongly involved with an inappropriate activation of the

[3] She had other encounters in the interim. (Ex. 4, pp. 44-49, 50-54.)

[4] Dr. Gershwin is currently a distinguished professor of medicine and the Jack and Donald Chia Professor of Medicine in the Rheumatology/Allergy and Clinical Immunology division of the University of California at Davis, where he previously served as chairperson of the Graduate Group in Immunology. (Ex. 14, p. 1.) Dr. Gershwin received his bachelor's degree from Syracuse University and his master's degree from the Centre for Astrophysics and Supercomputing. (*Id*.) He received his Doctor of Medicine from Stanford University and is currently licensed to practice medicine in California. (*Id.* at 1-2.) Dr. Gershwin is board certified in Internal Medicine with a subspecialty in Rheumatology and Allergy and Clinical Immunology. (*Id.* at 2.) In addition, he has published 72 books and monographs, 1039 experimental papers, 164 book chapters, 277 reviews, 40 guest editorials and book reviews, and five letters to the editors. (*Id.* at 8-138.)

innate immune system leading to the secondary dysregulation of the adaptive immune response that is now considered to be a central pathogenic feature of IIMs." (*Id*. (citing Thorsten Hornung & Joerg Wenzel, *Innate Immune-Response Mechanisms in Dermatomyositis: An Update on Pathogenesis, Diagnosis and Treatment,* 74 DRUGS 981 (2014) (Ex. 15); Sahil Khanna & Ann M. Reed, *Immunopathogenesis of Juvenile Dermatomyositis,* 41 MUSCLE NERVE 581 (2010) (Ex. 16); Kanneboyina Nagaraju & Ingrid E. Lundberg, *Polymyositis and Dermatomyositis: Pathophysiology,* 37 RHEUMATIC DISEASE CLINICS N. AM. 159 (2011) (Ex. 17)).) Dr. Gershwin indicates that dermatomyositis is a rare disease – too rare to be detected by epidemiologic study – that requires both a genetic predisposition and environmental factors to develop. (*Id*. at 2-3.)

Dermatomyositis is a disease of excessive activation of the innate immune system with strong expression of interferon (IFN)-regulated proteins directed to muscle and skin. (Ex. 13, p. 3 (citing J. Wenzel et al., *Evidence for a Role of Type I Interferons in the Pathogenesis of Dermatomyositis,* 156 BRIT. J. DERMATOLOGY 462 (2005) (Ex. 22); Steven A. Greenberg et al., *Interferon-α/β-Mediated Innate Immune Mechanisms in Dermatomyositis,* 57 ANNALS NEUROLOGY 664 (2005) (Ex. 23)).) Skin biopsies have shown mature CD4+ T cells producing IL-2, IFN-γ, and IL-4. (*Id*. (citing M. Caproni et al., *Infiltrating cells, Related Cytokines and Chemokine Receptors in Lesional Skin of Patients with Dermatomyositis,* 151 BRIT. J. DERMATOLOGY 784 (2004) (Ex. 26)).) Intermediate-sized vessels are affected by a compliment cascade leading to microangiopathy. (*Id*. (citing Marinos C. Dalakas, *Pathogenesis and Therapies of Immune-Mediated Myopathies,* 11 AUTOIMMUNITY REVS. 203 (2012) (Ex. 28)).) Antibodies directed against endothelial cells ultimately cause swelling, vacuolization, capillary necrosis, perivascular inflammation, ischemia, and ultimately destruction of muscle fiber. (*Id*. (citing Marinos C. Dalakas & Reinhard Hohlfeld, *Polymyositis and Dermatomyositis,* 362 LANCET 971 (2003) (Ex. 30)).) In this process, cytokines and chemokines released following the compliment activation recruit vascular cell adhesion molecules and activated T cells to the site of inflammation. (*Id*. at 3-4 (citing Dalakas & Hohlfeld, *supra,* at Ex. 30).) "Key cytokines in IIM are produced by Th1 (i.e. IFN-γ, IL-2, IL-12, TNF-α), TH2 (i.e. IL-4 and IL-13), Th17 (IL-17, IL-22, IL-23, IL-6), and Treg (i.e. IL-10, TGF-β) cells. IL-1 family cytokines such as Il-1 α and β are also altered in IIM sera and tissues." (*Id*. at 4 (citing E. M. Moran & F. L. Mastaglia, *Cytokines in Immune-Mediated Inflammatory Myopathies: Cellular Sources, Multiple Actions and Therapeutic Implications,* 178 BRIT. SOC'Y IMMUNOLOGY, CLINICAL & EXPERIMENTAL IMMUNOLOGY 405 (2014) (Ex. 33)).)

Regarding environmental factors, infectious agents are the most commonly associated with the induction of dermatomyositis. (Ex. 13, p. 4.) Dr. Gershwin suggests there are several proposed mechanisms. (*Id.* at 4-5.) First, the infectious agent could cause self-proteins to become novel neo-antigens. (*Id.* at 4.) Second, the infectious agent may expose an otherwise sequestered antigen to an immune response. (*Id.*) Third, there may be cross-reaction between the infectious agent and muscle antigens or other low affinity autoreactive cells. (*Id.* at 4-5.) Additionally, an aberrant cytokine response may facilitate a loss of tolerance in a genetically susceptible host. (*Id.* at 5.)

