In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: March 23, 2026

ALISA JETT-CRAWFORD,	*	PUBLISHED
Petitioner,	*	No. 21-2157V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Polymyalgia Rheumatica ("PMR"); Seronegative Rheumatoid Arthritis ("RA").
Respondent.	*	

Jessica Ann Wallace, Siri & Glimstad, LLP, Aventura, FL, for Petitioner.
Tyler King, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On November 10, 2021, Alisa Jett-Crawford ("Petitioner") filed a petition in the National Vaccine Injury Program[2] alleging that as a result of receiving an influenza ("flu") vaccine on November 11, 2018, she suffered polymyalgia rheumatica ("PMR") and appendicitis, requiring

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it 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 Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

surgical intervention.[3] Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating that "this case is not appropriate for compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 27).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[4] the undersigned finds that Petitioner has failed to provide preponderant evidence that the flu vaccination caused her illness. Thus, Petitioner has failed to satisfy her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is not entitled to compensation.

I. ISSUES TO BE DECIDED

First, the parties dispute whether Petitioner's diagnosis is PMR. Joint Sub. at 4. Respondent contends that the correct diagnosis is seronegative rheumatoid arthritis ("RA").[5] Id.

The parties also dispute all three Althen prongs. Joint Sub. at 4. Specifically, the parties dispute whether Petitioner has provided preponderant evidence that the flu vaccine can cause PMR and whether Petitioner provided preponderant evidence that flu vaccine did cause her PMR. Id. While the parties agree that Petitioner's symptom onset was November 19, 2018 (eight days post-vaccination), they dispute whether an eight-day onset is "an appropriate time interval" for PMR that resulted from the flu vaccine. Id.

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[3] While Petitioner alleged the flu vaccine caused her appendicitis and subsequent appendectomy in her petition, Petitioner does not include in this injury in her motion for a ruling on the record nor was it identified in the parties' joint submission. See Petitioner's Motion for a Ruling on the Record ("Pet. Mot."), filed Mar. 7, 2025, at 1 (ECF No. 91); Joint Submission ("Joint Sub."), filed Mar. 7, 2025, at 4 (ECF No. 88). Further, Petitioner's expert does not address appendicitis in his reports. See Pet. Exhibits ("Exs.") 25, 27. Thus, the undersigned finds that Petitioner no longer claims this injury and does not address appendicitis in this Decision.

[4] While the undersigned has reviewed all of the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[5] RA is "an autoimmune, inflammatory joint disease characterized by swelling, pain, and destruction of synovial joints." Resp. Ex. A, Tab 3 at 2 (Caitrin M. Coffey et al., Evidence of Diagnostic and Treatment Delay in Seronegative Rheumatoid Arthritis: Missing the Window of Opportunity, 94 Mayo Clin. Proc. 2241 (2019)). Approximately 20-25% of RA patients are seronegative, meaning the patients do not have serologic biomarkers specific to RA (i.e., rheumatoid factor ("RF") and anti-citrullinated peptide antibodies ("ACPAs")) "despite meeting clinical classification criteria for RA." Id.

II. BACKGROUND

A. Procedural History

On November 10, 2021, Petitioner filed a petition requesting compensation followed by medical records.[6] Petition; Pet. Exs. 1-23. The case was then assigned to the undersigned. Notice of Reassignment dated Apr. 26, 2022 (ECF No. 17). Respondent filed a Rule 4(c) report on September 2, 2022, arguing against compensation. Resp. Rept. at 1.

On February 13, 2023, Petitioner filed an expert report from Dr. M. Eric Gershwin. Pet. Ex. 25. On August 7, 2023, Respondent filed an expert report from Dr. Jonathan Miner. Resp. Ex. A.

The undersigned held a Rule 5 conference on September 26, 2023. Order dated Sept. 26, 2023 (ECF No. 51). The undersigned did not make a preliminary finding on diagnosis. Id. at 2. As to causation, she made no preliminary findings on Althen prong one but noted Dr. Gershwin's opinions were "confusing." Id. She did not find any alternative cause for Petitioner's condition. Id. The undersigned recommended the parties resolve the case through settlement. Id.

On February 12, 2024, Respondent advised he was not interested in settlement discussions. Resp. Status Rept., filed Feb. 12, 2024 (ECF No. 55). Petitioner proposed filing supplemental expert reports and then submitting the case for resolution through a ruling on the record. Order dated Apr. 19, 2024 (ECF No. 61). Respondent agreed with Petitioner's proposal and the undersigned set deadlines for the parties to file supplemental expert reports. Id.

On July 19, 2024, Petitioner filed a supplemental expert report from Dr. Gershwin. Pet. Ex. 27. Respondent filed a supplemental report from Dr. Miner on November 18, 2024. Resp. Ex. B.

Petitioner filed her motion for a ruling on the record on March 7, 2025. Pet. Mot. Respondent filed his responsive brief on May 6, 2025, and Petitioner filed a reply on June 11, 2025. Resp. Response to Pet. Mot ("Resp. Response"), filed May 6, 2025 (ECF No. 95); Pet. Reply to Resp. Response ("Pet. Reply"), filed June 11, 2025 (ECF No. 98).

This matter is now ripe for adjudication.

[6] Petitioner continued to file medical records throughout litigation.

B. Factual History

1. Stipulated Medical History[7]

The parties agreed to the following stipulated medical history in their Joint Submission. See Joint Sub. at 1-4.

Petitioner was a healthy 58-year-old woman with no history of chronic medical conditions prior to receiving the subject flu vaccine on November 11, 2018. Pet. Ex. 1 at ¶ 3; Pet. Ex. 2 at 4-12. Petitioner had a family medical history consisting of mesothelioma, dementia, hypertension, and coronary artery disease. Pet. Ex. 2 at 5. Petitioner was a former smoker, having smoked one pack of cigarettes per day off and on for thirty years before stopping in 2013. Id.

On November 11, 2018, Petitioner received a seasonal flu vaccine (Flucelvax quadrivalent). Pet. Ex. 3 at 5. Petitioner's onset of symptoms, including widespread body pain in her left wrist, shoulders, neck, hips, knees, and ankles began on or about November 19, 2018. Pet. Ex. 1 at ¶ 7. On December 7, 2018, during a phone call with her primary care provider ("PCP"), she was prescribed meloxicam.[8] Pet. Ex. 24 at 3.

On December 19, 2018, Petitioner was seen Dr. Nicole Akhaven at her PCP's office. Pet. Ex. 4 at 6. She reported a multiple month history of bilateral wrist pain, shoulder pain, hip pain, and ankle pain. Id. at 7. Following the physical examination, a recommendation was made to run an autoimmune workup. Id. at 8. It was noted that non-steroidal anti-inflammatory drugs ("NSAIDs"), including the prescription drug, Mobic, was ineffective at relieving Petitioner's pain. Id. Petitioner was prescribed a topical gel and recommended a trial of physical therapy. Id. at 9. She was advised to schedule a follow-up appointment to see her regular PCP, Dr. Edlira Maska, in January 2019. Id.

[7] While the stipulated medical history is taken from the parties' joint submission, the undersigned has made minor edits for style and accuracy and has added definitions of medical terms where appropriate.

[8] Meloxicam, the generic form of Mobic, is "a nonsteroidal antiinflammatory drug." Meloxicam, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30286 (last visited Feb. 17, 2026); see also Mobic, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=31788 (last visited Feb. 17, 2026).

Laboratory work from December 2018 revealed Petitioner had an elevated erythrocyte sedimentation rate ("ESR")[9] and C-reactive protein ("CRP")[10] but her RF[11] was normal, her antinuclear antibody[12] was negative, and her creatine kinase was normal.[13] Pet. Ex. 2 at 30.

On January 2, 2019, Petitioner returned to her PCP for a follow-up visit to address her continuing symptoms. Pet. Ex. 5 at 1. On January 29, 2019, Petitioner presented to the Emergency Department ("ED") at Shands at the University of Florida ("UF") with complaints of generalized body aches. Pet. Ex. 4 at 12. The differential diagnoses were PMR, osteoarthritis, and RA. Id. at 15. The ED physicians recommended 20 mg daily of prednisone for four weeks and additional laboratory work to check inflammatory markers, including ESR and CRP, and for surveillance of possible PMR. Id. at 16. At discharge, Petitioner's diagnosis was "arthralgia, unspecified joint." Id.

On February 20, 2019, Petitioner was seen by rheumatologist, Dr. Carolina Mejia Otero at the UF, Department of Medicine, Division of Rheumatology. Pet. Ex. 2 at 18. Following an examination, Dr. Otero provided her assessment and plan:

> [Petitioner] is a 58 [year old] Female coming for evaluation of joint pain. [Petitioner's] history very suggestive of PMR given sudden onset arthralgias and

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[9] ESR is "a measurement of the rate at which red blood cells . . . settle in saline solution or plasma over a specified time period." Erythrocyte Sedimentation Rate, Mosby's Manual of Diagnostic and Laboratory Tests 200 (6th ed. 2018). "ESR is a nonspecific test used to detect illnesses associated with acute and chronic infection, inflammation . . ., advanced neoplasm, and tissue necrosis or infarction." Id. at 199.

[10] CRP "is an acute-phase reactant protein used to indicate to indicate an inflammatory illness." C-Reactive Protein, Mosby's at 165.

[11] RF are "antibodies. . . [that] are found in the serum of about 80 percent of persons with classical or definite [RA] . . . [and] also occur in other connective tissue diseases and some infectious diseases." Rheumatoid Factor, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74591(last visited Mar. 17, 2026).

[12] Antinuclear antibodies are "antibodies directed against nuclear antigens . . . frequently found in [RA], scleroderma (systemic sclerosis), Sjögren syndrome, and mixed connective tissue disease." Antinuclear Antibodies, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited Mar. 17, 2026).

[13] ACPA testing done in December 2018 was also normal. Pet. Ex. 2 at 116-17. ACPAs are antibodies against cyclic citrullinated peptide, "a synthetic, citrulline-containing peptide with a cyclic structure," that are "highly specific for [RA]." Anti-CCP Antibody, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id= 56787 (last visited Mar. 20, 2026); Cyclic Citrullinated Peptide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=97140 (last visited Mar. 20, 2026).

