In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: March 16, 2026

MUHAMMAD ALQULISSI,	*	PUBLISHED
Petitioner,	*	No. 22-1511V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Rheumatoid Arthritis ("RA"); Causation-in-Fact.
Respondent.	*	

Sean Franks Greenwood, The Greenwood Law Firm, Houston, TX, for Petitioner.
Eleanor Hanson, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On October 13, 2022, Muhammad Alqulissi ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that an influenza ("flu") vaccination administered to him on November 11, 2019 "was the cause-in-fact" of his seronegative rheumatoid arthritis ("RA") and elevated erythrocyte sedimentation. Petition at

[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.

Preamble, ¶ 20 (ECF No. 1); Amended ("Am.") Petition at Preamble, ¶ 31 (ECF No. 22).[3] Respondent argued against compensation, stating that "this case is not appropriate for compensation under the terms of the Act." Respondent's Report ("Resp. Rept.") at 1, 6 (ECF No. 36) (emphasis omitted).

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 his flu vaccine caused his seronegative RA, and thus has not satisfied his 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

There are two issues in dispute. The first is whether Petitioner suffered a "medically recognized injury" identified as RA.[5] Joint Status Rept. ("Joint Submission"), filed Jan. 14, 2025, at 1 (ECF No. 73). The second dispute is causation, specifically the parties disagree as to whether Petitioner has provided preponderant evidence of all three Althen prongs. Id. at 1-2.

II. BACKGROUND

A. Medical Terminology

RA is a "systemic autoimmune disease characterized by inflammatory polyarthritis. The hallmark of RA is symmetric synovial proliferation and tenderness in multiple joints, particularly the small joints of the hands and feet." Petitioner's Exhibit ("Pet. Ex.") 55 at 1.[6] Approximately

[3] Petitioner's amended petition added Petitioner's pre-vaccination medical history and removed "seronegative." See Am. Petition.

[4] While the undersigned has reviewed all 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 [s]he does not explicitly reference such evidence in h[er] decision."); Simanski v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), aff'd, 601 F. App'x 982 (Fed. Cir. 2015); 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] Although his petition also alleged that he suffered elevated erythrocyte sedimentation, the parties appropriately refined the alleged injury as RA in their Joint Submission.

[6] Richard D. Brasington & Jonathan J. Miner, Clinical Features of Rheumatoid Arthritis, in Rheumatology 760 (Marc C. Hochberg et al. eds., 7th ed. 2019).

80% of patients with RA test positive for rheumatoid factor ("RF")[7] and/or anti-citrullinated peptide antibodies ("ACPAs").[8] Id.; Pet. Ex. 24 at 1.[9]

Diagnostic criteria promulgated by the American College of Rheumatology ("ACR") and the European League Against Rheumatism ("EULAR") in 2010 assess points based on an algorithm which includes the following findings or serology results: "active synovitis in at least one joint that cannot be better explained by another diagnosis," ACPAs, RF, abnormal inflammatory markers (erythrocyte sedimentation rate ("ESR")[10] and C-reactive protein ("CRP")),[11] and duration of symptoms of six weeks or greater. Pet. Ex. 23 at 1, 2 tbl.92.1.[12]

[7] RF antibodies are "antibodies directed against antigenic determinants, i.e., Gm, in the Fc region of the IgG class of immunoglobulins; these are found in the serum of about 80 percent of persons with classical or definite [RA]." Rheumatoid Factor, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74591 (last visited Mar. 12, 2026).

[8] 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. 12, 2026); Cyclic Citrullinated Peptide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=97140 (last visited Mar. 12, 2026).

[9] Chanchal Gera & Arti Muley, Classification Criteria for Seronegative Rheumatoid Arthritis Based on Rheumatologist's Practice and Experience, 19 Curr. Med. Issues 236 (2021).

[10] ESR is "the rate at which erythrocytes precipitate out from a well-mixed specimen of venous blood, measured by the distance the top of the column of erythrocytes falls in a given time interval under specified conditions; an increase in rate is usually due to elevated levels of plasma proteins, especially fibrinogen and immunoglobulins, which decrease the zeta potential on erythrocytes by dielectric shielding and thus promote rouleau formation. It is increased in monoclonal gammopathy, hypergammaglobulinemia due to inflammatory disease, hyperfibrinogenemia, active inflammatory disease, and anemia." Erythrocyte Sedimentation Rate, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=102146 (last visited Mar. 12, 2026).

[11] CRP is "a globulin that forms a precipitate with the somatic C-polysaccharide of the pneumococcus in vitro; it is the most predominant of the acute-phase proteins." C-Reactive Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100489 (last visited Mar. 12, 2026).

[12] Katherine P. Liao, Rheumatoid Arthritis: Classification and Epidemiology of Rheumatoid Arthritis, in Rheumatology, supra note 6, at 747-53.

The cause of RA is not known. See, e.g., Pet. Ex. 30 at 1;[13] Pet. Ex. 58 at 2 (explaining the "key biologic pathways that drive initial autoimmunity and then [] transition to a more pathogenic stated and clinically identifiable [RA] are not known");[14] Resp. Ex. A-2 at 1.[15] Risk factors include family history, female sex, smoking, obesity, lung disease, and periodontal inflammation. Pet. Ex. 23 at 3-5; Pet. Ex. 58 at 6 tbl.2; Resp. Ex. A-2 at 3-6. Genome studies have shown associations between seropositive RA and the human leukocyte antigen ("HLA") region (HLA-DR alleles).[16] Resp. Ex. A-20 at 3, 5, 7;[17] see also Pet. Ex. 30 at 1-2 (discussing the genetic basis of RA).

Studies have suggested that certain infectious pathogens may play a role in the etiology of RA, notably *Porphyromonas gingivalis* ("*P. gingivalis*") (periodontal disease), *Proteus mirabilis*, *Escherichia coli*, and Epstein Barr Virus ("EBV"). Pet. Ex. 63 at 6.[18] Other infectious agents have been associated with RA, including parvovirus, human immunodeficiency virus ("HIV"), and hepatitis B and C viruses. Id. It has also been suggested that in the context of infectious agents, molecular mimicry may play "a critical role" in the pathogenesis of RA. Id. Lastly, "[i]t should be noted that in sharp contrast, seronegative RA is enigmatic and distinct from seropositive RA." Id.

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[13] Ernest Choy, Understanding the Dynamics: Pathways Involved in the Pathogenesis of Rheumatoid Arthritis, 51 Rheumatology v3 (2012).

[14] Kevin D. Deane & V. Michael Holers, Rheumatoid Arthritis Pathogenesis, Prediction, and Prevention: An Emerging Paradigm Shift, 73 Arthritis & Rheumatol. 181 (2021).

[15] Cynthia S. Crowson, Epidemiology of, Risk Factors for, and Possible Causes of Rheumatoid Arthritis, UpToDate, https://www.uptodate.com/contents/epidemiology-of-risk-factors-for-and-possible-causes-of-rheumatoid-arthritis (last updated Apr. 30, 2019).

[16] HLA are "histocompatibility antigens governed by genes of the HLA complex (the human major histocompatibility complex), a region on the short arm of chromosome 6 containing several genetic loci, each having multiple alleles." Human Leukocyte Antigens, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56923 (last visited Mar. 12, 2026). HLA-DR loci "are [] associated with antigenic determinants on class II antigen molecules," which are "major histocompatibility antigens found only on immunocompetent cells, primarily B lymphocytes and macrophages; they are found on molecules consisting of two noncovalently bound chains." Class II Antigens, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56885 (last visited Mar. 12, 2026).

[17] Vivianne Malmström et al., The Immunopathogenesis of Seropositive Rheumatoid Arthritis: From Triggering to Targeting, 17 Nature Revs. Immunol.60 (2017).

[18] Manuel Rojas et al., Molecular Mimicry and Autoimmunity, 95 J. Autoimmun. 100 (2018).

B. Procedural History

Petitioner filed his petition on October 13, 2022. Petition. From October 2022 to April 2023, Petitioner filed medical records,[19] an affidavit from Petitioner, and an amended petition. Pet. Exs. 1-18; Am. Petition. This case was assigned to the undersigned in June 2023. Notice of Reassignment dated June 15, 2023 (ECF No. 26). Respondent filed his Rule 4(c) report arguing against compensation on October 24, 2023. Resp. Rept. at 1, 6.

From December 2023 to June 2024, Petitioner filed expert reports from Dr. David Axelrod and Respondent filed an expert report from Dr. Mehrdad Matloubian. Pet. Exs. 22, 53; Resp. Ex. A.

On October 1, 2024, pursuant to the parties' request, the undersigned held a Rule 5 conference. Rule 5 Order dated October 3, 2024 (ECF No. 66). The undersigned preliminarily found Petitioner's diagnosis to be seronegative RA. Id. at 2. The undersigned also preliminarily found onset to be November 15, 2019, within four days of vaccination. Id. However, the undersigned was unable to provide any preliminary opinions as to the Althen prongs. Id. Thereafter, Respondent indicated he wished to defend the case, and the parties requested a briefing schedule from the Court for resolution of entitlement on the record. Resp. Status Rept., filed Nov. 13, 2024 (ECF No. 68).

Petitioner filed a motion for a ruling on the record on February 3, 2025, and Respondent filed a response on May 9, 2025. Pet. Mot. for Ruling on the Record ("Pet. Mot."), filed Feb. 3, 2025 (ECF No. 77);[20] Resp. Response to Pet. Mot. ("Resp. Response"), filed May 9, 2025 (ECF No. 81). Petitioner did not file a reply.

This matter is now ripe for adjudication.

C. Factual History

1. Medical History[21]

Petitioner was thirty-six years old when he received a flu vaccination (Fluzone Quadrivalent) on November 11, 2019 at a CVS Minute Clinic in Houston, Texas. Pet. Ex. 13 at 11-12. Petitioner's medical history was significant for chronic microcytosis, fatty and mildly

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[19] Medical records were filed throughout litigation.

[20] Petitioner, *sua sponte*, filed a brief in support of a damages award along with his motion for a ruling on the record. See Pet. Supplemental Brief in Support of Damages Award, filed Feb. 3, 2025 (ECF No. 77-2). This brief was prematurely filed since entitlement to compensation was not yet adjudicated. As such, it is not relevant to the issues in dispute resolved by this Decision.

[21] This medical history is taken from Respondent's Responsive Brief as the undersigned finds it accurately reflects the medical records. See Resp. Response at 2-5. The undersigned has made edits, deleted less relevant entries, and included additional information and definitions.

enlarged liver, ultrasound suggestive of thyrotoxic goiter, recurrent acute tonsillitis, hematuria, and positive tests for *Helicobacter pylori* and gastroesophageal reflux disease ("GERD"). Pet. Ex. 6 at 2, 9, 11, 31, 53-54, 56.

On December 3, 2019, Petitioner saw orthopedist, Steven Nolan, M.D., with complaints of pain in his cervical spine, shoulders, hips, knees, and hands. Pet. Ex. 3 at 9. Petitioner stated he noticed pain and weakness in both knees and hips "on occasion," but his hips had improved, and that most of his pain was now in his right knee, which would occasionally give way. Id. Dr. Nolan noted that Petitioner had a previous X-ray on the right knee with normal results. Id. He assessed Petitioner with a "probable torn meniscus of the right knee" and referred Petitioner for a magnetic resonance imaging ("MRI"). Id.

Petitioner presented to his primary care physician ("PCP"), Chante Ellison-Hodges, M.D., on December 5, 2019, complaining of pain "all over joints for the past few weeks." Pet. Ex. 19 at 266. He reported "severe aching pain" in his wrists, fingers, knees, and feet that was worse in the morning. Id. He also had stiffness but no numbness or tingling. Id. Petitioner advised that his mother was diagnosed with RA, and that he was seen by an orthopedist about three weeks prior and given Motrin without relief. Id. at 267. A physical examination revealed swelling in the fingers, tenderness of the interphalangeal ("IP") joints of both thumbs and distal IP ("DIP") joints of both index fingers, as well as pain in all joints in both hands. Id. at 270. Dr. Ellison-Hodges ordered laboratory tests, referred Petitioner to rheumatology, and ordered dexamethasone tablets. Id.