He stresses that "[a] vaccination is designed to fool the body into thinking it is responding to an infection." (*Id.*)

Dr. Gershwin acknowledges that there are no reports in the literature of increased incidences of dermatomyositis after any large vaccination campaigns. (Ex. 13, p. 4.) However, he stresses that this is not surprising given the rarity of the condition, which he indicates has an estimated annual incidence of only 2 per million. (*Id.* (citing Peter N. Malleson et al., *The Incidence of Pediatric Rheumatic Diseases: Results from the Canadian Pediatric Rheumatology Association Disease Registry,* 23 J. RHEUMATOLOGY 1981 (1996) (Ex. 39)).) However, a mouse model study by Katsumata, et al., has shown that when genetically susceptible mice are immunized with the muscle autoantigen found in myositis – histidyl-tRNA synthase or "Jo-1" – they have auto antibodies 14 days post immunization that continue to increase for eight weeks and their muscle histopathology at eight weeks demonstrates inflammatory cells. (*Id*. at 5-6 (citing Yasuhiro Katsumata et al., *Species-Specific Immune Responses Generated by Histidyl-tRNA Synthetase Immunization are Associated with Muscle and Lung Inflammation,* 29 J. AUTOIMMUNITY 174 (2007) (Ex. 43)).) Further, he notes that the response to flu vaccination would have included production of interferons and, in particular, plasmacytoid dendritic cells. (*Id*. at 5-6 (citing Marie Wahren-Herlenius & Thomas Dorner, *Immunopathogenic Mechanisms of Systemic Autoimmune Disease,* 382 LANCET 819 (2013) (Ex. 45)).) He quotes Wahren-Herlenius, et al., for the further proposition that "[w]ith activation of dendritic cells, an autoimmune self-amplification loop would produce more interferon, promising and sustaining autoreactive responses, keeping activated T cells and B cells in a vicious cycle, and producing more autoantibodies." (*Id.* at 7 (quoting Wahren-Herlenius & Dorner, *supra,* at Ex. 45, p. 6).) Dr. Gershwin cites seven papers that he indicates demonstrate the plausibility of a linkage between vaccination and dermatomyositis. (*Id*. (citing (Arie Altman et al., *HBV Vaccine and Dermatomyositis: Is There an Association?,* 28 RHEUMATOLOGY INT'L 609 (2008) (Ex. 48); Clodoveo Ferri et al., *Polymyositis Following Pandemic Influenza A (H1N1) and 2009-10 Seasonal Trivalent Vaccines,* 2012 CASE REPORTS RHEUMATOLOGY 1 (2012) (Ex. 49); F.M. Jani et al., *Influenza Vaccine and Dermatomyositis,* 4 NEUROEPIDEMIOLOGY 125 (1985) (Ex. 50); Hedi Orbach et al., *Vaccines and Autoimmune Diseases of the Adult,* 9 DISCOVERY MED. 90 (2010) (Ex. 51); K. Perdan-Pirkmajer et al., *Autoimmune Response Following Influenza Vaccination in Patients with Autoimmune Inflammatory Rheumatic Disease,* 21 LUPUS 175 (2012) (Ex. 52); Natasa Toplak & Tadeg Avcin, *Influenza and Autoimmunity,* 1173 CONTEMP. CHALLENGES AUTOIMMUNITY 619 (2009) (Ex. 53); David C. Wraith et al., *Vaccination and Autoimmune Disease: What is the Evidence?,* 362 LANCET 1659 (2003) (Ex. 54)).)

Regarding petitioner's case, Dr. Gershwin proposes that she was genetically predisposed to dermatomyositis. (Ex. 13, p. 6.) However, he does not cite any evidence to support this suspicion apart from the fact that she developed dermatomyositis. In fact, he acknowledges that the medical records do not identify any underlying autoimmune or immunologic disease or infection that would have left her susceptible to development of dermatomyositis. (*Id*. at 1-2.) He does note, however,

that petitioner had a vitamin D deficiency, which he suggests is thought to be a predisposition to autoimmunity. (*Id*. at 1.)