> joint stiffness predominately of shoulder and hip girdle, in association with elevated inflammatory markers and appropriate age group for PMR. [Differential diagnoses] include peripheral inflammatory arthritis such as RA. Labs for RA were negative but cannot [rule out] seronegative RA given knees, ankles[,] and wrists involvement.

Id. at 22. Diagnoses were "arthralgia, unspecified joint" and PMR. Id.

Petitioner presented for a follow-up appointment regarding "possible PMR" with Dr. Otero on April 5, 2019. Pet. Ex. 2 at 23. The listed diagnosis was PMR versus "seronegative RA (worsening)." Id. at 25. Due to Petitioner's continued symptoms, she was advised to begin methotrexate,[14] a high-risk medication with the benefits and risks explained. See id.

On June 28, 2019, Petitioner was seen for a follow-up visit with Dr. Otero for "possible PMR." Pet. Ex. 2 at 26. The history stated that she had a sudden onset of symptoms of diffuse joint pain at the end of November 2018. Id. Petitioner's joint examination was normal. Id. at 30. Impression remained PMR versus seronegative RA. Id.

On or about August 7, 2019, Petitioner began having severe abdominal pain that led her to present to West Marion Community Hospital for evaluation. Pet. Ex. 6 at 15. Petitioner was discharged with a diagnosis of abdominal pain. Id. at 27-28. Due to continued abdominal pain, on August 8, 2019, Petitioner presented to the UF Spring Hill ED with complaints of abdominal pain. Pet. Ex. 2 at 31. Petitioner was discharged from West Marion Community Hospital the previous day, however, the pain did not subside, so she presented for a re-evaluation of these worsening symptoms. Id. After a physical examination and extensive diagnostic testing, Petitioner was diagnosed with appendicitis with perforation and required surgical intervention. Id. at 34-40. Petitioner underwent a laparoscopic appendectomy the same day. Id. at 49.

2. Other Relevant Medical Records

In addition to the above stipulated medical history, the undersigned finds the following medical records relevant.

At Petitioner's December 19, 2018 visit, Dr. Akhaven noted Petitioner had "[n]o history of recent viral illness" and documented a family history of rheumatological disease. Pet. Ex. 4 at

[14] Methotrexate is "a folic acid antagonist that acts by inhibiting synthesis of DNA, RNA, thymidylate, and protein . . . [that is] used as an antipsoriatic and antiarthritic in the treatment of . . . severe [RA] and psoriatic arthritis." Methotrexate, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30930 (last visited Feb. 17, 2026).

7. Petitioner's mother had systemic lupus erythematosus ("SLE"),[15] her sister had RA, and her brother has Sjögren's syndrome.[16] Id. On examination, Petitioner's left wrist had "visible mild edema" and was "tender to palpation." Id. at 8. Dr. Akhaven noted the examination showed "evidence of synovitis of the left wrist" and her assessment was polyarthralgia. Id.

On January 2, 2019, Petitioner was seen by Dr. Maska for follow-up of her diffuse joint pain. Pet. Ex. 9 at 102. Dr. Maska noted Petitioner's arthralgia had "improved after steroid therapy" and prescribed another week of prednisone at a lower lose. Id. at 104. Given her presentation and family history, Dr. Maska referred Petitioner to rheumatology. Id.

Laboratory work done at Petitioner's January 29, 2019 ED visits showed elevated ESR of 47 mm/hr (reference range is less than 20 mm/hr) and elevated CRP of 6.92 mg/L (reference range is 0.00 to 5.00 mg/L). Pet. Ex. 4 at 17-18.

Petitioner presented to Dr. Otero on February 20, 2019 with a chief complaint of joint pain. Pet. Ex. 2 at 18. The history of present illness noted

> [Petitioner] started to have symptoms at the end of [November 2018[17]] with sudden onset of diffuse joint pain involving mainly shoulders and hips but also knees, ankles, [and] wrists; noticed swelling in the [left] wrist and joint stiffness especially in shoulders and hips; she state[d] she could not lift up her shoulders or get out of a chair because of pain; pain seemed to be worse in the morning but also at night it used to hurt when she was trying to turn in bed. Tried NSAIDs without improvement; steroid course [prescribed] by PCP reportedly helped but symptoms [s]tarted to recur when she was on the lower doses of the taper and got

[15] SLE is "is a chronic, multisystem, inflammatory disorder of autoimmune etiology . . . . Manifestations may include arthralgias and arthritis, Raynaud syndrome, malar and other rashes, pleuritis or pericarditis, renal or central nervous system involvement, and autoimmune cytopenias." Kinanah Yaseen, Systemic Lupus Erythematosus, Merck Manual, https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/systemic-rheumatic-diseases/systemic-lupus-erythematosus-sle (last visited Mar. 17, 2026).

[16] Sjögren's syndrome is "a chronic, systemic, autoimmune, inflammatory disorder of unknown cause. It is characterized by dryness of the mouth, eyes, and other mucous membranes (sicca syndrome) due to lymphocytic infiltration of exocrine glands and secondary gland dysfunction." Kinanah Yaseen, Sjögren's Syndrome, Merck Manual, https://www.merckmanuals.com/professional/musculoskeletal-and-connective-tissue-disorders/systemic-rheumatic-diseases/sj%C3%B6gren-syndrome (last visited Mar. 17, 2026).

[17] The record stated symptoms began in "11/2019," however, this is an error as the visit occurred in January of 2019. Later visits confirm that Petitioner's symptoms began in November 2018. See Pet. Ex. 2 at 23, 26.

> worse when was off steroids prompting her to go to ER due to pain where she was [prescribed] 20 mg [prednisone] which has been able to control the symptoms.

Id. Dr. Otero discussed "the natural history of PMR and what to expect" with Petitioner. Id. at 22. The plan was to slowly taper prednisone and consider methotrexate if Petitioner's symptoms recurred during the taper. Id.

At the April 5, 2019 visit, Dr. Otero's assessment again noted that Petitioner's history was "very suggestive of PMR" but noted that seronegative RA could not be ruled out "given knees, ankles[,] and wrist involvement." Pet. Ex. 2 at 25. Petitioner's symptoms had recurred while receiving 10 mg of prednisone. Id. The plan was to check inflammatory markers and increase prednisone to 15 mg followed by a prednisone taper as well as begin methotrexate. Id.

At the June 28, 2019 visit, Dr. Otero's assessment once again noted that Petitioner's history was "very suggestive of PMR" but noted that seronegative RA could not be ruled out "given knees, ankles[,] and wrist involvement." Pet. Ex. 2 at 30. Petitioner's symptoms had recurred on 10 mg of prednisone, and she began methotrexate in April 2019 and since then had "been doing better." Id. The plan was to continue methotrexate, taper prednisone, and re-check inflammatory markers if she had any flares. Id. at 30-31.

On August 14, 2019, Petitioner returned to Dr. Maska for follow-up after her laparoscopic appendectomy and hospitalization. Pet. Ex. 2 at 61-64. Dr. Maska noted Petitioner's "prednisone and methotrexate were discontinued during her hospitalization and given the plan from her rheumatologist to eventually wean off of this medication[] she decided to self discontinue it for now." Id. at 62. Dr. Maska agreed with Petitioner's decision to discontinue prednisone and methotrexate. Id. at 64. He noted she had a follow-up visit with her rheumatologist "at the end of October" and could "discuss reinitiation of therapy if this is indicated."[18] Id. On November 5, 2019, Dr. Maska wrote a letter confirming Petitioner had been diagnosed with PMR and was being followed closely by Dr. Maska and rheumatology. Pet. Ex. 7 at 1.

On December 5, 2019, Petitioner presented to Dr. Maska for an annual visit. Pet. Ex. 2 at 66. She reported "intermittent joint pain" that she treated with ibuprofen about every 10 days. Id. Her physical examination was normal. Id. at 69-70. Petitioner's history of PMR was noted. Id. at 66. She declined flu and zoster vaccines. Id. at 71. Petitioner was referred to dermatology for a routine skin examination which found benign moles and photodamage in multiple areas. Id. at 71-75.

Approximately one year later, on December 9, 2020, Petitioner returned to Dr. Maska for her annual visit. Pet. Ex. 9 at 116. Petitioner was seen by both Dr. George Marek (resident) and Dr. Maska. Id. at 116-21. Dr. Marek noted Petitioner reported she was going to the gym five days a week and had intermittent joint pains that improved with ibuprofen. Id. at 116. She was

[18] It is not clear whether Petitioner attended this October 2019 visit with her rheumatologist. The visit is not documented in the filed medical records.

not taking any steroids. Id. Dr. Marek's assessment noted Petitioner's PMR symptoms were "stable and well controlled with ibuprofen occasionally." Id. at 120. Petitioner declined flu and zoster vaccinations, and she reported "that a flu vaccine set off her joint issues." Id. at 119.

On August 18, 2021, Petitioner presented to AdventHealth Ocala ED due to fever, cough, and headache for several days. Pet. Ex. 21 at 7-8. Upon examination, Petitioner's labs and chest X-rays were unremarkable but she tested positive (polymerase chain reaction test) for COVID-19. Id. Petitioner was prescribed antibiotics and prednisone 20 mg daily for five days and discharged. Id. at 9.

Petitioner received her first dose of the Moderna COVID-19 vaccine on September 4, 2021, and her second dose on October 2, 2021. Pet. Ex. 3 at 9-10. This was the last medical record submitted by Petitioner.

No additional relevant records were filed.

3. Petitioner's Affidavit

Petitioner filed one affidavit, executed November 15, 2021, addressing the onset of symptoms, her clinical course, and the impact of her condition on her life. Pet. Ex. 1.

Petitioner averred she was in good health, active, and "full of energy" with no chronic medical conditions or medications prior to her November 11, 2018 flu vaccine. Pet. Ex. 1 at ¶ 3.