The following day (December 6), Petitioner sent an email to Dr. Ellison-Hodges regarding questions about possible thalassemia.[22] Pet. Ex. 19 at 261. He had never previously been informed about thalassemia but had felt fatigued for the past three years and had shortness of breath when he exercised. Id. Laboratory testing to evaluate Petitioner for thalassemia was ordered. Id. Three days later (December 9), Petitioner emailed Dr. Ellison-Hodges to inquire about laboratory testing for systemic lupus erythematosus ("lupus"), stating that his mother and aunt were diagnosed with lupus. Id. at 248. Testing revealed Petitioner's antinuclear antibody ("ANA")[23] results were negative. Id. at 248, 271-72.

On December 10, 2019, Petitioner presented to rheumatologist, Padma Chimata, M.D., for an initial evaluation. Pet. Ex. 4 at 23. Petitioner reported that most of his symptoms started around the same time he received a flu vaccination and that he was worried that might have triggered something. Id. Petitioner advised his symptoms began one-to-two months ago, gradually starting with pain in his neck and shoulders, then hips, then knees and hands and feet.

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[22] Thalassemia is "a heterogeneous group of hereditary hemolytic anemias that have in common a decreased rate of synthesis of one or more hemoglobin polypeptide chains." Thalassemia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=49484 (last visited Mar. 12, 2026).

[23] ANA are "antibodies directed against nuclear antigens" that "are frequently found in [RA]." Antinuclear Antibodies, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56804 (last visited Mar. 12, 2026).

Id. Petitioner had joint pain in both hands, rated a 1/10, with redness, but no warmth or tenderness. Id. Dr. Chimata noted that Petitioner's CRP and ESR were abnormal,[24] and that prior to the onset of Petitioner's symptoms, he had a sore throat, "had taken flu shots [in] November," and had a strong family history of lupus and RA. Id. Dr. Chimata diagnosed polyarthritis, noting that Petitioner was on steroids. Id. at 24. Dr. Chimata prescribed sulindac (nonsteroidal anti-inflammatory) and famotidine. Id. at 25.

X-rays of Petitioner's hands, wrists, knees, and pelvis were performed on December 13, 2019, and were noted to be unremarkable. Pet. Ex. 4 at 29. X-rays of the hands/wrists did not show findings of inflammatory arthropathy. Id. X-ray of the bilateral knees revealed intact alignment and no evidence of inflammatory arthropathy. Id. The same was true of the pelvis X-ray. Id.

On January 8, 2020, Petitioner attended a follow-up appointment with Dr. Chimata to discuss test results. Pet. Ex. 4 at 20-22. Dr. Chimata diagnosed Petitioner with seronegative RA. Id. at 21. Petitioner's RF and ACPA were normal. Pet. Ex. 19 at 276; Pet. Ex. 4 at 44. Dr. Chimata prescribed methotrexate[25] and folic acid, refilled sulindac and famotidine, and advised Petitioner to follow up in four to six weeks. Pet. Ex. 4 at 21-22. Petitioner attended a follow-up appointment with Dr. Chimata on February 5, 2020, and no changes were noted. Id. at 17-19.

By June 11, 2020, when Petitioner returned to see Dr. Chimata, he reported he was "doing much better." Pet. Ex. 4 at 14. His laboratory results were also improved. Id. Petitioner denied any aching, pain, or joint stiffness, but complained of fatigue. Id. Petitioner's prescription for methotrexate was discontinued, and he was started on Arava.[26] Id. Dr. Chimata recommended he continue Sulindac as needed. Id.

Petitioner established care with Dr. Saiyada Mirza as his new PCP on June 24, 2020. Pet. Ex. 19 at 217-18. At that time, Petitioner reported malaise/fatigue, weight loss, and joint pain. Id. at 220. Dr. Mirza's assessment included elevated red blood cells, low mean corpuscular volume ("MCV"), hyperlipidemia, fatigue, and Vitamin D deficiency. Id. at 221-22. Petitioner was referred to hematology/oncology specialist, Monica Desai, M.D., for further evaluation of his MCV and red blood cell count, and for possible thalassemia. Id. at 154, 221-22.

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[24] His CRP (25; reference range 0-10 mg/L) and ESR (50; reference range 0-15 mm/hr) were elevated. Pet. Ex. 19 at 273, 276-77.

[25] Methotrexate is "a folic acid antagonist that acts by inhibiting synthesis of DNA, RNA, thymidylate, and protein" and is "used as an antipsoriatic and antiarthritic in the treatment of severe, recalcitrant, disabling psoriasis and severe [RA] and psoriatic arthritis." Methotrexate, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30930 (last visited Mar. 12, 2026).

[26] Arava (leflunomide) is "an immunomodulator that inhibits pyrimidine synthesis, used as a disease-modifying antirheumatic drug in treatment of [RA]." Leflunomide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=27821 (last visited Mar. 12, 2026).

Petitioner's next follow up with Dr. Chimata was September 9, 2020. Pet. Ex. 4 at 11. He was doing well on Arava with minimal stiffness in the mornings. Id. His anemia was also stable. Id. Dr. Chimata noted that Petitioner was seen by Dr. Desai who was also monitoring Petitioner for thalassemia. Id.

On September 22, 2020, Petitioner underwent an MRI of his left knee which revealed a chronic anterior cruciate ligament ("ACL") tear. Pet. Ex. 5 at 28.

Petitioner attended a tele-health visit with Dr. Mirza on November 2, 2020, with complaints of a sore throat and inflamed tonsils with pus. Pet. Ex. 19 at 132. He had discontinued taking Arava due to this acute infection on instruction from his rheumatologist. Id. at 133. Petitioner was diagnosed with tonsillitis and prescribed amoxicillin. Id. at 135-36.

On December 9, 2020, Petitioner attended a follow-up appointment with Dr. Chimata and was reportedly doing well on Arava. Pet. Ex. 4 at 8. Dr. Chimata advised Petitioner he would need to stop his medications for 90 days to plan a pregnancy, otherwise his treatment plan remained unchanged. Id. at 8-10. He returned to see Dr. Chimata on March 10, 2021 for refills and bloodwork. Id. at 5. He was noted to be stable. Id.

Petitioner received a Covid-19 vaccine on April 1, 2021. Pet. Ex. 5 at 1. He immediately developed symptoms, including fever, fatigue, chest pain, and sore throat. Pet. Ex. 19 at 90-91. He then began to develop abdominal pain, diarrhea, low grade fever, and chills three days post-vaccination, followed by sore throat and fever. Id. at 91. He was seen by his PCP on April 9, 2021, complaining of nasal congestion, sore throat, cough, and right-sided chest pain and had a temperature of 102.9ºF. Id. Physical examination was positive for erythema of the throat. Id. at 93. Petitioner tested positive for Covid-19. Id. at 120-21; Pet. Ex. 18 at 92. He received monoclonal antibody infusions due to his immunosuppressant therapy (Arava). Pet. Ex. 18 at 12-81; Pet. Ex. 4 at 2.

On May 4, 2021, Petitioner followed up with Dr. Chimata. Pet. Ex. 4 at 2. Petitioner reported his recent Covid vaccination and infection, for which he temporarily discontinued his Arava, as well as increased fatigue. Id. His pain level was a 0/10. Id. He received refills of his medications. Id.

Petitioner saw Dr. Mirza for his annual physical on June 10, 2021. Pet. Ex. 19 at 21. Petitioner reported that he recovered well from Covid-19. Id. On that date, it was documented that he continued to see Dr. Chimata for RA and that he took Arava daily. Id. at 21, 24. No musculoskeletal complaints were noted on review of systems or physical examination. Id. at 24-26. Assessment included seronegative RA. Id. at 27-28.

No additional relevant records have been filed.

2. Petitioner's Declaration[27]

Petitioner filed an undated declaration on October 14, 2022. Pet. Ex. 1. Petitioner averred that he was born November 16, 1982. Id. at ¶ 1. He received the flu vaccination at issue on November 11, 2019. Id. at ¶ 2. Approximately eight days later, he began to have "severe and painful muscle and joint stiffness all over [his] body." Id. at ¶ 3. His "arms, fingers, hands, knees, shoulders, hips, thighs, legs, and feet were so stiff [he] had difficulty moving them." Id. His condition became progressively worse. Id. Petitioner sought care from an orthopedic physician group and was diagnosed with a "possible torn meniscus." Id. at ¶¶ 4-5. Since this diagnosis did not explain his other pain, Petitioner sought a second opinion. Id. at ¶ 5.

On December 10, 2019, Petitioner saw rheumatologist Dr. Chimata and reported his symptoms and the progression of his illness. Pet. Ex. 1 at ¶ 7. In January 2020, Dr. Chimata diagnosed him with seronegative RA and elevated ESR. Id. at ¶ 9. She prescribed methotrexate and sulindac. Id. Petitioner expressed his concern that the flu vaccine triggered his RA. Id. His medication was changed to Arava and sulindac. Id. at ¶ 14.

Petitioner explained that he continues to suffer from "debilitating joint stiffness." Pet. Ex. 1 at ¶ 17. His illness "interferes with every aspect of his life." Id. Because Arava causes a risk of birth defects, in order to have another child, he will have to quit taking the medication. Id. Further, he is unable to "enjoy vacationing at ski resorts because [his] physical limitations are made worse in higher elevations and cold weather." Id. His "immune system in also highly compromised" and this makes him fearful of illnesses. Id. In conclusion, Petitioner averred that his "vaccination injury has made [him] undergo painful, stressful, and devasting life experiences" and "has deteriorated [his] quality of life." Id.

Petitioner maintained that he did not experience any of these symptoms prior to his flu vaccination, that his symptoms continued for more than six months, and continued to the day he executed the declaration. Pet. Ex. 1 at ¶ 18.

D. Expert Reports[28]

1. Petitioner's Expert, Dr. David Axelrod[29]

a. Background and Qualifications

Dr. Axelrod is a clinical immunologist board certified in internal medicine, adult rheumatology, and allergy and immunology. Pet. Ex. 22 at 1. He received his M.D. at the

[27] Although titled an affidavit, this document was not notarized, and therefore, it is referenced herein as a declaration.

[28] For the sake of brevity, the undersigned discusses only the most relevant portions of the expert reports and only the most pertinent references.

[29] Dr. Axelrod submitted two expert reports. Pet. Exs. 22, 53.

University of Michigan Medical School and his M.S. in Clinical Research Design and Statistical Analysis at the University of Michigan School of Public Health. Id.; Pet. Ex. 48 at 1. He then completed an internal medicine residency at University of Toronto School of Medicine and William Beaumont Hospital. Pet. Ex. 48 at 1. Dr. Axelrod completed a fellowship in clinical immunology, which included adult rheumatology, allergy and immunology, and medical laboratory immunology, at McGill University. Id. He also worked as a medical staff fellow in the clinical immunology laboratory at the National Institutes of Health ("NIH"). Id. Dr. Axelrod held academic appointments from 1982 to 2010, as well as other non-academic positions throughout his career before retiring in January 2018. Id. at 2. Although he is currently retired from patient care, as a clinician he was "involved with the diagnosis and treatment of individuals with drugs reactions (including to vaccines)." Pet. Ex. 22 at 1. Dr. Axelrod has authored or co-authored several publications. Pet. Ex. 48 at 3-4.

b. Diagnosis

Dr. Axelrod defined RA as an "autoimmune disease that may result in destruction of synovial joints, with resultant severe disability and premature mortality." Pet. Ex. 22 at 7 (emphasis omitted). Although joint damage is not usually seen in the early stages of RA, as the disease progresses, "the classification criteria tend to accumulate over time." Id. Criteria for diagnosis include "at least [one] joint with definite clinical synovitis (swelling)" that is "not better explained by another disease." Id.