Dr. Gershwin emphasizes that petitioner developed dermatomyositis post-vaccination without any evidence of any prior muscle disease and without any evidence of a viral infection at the time of onset. (Ex. 13, p. 1.) He interprets petitioner's biopsy as consistent with autoimmune myositis. (*Id*. at 2.) However, although petitioner had Anti-Jo-1 antibodies, Dr. Gershwin opines that "her disease was initiated by her innate immune system and there does not yet appear to be a clinically significant role for adaptive immunity." (*Id.* at 4 (citing Ann M. Reed & Floranne Ernste, *The Inflammatory Milieu in Idiopathic Inflammatory Myositis,* 11 CURRENT RHEUMATOLOGY REPORTS 295 (2009) (Ex. 35)).) Nonetheless, he proposes that the above-discussed Katsumata, et al., study, finding Jo-1 antibodies in mice by 14 days post-immunization, supports an appropriate temporal relationship because it is "similar to what occurred herein." (*Id*. at 5-6 (citing Katsumata et al., *supra,* at Ex. 43).)

b. Respondent's rheumatology expert, Christopher Mecoli, M.D.[5]

Dr. Mecoli agrees that petitioner has a form of IIM and more specifically concludes that petitioner has "anti-Jo1-positive idiopathic inflammatory myositis." (Ex. B, p. 3.) He notes that her medical records variously reference her condition as "myositis," "polymyositis," dermatomyositis," and "anti-synthase syndrome," but explains that all of these conditions fall under the larger umbrella of IIM. (*Id*.)

Dr. Mecoli stresses that the cause of IIM is unknown. (Ex. B, p. 4.) However, he agrees that infectious agents (bacteria, parasites, and viruses) are among the most commonly proposed causal agents, along with medications, ultraviolet light, and cancer. (*Id*. (citing C. Thompson et al., *The Pathogenesis of Dermatomyositis,* 179 BRIT. J. DERMATOLOGY 1256 (2018) (Ex. B, Tab 1); Brittany L. Adler & Lisa Cristopher-Stine, *Triggers of Inflammatory Myopathy: Insights into Pathogenesis,* 25 DISCOVERY MED. 75 (2018) (Ex. B, Tab 2)).) In light of the difficulties in studying IIM, causal assessments generally place an emphasis on temporality. (*Id*. at 4.) "Given the focus on infection, vaccines inevitably are considered as a potential environmental trigger." (*Id*.) However, this consideration has largely been limited to case reports, which should not be interpreted as proof of causation. (*Id*. (citing Joerg-Patrick Stubgen, *A Review on the Association Between Inflammatory Myopathies and Vaccination,* 13 AUTOIMMUNITY REVS. 31 (2014) (Ex. 41)).)

[5] Dr. Mecoli is a board certified rheumatologist. (Ex. A, p. 6.) He received his undergraduate degree from the College of New Jersey and his medical degree from Rutgers University. (*Id.* at 1.) He completed an internship and a residency at the University of Pennsylvania, and a fellowship in rheumatology at Johns Hopkins Hospital. (*Id.*) Dr. Mecoli is currently the Director of Research Operations at Johns Hopkins Myositis Center, the Director of Myositis at Precision Medicine Center Initiative, an assistant professor in the Division of Rheumatology at Johns Hopkins University, and an attending physician at Johns Hopkins Medical Center. (*Id.*) He has published 31 original research papers, a book chapter, and has been invited to review seven articles. (*Id.* at 1-4.)

Dr. Mecoli does not disagree with Dr. Gershwin regarding the general background of IIM but does not agree that his theory of causation is reliably supported. He suggests that "[t]he main argument appears to be that vaccines can elicit many of the same immunologic perturbations that are observed in patients with IIM, and therefore, given IIM occurred in close temporal proximity to the petitioner's influenza vaccination, there is likely a causal association." (Ex. B, pp. 4-5.) He disagrees that the Katsumata, et al., study cited by Dr. Gershwin is valuable, both because it is merely a mouse model and also because it did not involve the flu vaccine. (*Id*. at 5.) Dr. Mecoli indicates that the crux of his disagreement is Dr. Gershwin's suggestion that petitioner would have had a "unique" interferon signature that would have facilitated antigen presentation of her autoantigens to plasmacytoid dendritic populations leading to an inflammatory response in the skin and muscle. (*Id*. (quoting Ex. 13, p. 7).) While Dr. Mecoli agrees an interferon response to vaccination is a desired outcome, he stresses there is no evidence this happened in petitioner's case and no evidence this has led to IIM in any other patient. (*Id*.) Further, Dr. Mecoli stresses that, if vaccines could trigger this autoimmunity in susceptible individuals, then one might expect IIM patients to experience relapses or "flares" after vaccination; however, such flares have not been demonstrated. (*Id*. (citing Carla G. S. Saad et al., *Immunogenicity and Safety of the 2009 Non-Adjuvanted Influenza A/H1N1 Vaccine in a Large Cohort of Autoimmune Rheumatic Diseases,* 70 ANNALS RHEUMATIC DISEASES 1068 (2011) (Ex. B, Tab 5)).) Although he agrees the rarity of IIM complicates epidemiology, he notes that at least one study has purported to find no statistically significant evidence of increased incidences of post-vaccination dermatomyositis or polymyositis. (*Id*. at 6 (citing H. Orbach & A. Tanay, *Vaccines as a Trigger for Myopathies,* 18 LUPUS 1213 (2009) (Ex. B, Tab 6)).)

c. Respondent's immunology expert, James Moy, M.D.[6]