On November 11, 2018, Petitioner received a flu vaccine at a Publix pharmacy. Pet. Ex. 1 at ¶ 5. Eight days later, on November 19, 2018, Petitioner woke up with "pain all over [her] body" and "swelling in [her] left wrist, shoulders, neck, hips, and ankles." Id. at ¶ 7. She explained that her "pain and stiffness worsened" over the "next few weeks" and she was unable to relieve her pain with heating packs, ice packs, or over-the-counter medication, such as Advil or Tylenol. Id. at ¶¶ 8-9. She "became unable to dress/undress or bathe" herself and "couldn't even hold a glass of water without difficulty." Id. at ¶ 10. Petitioner explained she "had no idea what was happening" and felt "terrified." Id. at ¶ 11.

On December 7, 2018, Petitioner called her PCP for an appointment, and she was seen on December 19, 2018. Pet. Ex. 1 at ¶¶ 12-13. She was prescribed a topical gel and meloxicam; however, she "received no relief from the pain." Id. at ¶ 14. Her doctor then prescribed prednisone which provided "some relief." Id. at ¶¶ 14-15. However, when Petitioner tapered off prednisone, her "joint and muscle pain returned with a vengeance." Id. at ¶ 15. Petitioner returned to her PCP on January 2, 2019. Id. at ¶ 16. Petitioner averred that at the visit, she "mentioned to Dr. Maska that [she] was fine until [she] had the flu vaccine." Id. Petitioner was referred to a rheumatologist, and she made an appointment for March 2019. Id. at ¶ 17.

On January 29, 2019, Petitioner went to the emergency room after experiencing "horrific" pain. Pet. Ex. 1 at ¶ 17. Petitioner explained she was diagnosed with PMR, prescribed another round of steroids, and advised to follow-up with a rheumatologist. Id. The remainder of Petitioner's affidavit largely recounted her clinical course as documented in the medical records.

Id. at ¶¶ 18-25, 27-33. Petitioner noted that at her February 20, 2019 visit, she informed her rheumatologist that she "believed the PMR to be from the flu vaccine because [she] had been completely healthy before getting this vaccine." Id. at ¶ 18. Further, Petitioner stated that after she received her first dose of COVID-19 vaccine on September 4, 2021, her "joint and muscle pain . . . returned." Id. at ¶ 30. She explained that "since having COVID-19 and receiving the COVID-19 vaccine, [her] joint and muscle pain [has been] a daily struggle." Id. at ¶ 32. Finally, Petitioner explained that PMR has impacted both her "physical and mental health" and has "caused a severe strain on [her] marriage." Id. at ¶ 26.

C. Expert Reports

1. Petitioner's Expert, M. Eric Gershwin, M.D.[19]

a. Background and Qualifications

Dr. Gershwin is a Distinguished Professor of Medicine in the Division of Rheumatology, Allergy, and Clinical Immunology at the University of California, Davis School of Medicine. Pet. Ex. 42 at 1. He is board certified in internal medicine, rheumatology, and allergy and clinical immunology. Id. at 2. He completed his M.D. at Stanford University after which he completed an internship and residency in internal medicine at Tufts New England Medical Center and trained in immunology at the National Institutes of Health in Maryland. Id. at 1-2; Pet. Ex. 25 at 2. In his clinical practice, Dr. Gershwin has "seen many hundreds of patients" with PMR over his "50-year medical career." Id. Dr. Gershwin has also conducted "original research in rheumatology, allergy[,] and immunology." Id. Additionally, Dr. Gershwin has held various editor and reviewer positions on medical journals, and he has authored or coauthored over 1,000 publications during his career. Pet. Ex. 42 at 5-143.

b. Opinion

i. Diagnosis

Dr. Gershwin opined Petitioner's "diagnosis of PMR is well established on presentation and in terms of her response to therapy." Pet. Ex. 25 at 11. Dr. Gershwin described PMR as

> a clinical diagnosis which essentially requires people to be 50 years of age or older, have bilateral aching and stiffness of a month or more involving two or more areas of the body, most typically the neck, the shoulders, proximal regions of the arms, hips and proximal areas of the thighs; a[n] [ESR] greater than 40 mm/hour is typical and requires the exclusion of other diagnostic possibilities. Other criteria . . . include a rapid response to a Prednisone dosage of less than 20 mg/day.

[19] Dr. Gershwin submitted two expert reports. Pet. Exs. 25, 27.

Id. at 8 (citing Pet. Ex. 25, Tab 2);[20] see also Pet. Ex. 25, Tab 1 at 1 tbl. 1 (providing two sets of diagnostic criteria for PMR);[21] Pet. Ex. 25, Tab 10 at 3 tbl. 1 (providing the Bird-Wood criteria for PMR);[22] Pet. Ex. 30 at 5 tbl. 1 (providing EULAR/ACR provisional classification criteria for PMR).[23] Dr. Gershwin explained PMR occurs in approximately one of every 133 people over the age of 50 with disease incidence increasing after age 50 and peaking between 70 and 80 years old. Pet. Ex. 25 at 8 (citing Pet. Ex. 25, Tab 1).

Additionally, Salvarani et al. noted distal manifestations, such as "asymmetric peripheral arthritis (predominantly affecting the knees and wrists)," are present in "about of half of cases." Pet. Ex. 25, Tab 1 at 4. "When present, distal-limb symptoms may initially make it difficult to differentiate [PMR] from [RA] or other, similar syndromes." Id. at 9. The authors also noted that physical examination of patients with PMR "reveals little evidence of swelling or tenderness of proximal joints that could account for the patients' often marked symptoms." Id. at 3.

Dr. Gershwin acknowledged "a number of diseases . . . may imitate" PMR. Pet. Ex. 25 at 8. He also noted there was "no gold standard" for the diagnosis of PMR. Pet. Ex. 27 at 1; see also Pet. Ex. 30 at 5 ("There is no gold standard for diagnosing PMR, and . . . diagnosis of PMR can be challenging with a considerable number of conditions in the list of differential diagnoses."). Dr. Gershwin observed PMR is "closely related" to temporal arteritis (i.e. giant cell arteritis ("GCA")).[24] Pet. Ex. 25 at 8. He concluded that Petitioner's diagnosis of PMR "seems well established." Id.

Responding to Dr. Miner's (Respondent's expert) contention that seronegative RA was a more appropriate diagnosis, Dr. Gershwin acknowledged that PMR and seronegative RA have "striking similarities" with around 20% of patients having PMR that "evolves into what appears to be seronegative [RA]." Pet. Ex. 27 at 1. However, Dr. Gershwin opined that whether Petitioner "suffers from a similar and overlapping disease" (i.e., seronegative RA) did not change

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[20] Carlo Salvarani & Francesco Muratore, Clinical Manifestations and Diagnosis of Polymyalgia Rheumatica, UpToDate, https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-polymyalgia-rheumatica (last updated Jan. 13, 2021).

[21] Carlo Salvarani et al., Polymyalgia Rheumatica and Giant-Cell Arteritis, 347 NEJM 261 (2002).

[22] Makoto Wada et al., Giant Cell Arteritis with Polymyalgia Rheumatica Associated with Influenza Vaccination, 38 J. Dermatol. 1099 (2011).

[23] Ingrid E. Lundberg et al., An Update on Polymyalgia Rheumatica, 292 J. Intern. Med. 717 (2022).

[24] GCA "is an immune-mediated chronic vasculitis characterized by granulomatous inflammation in the walls of medium-sized and large arteries." Pet. Ex. 25, Tab 10 at 1. "One of the major complications of GCA is [PMR]" with "[a]proximately 50% of patients with GCA develop[ing] polymyalgic symptoms." Id. at 2.

his opinions in this case. Id. Dr. Gershwin asserted that while seropositive RA and seronegative RA are immunologically distinct disease, seronegative RA and PMR "may both stem from the same pathology." Id. (citing Pet. Ex. 28;[25] Pet. Ex. 29).[26]

However, the medical literature cited by Dr. Gershwin explained that "while PMR and [seronegative RA] have been considered components of the same disease process [,]" they "must be considered separate diseases with many similarities." Pet. Ex. 29 at 6. Of note, the authors identified different inflammatory cells present in PMR and seronegative RA. Id. at 5 ("PMR is characterised by the presence of macrophages and T lymphocytes, with few neutrophils, and no B cells or natural killer (NK) cells . . . [while] NK cells and B cells are present in the synovial fluids of [seronegative] RA patients.").

ii. Althen Prong One

Broadly, Dr. Gershwin opined that PMR can be caused by the flu vaccine triggering a local inflammatory response, followed by a systemic response in a genetically susceptible individual. Dr. Gershwin's immunological theory implicated the innate and adaptive immune system, autoinflammatory and autoimmune principles, and components of genetics and immune dysregulation (immunosenescence).

In his first report, Dr. Gershwin characterized PMR as "a disease of the innate immune system." Pet. Ex. 25 at 11. He explained that while the mechanism of PMR "remains enigmatic," it is an inflammatory disease and "is induced in part" by interleukin-6 ("IL-6"). Id. at 9-10. Dr. Gershwin noted that IL-6 is involved in both innate and adaptive immune responses. Id. at 10. And he opined the flu vaccine "may induce cytokine release." Id. (citing Pet. Ex. 25, Tab 19).[27]

Dr. Gershwin opined that Petitioner's vaccine-induced PMR was "consistent with a cytokine-driven process by her innate or first responder immune cells." Pet. Ex. 25 at 11. In his first report, he specifically explained that

> [i]n response to the flu vaccination, there is migration of the vaccine antigen to regional lymph nodes where it is processed by professional antigen presenting cells. This process includes production of cytokines by first responder cells.

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[25] Cengiz Korkmaz & Pinar Yildiz, Giant Cell Arteritis, Polymyalgia Rheumatica, and Late-Onset Rheumatoid Arthritis: Can They Be Components of a Single Disease Process in Elderly Patients?, 4 Eur. J. Rheumatol. 157 (2017).

[26] Ciro Manzo & Amir Emamifar, Polymyalgia Rheumatica and Seronegative Elderly-Onset Rheumatoid Arthritis: Two Different Diseases with Many Similarities, 4 Eur. Med. J. 111 (2019). This was also filed as Resp. Ex. B, Tab 1.

[27] Caroline Hervé et al., The How's and What's of Vaccine Reactogenicity, 4 NPJ Vaccines 39 (2019). This was also filed as Pet. Ex. 34.