Citing the 2010 ACR/EULAR diagnostic criteria, Dr. Axelrod explained that patients with a score less than six (out of ten) do not have definite RA, but subsequent assessments may establish criteria that are "fulfilled cumulatively over time." Pet. Ex. 22 at 7 (citing Pet. Ex. 23 at 2 tbl.92.1). These criteria are set forth below:

The target population for these criteria are individuals who:

1. Have at least 1 joint with definite clinical synovitis (swelling)
2. Have synovitis not better explained by another disease.

The classification criteria for Rheumatoid Arthritis and the associated score:

A. Joint involvement			
	a. 1 large joint	0	
	b. 2-10 large joints	1	
	c. 1-3 small joints	2	
	d. 4-10 small joints	3	
	e. >10 joints (at least 1 small joint)	5	
B. Serology			
	a. Negative RF and Negative ACPA	0	RF=Rheumatoid Arthritis
	b. Low-positive RF or low-positive ACPA	2	
	c. High-positive RF or high-positive ACPA	3	ACPA=α-citrullinated peptide
C. Acute Phase Reactants			
	a. Normal CRP and normal ESR	0	CRP=C-Reactive Peptide
	b. Abnormal CRP or abnormal ESR	1	ESR=Estimated Sedimentation Rate
D. Duration of symptoms			
	a. <6 weeks	0	
	b. ≥6 weeks	1	

Id. (citing Pet. Ex. 23 at 2 tbl.92.1).

Reviewing Petitioner's medical records, Dr. Axelrod noted that on December 10, 2019, Petitioner saw Dr. Chimata for joint pain. Pet. Ex. 22 at 8. Dr. Chimata noted wrist tenderness, tenderness at the metacarpophalangeal ("MCP") and proximal IP ("PIP") joints of the first, second, third, fourth and fifth digits, and swelling and tenderness at the metatarsophalangeal ("MTP") joints of both feet. Id. (emphasis omitted). Although Petitioner tested negative/normal

for RF and ACPA, he had elevated CRP and ESR levels. Id. Applying the 2010 ACR/EULAR criteria, Dr. Axelrod calculated a score of 7, based on swelling of at least one joint, no other disease implicated, more than 10 joints involved, elevated CRP and ESR, and duration of greater than six weeks. Id. Thus, Dr. Axelrod opined that Petitioner met the diagnostic criteria for RA. Id.; Pet. Ex. 53 at 5.

Dr. Axelrod further explained that Petitioner underwent a diagnostic work up, including labs and X-rays, which was negative for an "alternative cause" of his synovitis. Pet. Ex. 22 at 8-9. Dr. Axelrod acknowledged that while negative tests are not conclusive as to other causes, they "provide[] a low probability" of alternative causes. Id. at 9.

Moving to the fact that Petitioner had seronegative RA, Dr. Axelrod described relevant diagnostic criteria developed by Gera and Muley. Pet. Ex. 22 at 9 (citing Pet. Ex. 24). Gera and Muley explained that the diagnosis of seronegative RA is challenging because "chronic synovitis is nonspecific" and found in many diseases. Pet. Ex. 24 at 1. Due to this difficulty, and the need to identify patients with seronegative RA early to institute treatment, the authors developed classification criteria. Id. at 1-2. They sent questionnaires to rheumatologists to obtain information about how they made the diagnosis of seronegative RA in their patients. Id. The responses of 50 rheumatologists were analyzed and used to develop recommended diagnostic criteria, which require (1) chronic inflammatory polyarthritis[30] (five or more joints on examination), (2) elevated ESR and/or CRP, (3) absence of RF and ACPAs, and (4) one to three of the following: (a) absence of spondyloarthropathy,[31] (b) synovitis confirmed by ultrasonography or MRI, (c) absence of clinical features of connective tissue disease and negative ANA, and (d) absence of hepatitis C virus, hepatitis B surface antigen, HIV, tuberculosis, and leprosy. Id. at 3-4.

Dr. Axelrod opined Petitioner met the Gera and Muley diagnostic criteria. Pet. Ex. 22 at 10. Petitioner had chronic inflammatory polyarthritis in more than five[32] joints, he had elevated CRP and ESR, he was negative for RF and ACPAs, and his records did not suggest spondylarthritis, psoriasis, or connective tissue disease, his ANA was negative, and he did not have hepatitis B or C, HIV, tuberculosis, or leprosy. Id.

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[30] Polyarthritis is "inflammation of several joints together." Polyarthritis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=40045 (last visited Mar. 12, 2026).

[31] Spondyloarthropathy is the "disease of the joints of the spine." Spondyloarthropathy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=46736 (last visited Mar. 12, 2026).

[32] Dr. Axelrod's report stated Petitioner had "chronic inflammatory polyarthritis with documented joint swelling of more than *4* joints on examination." Pet. Ex. 22 at 10 (emphasis added). This appears to be a typographical error. If it is not, this fact is not determinative to the undersigned's Decision.

Respondent's expert, Dr. Matloubian, questioned the diagnosis of RA due to Petitioner's "atypical presentation." Resp. Ex. A at 7-9. In response, Dr. Axelrod referenced portions of Petitioner's medical records showing the presence of joint swelling of the fingers, tenderness of the IP joints of thumb and index fingers, and elevated CRP and ESR inflammatory markers. Pet. Ex. 53 at 1. Petitioner had morning stiffness, especially in his feet and neck, indicating inflammation. Id. Additionally, Petitioner had swelling of the knee and MTP joints of both feet. Id. Dr. Axelrod also observed that Dr. Chimata documented that Petitioner's small and medium joints were involved, which is "typical of [RA]" and not osteoarthritis. Id.

Another point raised by Dr. Matloubian was the acute nature of Petitioner's onset, which he opined was not consistent with RA. Resp. Ex. A at 9. Dr. Axelrod disagreed and cited Brasington and Miner, who noted that 10-25% of RA patients experience sudden onset instead of gradual progression. Pet. Ex. 53 at 2 (citing Pet. Ex. 55 at 6 ("The clinical course of RA follows an onset of disease that may be abrupt and acute, gradual and insidious, or subacute between these extremes. A gradual onset is most common (at least 50% of cases); a sudden onset is much less common (10%–25%).")).

Dr. Matloubian also questioned Petitioner's complaints of muscle pain, which he stated was not typical of RA. Resp. Ex. A at 7. In response, Dr. Axelrod explained that RA can begin with "an extra-articular or non-articular manifestation, such as myalgia (pain in the muscles)." Pet. Ex. 53 at 2 (citing Pet. Ex. 55 at 6 (noting RA "may [] start as an extraarticular or nonarticular manifestation")).

Dr. Axelrod also disagreed with Dr. Matloubian's opinion that that fact Petitioner's symptoms did not worsen when he stopped taking Arava undermined the diagnosis of RA. Resp. Ex. A at 8-9; Pet. Ex. 53 at 2. Petitioner held Arava during periods he was ill with acute streptococcal tonsilitis (diagnosed on November 2, 2020) and Covid-19 (April 2021). Resp. Ex. A at 8-9; Pet. Ex. 53 at 2. Dr. Axelrod noted, however, that subsequent records show Petitioner restarted Arava once his fever and infection symptoms subsided and he has continued to require Arava, which he opined is evidence that Petitioner continued to require treatment for his RA. Pet. Ex. 53 at 2.

c. Causation

i. Althen Prong One

Dr. Axelrod offered two mechanistic theories of causation. The first is "[v]accine induced cytokine damage to the synovium." Pet. Ex. 22 at 11. In his first expert report, Dr.

Axelrod did not discuss this theory and instead referenced six papers, discussed in more detail below.[33] Id.

Choy explained the cause of RA is "unknown." Pet. Ex. 30 at 1. The pathogenesis is complex, involving "T cells, B cells[,] and the orchestrated interaction of pro-inflammatory cytokines" including TNF-α and IL-6. Pet. Ex. 30 at 12. "These cytokines are messengers that activate and differentiate effector cells that cause local and systemic symptoms associated with the disease." Id. at 7. IL-17 was also identified as a "potent cytokine that promotes synovitis." Id. at 1. Choy detailed the complex process whereby bones are destroyed in RA. Id. at 5. Choy did not identify vaccines or the flu vaccine in his paper, nor specifically as a cause of RA or a cause of cytokine induction.

Continuing with cytokines, Foti[34] described them as "cell-signaling proteins that affect the biological function of cells and processes such as inflammation, a variety of immune responses, and [] wound healing activity." Pet. Ex. 27 at 1. Foti provided a brief description of cytokine activity in RA and discussed the effectiveness of agents that block certain cytokines. Id. at 19-23. Biologic agents that block cytokines IL-6 and TNF-α have had some success in the treatment of RA; however, the results are not uniform. Id. at 19-21. Approximately "one-third of patients with RA treated with anti-TNFα agents did not respond." Id. Foti did not discuss vaccines or offer an opinion about whether vaccines can induce or activate cytokines to cause RA.

Yamaguchi et al.[35] examined the role of IL-6 in synovial fluid following the "induction of ischemic osteonecrosis" in children with ischemic osteonecrosis of the femoral head. Pet. Ex. 29 at 1, 8. Hip MRIs revealed the presence of synovitis and additional studies showed elevated

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[33] Some studies were less relevant than others. Papoudas et al. described a novel way to study complex concepts related to gene position (i.e. linear proximity), gene regulation, and gene expression, "through the combination of topological and functional information into bipartite networks." Pet. Ex. 28 at 1 (Stylianos Mavropoulos Papoudas et al., Monitoring the Prolonged Tnf Stimulation in Space and Time with Topological-Functional Networks, 18 Comput. & Struct. Biotech. J. 220 (2020)). The study used RNA sequencing of "sustained [tumor necrosis factor ("TNF")] stimulation of mouse synovial fibroblasts" to "identify unexpected regulatory changes taking place in the cells" after "prolonged exposure of fibroblasts to [TNF]." Id. at 1-3. The authors do not suggest that this experiment, or the use of prolonged exposure of fibroblasts to TNF was meant to simulate the effects of vaccination. See id. at 1-9. Instead, the goal of the study was to determine whether the "bipartite networks" described provided "insight on the functional modularity of gene expression profiles" and "genome organization." Id. at 9.

[34] Maria Foti, Introduction to Cytokine as Tissue Regulators in Health and Disease, in Cytokine Effector Functions in Tissues 3 (Maria Foti & Massimo Locati eds., 2017).

[35] Ryosuke Yamaguchi et al., HIF-1-Dependent IL-6 Activation in Articular Chondrocytes Initiating Synovitis in Femoral Head Ischemic Osteonecrosis, 98 J. Bone & Joint Surgery 1122 (2016).

IL-6 in synovial fluid. Id. at 8. The study suggested a "link between the induction of femoral head ischemia and the production of IL-6 from articular chondrocytes due to HIF-1α activation, which stimulates an inflammatory response from synovial cells." Id. However, Dr. Axelrod does not explain how ischemia resulting in death and necrosis of the femoral head, leading to the production of IL-6 activation, is comparable to vaccination. See Pet. Ex. 22 at 11. There is no suggestion here that RA involves an ischemic mechanism which leads to bone death, thus stimulating cytokine activation.

Moving to the flu vaccine, Dr. Axelrod cited Christian et al.,[36] a study that compared cytokine responses after vaccination in 28 pregnant and 28 non-pregnant women. Pet. Ex. 26 at 1. The study showed there were significant increases in cytokine IL-6, and an increase in TNF-α, with peak responses at day one post-vaccination. Id. There were no differences in the cytokine responses between pregnant and non-pregnant women. Id. From a clinical viewpoint, the results were "consistent with prior data showing that inflammatory responses to [trivalent flu] vaccination [were] mild and transient in pregnant women." Id. at 6. The authors did not address any association between the flu vaccination and induction of RA.