Dr. Moy asserts that Dr. Gershwin "has not given any specific medical theory on mechanisms" as to how the flu vaccine can cause dermatomyositis. (Ex. D, p. 3.) Regarding Dr. Gershwin's observation that the response to the flu vaccine leads to production of interferons, Dr. Moy characterizes this as a "broad, general statement" that does not explain how the flu vaccine would lead to dermatomyositis. (*Id*.) Dr. Moy indicates that the Wahren-Herlenius publication Dr. Gershwin quotes addresses environmental factors including drugs, chemicals, infectious agents, cigarettes, and

[6] Dr. Moy is a board certified in allergy and immunology and pediatrics. (Ex. C, p. 2.) He received his undergraduate degree from Northwestern University and his medical degree from the University of Illinois College of Medicine. (*Id.* at 1.) He completed a residency in pediatrics at the University of Minnesota and a fellowship in allergy and immunology at Rush-Presbyterian-St. Luke's Medical Center. (*Id.*) Dr. Moy is currently an associate professor of Immunology/Microbiology, Pediatrics, and Internal Medicine, an associate program director of the Allergy and Immunology Fellowship, and an associate director of Immunology Discipline at Rush Medical College. (*Id.* at 1-2.) He has "evaluated and managed over 300 pediatric, adolescent and young adult patients with autoimmune diseases and disorders" including "lupus erythematosus, juvenile idiopathic arthritis, . . . juvenile dermatomyositis, Kawasaki disease, scleroderma, inflammatory bowel diseases, . . . and celiac disease." (Ex. D, p. 1.) At the time he submitted his CV, he had authored 53 publications, 43 abstracts, and 8 book chapters. (Ex. C, pp. 7-17.)

ultraviolet radiation, can precipitate autoimmunity, but not vaccinations. (*Id*. at 4 (citing Wahren-Herlenius & Dorner, *supra,* at Ex. 45, pp. 6 (Figure 5), 8 (Table 2)).)

Dr. Moy agrees with Dr. Gershwin's general discussion of the roles of both the innate and adaptive immune responses in autoimmune disease but finds the proposed link to the flu vaccine lacking any reliable or persuasive evidence. (Ex. D, p. 4.) Regarding the seven specific citations Dr. Gershwin cited to demonstrate the plausibility of this purported link, Dr. Moy addresses each in turn and observes the following: Three of Dr. Gershwin's citations (Altman et al., *supra,* at Ex. 48; Ferri et al., *supra,* at Ex. 49; Jani et al., *supra,* at Ex. 50) are merely case reports, which "are not scientific evidence of cause and result." (*Id.* (citing Trygve Nissen & Rolf Wynn, *The Clinical Case Report: A Review of its Merits and Limitations,* 7 BIOMED. CENT. RSCH NOTES 1 (2014) (Ex. D, Tab 2)).) Moreover, the case report at Exhibit 48 involves a different vaccine, further reducing its significance. (*Id*. (citing Altman et al., *supra,* at Ex. 48).) Petitioner's Exhibit 51 is a study from which the authors concluded that no statistically significant increase in polymyositis or dermatomyositis was observed after mass vaccination. (*Id*. (citing Orbach et al., *supra,* at Ex. 51, p. 3).) Petitioner's Exhibit 52 is a study that found only "transient changed in antibody production" in both autoimmune patients and healthy subjects following flu vaccination, but no exacerbation of disease in autoimmune patients or new onset of autoimmune disease in healthy patients. (*Id.* at 5 (citing Perdan-Pirkmajer et al., *supra,* at Ex. 52).) Petitioner's Exhibits 53 and 54 discuss the flu vaccine as a cause of autoimmunity in the context of GBS and Henoch-Schoenlein purpura, but not dermatomyositis. (*Id.* (citing Toplak & Avcin, *supra,* at Ex. 53; Wraith et al., *supra,* at 54).)

Regarding the details of this petitioner's history, Dr. Moy places onset of petitioner's dermatomyositis on November 17, 2017, about three days post-vaccination. (Ex. D, p. 5.) He therefore contends that the Katsumata, et al., mouse study, which observed autoantibodies at 14 days, does not support medically appropriate timing of onset in this case. (*Id*. (citing Katsumata et al., *supra,* at Ex. 43).) Dr. Moy also asserts that Dr. Gershwin is inconsistent in suggesting that there was no significant clinical role for petitioner's adaptive immune response given that petitioner did have anti-Jo-1 antibodies. (*Id.*) This implies that her condition was at least in part a consequence of her adaptive immune response. (*Id.*) Dr. Moy opines that there is not reliable evidence that the flu vaccine did cause petitioner's dermatomyositis and further that the timing of onset is too soon after vaccination to implicate her vaccination. (*Id*. at 6.)

V. Discussion

a. *Althen* prong one

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford ex rel. Pafford v. Sec'y of Health & Human Servs.,* 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (quoting *Pafford ex rel. 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,* 35 F.3d at 548-49. Petitioner 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 ex rel. Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (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 [her] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'" *Boatmon,* 941 F.3d at 1359 (quoting *Moberly ex rel. Moberly,* 592 F.3d 1315, 1322 (Fed. Cir. 2010); *Knudsen*, 35 F.3d at 548-49).