> However, as in other patients with PMR, the effect of first responder cells, is on the articular tissue and muscles. There is an enormous diversity of the human immune response that makes events probable, although rare. PMR is a disease of the innate immune system.

Id. at 11.

Dr. Gershwin also analogized the pathogenesis of PMR to GCA and observed the diseases were "closely related." Pet. Ex. 25 at 8. Dr. Gershwin provided a lengthy excerpt discussing the role of T cells, NK cells and B cells in the pathogenesis of both PMR and GCA. Id. at 8-9 (quoting Pet. Ex. 25, Tab 11 at 2).[28]

Further, Dr. Gershwin invoked immunosenescence (i.e., aged related changes in immunity) and genetic susceptibility as relevant to the development of PMR following vaccination. Pet. Ex. 25 at 9-10. Dr. Gershwin asserted there "is an enormous degree of genetic variation in the immune response." Id. at 9. He then discussed "studies of the genetic basis of immune activation following wild type measles vaccination in healthy individuals." Id. Petitioner did not receive a measles vaccine.

In his second report, Dr. Gershwin modified his immunological theory by including references to TH17 cells[29] and adaptive immunity. Pet. Ex. 27. He explained that

> PMR is initiated by an excessive innate immune response in a genetically susceptible host; a host that is "normally" immune dysregulated as part of aging or immunosenescence. PMR never occurs in young patients. Over time . . . an adaptive response becomes part of the pathologic process. TH17 cells are a limb of adaptive immunity.

Id. at 1.

In support of his modified theory, he cited a recent paper by Hysa et al.[30] Pet. Ex. 27 at 1 (citing Pet. Ex. 31). Hysa et al. conducted a literature search for publications related to immune

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[28] Miguel Gonzalez-Gay & Trinitario Pina, Giant Cell Arteritis and Polymyalgia Rheumatica: An Update, 17 Curr. Rheuatol. Rep. 1 (2015).

[29] TH17 are a type of differentiated T helper cells that "promote tissue inflammation." Peter J. Delves, Cellular Components of the Immune System, Merck Manual, https://www.merck manuals.com/professional/immunology-allergic-disorders/biology-of-the-immune-system/cellular-components-of-the-immune-system (last visited Mar. 18, 2026). T helper cells "secrete[] several cytokines" with TH17 producing IL-17, IL-21, and IL-22. Id.

[30] Elvis Hysa et al., Immune System Activation in Polymyalgia Rheumatica: Which Balance Between Autoinflammation and Autoimmunity? A Systematic Review, 21 Autoimmun. Rev. 102995 (2022).

system activation in PMR and identified 52 papers which were reviewed and summarized. Pet. Ex. 31 at 2. At the outset, Hysa et al. acknowledged that the "pathophysiological mechanisms of PMR are only partially understood." Id. The paper discussed numerous mechanisms. Id. at 2-5.

Relevant to the innate immune system, the following pathological mechanisms of the innate immune system were reported by Hysa et al.: "hyper-activity of neutrophils and monocytes, expressing toll-like receptor 7 in active disease, . . . impaired phagocytosis and endothelial dysfunction," and elevated innate T cells. Pet. Ex. 31 at 1-3. Regarding cytokines, elevated levels of IL-6 were reported. See id. at 4, 6.

Relative to the adaptive immune system, abnormalities were described by Hysa et al. in the way that T cells were "polarized . . . towards T helper 1 and 17 phenotypes," there was evidence of immunosenescence and a "downregulated immunoregulatory response" as well as alteration of peripheral B cells. Pet. Ex. 31 at 1, 3-4. The authors noted that "very recent data suggest that B cells might play a key role in PMR pathophysiology." Id. at 6. Interaction between innate and adaptive immune response "was documented by synovial infiltration of macrophages and T cells." Id. at 1. Multiple autoantibodies were detected in patients with PMR, but none of them correlated with disease or antigens from "environmental triggers." Id.

At the beginning of the paper, Hysa et al. questioned whether PMR is an autoinflammatory or autoimmune condition. Pet. Ex. 31 at 1-2. They concluded that PMR "might be regarded as an inflammatory immune-mediated disease with mixed mechanisms in a background of genetic and epigenetic factors." Id. at 7. Further research was recommended to determine whether the "base of the disease is elicited by specific triggers on a predisposing setting." Id.

Due to their inability to better address the pathogenesis of PMR and its immune processes, which they described as "still hidden," Hysa et al. identified five unanswered questions:

> [1] Are the detected peripheral innate cells active key players or the reflection of systemic inflammation in PMR?
> [2] To what extent [do] innate cells influence T- and B-cell activation in PMR pathogenesis?
> [3] How central is T cell autoreactivity in PMR development considering the associations of PMR with ageing and immune checkpoint inhibitors?
> [4] If B cells do not produce pathogenic antibodies, how do they participate in PMR active disease?
> [5] Are the detection of peripheral autoantibodies and local interferon-associated proteins hints that PMR is an antigen-induced disease?

Pet. Ex. 31 at 7. Hysa et al. did not conclude that PMR was driven by a cytokine induced mechanism or identify vaccines as a potential environmental trigger. See id.

Addressing the role of antigens in the development of PMR, Dr. Gershwin agreed "that no autoantigen has been identified." Pet. Ex. 27 at 1. However, he opined that "the footprint left

by the immune system argues there is a continued role for adaptive immunity." Id. (citing Pet. Ex. 31).

Turning back to his cytokine theory, Dr. Gershwin described how "vaccination leads to cytokine release." Pet. Ex. 27 at 7-10. He provided lengthy excerpts from Chatziandreou et al.,[31] Hervé et al., and Wu et al.[32] that discussed the concepts of cytokine storms, systemic reactogenicity, and loss of tolerance. Id. (citing Pet. Exs. 33-35). This part of Dr. Gershwin's report was difficult to follow and the references not on point. Overall, the references provide general information about cytokines and their role in immune responses, but they do not provide support for the proposition that the flu vaccination can induce cytokines to cause disease pathology that presents as PMR. Chatziandreou et al. did not suggest that the findings relative to cytokines induce pathology presenting as PMR. See Pet. Ex. 33. Reactogenicity, the "subset of reactions that occur soon after vaccination," described by Hervé et al., includes manifestations like injection-site pain and redness, as well as general symptoms of headache, fever, and fatigue. Pet. Ex. 34 at 1. Hervé et al. did not describe how cytokine induction following administration of the flu vaccine can cause PMR. See id. Wu et al. discussed "paradigms by which cytokines maintain or break immune tolerance." Pet. Ex. 35 at 1. The paper did not suggest that flu vaccinations play a role in cytokines leading to a "break in immune tolerance" or "dysregulated immune system." See id. Further, Dr. Gershwin did not provide foundational evidence demonstrating that these articles were relevant to Petitioner's condition. For example, while Dr. Gershwins described the concept of cytokine storms, there is no evidence that Petitioner experienced cytokine storm,[33] a severe life-threatening event. Pet. Ex. 27 at 8-9.

Responding to Dr. Miner's assertion that IL-6 increases after vaccination but "falls several days later," Dr. Gershwin asserted that Dr. Miner "fail[ed] to distinguish the imitation of an immune response from the perpetual and sustained cytokine dysregulation that occurs because of the onset of disease." Pet. Ex. 27 at 4. Dr. Gershwin cited to Roth et al.[34] in support. Id. (citing Pet. Ex. 32). Similar to the articles discussed above, Roth et al. provided general information about cytokines and their role in immune responses to vaccination. See Pet. Ex. 32.

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[31] Nikolaos Chatziandreou et al., Macrophage Death Following Influenza Vaccination Initiates the Inflammatory Response That Promotes Dendritic Cell Function in the Draining Lymph Node, 18 Cell Rep. 2427 (2017).

[32] Jie Wu et al., Cytokine Regulation of Immune Tolerance, 2 Burns & Trauma 11 (2014).

[33] Cytokine storm refers to "a profound systemic oversecretion of cytokines" resulting in "local tissue and organ damage, and systemic symptoms." Inst. of Med., Adverse Events Associated with Childhood Vaccines: Evidence and Causality 75 (Kathleen Stratton et al. eds., 2012). Cytokine storms "may follow infections or other types of massive immune activation including bacterial sepsis, avian [flu], acute respiratory distress syndrome, hemophagocytic lymphohistiocytosis, and macrophage activation syndrome." Id. at 75-76.

[34] Gillie A. Roth et al., Designing Spatial and Temporal Control of Vaccine Responses, 7 Nat. Rev. Mater. 174 (2022).

The authors state that "[i]mmediately following vaccine administration, local innate cells release cytokines into the circulation to enable a coordinated response." Id. at 4 fig. 1. Adjuvants and their effect on cytokine production are discussed, but the flu vaccine here did not contain an adjuvant.

Additionally, Dr. Gershwin provided several case reports of PMR and/or GCA following vaccination. Pet. Ex. 25 at 9 (citing Pet. Ex. 25, Tabs 3-10).[35]

Brown and Bertough,[36] an older article published in 1994, reported on three patients, one of which developed PMR after vaccination (the other two patients were diagnosed with systemic lupus and RA). Pet. Ex. 25, Tab 3 at 1. The PMR patient developed symptoms three weeks after flu vaccination. Id. He began treatment with prednisone, which was later weaned and discontinued, and his condition resolved within nine months after which he had no symptoms and required no treatment. Id. The authors did not offer a causal theory to explain how the flu vaccine could cause this reaction, and they did not discuss cytokines.

Soriano et al.[37] described 10 female patients who developed PMR/GCA, or both, following flu vaccination in Italy. Pet. Ex. 25, Tab 9 at 2, 2 tbl. 1. Two were diagnosed with PMR. Id. One patient developed PMR one month after flu vaccination, while another patient had onset two months after flu vaccination and experienced a PMR relapse two weeks after a subsequent flu vaccination two years later. Id. Regarding causal mechanisms, the authors discussed adjuvants as potential "triggers of autoimmunity" and suggested that adjuvants could be "a possible expression" of autoimmunity/inflammatory syndrome induced by adjuvants

[35] The paper by Saadoun et al. is in French (only the abstract is in English); thus, the undersigned is unable to review it or verify the information contained therein. See Pet. Ex. 25, Tab 8 (D. Saadoun, Vascularites Postvaccinales: À Propos de Trois Observations, 22 Rev. Méd. Interne 172 (2001)). Therefore, it is not discussed.