Although Dr. Axelrod cited the above papers in support of his proffered cytokine theory, he did not offer any opinions based on these papers in his first report. See Pet. Ex. 22 at 11. For example, he did not opine that the flu vaccine produces or activates cytokines such that they cause or significantly contribute to the induction of RA. See id. Further, he did not state that he held his opinions to a preponderant or more likely than not standard. See generally id. In his second report, relevant to cytokines, Dr. Axelrod stated that "[w]ith regard to damage to the synovia, it is possible that the elevated cytokine levels caused damage to the synovia." Pet. Ex. 53 at 6 (citing Pet. Ex. 22 at 11). Regarding his opinions related to his cytokine theory, Dr. Axelrod did not state that he held the opinions to a preponderant standard. See generally Pet. Exs. 22, 53.

The second mechanism offered by Dr. Axelrod is molecular mimicry. Pet. Ex. 22 at 12-16. Dr. Axelrod cited papers that discuss molecular mimicry, some of which are general in nature and some that are more specific; however, none address the question of whether the flu vaccine can cause RA via molecular mimicry. See id. He referenced Xia et al.[37] who "showed that [flu] hemagglutinin [("HA")[38]] could inhibit binding of type II collagen to the T-cell receptor, suggesting a possible role of [flu] [HA] in the activation of autoreactive T-cells[] in

[36] Lisa M. Christian et al., Serum Proinflammatory Cytokine Responses to Influenza Virus Vaccine Among Women During Pregnancy Versus Non-Pregnancy, 70 Am. J. Reprod. Immunol. 45 (2013).

[37] L. Xia et al., Altered Influenza Virus Haemagglutinin Peptides Inhibit T Cell Responses to Type II Collagen in Rheumatoid Arthritis, 64 Ann. Rheum. Dis. 1790 (2005).

[38] HA is "an agglutinin, e.g., an antibody or lectin, that agglutinates erythrocytes." Hemagglutinin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=21684 (last visited Mar. 12, 2026).

[RA]." Id. at 12 (citing Pet. Ex. 34). Xia et al. did not study the flu vaccine; they examined the effect of altered flu virus HA with respect to T-cell receptors/T-cell responses to type II collagen in RA. See Pet. Ex. 34. Dr. Axelrod did not explain how the study is comparable to vaccination. See Pet. Ex. 22 at 12. Further, Dr. Axelrod characterized the conclusion he derived from Xia et al. as "possible" and not likely or probable. Id.

Next, Dr. Axelrod suggested "there is an expectation that vaccine peptides are likely to align with human peptides, including those that act as target autoantigens for [RA]." Pet. Ex. 22 at 12. However, Dr. Axelrod did not provide foundational literature which suggested that vaccines in general, or the flu vaccine specifically, can induce RA via peptide homology.

In lieu of articles that discuss a role for vaccines in the pathogenesis of RA, Dr. Axelrod generally referenced articles about molecular mimicry. Pet. Ex. 22 at 12-13. Johnson et al.[39] define autoimmune illnesses as rare conditions "in which immune responses to specific self-antigens contribute the ongoing tissue damage." Pet. Ex. 31 at 1. These are adaptive immune responses, "usually directed against exogenous antigens—but its targets are autoantigens." Id. Although the foreign antigens may be structurally different from self-antigens, they may share "significant similarity in focal regions," which may allow for cross-reactive responses. Id. at 6. This is a broad general statement about molecular mimicry that does not speak to vaccination or vaccine-induced RA.

Next, Dr. Axelrod cited Kanduc et al.[40] for the proposition that "viruses (including [flu] virus) share proteomes (peptides) with the human proteome." Pet. Ex. 22 at 12 (citing Pet. Ex. 32). Kanduc et al. wrote molecular mimicry is a hypothesis, suggesting that when viruses (and bacteria) share proteins with a host, "an immune response against the infectious agent may result in formation of cross-reacting antibodies that bind the shared epitopes on the normal cell and result in the auto-destruction of the cell." Pet. Ex. 32 at 11. Dr. Axelrod similarly cited Trost et al.[41] for the proposition that "bacteria share proteomes with the human proteome." Pet. Ex. 22 at 12 (citing Pet. Ex. 33).

Dr. Axelrod cited Rojas et al., which discussed molecular mimicry and autoimmunity in various autoimmune diseases. Pet. Ex. 22 at 12 (citing Pet. Ex. 63). Although the authors discussed RA, they do not discuss vaccines in the context of RA or otherwise provide an opinion about any association between vaccines, and particularly the flu vaccine, and RA. Dr. Axelrod did not explain how the paper was relevant to RA. Dr. Axelrod similarly cited a review of

[39] Tory P. Johnson et al., Mechanisms of Autoimmunity, in Clinical Immunology: Principles and Practice 649 (Robert R. Rich et al. eds., 6th ed. 2022).

[40] Darja Kanduc et al., Massive Peptide Sharing Between Viral and Human Proteomes, 29 Peptides 1755 (2008).

[41] Brett Trost et al., Bacterial Peptides Are Intensively Present Throughout the Human Proteome, 1 Self/Nonself 71 (2010).

molecular mimicry from Martins et al.,[42] which does not mention or discuss RA, nor did Dr. Axelrod explain its relevance to RA. Id. (citing Pet. Ex. 35).

Dr. Axelrod cited other papers about T-cell function and recognition, antigen presentation, and major histocompatibility complex ("MHC") molecules[43] (including discussion of the design and effectiveness of vaccines), the optimum length of peptides, and other related subjects, but these papers did not discuss how the flu vaccine could cause RA by the mechanism of molecular mimicry. See Pet. Ex. 36;[44] Pet. Ex. 37;[45] Pet. Ex. 38;[46] Pet. Ex. 39;[47] Pet. Ex. 40.[48]

More specific to RA, Dr. Axelrod cited Yoshida et al.,[49] stating that "the authors found evidence to suggest that human Type II Collagen acts as an autoantigen" in RA patients. Pet. Ex. 22 at 14 (citing Pet. Ex. 41); see also Pet. Ex. 53 at 7. Yoshida et al. explained that RA is "associated with citrullination, which is the conversion of arginine residues into citrulline residues." Pet. Ex. 41 at 1. Citrullination is important in protein degradation, "as molecular interactions that are mediated by positively charged arginine residues are lost upon citrullination." Id. The study showed that type II collagen was citrullinated in joints affected by RA, and that these patients had autoantibodies to citrullinated type II collagen. Id. at 1, 4.

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[42] Yuri Chaves Martins et al., Visiting Molecular Mimicry Once More: Pathogenicity, Virulence, and Autoimmunity, 11 Microorganisms 1 (2023).

[43] MHC is "the genes determining the major histocompatibility antigens, in all species a group of closely linked multiallelic genes located in a small region on one chromosome." Major Histocompatibility Complex, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=66341 (last visited Mar. 12, 2026).

[44] Abul K. Abbas et al., Cellular and Molecular Immunology 117-44 (9th ed. 2018).

[45] Andrea J. Sant, Overview of T-Cell Recognition: Making Pathogens Visible to the Immune System, in Clinical Immunology: Principles and Practice 93 (Robert R. Rich et al. eds., 5th ed. 2019).

[46] J. Linsday Whitton et al., Molecular Analyses of a Five-Amino-Acid Cytotoxic T-Lymphocyte (CTL) Epitope: An Immunodominant Region Which Induces Nonreciprocal CTL Cross-Reactivity, 63 J. Virol. 4303 (1989).

[47] Bernhard Hemmer et al., Minimal Peptide Length Requirements for CD4+ T Cell Clones—Implications for Molecular Mimicry and T Cell Survival, 12 Int'l Immunol. 375 (2000).

[48] Sune Frankild et al., Amino Acid Similarity Accounts for T Cell Cross-Reactivity and for "Holes" in the T Cell Repertoire, 3 PLoS ONE e1831 (2008).

[49] Mamoru Yoshida et al., Autoimmunity to Citrullinated Type II Collagen in Rheumatoid Arthritis, 16 Mod. Rheumatol. 276 (2006).

"Immunocomplexes[50] composed of fragments of citrullinated type II collagen and autoantibodies were formed and deposited in the inflamed articular synovium of RA patients." Id. at 4. Whether these immunocomplexes played a role in RA disease pathogenesis was not determined. Id. at 4-5.

Employing the UniProtKB database,[51] Dr. Axelrod compared protein sequence alignment between HA from two flu strains in the vaccine administered to Petitioner[52] with peptides from human synovial membrane (Trefoil factor family peptide or TFF3)[53] and peptides from rabbit synovial membranes (using "human equivalents").[54] Pet. Ex. 22 at 14-16, apps. A1-A7; Pet. Exs. 43-44. He identified seven examples of amino acid alignment between the flu B strain of the flu vaccine and peptides from human equivalents (of peptides from the synovial membranes of rabbits). Pet. Ex. 22 at 15-16, apps. A1-A7. The amino acid sequences varied in length (from 3 to 13 consecutive amino acids). Id. Based on his research, Dr. Axelrod concluded that the flu

[50] Immunocomplexes, also known as antigen-antibody complexes, are "formed by the noncovalent binding of an antibody and an antigen. Complexes of antibodies belonging to certain immunoglobulin classes may activate complement. Antigen-antibody complexes are mediators of type III immune responses (Arthus reactions, serum sickness, and immune complex diseases)." Antigen-Antibody Complex, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=66292 (last visited Mar. 12, 2026).

[51] "The UniProt Knowledgebase (UniProtKB) is the central hub for the collection of functional information on proteins, with accurate, consistent[,] and rich annotation. In addition to capturing the core data mandatory for each UniProtKB entry (mainly, the amino acid sequence, protein name or description, taxonomic data[,] and citation information), as much annotation information as possible is added." UniProtKB, https://www.uniprot.org/help/uniprotkb (last modified Nov. 25, 2025).

[52] Dr. Axelrod provided the following chart for reference.

Hemagglutinin from Influenza strain	UniProtKB Entry ID
A/Brisbane/02/2018 (H1N1)pdm09-like	
A/Kansas/14/2017 (H3N2)-like	
B/Colorado/06/2017-like (Victoria lineage)	A0A1X9RX59
B/Phuket/3073/2013-like (Yamagata lineage)	**A4D5Q0**

Pet. Ex. 22 at 14. For more information on UniProtKB and entry identifiers, see UniProtKB, https://www.uniprot.org/help/uniprotkb (last modified Nov. 25, 2025).

[53] See Pet. Ex. 43 (Judith Popp et al., Human Synovia Contains Trefoil Factor Family (TFF) Peptides 1–3 Although Synovial Membrane Only Produces TFF3: Implications in Osteoarthritis and Rheumatoid Arthritis, 20 Int'l J Mol. Scis. 1 (2019)).

[54] See Pet. Ex. 44 (Weilin Liu et al., Differential Proteomics of the Synovial Membrane Between Bilateral and Unilateral Knee Osteoarthritis in Surgery-Induced Rabbit Models, 14 Mol. Med. Reps. 2243 (2016)).

vaccine shared "similar consecutive amino acid sequences with synovial antigens." Id. at 16. Dr. Axelrod opined this data provided "scientific evidence to link the [flu] vaccination received by [Petitioner] and an immune reaction to synovial autoantigens, including Type II Collagen that is considered an autoantigen for [RA]." Pet. Ex. 53 at 4.

Regarding molecular mimicry, Dr. Axelrod did not state that he held his opinions to a preponderant or more likely than not standard.

In his second report, Dr. Axelrod responded to Dr. Matloubian's critique of his opinions. Regarding Dr. Axelrod's cytokine theory, Dr. Matloubian's primary criticism was that the flu vaccine here (inactivated and non-adjuvanted) does not cause "persistent or high level[s] of cytokine production," and therefore, it would be unlikely for it to cause joint damage. Resp. Ex. A at 12. In response, Dr. Axelrod cited Christian et al., which he asserted showed that the flu vaccine did cause elevated cytokine levels. Pet. Ex. 53 at 6 (citing Pet. Ex. 26). Dr. Axelrod opined that "it is possible that the elevated cytokine levels cause[s] damage to the synovia." Id.