In this case, the parties' experts all effectively agree regarding the pathogenesis of IIM generally. That is, both parties agree that IMMs are likely autoimmune conditions involving a combination of genetic and environmental factors, but for which specific causes are unknown or, as Dr. Gershwin puts it, "enigmatic." (Ex. 13, p. 3; *see also* Ex. B, pp. 3-4; Ex. D, p. 2, 3.) There is also no dispute that infectious antigens are among the suspected causes and that anti-Jo-1 antibodies can be implicated as the autoimmune pathway as Dr. Gershwin asserts. (Ex. 13, pp. 4-5; *see also* Ex. B, p. 3-4; Ex. D, pp. 2-3.) However, none of this implicates vaccinations as a cause of IMMs. Thus, the crux of Dr. Gershwin's theory is his juxtaposition of two concepts: (1) that the initial inflammatory aspect of IMMs is mediated by interferons and (2) that the flu vaccine is known to stimulate interferons. (Ex. 13.) Combining these two concepts, Dr. Gershwin asserts that it is plausible to link the flu vaccine to IMM. (Ex. 13, p. 7.) However, as Dr. Moy submits, even accepting both concepts as true, this latter assertion is a broad and generic statement that merely invokes the immune response that the flu vaccine is *intended* to have. (Ex. D, pp. 3-4.) Prior decisions have concluded that this type of reasoning is not persuasive, without more, as a theory of causation. *E.g. Cordova v. Sec'y of Health & Human Servs*., No. 17-1282V, 2021 WL 3285367, at *17 (Fed. Cl. Spec. Mstr. June 23, 2021) (observing that "[e]vidence that a vaccine causes an immune response – the intended function of any vaccine – does not amount to a showing that this response is, or can become, pathologic, absent additional proof linking to evidence of the vaccine . . ."). Accordingly, petitioner's *Althen* one presentation turns on the evidence underlying Dr. Gershwin's statement that a link between dermatomyositis and the flu vaccine is plausible and how effectively Dr. Gershwin marries the largely undisputed general understanding of interferon-mediated inflammation leading to autoimmunity and IIM with the very modest evidence implicating vaccines in that process. Respondent's experts persuasively assert that on this record, Dr. Gershwin's assertion that the flu vaccine has been plausibly linked to IMMs is virtually unsupported.

As discussed above, Dr. Gershwin has cited seven specific publications that he asserts support the plausibility of the flu vaccine as a cause of IIMs, each of which respondent's experts, and in particular Dr. Moy, have sought to rebut. (*Compare* Ex. 13, pp. 6-7 *and* Ex. D, pp. 4-5.) In addition to several case reports (by Altman, et al, Ferri, et al, and Jani, et al, respectively), Dr. Gershwin cites two review papers, one by Orbach et al., and one by Wraith, et al., addressing vaccines as a potential cause of

autoimmunity more broadly (Orbach et al., *supra,* at Ex. 51, Wraith et al., *supra,* at 54), a paper by Toplak and Avcin addressing influenza infection and autoimmunity (Toplak & Avcin, *supra,* at Ex. 53), and a study by Perdan-Pirkmajer, et al., addressing the flu vaccine and inflammatory rheumatic diseases (Perdan-Pirkmajer et al., *supra,* at Ex. 52). However, apart from the case reports, none of these publications purport to directly implicate the flu vaccine as a cause of IIMs. To the extent they could theoretically provide circumstantial support even without being facially relevant, Dr. Gershwin has not actually explained what in these papers actually supports his opinion. (See Ex. 13, p. 7.) In fact, his report seems to acknowledge that the proposed link is asserted without yet being supported. Specifically, Dr. Gershwin indicates in reference to these citations that "[t]he plausibility of a linkage between vaccination and [dermatomyositis] is also noted from a variety of literature that has attempted to show causation and [] which discusses the theoretical basis to link an association, albeit with the limitations of power calculations discussed above." (*Id*.)