[36] M.A. Brown & J.V. Bertouch, Rheumatic Complications of Influenza Vaccination, 24 Aust. NZ J. Med. 572 (1994).

[37] A. Soriano et al., Giant Cell Arteritis and Polymyalgia Rheumatica After Influenza Vaccination: Report of 10 Cases and Review of Literature, 21 Lupus 153 (2012). Of note, Soriano et al. also provided a table of cases reported in the literature. See Pet. Ex. 25, Tab 9 at 4, tbl. 2. Some of the papers identified have been filed in this case and are discussed herein.

("ASIA").[38] Id. at 1, 4. Again, a theory based on adjuvants lacks foundational relevance here, since there is no evidence that the flu vaccine at issue contained an adjuvant.[39]

Liozon et al.[40] described a 91-year-old-women who developed PMR approximately two weeks after flu vaccination. Pet. Ex. 25, Tab 4 at 2. She developed high fever, chills, rash and fatigue the day after flu vaccination (her first flu vaccine). Id. Urine culture was positive, and antibiotics were prescribed. Id. She continued to have fever, and 13 days after vaccination developed muscle pain, joint stiffness, and reduced range of motion and "pulsatile temporal arteries." Id. Inflammatory markers ESR and CRP were elevated. Id. She was diagnosed with PMR and treated with prednisone, which was later successfully tapered over the following three-month period. Id. The mechanism proposed was an "interaction between vaccine (or egg protein)[41] antigens and native antibodies [which] may have generated immune complexes which led to the vasculitis syndrome." Id.; see also Pet. Ex. 25, Tab 6 at 12 (reporting on a case of PMR following flu vaccination and theorizing that "antigenic properties of [flu vaccine] (viral or egg protein) interacting with native antibodies might induce an abnormal immune response, generating immunocomplexes that could lead to vessel inflammation"). The authors concluded that the flu vaccine was "possibly" associated with PMR. Pet. Ex. 25, Tab 4 at 2.

A second article by Liozon et al.[42] described a systematic literature review from 1994 to 2014 and identified 13 patients[43] who developed PMR following flu vaccination. Pet. Ex. 25, Tab 20 at 5. Like Soriano et al., the authors suggested that post-flu vaccine PMR could represent

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[38] Special masters have routinely rejected ASIA as a "credible or scientifically-reliable casual theory." Trollinger v. Sec'y of Health & Hum. Servs., No. 16-473V, 2023 WL 2521912, at *10 n.20 (Fed. Cl. Spec. Mstr. Feb. 17, 2023); Monzon v. Sec'y of Health & Hum. Servs., No. 17-1055V, 2021 WL 2711289, at *8 n.6 (Fed. Cl. Spec. Mstr. June 2, 2021) ("The ASIA theory for adjuvant-induced autoimmunity has never been deemed medically reliable in any prior Program cases."); Suliman v. Sec'y of Health & Hum. Servs., No. 13-993V, 2018 WL 6803697, at *27 (Fed. Cl. Spec. Mstr. Nov. 27, 2018) ("No special masters have ever found ASIA or ASIA-like theories to be persuasive.").

[39] Flucelvax 2018-2019 Formula was an inactivated vaccine, containing four flu strains. Pet. Ex. 41 at 1, 10-11. There is no indication that it contained an adjuvant. See id.

[40] Eric Liozon et al., Polymyalgia Rheumatica Following Influenza Vaccination, 48 J. Am. Geriatr. Soc. 1533 (2000).

[41] The vaccine at issue here was not derived from egg protein. See Pet. Ex. 41 at 10-11.

[42] Eric Liozon et al., Giant Cell Arteritis or Polymyalgia Rheumatica After Influenza Vaccination: A Study of 12 Patients and a Literature Review, 20 Autoimmun. Rev. 102732 (2021).

[43] These patients include cases reported in medical articles, some of which are duplicative of the other case reports described herein. See Pet. Ex. 25, Tab 20 at 4 tbl. 3.

a form of the ASIA syndrome (with "an external trigger such as an adjuvant."). Id. at 5-6. They also questioned the role of the HLA DRB1*13:01 allele.[44] Id.

Dr. Gershwin also noted there was a case report of PMR relapse following flu vaccination. Pet. Ex. 25 at 11 (citing Pet. Ex. 25, Tab 16).[45] Bassendine and Bridge described a woman with PMR, described as severe due to very elevated CRP (141.7 mg/L) who was in remission but suffered a relapse following receipt of a flu vaccine that contained an adjuvant. Pet. Ex. 25, Tab 16 at 2. The authors suggested that the ASIA syndrome may be the causal mechanism. Id.

Other case reports discussed either GCA or PMR in association with GCA. For example, Wada et al. reported on a previously healthy 70-year-old women developing GCA with PMR one day after flu vaccination. Pet. Ex. 25, Tab 10 at 3; see also Pet. Ex. 25, Tab 7 (reporting a case of GCA three weeks after flu vaccination).[46] Finally, Mader et al. reported on three cases of systemic vasculitis following flu vaccination. Pet. Ex. 25, Tab 5 at 1.[47]

Additionally, Dr. Gershwin reported that in a "large series of nearly 1,800 adverse events, PMR was found in 9.2% of patients." Pet. Ex. 25 at 11 (citing Pet. Ex. 25, Tab 15).[48] Felicetti et al. provided an overview of vasculitis as an adverse event reported in three databases, including

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[44] "The human leukocyte antigen (HLA) system (the major histocompatibility complex [MHC] in humans) is an important part of the immune system and is controlled by genes located on chromosome 6." Peter J. Delves, Human Leakocyte Antigen (HLA) System, Merck Manual, https://www.merckmanuals.com/professional/immunology-allergic-disorders/biology-of-the-immune-system/human-leukocyte-antigen-hla-system (last visited Mar. 19, 2026). HLA alleles "defined by DNA sequencing are named to identify the gene, followed by an asterisk, numbers representing the allele group (often corresponding to the serologic antigen encoded by that allele), a colon, and numbers representing the specific allele" with "[s]ome autoimmune disorders are linked to specific HLA alleles." Id.

[45] Margaret Bassendine & Simon Bridge, Relapse of Polymyalgia Rheumatica Following Adjuvanted Influenza Vaccine: A Case-Based Review, 7 Eur. J. Rheumatol. 37 (2020).

[46] Carlos Perez et al., Giant Cell Arteritis After Influenza Vaccination, 160 Arch. Intern. Med. 2677 (2000).

[47] Reuven Mader et al., Systemic Vasculitis Following Influenza Vaccination: Report of 3 Cases and Literature Review, 20 J. Rheumatol. 1429 (1993).

[48] Patrizia Felicetti et al., Spontaneous Reports of Vasculitis As an Adverse Event Following Immunization: A Descriptive Analysis Across Three International Databases, 34 Vaccine 6634 (2016).

the Vaccine Adverse Event Reporting System ("VAERS").[49] Pet. Ex. 25, Tab 15 at 1. Reports of vasculitis following vaccinations (not limited to flu vaccines) between 2003 and 2014 were reviewed. Id. Just more than half of the reports were in children. Id. at 2-3. The most common types of vasculitis reported were Henock-Schoenlein Purpura and Kawasaki Disease, reported at 19.1% and 16.1%, respectively. Id. at 3. PMR made up 9.2% of the reports. Id.

Finally, Dr. Gershwin discussed a "recent retrospective study" from Falsetti et al.[50] Pet. Ex. 27 at 10 (citing Pet. Ex. 36). The authors reviewed 58 cases of PMR from a single rheumatology care setting in Italy between 2003 and 2017. Pet. Ex. 36 at 2. The authors noted that over the 20-year study period, 26% of their patients (15 patients) had a connection with an environmental trigger, including six who had recently received a vaccine. Id. Of these, four received a flu vaccine and two received tetanus toxoid vaccines. Id. at 3. The authors suggested immune senescence and "genetic polymorphisms" could be explain the link between vaccination and PMR/GCA. Id. at 2. The authors also referenced "innate immune dysregulation in the elderly, with excessive and prolonged production of pro-inflammatory cytokines." Id. The authors concluded that given "the notably high number of years encompassed by our retrospective analysis, a direct correlation with any specific subtype of vaccine, which varies year per year, can be excluded." Id. at 4. The authors recommended further study but concluded that in spite of progress in the treatment of PMR/GCA, the "etiology remains an intriguing matter of debate." Id.

Addressing epidemiological studies, Dr. Gershwin opined that any "[e]pidemiologic studies to specifically address PMR and flu vaccine would have to include a sufficient number of subjects in [Petitioner's] age group and have sufficient power to address rare events." Pet. Ex. 25 at 10. In his second report, Dr. Gershwin provided a lengthy power calculation to illustrate the concept that very large sample sizes (such as 4,669,012 subjects over the age of 50) would be needed for statistically significant detection of vaccination as an "environmental factor that can stimulate . . . PMR." Pet. Ex. 27 at 4-6. He explained that "classical epidemiology" will "fail without appropriate power calculations." Id. at 4. He further opined "[e]pidemiology is forced to overlook the potential interaction where multiple factors could interact in a non-linear manner to influence the development of PMR." Id. at 6.

In both of his expert reports, Dr. Gershwin acknowledged that the cause and immunological mechanism of PMR are not known. Pet. Ex. 25 at 10; Pet. Ex. 27 at 1. In addition to the paper by Hysa et al., other articles cited by Dr. Gershwin stated that the cause of

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[49] The Vaccine Adverse Event Reporting System, or VAERS, is "a national early warning system to detect possible safety problems in U.S.-licensed vaccines. . . . . VAERS is a passive reporting system . . . [and] is not designed to determine if a vaccine caused a health problem, but is especially useful for detecting unusual or unexpected patterns of adverse event reporting that might indicate a possible safety problem with a vaccine." About VAERS, U.S. Dep't Health & Hum. Servs., https://vaers.hhs.gov/about.html (last visited Mar. 17, 2026).