Dr. Axelrod also offered rebuttal opinions to Dr. Matloubian's criticisms of his molecular mimicry theory. One criticism by Dr. Matloubian was that of the seven peptides proposed, "only vimentin and collagen have been identified as targets of autoreactive T-cells in [RA]." Pet. Ex. 53 at 8; Resp. Ex. A at 15. Even so, Dr. Axelrod opined "[i]f attacked by the adaptive immune response, damage with inflammation to the synovium would be an outcome." Pet. Ex. 53 at 8.

Another concern by Dr. Matloubian about Dr. Axelrod's sequence research was that TFF3 has not been identified an antigen that causes disease in RA. Resp. Ex. A at 15. Further, Dr. Axelrod failed to indicate the beginning and ending point of his proposed peptides. Dr. Matloubian also took issue with Dr. Axelrod's failure to account for how "peptides are generated and presented to T cells," which renders "comparing simple sequence homology immunologically meaningless." Id. While the undersigned does not summarize the details of the experts' discussions on the details of these immunological points, the following quote by Dr. Axelrod is notable. Dr. Axelrod stated, "[p]erhaps the limited probability that similar sequences will align with the central position of autoreactive T-cell receptors contributes to the safety of the vaccines." Pet. Ex. 53 at 10.

In response to Dr. Matloubian's opinion that there was "no evidence of an association" between the flu vaccine and RA, Dr. Axelrod did not identify case reports or other articles that discussed any such association. Instead, he identified exhibits that he referenced in his first report that discussed cytokines and molecular mimicry. Pet. Ex. 53 at 3 (citing Pet. Ex. 22 at 11 (relative to cytokines), 12-14 (relative to molecular mimicry)). Dr. Axelrod reiterated that he provided evidence of the "molecular basis for associating the sequences of [HA]" based on his research of similar amino acid sequences. Id.

In response to Dr. Matloubian's opinion that infections have not been shown to induce RA, Dr. Axelrod cited Mankia et al.,[55] who reported an association between *P. gingivalis* bacterial infection periodontal disease and RA. Pet. Ex. 53 at 5 (citing Pet. Ex. 54 at 1). Dr. Axelrod did not explain, however, how *P. gingivalis* infection and periodontal disease and the association between this condition and RA provides evidence in support of an association between the flu vaccine and RA.

Next, regarding the studies cited by Dr. Matloubian supporting the safety profile of the flu vaccine, Dr. Axelrod agreed that the inactivated flu vaccination "appears to be safe," but explained this finding does not "preclude an individual from developing [RA] as a result of [the] [] vaccine." Pet. Ex. 53 at 4. He discussed the limitations of epidemiology studies, bias, sample size, and Type I and Type II errors, concluding that the "absence of evidence, [is] not evidence of absence." Id. (emphasis omitted).

Specifically related to the study by Ray et al.,[56] Dr. Axelrod stated that the authors "found that [flu] vaccination measurably increased the risk of developing [RA]." Pet. Ex. 53 at 6 (citing Pet. Ex. 61). However, this statement appears to be inaccurate. Ray et al. reported that "[i]n [their] initial cohort analysis, [they] found a possible association between RA and [the] [flu] vaccine in the previous 180 or 365 days, but the extended case-control analysis, bringing in more data, did not demonstrate this increase." Pet. Ex. 61 at 5.

Dr. Axelrod also quoted Ray et al. stating "[i]ndividual cases of RA and other autoimmune disease occurring after vaccination have been reported for years." Pet. Ex. 53 at 6 (quoting Pet. Ex. 61 at 4). The quote is accurate, but the references cited and examples following the quote do not describe the flu vaccine and RA; instead, the references describe the flu vaccine and Guillain-Barré Syndrome ("GBS"), the live virus rubella vaccine and arthritis, hepatitis vaccination and RA, and hepatitis B vaccination and Sjogren's syndrome. See Pet. Ex. 61 at 4-5. Ray et al. did not find or report that the flu vaccine was causally associated with RA.

Dr. Matloubian asserted that "RA-related autoimmunity and inflammation are present long before [disease] onset," referencing a 2019 review from Deane and Holers.[57] Resp. Ex. A at 9 (citing Resp. Ex. A-6) (emphasis omitted). Dr. Axelrod disagreed, characterizing these "immunological changes" as "susceptibility factors" that occur in individuals who "may or may not develop [RA]." Pet. Ex. 53 at 4-5. Dr. Axelrod cited a 2021 paper by Deane and Holers that stated most people who develop seropositive RA have elevated RA-related antibodies for a

[55] Kulveer Mankia et al., Prevalence of Periodontal Disease and Periodontopathic Bacteria in Anti-Cyclic Citrullinated Protein Antibody-Positive At-Risk Adults Without Arthritis, 2 JAMA Network Open 1 (2019).

[56] Paula Ray et al., Risk of Rheumatoid Arthritis Following Vaccination with Tetanus, Influenza and Hepatitis B Vaccines Among Persons 15–59 Years of Age, 29 Vaccine 6592 (2011). This study was also cited as Resp. Ex. A-9.

[57] Kevin D. Deane & V. Michael Holers, The Natural History of Rheumatoid Arthritis, 41 Clin. Ther. 1256 (2019).

period prior to the clinical manifestation of symptoms, suggesting not all people with RA-related antibodies develop RA. Id. at 5 (citing Pet. Ex. 58). Here, it seems both experts agree, although Dr. Axelrod referenced this "pre-RA" phase as a susceptibility, whereas Dr. Matloubian viewed it as a pre-clinical phase of the illness.

Relative to molecular mimicry, Dr. Matloubian cited Rose,[58] who stated that although there are examples of molecular mimicry triggering "a defined model of autoimmune disease in experimental animals," these occurrences are "still relatively rare in humans." Pet. Ex. 53 at 7 (quoting Resp. Ex. A-19 at 1); see also Resp. Ex. A at 14. Examples of molecular mimicry in humans include rheumatic fever and *Streptococcus pyogenes*, GBS and *Campylobacter jejuni*, and possibly narcolepsy and the flu vaccine. Id. at 3. Dr. Axelrod does not take issue with Dr. Rose's conclusion that these are rare events or the fact that evidence of homology supporting molecular mimicry is often lacking. See id. at 4.

Dr. Matloubian cited a paper authored by Malmström et al., who reported that T-cell activation by an antigen from the flu virus was similarly activated in both normal patients and RA patients (seropositive). Resp. Ex. A at 14-15; Pet. Ex. 53 at 7 (citing Resp. Ex. A-20 at 7). This finding weighs against Dr. Axelrod's theory of molecular mimicry, and in response, Dr. Axelrod cited Xia et al. for the suggestion that there is a "possible" role of flu virus HA "in the activation of autoreactive T-cells" in RA because Xia et al. showed flu HA could inhibit binding of Type II collagen to the T-cell receptor. Pet. Ex. 53 at 7 (citing Pet. Ex. 34). Dr. Axelrod also cited Yoshida et al., who "found evidence to suggested human Type II Collagen acts as an autoantigen in [RA]." Id. (citing Pet. Ex. 41). Dr. Axelrod stated, "[t]hese studies suggest that peptides derived from [flu] [HA] can activate autoreactive T-cells to participate in the development of [RA]." Id.

ii. Althen Prong Two

Dr. Axelrod opined that Petitioner has RA, and thus, he had the "susceptibilities required" to develop RA including "the presence of synovial autoreactive T-cells [in] his peripheral immune system." Pet. Ex. 22 at 16; see also Pet. Ex. 53 at 5. According to Dr. Axelrod, Petitioner received the flu vaccine which contained flu HA that "share[d] similar consecutive amino acid sequences with synovial antigens." Pet. Ex. 22 at 16. "The [flu] [HA] activated [Petitioner's] autoreactive T-cells, allowing those cells to escape his tolerance mechanisms and attack his synovial cells, resulting in synovitis, with the clinical picture of [RA]." Id.

As it relates to molecular mimicry, Dr. Axelrod asserted that "Type II Collagen acts as an autoantigen" in RA, and that both "citrullinated and non-citrullinated type antibodies" have been found in "serum and synovium" of RA patients. Pet. Ex. 22 at 14. Thus, "[i]t is not inconceivable that [Petitioner] has these antibodies in his synovial tissue, without being able to detect them in his serum." Id.

[58] Noel R. Rose, Negative Selection, Epitope Mimicry and Autoimmunity, 49 Curr. Op. Immunol. 51 (2017).

Regarding the cytokine theory, Dr. Axelrod opined that the flu vaccine increased Petitioner's cytokine levels, which "may have caused his pain symptoms in the hours and days" following vaccination. Pet. Ex. 22 at 16. The increased cytokine levels caused "some damage to his synovium, with release of synovial antigens, which activated his autoreactive T-cells to his synovium," resulting in synovitis and RA. Id. Although the sequences identified by Dr. Axelrod (in his searches for homology between HA and human proteins) cannot be tested to determine whether they would result in T-cell activation, Dr. Axelrod opined that Petitioner "did develop [RA] following his November 11, 2019 [flu] vaccination. Therefore, he must have presented [flu] [HA] peptides to autoreactive T-cells, if his [RA] was induced by the [flu] vaccination."[59] Pet. Ex. 53 at 10.

Dr. Axelrod opined that, other than vaccination, Petitioner did not have an alternate cause for his RA. Pet. Ex. 22 at 17. While Dr. Chimata documented on December 10, 2019, that Petitioner had a sore throat about the time of vaccination (November 11, 2019), the more contemporaneous records (December 3, 2019 and December 5, 2019) do not reference complaints of a sore throat. Id.; Pet. Ex. 53 at 2. Records from December 3 and December 5 do not document complaints of a sore throat or reveal a physical examination consistent with a sore throat. Pet. Ex. 22 at 17. The records also do not show that Petitioner had inflammation, lymphadenopathy, or receive diagnostic testing or treatment for a sore throat or infection. Id. Thus, Dr. Axelrod concluded Petitioner did not have an alternative cause for his synovitis or RA. Id.; Pet. Ex. 53 at 2, 12-15.

Dr. Axelrod disagreed with Dr. Matloubian's opinion that Petitioner had a viral infection which led to transient inflammatory arthritis. Pet. Ex. 53 at 2. Further, Dr. Axelrod stated there was no evidence that Petitioner had an infection until November 2, 2020, almost one year after vaccination, when he had *streptococcal* tonsillitis. Id.

Relevant to Althen prong two, Dr. Axelrod did not state that he held his opinions to a preponderant standard. See Pet. Exs. 22, 53.

iii. Althen Prong Three

Dr. Axelrod opined that Petitioner developed RA "abruptly, without laboratory evidence of inflammation prior to the onset of his clinical disorder." Pet. Ex. 53 at 5, 15. Whatever caused Petitioner's RA "likely [occurred] in proximity to the time" that he first developed symptoms of RA. Id. at 15. Dr. Axelrod opined that if Petitioner's onset of RA was one or two days after vaccination, it was consistent with "cytokine release following vaccination." Id. If, however, onset was four or eight days after vaccination, onset was "consistent with a secondary adaptive immune response." Id. He agreed that the notes regarding time intervals in Petitioner's records closer in time to vaccination are "consistent with a secondary adaptive immune response" (molecular mimicry). Id. at 6.

[59] Dr. Axelrod and Dr. Matloubian engaged in a significant colloquy about what is required for molecular mimicry and T-cell activation. See Pet. Ex. 53 at 10-11; Resp. Ex. A at 14-21. A summary of that discussion is omitted for the sake of brevity.

Petitioner had pain in his spine, shoulders, hips, knees and hands eight days after vaccination, on November 19, 2019, according to Petitioner's affidavit. Pet. Ex. 22 at 21 (citing Pet. Ex. 1). Dr. Axelrod opined "[t]his time interval [was] a bit short for a primary adaptive immune response," but "consistent with a secondary adaptive immune response" to the flu vaccine administered November 11, 2019. Id.