Upon my own review, neither Orbach, et al., nor Wraith, et al., implicate vaccinations as a cause of IIMs. Wraith, et al., lists dermatomyositis as a confirmed autoimmune condition, but not as an autoimmune condition known to be caused by vaccinations. (Wraith et al., *supra,* at Ex. 54, pp. 2-3 (*Compare* Panel 1 *and* Panel 3).) Orbach, et al., explain that there have been "sporadic" case reports reporting post-vaccination myopathies, but stressed that "[t]here is no statistically significant increase in the incidence of polymyositis or dermatomyositis after any mass vaccination." (Orbach et al., *supra,* at Ex. 51, p. 3.) Toplak and Avcin do not discuss IMMs *at all*. (Toplak & Avcin, *supra,* at Ex. 53.) Perdan-Pirkmajer examined potential autoimmune responses following seasonal flu vaccination among 218 patients already suffering rheumatologic conditions. (Perdan-Pirkmajer et al., *supra,* at Ex. 52, pp. 1-2.) The study reported transient changes in autoantibodies post-vaccination, especially ANA. (*Id*. at 1.) However, the study did not correlate these findings to clinical outcomes. Moreover, out of a study population of 218 patient with rheumatologic conditions, only one patient had dermatopolymyositis. (*Id.* at 2.) Although the authors note having screened some patients for anti-Jo-1 antibodies, no findings were reported relative to that antibody. (*Id.* at 3.) Although a specific autoantibody has not been definitively identified for dermatomyositis (Hornung & Wenzel, *supra,* at Ex. 15, p. 3; Khanna & Reed, *supra,* at Ex. 16, p. 6 (Table 1)), Dr. Gershwin specifically singled out the anti-Jo-1 antibody as having been experimentally supported as leading to the development of dermatomyositis. (Ex. 13, pp. 5-6 (citing Katsumata et al., *supra,* at Ex. 43).) Moreover, he cited literature indicating that the anti-Jo-1 antibody is "strongly associated" with the interferon pathway to autoimmunity he has advanced in this case. (Nagaraju & Lundberg, *supra,* at Ex. 17, p. 5.)

This leaves only the case reports as support for Dr. Gershwin's theory. However, as Dr. Moy explains, case reports "are not scientific evidence of cause and result." (Ex. D, p. 4 (citing Nissen & Wynn, *supra,* at Ex. D, Tab 2).) Although case reports are not entirely without evidentiary value, they are not strong evidence without more. *E.g. Crutchfield v. Sec'y of Health & Human Servs.*, No. 09-39V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) ("single 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); *see also Paluck v. Sec'y of Health & Human Servs.*, 104 Fed. Cl. 457, 475 (2012) (indicating that case reports "'do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value'. . . [but] 'the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight."), ***aff'd***, 786 F.3d 1373 (Fed. Cir. 2015)). Additionally, one of the case reports cited by petitioner involves a different vaccine, further reducing its significance. (Altman et al., *supra,* at Ex. 48).)

To be clear, both parties have filed literature that acknowledges that vaccines have been hypothesized as a cause of IIM in at least some rare instances. (Orbach & Tanay, *supra,* at Ex. B, Tab 6; Stubgen, *supra,* at Ex. 41.) However, when considering this literature holistically, it is apparent from these discussions that the purported link has not been substantiated. In that regard, Dr. Gershwin asserts that dermatomyositis is too rare to reasonably expect it to be detected by epidemiology. (Ex. 13, pp. 2-3.) Respondent's experts likewise agree that dermatomyositis is a rare enough condition that epidemiology must be approached with caution. (Ex. B, p. 3.) That is, both parties' experts conclude would be very challenging to develop a study sufficiently powered to detect post-vaccination incidences of dermatomyositis. (*Id.* at 4; Ex. 13, pp. 2-3.) And, of course, petitioners in this program are not obligated to come forward with epidemiologic support in order to meet *Althen* prong one. *See Andreu,* 569 F.3d at 1378-79 (citing *Capizzano,* 440 F.3d at 1325-26).

However, that is not to say that all epidemiology is irrelevant. Special masters may consider whether available epidemiology undermines a petitioner's claim. *D'Tiole v. Sec'y of Health & Human Servs.*, 726 F. App'x 809, 811 (Fed. Cir. 2018) (citing *Andreu*, 569 F.3d at 1379 ("Although *Althen* and *Capizzano* make clear that a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the Special Master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury."). In this case, Dr. Gershwin has cited a review paper that discusses two types of studies that are not affected by the issue of the rarity of dermatomyositis in the general population. First, retrospective study has examined whether patients diagnosed with dermatomyositis had had recent immunizations. (Stubgen, *supra,* at Ex. 41, p. 3.) Vaccination was not identified as an important trigger for dermatomyositis. (*Id*.) Second, a prospective study also looked at the effects of the flu vaccination on individuals already suffering dermatomyositis. (*Id.* at 6.) This study determined that the flu vaccine has no short-term effect on the disease course of those already suffering dermatomyositis. (*Id*.) While these types of studies obviously still have limitations, they do not depend on having a large enough scale to overcome the rarity of dermatomyositis in the general population, overcoming the specific issue raised by Dr. Gershwin to discount epidemiologic study of dermatomyositis. Accordingly, I find it reasonable to consider these studies as some evidence weighing against petitioner's theory.