[50] Paolo Falsetti et al., Polymyalgia Rheumatica Following Infective Triggers of Vaccinations: A Different Subset of Disease?, 58 Reumatologia 76 (2020).

PMR is not known. See, e.g., Pet. Ex. 25, Tab 2 at 2 ("The cause of [PMR] is unknown."); Pet. Ex. 25, Tab 16 at 1 ("The etiology and pathogenesis of PMR remain obscure."); Pet. Ex. 25, Tab 20 at 1 ("The origin of the disease in unknown."); Pet. Ex. 30 at 12 ("Further studies on pathophysiology are needed to better understand the disease mechanisms.").

iii. Althen Prongs Two and Three

Addressing Althen prong two, Dr. Gershwin opined there was a "logical sequence of cause and effect showing the vaccination was the reason for the injury." Pet. Ex. 25 at 11. He explained,

> Following a vaccination, there is migration of the vaccine antigen to regional lymph nodes where it is processed by professional antigen presenting cells. This process includes production of cytokines, recruitment, and activation of both antigen specific and antigenic non-specific immune cells. Such cells migrate throughout the body as part of the body's systemic ability to protect itself. Antibodies are produced, such a sequence is normal and part of the vaccination process. It was not different in the case of [Petitioner] as anyone else. However, it was the abnormal innate immune response that targeted its articular tissue and muscles that was unique to [Petitioner].

Id. He noted this "abnormal activation occurred" after her flu vaccination "without any other antecedent immunological challenges" such as an infection. Id.

Turning to Althen prong three, in his first report, Dr. Gershwin opined that Petitioner had an "innate immune response approximately one week after vaccination." Pet. Ex. 25 at 11. He stated that the "kinetics of appearance of her complaints are consistent with a normal timing of an immune response following a vaccination." Id.

Responding to Dr. Miner's criticism that a one-week onset was inconsistent with an innate immune response, Dr. Gershwin agreed that "innate responses occur rapidly, within several days." Pet. Ex. 27 at 3. However, Dr. Gershwin opined that innate immune responses "do not peak immediately" and asserted "the onset of an innate response is not the same an initiation of the first clinical signs and symptoms." Id. at 3-4. Additionally, while Dr. Gershwin opined that innate and adaptive immune responses are involved in the immune mechanism of PMR, he did not address the impact of an adaptive immune response as it relates to onset. See id. at 1.

2. Respondent's Expert, Jonathan Miner, M.D., Ph.D.[51]

a. Background and Qualifications

Dr. Miner is an Associate Professor of Medicine in the Division of Rheumatology at University of Pennsylvania, Perelman School of Medicine with a joint appointment to the

[51] Dr. Miner filed two expert reports. Resp. Exs. A-B.

Department of Microbiology. Resp. Ex. A at 1. He is board certified in rheumatology and internal medicine. Resp. Ex. A, Tab 9 at 2.[52] Dr. Miner received a Ph.D. in biochemistry as well as an M.D. from University of Oklahoma. Id. at 1. He completed an internal medicine residency and rheumatology fellowship at Washington University in St. Louis and Barnes-Jewish Hospital. Id. He also received "extensive training" in microbiology and immunology. Resp. Ex. A at 2. In his clinical practice, he has "cared for many hundreds of patients with rheumatologic diseases." Id. at 3. In addition to his clinical practice, Dr. Miner is also the chair of Gene Therapy and Vaccines Graduate Program at the University of Pennsylvania. Resp. Ex. A, Tab 9 at 1. His research is focused on "the intersection of autoimmunity and antiviral immunity." Resp. Ex. A at 1. Additionally, Dr. Miner has served as an editor or reviewer for several medical journals and he has authored or co-authored numerous publications. Resp. Ex. A, Tab 9 at 2-3, 7-11.

b. Opinion

i. Diagnosis

Dr. Miner opined Petitioner's diagnosis was "more likely" seronegative RA as her clinical history was "more consistent with seronegative RA than with PMR." Resp. Ex. A at 3. While PMR and seronegative RA have overlapping symptoms, Dr. Miner opined they "are distinct diseases." Id.; Resp. Ex. B at 2; see also Pet. Ex. 29 (comparing PMR and seronegative RA).

Dr. Miner noted PMR typically affects patients over 60 years old and involves the shoulder and hip girdle with patients "typically respond[ing] promptly to low doses of prednisone." Resp. Ex. A at 3. Treatment with immunosuppression, such as methotrexate or an antibody that blocks the IL-6 receptor, is sometimes required. Id. Dr. Miner explained that while patients with PMR "can develop stiffness and swelling in their peripheral joints including the hands and wrists, PMR is more often associated with the absence of significant peripheral joint involvement." Id. Accordingly, Dr. Miner opined "[i]nvolvement peripheral joints, including the knees and wrists, argues against a diagnosis of PMR." Id.

RA is "an autoimmune, inflammatory joint disease characterized by swelling, pain, and destruction of synovial joints." Resp. Ex. A, Tab 3 at 2. Unlike PMR, RA "frequently involves the fingers, wrists, knees, and various other peripheral joints, in addition to shoulder and hip pain." Resp. Ex. A at 3. Further, Dr. Miner opined RA tends to "progressively involve additional joints, as was the case here." Id. Next, Dr. Miner explained patients with seronegative RA have negative RF and ACPAs. Id. (citing Resp. Ex. A, Tab 3). Approximately 20% of RA patients have seronegative RA. Id. Due to the similarities of PMR and seronegative RA, retrospective studies have shown that more than 20% of patients diagnosed with PMR are later diagnosed with RA. Resp. Ex. B at 2; see also Pet. Ex. 29 at 6.

[52] Dr. Miner's C.V. is identified as Resp. Ex. A, Tab 9 in Respondent's exhibit list; however, the filed C.V. is labeled as Resp. Ex. B. The undersigned will refer to Dr. Miner's C.V. as Resp. Ex. A, Tab 9.

Finally, Dr. Miner noted that Petitioner's treating rheumatologist, Dr. Otero, included a differential diagnosis of PMR versus seronegative RA "in nearly all of her notes." Resp. Ex. A at 3. For the above discussed reasons, Dr. Miner concluded Petitioner's medical history was "more consistent with seronegative RA than with PMR." Id.

ii. Althen Prong One

While Dr. Miner opined seronegative RA was the appropriate diagnosis in this case, he provided vaccine causation opinions for both diagnoses under consideration—PMR and seronegative RA. Resp. Ex. A at 3.

Dr. Miner summarized the first part of Dr. Gershwin's theory as "invok[ing] IL-6 and an innate immune mechanism as the immunological mechanisms of disease." Resp. Ex. A at 4. Dr. Miner opined that this theory of vaccine-induced PMR or seronegative RA is "speculative." Id. at 5. Specifically, Dr. Miner disagreed with Dr. Gershwin's contention that the flu vaccine could cause "sustained IL-6 elevation" after vaccination. Id. Dr. Miner agreed that IL-6 is thought to be involved in the pathogenesis of both PMR and RA. Id. at 4. However, he disagreed that "vaccines cause prolonged cytokine elevation leading to disease." Resp. Ex. B at 3.

Relying on Tsai et al.,[53] Dr. Miner explained that while the flu vaccine causes "transient elevation of IL-6 levels," these "return to baseline within days." Resp. Ex. A at 4-5 (citing Resp. Ex. A, Tab 7); Resp. Ex. B at 3. The study by Tsai et al. examined six inflammatory markers (including IL-6) following flu vaccination. Resp. Ex. A, Tab 7 at 2. Blood samples were collected from 22 participants on days zero, one, three, and seven. Id. Vaccination had the "most pronounced effect" on IL-6 resulting "in a 58% increase in IL-6 on day [one] from the normalized mean IL-6 baseline measurement; levels then returned to baseline by day [three]." Id. at 4, 4 tbl. 1. The authors concluded that the flu vaccine caused a "small and transient" change in inflammatory markers." Id. at 5. Dr. Miner concluded that Tsai et al. "controverts the claims of prolonged vaccine-induced cytokine elevation." Resp. Ex. B at 3. He further opined that the article demonstrated cytokine elevation following vaccination occurs "at much lower levels than occur in PMR." Id.

Addressing Dr. Gershwin's second report, Dr. Miner criticized Dr. Gershwin's modified theory as circular. Resp. Ex. B at 3. Dr. Miner explained that Dr. Gershwin described elevated prolonged cytokines as both the result of vaccination and the result of PMR itself. Id.

Next, Dr. Miner explained that Dr. Gershwin emphasized B cells are relevant in the cause of PMR and that they are "driven by antigen," implying "that adaptive immunity in being invoked." Resp. Ex. A at 4. Addressing the role of T and B cells, Dr. Miner opined "T cells and B cells as well as antigen-specific immune responses are thought to play a key role" in the pathogenesis of RA as part of an adaptive immune response. Id. However, Dr. Miner

[53] Michael Tsai et al., Effect of Influenza Vaccine on Markers of Inflammation and Lipid Profile, 145 J. Lab. & Clin. Med. 323 (2005).

questioned the role of T cells and B cells in the pathogenesis of PMR. Id. He explained that while "B cell depletion and blockade of T cell co-stimulation are commonly used and effective in the treatment of RA[,]" these same treatments are not thought to be effective against PMR. Id.

Dr. Miner also asserted that the underlying immunological mechanism in this particular case could not be determined as Petitioner was not prescribed targeted therapies against IL-6 receptors, T cells, or B cells. Resp. Ex. A at 3. He further opined that the "[e]ffectiveness of immune-targeted therapies can highlight the role of a particular immunological factor of in disease pathogenesis and therefore support or undermine an immunological theory of causation." Resp. Ex. B at 2. For example, "[i]f B cells were key players in PMR, then B cell depletion could be used to treat the disease, as is done in RA." Id. However, B cell depletion is not approved for the treatment for PMR. Id.