On December 5, 2019, Petitioner reported he had experienced widespread pain for the past few weeks, which would place onset approximately November 15, four days post-vaccination. Pet. Ex. 22 at 21. Dr. Axelrod opined that a time interval of four days is also "consistent with a secondary adaptive immune response" to his flu vaccination. Id.

Related to the adaptive immune response and molecular mimicry, Dr. Axelrod referenced several papers[60] showing that after a "booster" vaccination,[61] the secondary adaptive immune response, characterized by increased antibody levels (of a "greater magnitude than the primary adaptive immune response"), begins at two to four days post-vaccination, increasing through eight days, and remaining elevated from 14 to 22 days. Pet. Ex. 22 at 18-20.

In the alternative, using Dr. Chimata's note on December 10, 2019, Dr. Axelrod opined that Petitioner had experienced pain for four weeks, which would place onset one or two days after vaccination. Pet. Ex. 22 at 21. This time frame, according to Dr. Axelrod, "is too short for a secondary adaptive immune response," but consistent with an innate immune response and "symptoms expected from the elevated cytokines." Id. Dr. Axelrod concluded that Petitioner developed an "innate immune response with elevated cytokines that caused his early symptoms and activated his autoreactive T-cells (to the synovia), driving them out of the control of his immune tolerance mechanisms, allowing him to cause immune damage to his synovia, with resultant synovitis and a clinical picture of [RA]." Id. at 22.

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[60] See Pet. Ex. 36; Pet. Ex. 45 (John J. Miller et al, The Speed of the Secondary Immune Response to Tetanus Toxoid with a Review of War Reports and Observations on Simultaneous Injection of Toxoid and Antitoxin, 3 Pediatrics 64 (1949)); Pet. Ex. 46 (Asia Wyatt & Doron Levy, Modeling the Effect of Memory in the Adaptive Immune Response, 82 Bull. Math. Biol. 124 (2020)).

[61] A booster response, or a secondary immune response, refers to "the immune response occurring on the second and subsequent exposures to an antigen; compared to a primary immune response, the lag period is shorter, the peak antibody titer is higher and lasts longer, IgG production predominates, the antibodies produced have a higher affinity for the antigen, and a much smaller dose of the antigen is required to initiate the response." Immune Response, Secondary, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=103686 (last visited Mar. 12, 2026). Dr. Axelrod did not provide evidence that the flu vaccine at issue here was a "booster vaccination." He did not explain how or why his discussion of a booster vaccination and/or secondary adaptive immune response was relevant here.

Dr. Axelrod cited a paper about cytokines produced in the context of flu infection, not vaccination. See Pet. Ex. 25.[62] Hayden et al. showed that when humans are infected with the flu A virus, they release cytokines IL-6 and TNF-α (found in serum) in the first three days, which "correlate[] directly with viral titers, temperature, mucus production, and symptoms scores" during this timeframe. Id. at 1.

Dr. Axelrod also cited studies showing that cytokines IL-6 and TNF-α peak one day after vaccination and up to seven days when there is a "shift from an acute inflammatory response to a more generalized function of the immune system." Pet. Ex. 22 at 18 (citing Pet. Exs. 26, 28) (emphasis omitted). He also cited Yamaguchi et al. for the proposition that IL-6 induced other proinflammatory cytokines (IL-1 β and TNF-α), "with increased proliferation of synovial cells at 24 and 48 hours." Id. (citing Pet. Ex. 29). Based on these studies, Dr. Axelrod further opined that it was "possible" that "elevated cytokine levels caused damage to the synovia." Pet. Ex. 53 at 6 (citing Pet. Ex. 22 at 11).

In summary, Dr. Axelrod opined that the onset of Petitioner's symptoms was consistent with an innate response and a secondary adaptive immune response to the flu vaccination. Pet. Ex. 22 at 22. Dr. Axelrod further opined that "[Petitioner] developed [RA] following his [flu] vaccination of November 11, 2019. Therefore, whatever caused his [RA] was likely in proximity to the time that he developed the first symptoms of [RA]" and "[t]he only new environmental exposure prior to the onset of [RA] was the [flu] vaccination." Pet. Ex. 53 at 15.

Regarding causation, Dr. Axelrod did not state in either of his expert reports that he held his opinions to a preponderant evidence or "more likely than not" standard. See Pet. Exs. 22, 53.

2. Respondent's Expert, Dr. Mehrdad Matloubian[63]

a. Background and Qualifications

Dr. Matloubian is a "physician-scientist with basic science training in virology and immunology and clinical training in adult rheumatology." Resp. Ex. A at 1. He is board certified in rheumatology and internal medicine. Resp. Ex. B at 1-2. He received his M.D. as well as a Ph.D. in virology/immunology from University of California, Los Angeles. Resp. Ex. A at 1; Resp. Ex. B at 1. Since 2001, he has taught at the University of California, San Francisco where he is now a Professor. Resp. Ex. B at 2. Dr. Matloubian's research for the past 20 years has been focused on "innate and adaptive immune responses, including those of T and B cells, to acute and chronic viral infections." Resp. Ex. A at 1. "[His] areas of expertise include T and B cell responses, especially to viruses as well as factors that regulate lymphocyte circulation and trafficking." Id. He has published numerous peer-reviewed articles in these areas. Id.; see Resp. Ex. B at 10-17. "As an immunologist and board-certified rheumatologist who actively evaluates

[62] Frederick G. Hayden et al., Local and Systemic Cytokine Responses During Experimental Human Influenza A Virus Infection, 101 J. Clin. Invest. 643 (1998).

[63] Dr. Matloubian submitted one expert report. Resp. Ex. A.

and treats patients with complex autoimmune diseases[,] . . . [Dr. Matloubian] [is] qualified to address both diagnostic and immunological issues regarding these diseases." Resp. Ex. A at 1.

b. Diagnosis

Before offering an opinion about diagnosis, Dr. Matloubian summarized Petitioner's pre-vaccination history. Resp. Ex. A at 1-2, 6. Before vaccination, based on available records from Saudi Arabia, Petitioner took "multiple rounds" of antibiotics from 2016 through 2018. Id. at 2, 6. He also had abnormalities in his lungs seen on chest X-rays in 2017. Id.

Petitioner received the flu vaccine at issue on November 11, 2019. Resp. Ex. A at 2, 6. He had documented swelling and tenderness in his thumbs and DIP joints of his index fingers within three weeks of his flu vaccination, and steroids were prescribed. Id. at 6 (citing Pet. Ex. 19 at 270). Petitioner also had elevated inflammatory markers (ESR and CRP). Id. (citing Pet. Ex. 19 at 272-77). Subsequently, Petitioner was seen by rheumatologist Dr. Chimata, who noted joint tenderness in the hands and a swollen joint in the foot. Id. (citing Pet. Ex. 4 at 24). Dr. Chimata diagnosed Petitioner with seronegative RA and prescribed treatment with methotrexate followed by Arava, resulting in a favorable result. Id.

Dr. Matloubian questioned Petitioner's diagnosis of seronegative RA. Resp. Ex. A at 6-9. When seen by Dr. Nolan on December 3, 2019, Petitioner's physical examination did not reveal swollen joints. Id. at 7 (citing Pet. Ex. 3 at 8). Two days later, on December 5, Petitioner was seen by Dr. Hodges who noted that Petitioner had swelling and tenderness in the small joints of his hands. Id. (citing Pet. Ex. 19 at 270). Petitioner had elevated inflammatory markers, but these were "nonspecific." Id. Dr. Matloubian explained that because the lab results were non-specific, the results "do not distinguish between inflammation due to a recent infection or an autoimmune inflammatory process." Id. On December 10, 2019, while Petitioner was taking steroids, Petitioner was seen by Dr. Chimata who documented "only one swollen small joint of the foot" and a small knee effusion. Id. (citing Pet. Ex. 4 at 24). Dr. Matloubian also noted these findings were non-specific, though he agreed they "could be consistent with an inflammatory arthritis, such as RA." Id.

In addition to findings that were non-specific, Dr. Matloubian believed that Petitioner's presentation was "atypical" of RA. Resp. Ex. A at 7. Petitioner reported severe muscle pain, which Dr. Matloubian opined is not typical in RA since RA primarily affects joints and not muscles. Id. Another unusual feature of Petitioner's presentation was that his pain began in his neck, shoulders, and hips, before it moved to his hands, knees, and feet. Id. According to Dr. Matloubian, most patients report symptoms in the small joints before larger joints. Id. (citing Resp. Ex. A-3 at 2, 4, 9).[64] Next, Dr. Matloubian noted that when seen by Dr. Hodges on December 5, 2019, Petitioner had tenderness in the DIP joints of the index fingers. Id. at 8. Dr. Matloubian opined this is not characteristic of RA, since it "rarely, if ever, involves the [DIP] joints." Id. (citing Resp. Ex. A-3 at 4-5).

[64] James R. O'Dell et al., Rheumatoid Arthritis, in Current Diagnosis & Treatment in Rheumatology (John Imboden et al. eds., 3d ed. 2013).

Due to Petitioner's "atypical presentation," and because he had an antecedent sore throat, Dr. Matloubian suggested Petitioner "possibly had a virus associated transient arthritis that was labeled as seronegative RA." Resp. Ex. A at 8. Dr. Matloubian opined that Petitioner responded well to Arava and did well when he discontinued Arava, suggesting his condition was transient. Id. Moreover, Petitioner was prescribed 10 mg per day, and Dr. Matloubian opined that the "typical dose" for RA is 20 mg per day. Id. at 23. As to whether Petitioner was able to discontinue Arava, Dr. Matloubian noted he only reviewed Petitioner's medical records through June 2021 and had not reviewed updated records, and thus, he acknowledged his opinions were limited in time to events occurring up until June 2021.[65] Id. at 8. He issued a caveat and noted that if updated records showed that Petitioner developed a recurrence of symptoms after discontinuing immunosuppressive theory, this would support a diagnosis of chronic inflammatory arthritis (RA). Id.

Dr. Matloubian did not dispute Dr. Axelrod's opinion that Petitioner satisfied the ACR/EULAR 2010 criteria for the diagnosis of RA, "especially since he was observed to have at least one swollen joint on physical examination." Resp. Ex. A at 9. However, Dr. Matloubian opined that the diagnosis of seronegative RA is "challenging" and "requires careful longitudinal observation for confirmation." Id. While he believed it was "possible" that Petitioner was correctly diagnosed, Petitioner's atypical presentation combined with the lack of recurrence of symptoms when Arava was discontinued, "suggest[ed] a more likely transient inflammatory arthritis." Id.

c. Causation

i. Athen Prong One

Dr. Matloubian disagreed that the flu vaccine is "associated with the development [] of RA." Resp. Ex. A at 10. He explained that the causes of RA are not known; however, "established and evolving research" suggests there is a "pre-RA" phase and that "RA-related autoimmunity and inflammation are present long before" onset of symptoms. Id. at 9 (quoting Resp. Ex. A-6 at 2) (emphasis omitted). Thus, there is a "breakdown of tolerance and [] autoimmunity" years before swollen joints develop. Id. (emphasis omitted).