Notably, prior petitioners have failed to come forward with preponderant evidence demonstrating that the flu vaccine can cause dermatomyositis. *Whelan v. Sec'y of Health & Human Servs*., No. 16-1174V, 2019 WL 1061473 (Fed. Cl. Spec. Mstr. Jan. 28, 2019); *McDaniel v. Sec'y of Health & Human Servs*., No. 17-1322V, 2023 WL 4678688 (Fed. Cl. Spec. Mstr. June 26, 2023). However, I am also aware of one prior case in which a petitioner was found entitled to compensation for dermatomyositis caused-in-fact by a flu vaccine based on a theory similar to the one presented in this case.[7] *Ulysse v. Sec'y of Health & Human Servs*., No. 15-451V, 2022 WL 2115248 (Fed. Cl. Spec. Mstr. May 19, 2022). A critical factor in the resolution of that case was the degree to which the petitioner's theory of causation was unchallenged by respondent. *Id.* at 19. The special master noted in conclusion that "[i]n a different case, with a more substantive opposition marshaled by Respondent, the outcome would likely have been different." *Id*. at 20. An additional case found a petitioner entitled to compensation for a minor child's juvenile dermatomyositis caused by a simultaneous administration of DTaP, MMR, Polio, and varicella vaccinations. *Rodriguez ex rel. C.R. v. Sec'y of Health & Human Servs*., No. 13-253V, 2017 WL 5563419 (Fed Cl. Spec. Mstr. Oct. 26, 2017). However, that case involved different vaccines and other cases have suggested that juvenile dermatomyositis is a distinguishable condition. *Id.*; *McDaniel,* 2023 WL 4678688, at *32 n. 62.

Thus, considering all of the above and the record as a whole, the record of this case best reflects the conclusion that it may be possible, but not preponderantly established, that the flu vaccine could be a cause of dermatomyositis. Thus, petitioner has not met her burden of proof under *Althen* prong one. Under *Althen* prong one, petitioner must come forward with more than a merely possible theory of causation.[8] *Boatmon,* 941 F.3d at 1360.

a. *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; *Grant*, 956 F.2d at 1148. Medical records are generally viewed as particularly trustworthy evidence. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir.

[7] Myositis is also sometimes raised as part of an overlapping presentation inclusive of other rheumatologic conditions. These cases have also been resolved to mixed results for petitioners. Compare *Rodd v. Sec'y of Health & Human Servs*., No. 13-122V, 2015 WL 8489035 (Fed. Cl. Spec. Mstr. Nov. 13, 2015) (implicating the flu vaccine in an overlap syndrome inclusive of Sjogren's Syndrome, carpal tunnel syndrome, polymyositis, and inflammatory arthritis) *and Suliman v. Sec'y of Health & Human Servs.,* 13-993V, 2018 WL 6803697 (Fed. Cl. Spec. Mstr. Nov. 27, 2018) (finding against the petitioner in case involving polymyalgia rheumatica ("PMR") and myositis allegedly caused by a Tdap vaccine). These other types of cases are not easily compared to the instant case given the interplay of the different conditions and diagnoses.

[8] In his motion response, respondent stresses Dr. Gershwin's use of the word "plausible" to describe the link between the flu vaccine and dermatomyositis. (ECF No. 43, p. 16.) Although respondent is correct, my conclusion that petitioner's theory is merely plausible derives from my own review of the record evidence. It does not turn on Dr. Gershwin's specific use of the term plausible in his report.

1993). 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 ex rel. Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n. 67 (2009) ("there 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 v. Sec'y of Health & Human Servs.,* 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines,* 940 F.2d at 1528).

Although Dr. Ozeri opined that petitioner's condition was vaccine caused, his stated rationale for so opining was the concept of autoimmune syndrome induced by adjuvants or "ASIA." (*See* Ex. 5, p. 105.) ASIA has been the subject of substantial criticism and has been repeatedly rejected as a viable theory of causation in this program. *See J.F. v. Sec'y of Health & Human Servs.,* No. 13-799V, 2022 WL 5434214 (Fed. Cl. Spec. Mstr. Sept. 9, 2022); *D'Angiolini, v. Sec'y of Health & Human Servs.,* No. 99-578V, 2014 WL 1678145 (Fed. Cl. Spec. Mstr. Mar. 27, 2014), *mot. for rev. denied,* 122 Fed. Cl. 86 (2015); *Rowan v. Sec'y of Health & Human Servs.,* No. 10-272V, 2014 WL 7465661 (Fed. Cl. Spec. Mstr. Dec. 8, 2014), *aff'd,* 2015 WL 3562409 (Fed. Cl. May 18, 2015). In fact, ASIA has specifically been rejected as a theory purporting to link myositis to vaccination. *Suliman*, 2018 WL 6803697, at *25-28. Although treating physician opinions are often given significant weight, such opinions are only as sound as their bases. *See Isaac v. Sec'y of Health & Human Servs.,* No. 08-601V, 2012 WL 3609993, at *24 (Fed. Cl. Spec. Mstr. July 30, 2012). Moreover, respondent stresses that the flu vaccine petitioner received was not adjuvanted. (ECF No. 43, p. 22.) Accordingly, without further elaboration or substantiation of his opinion, Dr. Ozeri's mere invocation of ASIA does not preponderantly support petitioner's claim. Conspicuously, petitioner's expert, Dr. Gershwin, did not rely on ASIA in formulating his own theory of causation even though some of the literature he cited does discuss the ASIA hypothesis with respect to post-vaccination autoimmunity more broadly. (*E.g*. Stubgen, *supra,* at Ex. 41, p. 6; Perdan-Pirkmajer et al., *supra,* at 52, p. 8.)