Next, Dr. Miner criticized the power calculation provided by Dr. Gershwin. Resp. Ex. B at 3. First, Dr. Miner challenged that assumption that "achieving statistical significance equates to a clinically relevant finding." Id. Next, Dr. Miner argued that Dr. Gershwin's power calculation "assume[d] that each risk factor operates independently" when "epidemiological evidence demonstrates that health outcomes result from complex interactions among multiple factors, and not from isolated risks." Id. Finally, Dr. Miner opined that Dr. Gershwin's premise (that large sample sizes are needed to detect a weak effect) was "moot" as there is "no strong evidence linking vaccination to PMR or RA." Id. at 4.

Turning to the medical literature, Dr. Miner stated he "performed numerous, repeated searches of the medical literature for associations between [flu] vaccinations and RA or PMR" and concluded there was "no strong basis in the medical literature to invoke vaccination" as casual. Resp. Ex. A at 4-5. Further, Dr. Miner criticized Dr. Gershwin's reliance on Falsetti et al. and opined it was "not compelling evidence for a link between" vaccination and disease. Resp. Ex. B at 1. Falsetti et al. reported on six out of 58 patients with PMR who described vaccination as a "environmental trigger" for their disease. Id. Dr. Miner explained that patients often "misattribute their disease" and underestimate the role of "strong and established risk factors." Id. Next, Dr. Miner noted that in a cohort of 58 patients with PMR who are in an age range that receive one to two seasonal vaccines per year, six or more patients would be expected to coincidentally receive a vaccine in the weeks or months before developing disease. Id. Thus, Dr. Miner concluded the paper did "not provide meaningful support for the idea that a vaccine is likely to cause" PMR. Id. at 2.

Finally, Dr. Miner opined that an association between flu vaccine and PMR or RA was "not accepted by the rheumatology community." Resp. Ex. A at 3-4, 5. Dr. Miner noted vaccination is recommend for patients with a history of inflammatory arthritis, such as PMR or RA. Resp. Ex. B at 3; Resp. Ex. A at 3-4 (citing Resp. Ex. A, Tab 6).[54] Dr. Miner concluded that the recommendation that patients with existing PMR receive seasonal flu vaccines "argues

[54] Anne E. Bass et al., 2022 American College of Rheumatology Guideline for Vaccinations in Patients with Rheumatic and Musculoskeletal Diseases, 75 Arthritis & Rheumatol. 333 (2023).

strongly against the notion that vaccines can induce chronic arthritic condition" such as PMR. Resp. Ex. A at 5.

iii. Althen Prongs Two and Three

Dr. Miner disagreed that vaccination "cause[d] disease in this case." Resp. Ex. B at 4. Instead, he opined that Petitioner had genetic risk factors, including her family history of RA, SLE, and Sjögren's syndrome. Resp. Ex. A at 3.

In addition to genetic risk factors due to her family history, Dr. Miner identified smoking as a risk factor, noting that Petitioner had a prior 30-year history of smoking. Resp. Ex. A at 3 (citing Pet. Ex. 4 at 14); Resp. Ex. B at 1, 3-4. In support, Dr. Miner cited Kang et al.,[55] who analyzed data from two ongoing longitudinal cohort studies and identified 574 cases of PMR in the 69,109 study participants. Resp. Ex. A, Tab 5 at 1. While there was no associated risk with a history of any smoking or current smoking, the authors found a 20-year smoking history was "associated with modestly higher risk of incident PMR among women." Id. at 1-2.

Further, Dr. Miner opined that smoking (unlike vaccination) is associated with chronically elevated IL-6. Resp. Ex. A at 5 (citing Resp. Ex. A, Tab 8).[56] Using data from a cohort of patients with head and neck cancer, Cottin et al. identified eight factors (including tobacco use) independently associated with serum IL-6 levels. Resp. Ex. A, Tab 8 at 1. The authors concluded "[t]he dose-response relationship between lifetime smoking and IL-6 serum levels suggested a causal role of tobacco exposure on IL-6 production." Id. "IL-6 serum levels increased with longer duration of cigarette smoking. . . . Among former cigarette smokers, IL-6 levels decreased with the number of years since quitting." Id. at 4; see also id. at 7 tbl. 4 (results of a "multivariate analyses showing lifetime cigarette consumption variables associated with IL-6 serum levels").

Addressing Althen prong three, Dr. Miner opined that Petitioner's one week onset was "inconsistent" with Dr. Gershwin's proposed immunological theory. Resp. Ex. A at 4. Dr. Miner explained that innate response occurs "immediately or within a few days." Id. He again noted that Tsai et al. showed IL-6 returned to base levels within three days of vaccination rather than remaining elevated or peaking one week after vaccination as would be expected under Dr. Gershwin's proposed theory. Id. (citing Resp. Ex. A, Tab 7). Dr. Miner explained a seven-day onset would be more consistent with an antibody or adaptive response than with an innate response. Id. at 5.

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[55] Jae Hee Kang et al., Cohort Study of Cigarette Smoking and the Risk of Developing Polymyalgia Rheumatica Among Women [abstract], 74 Arthritis & Rhematol. Suppl. 9 (2022).

[56] Sylvine Carrondo Cottin et al., Predictors of Circulating Interleukin-6 Levels in Head and Neck Cancer Patients, 3 Cancers Head & Neck 1 (2018).

II. LEGAL FRAMEWORK

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, a petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding her claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

Medical records, specifically contemporaneous medical records, are presumed to be accurate and generally "warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992)), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. den'd, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to §

13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.) but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that she suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege she suffered a Table Injury, she must prove a vaccine actually caused her injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in her favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

III. ANALYSIS

A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury[,]" determining facts relating to the claimed injury can be significant in a case where diagnosis is not clear. Id. Here, the parties dispute diagnosis, and so it is appropriate to first resolve that issue.

The undersigned finds preponderant evidence to support a finding that Petitioner's diagnosis is PMR. While medical records show that while both PMR and seronegative RA were considered as differential diagnoses, the weight of the evidence tilts in favor of an ultimate diagnosis of PMR. In February, April, and June 2019, Petitioner's treating rheumatologist, Dr. Otero, assessed Petitioner with a history "very suggestive of PMR," but noted that seronegative RA could not be ruled out. See Pet. Ex. 2 at 22, 25, 30. The phrase "very suggestive" implies that Dr. Otero favored the diagnosis of PMR over seronegative RA.

Subsequently, Petitioner saw her PCP, Dr. Maska, and after June 2019, her condition was consistently referred to as PMR. In August 2019, Dr. Maska documented Petitioner had weaned herself off her medication for PMR consistent with the plan agreed to by her rheumatologist. See Pet. Ex. 2 at 62. Dr. Maska agreed with Petitioner's decision to discontinue prednisone and methotrexate. Id. at 64. In November 2019, Dr. Maska wrote a letter confirming Petitioner's diagnosis of PMR. When Dr. Maska saw Petitioner for her annual visit in December 2020, Petitioner's PMR was referenced, and her symptoms were "stable and well controlled" with ibuprofen. Pet. Ex. 9 at 120.

In summary, in the Spring of 2019, Petitioner's rheumatologist characterized Petitioner's presentation as "very suggestive" of PMR. After June 2019, Dr. Maska consistently referred to Petitioner's diagnosis as PMR.

Here, the undersigned gives weight to the statements of Petitioner's treating physicians as they are "in the best position" to determine Petitioner's injury. See Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326; Cucuras, 993 F.2d at 1528 (noting contemporaneous medical records, "in general, warrant consideration as trustworthy evidence").

Petitioner's expert, Dr. Gershwin, opines that Petitioner's diagnosis is PMR. Dr. Gershwin's opinions are consistent with those above, as set forth in Petitioner's medical records. Respondent's expert, Dr. Miner, opined that Petitioner's symptoms were more consistent with seronegative RA. The undersigned resolves this dispute by considering all the records, including the later records by Dr. Maska, the fact that Petitioner was able to discontinue her medications, and because she was not later diagnosed with RA (as occurs in about 20% of patients initially diagnosed with PMR). Thus, the undersigned finds Petitioner has shown by preponderant evidence that her diagnosis is PMR.

B. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds that Petitioner has failed to prove a causal mechanism by preponderant evidence as required under Althen prong one for the following reasons.

First, although Dr. Gershwin's reports are difficult to follow, in his first report, he advances a theory implicating cytokine production, especially IL-6, in the context of genetic variation rendering an individual susceptible to developing pathology. Dr. Gershwin opines cytokine production "reaches its peak within [five to seven] days of vaccination in a normal host." Pet. Ex. 25 at 10. However, Dr. Gershwin did not provide evidence that cytokines, especially IL-6, induced by the flu vaccine can cause pathology. Regarding this part of the theory, the undersigned finds Dr. Miner's opinions more persuasive. While Dr. Miner agrees that IL-6 is involved in the pathogenesis of PMR, the flu vaccine induces only transient and not sustained increases in cytokines. Dr. Miner's supports his opinion with the findings described by Tsai et al., showing IL-6 peaks on day one and returns to baseline by day three. Petitioner did not refute this evidence. In response, Dr. Gershwin suggests that PMR is initiated by "an excessive innate immune response" but he does not provide foundational support for this proposition as it relates to flu vaccination. He cites Roth et al., which describes the roles played by cytokines after vaccination. However, the paper does not support Dr. Gershwin's opinions or but instead bolsters Dr. Miner's opinions and the findings reported in Tsai et al. Based on a review of all the evidence filed herein, the undersigned does not find preponderant evidence supports Petitioner's cytokine theory of vaccine-induced PMR.