In support, Dr. Matloubian cited studies showing that "RA-related autoantibodies are detectable . . . 3-5 years before" symptoms are "clinically detectable." Resp. Ex. A at 9 (quoting Resp. Ex. A-6 at 2) (emphasis omitted); see also Pet. Ex. 31 at 1 ("[S]ignificant recent data from longitudinal studies have demonstrated that the onset of autoimmune responses and the development of clinical symptoms are separated in time."). Deane and Holers wrote that "[i]t is now well established that RA develops in a series of phases." Resp. Ex. A-6 at 1. The first phase is characterized by genetic and/or environmental risk factors, "in the absence of detectable systemic autoimmunity" such as autoantibodies or inflammatory markers. Id. The second phase,

[65] No additional medical records were filed documenting treatment related to RA after June 2021. Records from Houston Methodist Oncology Group after June 2021 were filed, but do not speak to the issue of whether Petitioner continued to take Arava or have a recurrence of symptoms if he discontinued Arava. See generally Pet. Ex. 21.

or propagation phase, is notable for "autoimmunity, inflammation, and symptoms." Id. This leads to "clinically detectable inflammatory arthritis . . . classified as RA." Id. Studies have shown that '"RA-related autoantibodies are detectable in the circulation a mean of 3-5 years" before symptoms of inflammatory arthritis occur. Id. at 2. Based on this current knowledge, Dr. Matloubian opined "it is [] highly likely that factors that trigger and propagate RA-related autoimmunity [and] drive the transition from pre-RA to [RA] are acting years before diagnosis." Resp. Ex. A at 10 (emphasis omitted). Smoking has been identified as a major risk factor, "which is thought to act years before transition to clinical RA." Id.

Further, Dr. Matloubian stated that acute infections have not been shown to induce RA. Resp. Ex. A at 10. He acknowledged that there are references to acute infections in the literature, but opined there is no conclusive evidence that they trigger RA. Id. Dr. Matloubian further opined that based on currently available literature, the flu virus has not been shown to cause RA. Id. (citing Resp. Ex. A-4 (listing viruses that cause arthritis and not listing the flu virus);[66] Resp. Ex. A-8 (same)).[67] For these reasons, "RA is not a post-infectious disease." Id.

After opining that infections are not thought to cause RA, Dr. Matloubian opined there is no association between vaccines and RA, specifically the flu vaccine and RA, and discussed studies in support. Resp. Ex. A at 10-11. Ray et al. conducted a large study of one million Kaiser Permanente Northern California patients and found no association between hepatitis B, tetanus, and flu vaccinations and RA. Pet. Ex. 61 at 1, 4. The study employed a retrospective chart review with cohort and case-control analysis (of ages 15 to 59) from 1997 to 1999. Id. at 1. A "possible association" between the flu vaccination and RA was seen in the range of 180 and 365 days after vaccination in the cohort analysis, but in the larger case-control analysis, this association was not seen. Id.

Fomin et al.,[68] a small study with case controls, studied the efficacy of the flu vaccine given to patients with RA being treated with disease modifying drugs. Resp. Ex. A-10 at 1. Vaccination did not cause significant worsening of symptoms or adversely affect inflammatory markers. Id. at 4.

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[66] Terry L. Moore & Reema Syed, Viruses That Cause Arthritis, UpToDate, https://www.uptodate.com/contents/viruses-that-cause-arthritis?search=viral+arthritis&topicRef=5614&source=see_link (last updated Nov. 27, 2018).

[67] Terry L. Moore, Viral Arthritis: Approach to Evaluation and Management, UpToDate, https://www.uptodate.com/contents/viral-arthritis-approach-to-evaluation-and-management?search=viral+arthritis&topicRef=5584&source=see_link (last updated Feb. 11, 2019).

[68] I. Fomin et al., Vaccination Against Influenza in Rheumatoid Arthritis: The Effect of Disease Modifying Drugs, Including TNFα Blockers, 65 Ann. Rheum. Dis. 191 (2006).

Bengtsson et al.[69] conducted a larger study of 1,998 Swedish RA patients with matched controls (2,252) and found no increased risk of RA after vaccinations, including the flu, tetanus, diphtheria, and hepatitis vaccinations. Resp. Ex. A-11 at 1-2. The study group included patients with and without ACPAs. Id. Vaccinations did not increase the risk of RA in a control group or in patients with a history of smoking who are at higher risk of developing RA. Id. at 2.

And Westra et al.[70] showed that vaccination is effective and safe in patients with autoimmune inflammatory rheumatic diseases to prevent vaccine preventable diseases, even in patients being treated with immunomodulatory drugs. Resp. Ex. A-14 at 8.

Based on these studies and others, the ACR in 2015 issued guidelines for patients with RA who were beginning or receiving disease modifying treatment or biologic agents, recommending use of killed vaccines (like the flu vaccine) and live attenuated vaccines (herpes zoster vaccine). Resp. Ex. A-15 at 17 fig.8, 18.[71]

In summary, based on multiple studies, Dr. Matloubian concluded that "no association has been found" between vaccinations, including the flu vaccination, and RA. Resp. Ex. A at 11.

Next, Dr. Matloubian addressed Dr. Axelrod's theories based on cytokines and molecular mimicry. Starting with the cytokine theory, especially IL-6 and TNF-α, Dr. Matloubian opined that the literature does not support such a theory. Resp. Ex. A at 12. Christian et al. showed that while there were post-vaccination elevations of cytokines IL-6 and TNF-α, the peak occurred one day after vaccination and was transient. Id. (citing Pet. Ex. 26 at 5). No safety issues were associated with the increased cytokines. See id. (citing Pet. Ex. 26 at 6). Thus, Dr. Matloubian opined the findings by Christian et al. do not support the idea that an inactivated non-adjuvanted flu vaccine causes levels of cytokines that lead to joint damage. Id.

Dr. Matloubian also commented on Papoudas et al., who studied TNF-α effects on mouse synovial cells and showed that it changed expression of proteins in these cells. Resp. Ex. A at 12 (citing Pet. Ex. 28 at 2). However, Dr. Matloubian explained the study did not show that low or transient levels of TNF-α can cause joint injury. Id.

Dr. Matloubian disagreed that Yamaguchi et al. supported Petitioner's cytokine theory. Resp. Ex. A at 13. Yamaguchi et al. studied the role of IL-6 in synovial fluid in children with ischemic osteonecrosis of the femoral head. Pet. Ex. 29 at 1, 8. Hip MRIs revealed the presence of synovitis and additional studies showed elevated IL-6 in synovial fluid. Id. at 8. The study

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[69] Camilla Bengtsson et al., Common Vaccinations Among Adults Do Not Increase the Risk of Developing Rheumatoid Arthritis: Results from the Swedish EIRA Study, 69 Ann. Rheum. Dis. 1831 (2010).

[70] Johanna Westra et al., Vaccination of Patients with Autoimmune Inflammatory Rheumatic Diseases, 11 Nat. Rev. Rheumatol. 135 (2015).

[71] Jasvinder A. Singh et al., 2015 American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis, 68 Arthritis Care & Rsch. 1 (2016).

suggested a "link between the induction of femoral head ischemia and the production of IL-6 from articular chondrocytes." Id. Dr. Matloubian opined that the study does not support vaccine causation due to cytokine effects but instead shows that surgically induced injury that causes joint damage can lead to inflammation that involves the production of IL-6. Resp. Ex. A at 13.

Hayden et al. studied cytokine responses to flu A virus infection and showed that IL-6 peaked at two days and returned to baseline between four to eight days after infection. Resp. Ex. A at 12 (citing Pet. Ex. 25 at 4). TNF-α peaked three days after infection and returned to baseline between days four to eight. Id. (citing Pet. Ex. 25 at 4). If Petitioner's cytokine theory was likely, Dr. Matloubian opined that RA would be commonly associated with flu infections, but it is not. Id.

While Dr. Matloubian agreed that IL-6 and other proinflammatory cytokines may be elevated during infections, after surgery, and in patients with certain autoimmune illnesses, these instances or illnesses "differ in their pathogenic mechanism." Resp. Ex. A at 13. Further, he opined that elevated IL-6 levels "seem[] to be a consequence of multiple disease processes," and "not the triggering cause." Id.

Moreover, Dr. Matloubian explained that if Dr. Axelrod's cytokine hypothesis—that vaccinations (including the flu vaccine) increase IL-6 leading to disease or worsening of symptoms—was well founded, then ACR and others in the medical community would not recommend vaccinations (including the flu vaccine) to patients with RA and other autoimmune diseases. Resp. Ex. A at 13. For support, Dr. Matloubian cited Westra et al., who showed that vaccination is effective and safe in patients with autoimmune inflammatory rheumatic diseases to prevent vaccine preventable diseases, even in patients being treatment with immunomodulatory drugs. Resp. Ex. A-14 at 8. Dr. Matloubian also cited to 2015 ACR guidelines recommending vaccinations, including inactivated killed vaccines (flu) and live attenuated vaccines (herpes zoster), to RA patients following review of numerous studies. Resp. Ex. A-15 at 17 fig.8, 18.

Regarding Dr. Axelrod's molecular mimicry theory, Dr. Matloubian summarized Petitioner's theory: RA was caused by activation of flu specific T cells, which through the mechanism of molecular mimicry, "recognized joint associated self-antigens" and induced seronegative RA. Resp. Ex. A at 14 (citing Pet. Ex. 22 at 16, 22). More specifically, Dr. Axelrod suggested that the flu vaccine "share[d] similar consecutive amino acid sequences with synovial antigens. The [flu vaccine] [HA] activated [] autoreactive T cells, allowing those cells to escape [] tolerance mechanisms and attack [] synovial cells," causing synovitis and resulting in RA. Id. (citing Pet. Ex. 22 at 16). Dr. Matloubian disagreed with this theory.

First, although natural infections have been shown to cause autoimmune illness via molecular mimicry, the natural flu virus infection has not been demonstrated to cause RA. Resp. Ex. A at 14. Since there is no cause-and-effect link between the flu infection and RA, Dr. Matloubian argued it is not probable ("exceedingly improbable") that antigens from the natural infection contained in the flu vaccine could cause RA via molecular mimicry. Id. (emphasis omitted). He explained that flu antigens are not thought to be associated with RA. Id. In fact, this is why research studies about autoreactive T cells in RA patients use antigens derived from the flu virus as a control. Id. (citing Resp. Ex. A-20 at 7 (noting studies have shown "reactivity

to an unrelated antigen (from [flu] virus) was similar between healthy controls and patients with RA")).

Second, Dr. Matloubian noted he is not aware of any reliable evidence in medical literature or from his experience caring for RA patients, suggesting that RA occurs after flu infections or flu vaccination. Resp. Ex. A at 10, 14. As described above, based on his review of the relevant studies, Dr. Matloubian concluded that "multiple controlled studies have shown that immunizations do not lead to development of RA . . . [or] lead to exacerbation of symptoms in those who already suffer from this disease." Id. at 11.

Moving to Dr. Axelrod's sequence homology research, Dr. Matloubian raised several concerns. Of the peptide sequences identified, Dr. Matloubian stated that "only vimentin and collagen have been identified as targets of autoreactive T cells in RA." Resp. Ex. A at 15. There was no evidence provided by Dr. Axelrod to show that the other proteins identified are "biologically relevant to the pathogenesis of RA." Id.

Next, Dr. Matloubian opined that simply having homologous protein sequences between pathogen and host is not sufficient to achieve molecular mimicry. Resp. Ex. A at 15 (citing Pet. Ex. 35 at 5). Without information about "how peptides are generated and presented to T cells," it is not meaningful to simply compare sequence homology.[72] Id. Therefore, showing that the flu vaccine shares short sequences of amino acids with the synovium "does not constitute evidence for molecular mimicry." Id. at 16, 18-20.

Further, Dr. Matloubian takes issue with Dr. Axelrod's suggestion that molecular mimicry can occur with similarity as opposed to complete identity. Resp. Ex. A at 18. According to Dr. Matloubian, even one amino acid difference in a peptide sequence can significantly affect the ability of the peptide to activate T cells. Id. Thus, he disagreed that similar but not identical sequences support molecular mimicry for T cells.[73] Id. at 18-19. Moreover, the critical and identical amino acids must be "at a specific position within a peptide." Id. at 19-20.

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[72] For Dr. Matloubian's discussion of how peptides are presented to T cells and what is required for molecular mimicry to occur, see Resp. Ex. A at 15-22.