Dr. Gershwin's causal assessment also faulters insofar as he has not preponderantly established that the flu vaccine can cause dermatomyositis at all. Additionally, his opinion suffers due to internal inconsistency. As Dr. Moy points out, despite explaining dermatomyositis as involving an inflammatory response leading to an autoimmune response involving anti-Jo-1 antibodies, Dr. Gershwin puzzlingly disclaimed any role for petitioner's documented anti-Jo-1 antibodies in her own disease process. (Ex. D, p. 5.) This appears likely to be an effort to sidestep any need to substantiate a more direct causal relationship between petitioner's vaccination and her ultimate antibody response. Dr. Gershwin was never more specific than to assert that vaccinations mimic infections and that there are a variety of theories to suggest how an infectious agent may induce dermatomyositis. (Ex. 13, pp. 4-5.) However, respondent's experts reasonably concluded that petitioner's anti-Jo-1 antibodies likely

suggest her condition was least partly the result of an adaptive immune response. (Ex. D, p. 5.) Especially given the nature of the evidence Dr. Gershwin presented to support his theory of causation, which among other things "strongly associated" anti-Jo-1 antibodies to the interferon pathway to autoimmunity in dermatomyositis, this sidestepping by Dr. Gershwin reduces his causal opinion to one merely recognizing the presence of timing coincident to vaccination. Such opinions do not preponderantly support a cause-in-fact claim. *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149).

For all these reasons, petitioner has not met her preponderant burden of proof under *Althen* prong two.

a. *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 disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008).

Petitioner has submitted three publications that include five case reports of individual patients experiencing IIM subsequent to vaccination. Among those five case reports, one patient initially had a fever and cough a "few" days post-vaccination and then later developed additional symptoms of IIM such as muscle weakness two weeks later. (Ferri et al., *supra,* at Ex. 49, p. 1.) Otherwise, the remaining four cases identify onset ranging from one week to one-month post-vaccination. (*Id.*; Altman et al., *supra,* at 48; Jani et al., *supra,* at 50.) Similarly, the review paper cited by Dr. Gershwin identifies two case reports of either dermatomyositis or polymyositis occurring within two days of vaccination, but otherwise lists 20 other case reports placing onset at between five days and six weeks post-vaccination. (Stubgen, *supra,* at Ex. 41, p. 3 (Table 1).) Thus, these case reports on the whole tend to suggest that onset of vaccine-caused dermatomyositis – if such a thing were to exist – would likely occur at least one-week post-vaccination. In this case, however, Dr. Gershwin asserts that onset of petitioner's own condition arose just two days post-vaccination.

In seeking to assert that onset of petitioner's dermatomyositis occurred during a timeframe within which it is reasonable to infer vaccine causation, Dr. Gershwin relies on a mouse model study by Katsumata, et al., that found Jo-1 antibodies in mice by 14 days post-immunization. (Ex. 13, pp. 5-6 (citing Katsumata et al., *supra,* at Ex. 43).) There are two issues with this reliance. First, as discussed under *Althen* prong two, above, Dr. Gershwin disclaimed any causal role for petitioner's own documented anti-Jo-1 antibodies. (Ex. 13, pp. 6-7.) Accordingly, Dr. Gershwin has not substantiated that this antibody finding by Katsumata, et al., has any relevance to the timing of onset based on Dr. Gershwin's own assessment of the cause of petitioner's condition. Second, the Katsumata study authors appear to indicate that it is the difference in

antibody response between ten days and eight weeks that permits them to conclude that a cross-reactive antibody response had developed during that period. (Katsumata et al., *supra,* at Ex. 43, pp. 6, 16 (Fig. 4).) Indeed, Dr. Gershwin cites this progression and corresponding findings on muscle histopathology, as the basis for opining that a disease process has set in. (Ex. 13, pp. 5-6.) However, the study authors confirm that in the course of the study subject mice were sacrificed for sample harvesting after ten days at the earliest. (Katsumata et al., *supra,* at Ex. 43, pp. 3-4.) Thus, it is not apparent that the Katsumata study has any ability in itself to suggest what, if any, disease process occurred prior to ten days. These study results remain consistent with the great majority of the above-discussed case reports that generally place onset between one-week and six-weeks post vaccination.

None of this evidence necessarily *rules out* a two to three-day latency as medically appropriate to infer vaccine-causation. However, it does not preponderantly *support* a temporal relationship between petitioner's vaccination and onset of her dermatomyositis. For his part, respondent's immunology expert, Dr. Moy, did opine that petitioner's condition arose too soon after vaccination to implicate her vaccine as a cause. (Ex. D, p. 6.)

For all these reasons, petitioner has not met her preponderant burden of proof under *Althen* prong three.

VI. Conclusion

After weighing the evidence of record within the context of this program, and for all the reasons discussed herein, petitioner has not demonstrated by preponderant evidence that her condition was caused by her November 14, 2017 flu vaccination. Accordingly, this case is dismissed.[9]

IT IS SO ORDERED.

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

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