Second, the undersigned's finding in regarding Petitioner's cytokine theory is consistent with prior Vaccine Act cases. Other special masters have also rejected the same or similar cytokine theories for a wide range of alleged injuries, including PMR. See, e.g., Giesbrecht v. Sec'y of Health & Hum. Servs., No. 16-1338V, 2023 WL 2721578, at *7 (Fed. Cl. Spec. Mstr. Mar. 30, 2023) (rejecting Dr. Gershwin's theory that "an innate immune response involving cytokine production" caused the petitioner's PMR); Sciortino v. Sec'y of Health & Hum. Servs.,

No. 22-99V, 2024 WL 4579389, at *13 (Fed. Cl. Spec. Mstr. July 24, 2024) (finding Dr. Gershwin's proposed cytokine driven process unpersuasive and denying petitioner's vaccine-induced PMR claim); Landis v. Sec'y of Health & Hum. Servs., No. 15-1562V, 2019 WL 7844617, at *11 (Fed. Cl. Spec. Mstr. Aug. 20, 2019) (citing cases and rejecting theory that cytokines cause osteoarthritis); Baron v. Sec'y of Health & Hum. Servs., No. 14-341V, 2019 WL 2273484, at *18-19 (Fed. Cl. Spec. Mstr. Mar. 18, 2019) (rejecting theory that cytokines cause anti-NMDA encephalitis); Langley v. Sec'y of Health & Hum. Servs., No. 17-837V, 2022 WL 897959, at *15 (Fed. Cl. Spec. Mstr. Mar. 3, 2022) (rejecting theory that cytokines can cause an anxiety disorder and citing cases).

Specific to the flu vaccine and PMR, there are two cases on point. See Giesbrecht, 2023 WL 2721578; Sciortino, 2024 WL 4579389. In both cases, the petitioners offered Dr. Gershwin as their expert, and like here, he offered a theory based on "an innate immune response involving cytokine production" which was found unpersuasive. Giesbrecht, 2023 WL 2721578, at*7; Sciortino, 2024 WL 4579389, at *9, 13. In Sciortino, the Chief Special Master explained Dr. Gershwin's theory

> relies heavily on a cytokine-drive process that conflates innate and [adaptive] immune phases, but largely focusing on the vaccine's initial stimulation of cytokine production—but without a persuasive or reliable showing that this initial upregulation of cytokines is likely to instigate a disease process that will involve many other aspects of the immune response.

Sciortino, 2024 WL 4579389, at *13. The undersigned agrees with the reasoning and conclusions of the Chief Special Master in Sciortino and finds it applicable to the present case.

The third reason explaining the undersigned's decision relates to opinions set forth in Dr. Gershwin's second expert report, where in addition to the innate immune response, he implicates the adaptive immune response, citing the illustration by Hysa et al. Dr. Gershwin quotes Hysa et al. stating that PMR is "an inflammatory immune-mediated disease with mixed mechanisms in a background of genetic and epigenetic factors together with immunological and endocrine senescence." Pet. Ex. 27 at 1 (citing Pet. Ex. 31 at 1). Hysa et al. discuss a host of mechanisms derived from a systematic literature search. The paper and illustration suggest that PMR is a complex process that begins with an unknown antigen and involving reactive autoantibodies. See Pet. Ex. 31 at 4 fig. 2.

Dr. Gershwin acknowledges there is no evidence of an antigen (and he "agrees that no autoantigen has been identified"), but he opines that "the footprint left by the immune system argues there is a continued role for adaptive immunity." Pet. Ex. 27 at 1. The undersigned finds that Dr. Gershwin does not adequately explain how components of the vaccine, or any antigens in the vaccine, activate the adaptive immune system to trigger PMR.[57]

[57] Of note, in Sciortino, Dr. Gershwin's theory was described as confusing, because while he focused on the induction of a cytokine response, he also opined that PMR is antigen-driven, implicating the adaptive immune response. Sciortino, 2024 WL 4579389, at *13. The same is true here.

Overall, Dr. Gershwin offers a broad theory that implicates both the innate and adaptive immune system, in a genetically susceptible host, who is older and subject to immnosenescence and immune dysregulation. Hysa et al. provides a comprehensive overview of peer reviewed studies and publications related to the pathogenesis of PMR. In conclusion, the authors identify five basic questions which require answers before the cause of PMR can be understood. These unanswered questions may explain why Dr. Gershwin's expert reports are difficult to follow and lack foundational support. The lack of knowledge about the pathogenesis of PMR explains the difficulty faced by Dr. Gershwin in offering a theory. He has culled pieces of information from various studies and tried to put them together to form a theory. However, this approach of casting a broad net renders Dr. Gershwin's opinions less persuasive overall. See Baron, 2019 WL 2273484, at *17 ("Although Petitioners . . . do not need to provide the specific components of the mechanism by which the vaccine[] at issue can cause [the alleged injury], they do need to propose something more than taking a vague 'kitchen sink' approach . . . . Petitioners have listed many possibilities but have not identified a sound and reliable explanation that can be applied to the vaccines and injury in this case.").

Fourth, Dr. Gershwin does not state in his expert reports that he holds his opinions to a preponderant, or more likely than not, standard. His reports are silent as to the applicable standard. Causation must be established by a preponderance of the evidence, and a possible theory or mechanism is insufficient to establish causation by a preponderance of the evidence. See Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Waterman, 123 Fed. Cl. at 573-74 (denying petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard); de Bazan, 539 F.3d at 1315.

The fifth and last reason for the undersigned's finding is that caselaw does not generally support a finding that PMR is a vaccine-related illness. Special masters have routinely found that vaccines, including flu as described above, do not cause PMR. See Sciortino, 2024 WL 4579389, at *1 (denying entitlement in flu/PMR); Wilkinson v. Sec'y of Health & Hum. Servs., No. 18-1829V, 2024 WL 3857696, at *1 (Fed. Cl. Spec. Mstr. July 22, 2024) (same); Giesbrecht, 2023 WL 2721578, at *1 (same); Kelly v. Sec'y of Health & Hum. Servs., No. 17-1475V, 2022 WL 17819157, at*1 (Fed. Cl. Spec. Mstr. Oct. 12, 2022) (same); C.P. v. Sec'y of Health & Hum. Servs., No. 14-917V, 2019 WL 5483621, at *1 (Fed. Cl. Spec. Mstr. Aug. 21, 2019) (denying entitlement in flu/PMR and/or seronegative RA case); Van Dycke v. Sec'y of Health & Hum. Servs., No. 18-106V, 2023 WL 4310701, at *1, *26 (Fed. Cl. Spec. Mstr. June 7, 2023) (denying compensation in a Tdap vaccine and PMR/GCA case and noting that "[w]hile the mechanisms may differ, GCA/PMR has been rejected as a vaccine related injury due to insufficient evidence to support causation"); Munoz v. Sec'y of Health & Hum. Servs., No. 21-1369V, 2024 WL 4113486, at *1 (Fed. Cl. Spec. Mstr. Aug. 12, 2024) (denying Tdap/PMR claim), mot. for rev. denied, 174 Fed. Cl. 276 (2024); Suliman, 2018 WL 6803697, at *1 (same); Thompson v. Sec'y of Health & Hum. Servs., No. 18-1217V, 2023 WL 9053982, at *1 (Fed. Cl. Spec. Mstr. Dec. 5, 2023) (denying a Prevnar/PMR claim).

While the undersigned is not required to agree with her colleagues or find the reasoning of other cases controlling, she finds the above cases are well reasoned and inform her opinion about the present case. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

For all these reasons, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to the first Althen prong.

2. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as 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.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court").

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds that Petitioner has failed to prove Althen prong two by preponderant evidence because her clinical course is not consistent with Dr. Gershwin's theory of cytokine induction of disease. There is no evidence that Petitioner had an abnormal or excessive cytokine response consistent with Dr. Gershwin's cytokine theory. In her affidavit, Petitioner did not describe any symptoms until eight days after vaccination. Tsai et al. shows that after the flu vaccination, IL-6 peaks at day one and returns to baseline at day three. During days one to three post-vaccination, Petitioner did not describe symptoms.

Regarding an alternative cause, Dr. Miner opined that Petitioner had risk factors for PMR, including her family history and 30-year smoking history. He explained that smoking is associated with chronically elevated IL-6 levels. The undersigned reviewed the papers cited in support of Dr. Miner's opinions, noting that Cottin et al. found that "among former smokers, IL-6 levels decreased with the number of years since quitting." Resp. Ex. A, Tab 8 at 1. Petitioner

quit smoking in 2013, and she developed PMR in 2018. While smoking may have increased her risk, based on the evidence here, the undersigned is not able to conclude that Petitioner's history of smoking was a potential cause of her PMR.

Because Petitioner has not proven Althen prong one, and she has not shown that her clinical course was consistent with the theory of induction propounded by Dr. Gershwin, the undersigned finds that she has failed to prove Althen prong two by preponderant evidence.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. 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-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542. Thus, prong three contains two parts. First, Petitioner must establish the "timeframe for which it is medically acceptable to infer causation" and second, they must demonstrate that the onset of the disease occurred in this period. Shapiro, 101 Fed. Cl. at 542-43.

Here, the date of onset is not in dispute. The parties stipulated that Petitioner's PMR began on November 19, 2018, eight days after vaccination. However, the parties dispute whether an eight-day onset is temporally appropriate.

Dr. Gershwin initially opined that Petitioner had an "innate immune response approximately one week after vaccination" which was "consistent with a normal timing of an immune response after vaccination." Pet. Ex. 25 at 11. When this time frame was described by Dr. Miner as inappropriate, Dr. Gershwin agreed that "innate immune responses occur rapidly, within several days." Pet. Ex. 27 at 3. But he countered that "the onset of an innate response is not the same as initiation of the first clinical signs and symptoms." Id. at 3-4. Relying on Tsai et al., Dr. Miner opined that Petitioner's onset was inconsistent with Dr. Gershwin's theory.

Dr. Gershwin advances a cytokine induction theory which both experts agree would occur within several days of vaccination. Based on Tsai et al., IL-6 peaks on day one and returns to baseline on day three. Here, there is no evidence that Petitioner had any symptoms of excessive or aberrant cytokine activation during those days. Although Dr. Gershwin maintains that onset of an innate response is not the same as onset of symptoms, he has not explained how an excessive or aberrant cytokine response would be clinically silent until eight days after vaccination. Therefore, Dr. Gershwin's statement in this regard is not persuasive.

Based on the evidence, the undersigned finds that Petitioner has not proven by preponderant evidence that the onset of her symptoms is consistent with a cytokine-induced innate immune response. Thus, Petitioner has failed to prove Althen prong three.

IV. CONCLUSION

The undersigned extends her sympathy to Petitioner for the pain and suffering she experienced due to her illness. The undersigned's Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master