[73] For support of this aspect of Dr. Matloubian's opinion, see Resp. Ex. A-23 (Mark A. Daniels et al., Thymic Selection Threshold Defined by Compartmentalization of Ras/MAPK Signalling, 444 Nature 724 (2006)); Resp. Ex. A-24 (Amitabh Gaur et al., Amelioration of Relapsing Experimental Autoimmune Encephalomyelitis with Altered Myelin Basic Protein Peptides Involves Different Cellular Mechanisms, 74 J. Neuroimmunol. 149 (1997)); Resp. Ex. A-25 (Nathan Karin et al., Reversal of Experimental Autoimmune Encephalomyelitis by a Soluble Peptide Variant of a Myelin Basic Protein Epitope: T Cell Receptor Antagonism and Reduction of Interferon γ and Tumor Necrosis Factor α Production, 180 J. Exp. Med. 2227 (1994)); Resp. Ex. A-26 (Vijay K. Kuchroo et al., A Single TCR Antagonist Peptide Inhibits Experimental Allergic Encephalomyelitis Mediated by a Diverse T Cell Repertoire, 153 J. Immunol. 3326 (1994)).

Dr. Axelrod cited Xia et al., stating they showed "a possible role of [flu] [HA] in the activation of autoreactive T-cells[] in RA." Resp. Ex. A at 21 (quoting Pet. Ex. 22 at 12, 14) (citing Pet. Ex. 34); see also Pet. Ex. 53 at 7. Dr. Matloubian opined Dr. Axelrod's interpretation of Xia et al. as a "misinterpretation of the findings" for several reasons. Resp. Ex. A at 21. First, the study did not suggest molecular mimicry between flu HA and collagen peptides. Id. at 22. Second, "[t]he residues of a peptide that bind to MHC molecules are distinct from those that are recognized by T cells." Id. (quoting Pet. Ex. 36 at 25). Dr. Matloubian explained that "just because two peptides bind to the same MHC/HLA molecule, does not mean that they are going to be recognized by the same T cell" or constitute a molecular mimic of each other. Id. According to Dr. Matloubian, Xia et al. "took advantage of this fact and altered the [flu] HA peptide so that it [could] bind to HLA-DR1/4 but not be recognized by T cells." Id. The altered peptide ligand[74] binds to "a specific MHC/HLA molecule with high affinity," thus preventing the binding by a pathogenic self-peptide, like collagen, to prevent T cells that recognize the autoantigens from being activated. Id. at 22, 23, fig. 6.

Dr. Matloubian summarized that

> Dr. Axelrod opine[d] that Petitioner "developed RA due to vaccine induced cytokine damage to joints leading to activation of autoreactive T cells as well as through molecular mimicry between [flu] HA and components of the joint. However, natural infection with [flu] virus induces higher and more prolonged levels of cytokines . . . than vaccination, yet, it has not been established to cause RA. Since [flu] vaccines contain the same antigens as the wild-type [flu] virus, it is unlikely that [the flu] vaccination would induce RA through molecular mimicry when the [flu] virus itself does not. Moreover, . . . peptide sequence homologies and even extensive identities do not reliably demonstrate molecular mimicry even under a more likely than not standard.

Resp. Ex. A at 25.

ii. Althen Prong Two

Dr. Matloubian opined that Petitioner's inflammatory arthritis diagnosed as seronegative RA was not caused by his flu vaccination. Resp. Ex. A at 25. He further opined that "[t]he cause of RA is not known and [he] [did] not know what caused [P]etitioner's inflammatory arthritis." Id. at 23.

While Dr. Matloubian does not know the cause of Petitioner's RA, he discussed a possible alternate cause for his illness: a viral infection that led to a "transient virus associated

[74] A ligand is "a molecule that binds to another molecule, used especially to refer to a small molecule that binds specifically to a larger molecule, e.g., an antigen binding to an antibody, a hormone or neurotransmitter binding to a receptor, or a substrate or allosteric effector binding to an enzyme." Ligand, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=28261 (last visited Mar. 12, 2026).

inflammatory arthritis" instead of seronegative RA. Resp. Ex. A at 23. This opinion, however, was not stated as more likely than not, but as a "possibility." Id.

Dr. Matloubian referenced medical records documenting that Petitioner had a sore throat prior to his vaccination. Resp. Ex. A at 22. Although he discussed the possibility of a viral infection, Dr. Matloubian was careful to explain that Petitioner's RA was not caused by infection. See id. at 22-24. However, Dr. Matloubian described a growing body of evidence suggesting that the gut microbiome[75] may be involved in the development of RA. Id. at 24. Antibiotics can disrupt the "equilibrium of the gut microbiome, which can then indirectly affect the immune system." Id. Petitioner's medical records show that he was treated with "multiple courses of antibiotics," presumably for infections prior to the onset of his RA. Id. And "alteration of the gut microbiome by repeated antibiotic usage" may affect self-tolerance in susceptible people. Id. Resulting imbalance of "intestinal flora composition of patients with preclinical and diagnosed RA indicate that the imbalance of intestinal flora may have an important impact on the induction and persistence of RA." Id. (quoting Resp. Ex. A-30 at 2).[76] Regardless, Dr. Matloubian opined there is no reliable evidence to suggest that either the flu vaccine or an acute flu infection can cause RA. Id.

Dr. Matloubian concluded that Petitioner's illness "more likely than not was not a consequence of his [flu vaccine]." Resp. Ex. A at 25.

iii. Althen Prong Three

Dr. Matloubian opined that "RA develops for several years before symptoms manifest." Resp. Ex. A at 25. Therefore, he opined that, more likely than not, Petitioner developed RA before his flu vaccination on November 11, 2019. Id.

Regarding clinical symptom manifestation, Dr. Matloubian cited Petitioner's affidavit and excerpts from the medical records which provide inconsistent references to symptom onset. Resp. Ex. A at 24-25. Beginning with his affidavit, Petitioner averred he developed muscle and joint pain approximately eight days after his flu vaccination. Id. at 24 (citing Pet. Ex. 1 at 1).

Then, the medical records show Petitioner called Dr. Nolan requesting a prescription for Motrin on November 25, 2019, approximately 14 days post-vaccination. Resp. Ex. A at 24 (citing Pet. Ex. 15 at 6).

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[75] Dr. Matloubian defined the microbiome as "the collection of all microbes, especially bacteria that constantly live on our external and internal body surfaces that are open to the environment." Resp. Ex. A at 24.

[76] Yanhui Peng et al., Associations Between Rheumatoid Arthritis and Intestinal Flora, with Special Emphasis on RA Pathologic Mechanisms to Treatment Strategies, 188 Microb. Pathog. 1 (2024).

On December 5, 2019, Dr. Hodges took a history that noted Petitioner had joint pain for the "past few weeks," which would place onset approximately November 19, or eight days post-vaccination. Resp. Ex. A at 24 (quoting Pet. Ex. 19 at 266).

However, on December 10, Dr. Chimata's history referenced symptoms for the "last 4 weeks" and "for the past 1-2 months." Resp. Ex. A at 24 (quoting Pet. Ex. 4 at 23). This would place onset in a range from approximately October 10, 2019 (two month) to November 12, 2019 (four weeks). Id. This range begins pre-vaccination and ends one day after vaccination. Id.

In summary, Dr. Matloubian opined that Petitioner's onset is not clear. Resp. Ex. A at 25. However, based on the current understanding of the illness, "more likely than not," Petitioner's RA onset predated his flu vaccination. Id.

III. DISCUSSION

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 his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, 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

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his 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). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 13(b)(1)(A).

Contemporaneous medical records, "in general, 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).

However, there are situations in which compelling oral 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) ("Written 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 to medical records, special masters are not rigidly bound 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) (noting 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 he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he 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 he suffered a Table Injury, he must prove a vaccine he received caused his 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 his 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 a 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 undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his 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.

IV. 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. Id. Here, the parties disagree as to diagnosis. The undersigned finds that preponderant evidence supports Petitioner's diagnosis of seronegative RA. There are several reasons for this finding.

First, Petitioner's treating rheumatologist made a diagnosis of seronegative RA. Petitioner saw his rheumatologist, Dr. Chimata, for an initial evaluation in December 2019. Dr. Chimata noted that Petitioner's CRP and ESR were abnormal. Physical examination revealed wrist tenderness and tenderness of the MCP and PIP joints of the fingers without involvement of the DIP joints. Petitioner had a small knee effusion. He also had tenderness and swelling of the MTP joints of both of his feet. Testing for RA-related antibodies was ordered. At a follow-up visit in January 2020, Dr. Chimata diagnosed Petitioner with seronegative RA. Dr. Chimata continued to see Petitioner at six months intervals, and the diagnosis of seronegative RA remained consistent. The latest relevant records show that in June 2021, Petitioner continued to see Dr. Chimata for his RA and continued to take Arava daily. See Pet. Ex. 19 at 21, 24.

Here, the undersigned gives weight to the statements of Petitioner's treating physician as she is "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").

Second, Dr. Axelrod was persuasive in showing why Petitioner's diagnosis of seronegative RA was appropriate under the relevant criteria. Applying Dr. Chimata's physical examination findings and Petitioner's lab results to the 2010 ACR/EULAR criteria, Dr. Axelrod calculated a score of seven, which was based on swelling of at least one joint, involvement of more than 10 joints, elevated CRP and ESR, symptoms lasting longer than six weeks, and no other disease implicated, and he concluded that Petitioner met the requirements for RA.

Furthermore, Dr. Matloubian did not dispute Dr. Axelrod's opinion that Petitioner satisfied the ACR/EULAR 2010 criteria for the diagnosis of RA. See Resp. Ex. A at 9.

Using the Gera and Muley classification criteria for seronegative RA and applying the physical examination findings and labs for Petitioner also shows that Petitioner's diagnosis of seronegative RA was appropriate due to documented swelling of five or more joints, elevated CRP and ESR, and absence of infection, psoriasis, or other disqualifying conditions.

Further, although Dr. Matloubian questioned the diagnosis of seronegative RA due to Petitioner's nonspecific findings and atypical presentation, he agreed that Dr. Chimata's initial physical examination findings could be consistent with RA.

Moreover, Dr. Matloubian's opinion suggesting that Petitioner had a transitory inflammatory arthritis instead of RA was based on a caveat. Dr. Matloubian's opinion was based on the assumption that after June 2021, Petitioner did not continue to require Arava for treatment of his symptoms. Dr. Matloubian did not review records after that date, and no relevant records were filed that reflect care after that date. Thus, while there is a question about whether Petitioner continued to take medication for his RA, this question does not negate the fact that Petitioner required medication for his illness beginning in December 2019, and that he continued to take the medication diagnosis for his RA through June 2021.

For the above reasons, the undersigned finds Petitioner has proven by preponderant evidence that his appropriate diagnosis was seronegative RA.

B. Causation

1. Althen Prong One

Under Althen prong o, 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, 223 (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 Petitioner failed to provide preponderant evidence of a sound and reliable theory to explain how the flu vaccination can cause RA.

First, the undersigned finds there is not preponderant evidence of a causal association between the flu vaccine and RA. Petitioner did not file a case report, a case series study, or other medical literature or evidence showing that the flu vaccine can cause RA. Studies filed and referenced by Respondent also did not show an association between the flu vaccine and RA. For example, Ray et al. conducted a study involving medical chart review of approximately one million patients with RA from 1997 to 1999 and found a "possible association" between flu vaccination and RA with onset periods between six months and one year (180 days to 365 days) in their cohort analysis but no association in their larger case-control analysis. Bengtsson et al., in a large study of 1,998 Swedish RA patients with matched controls (2,252), found no increased risk of RA after vaccinations, including the flu vaccination. Fomin et al. conducted a small study on the efficacy of the flu vaccine in RA patients and noted no significant worsening of symptoms or adversely affect inflammatory markers were seen. Similarly, Westra et al. showed vaccinations are effective and safe in patients with autoimmune inflammatory rheumatic diseases.

Although a petitioner need not make a specific type of evidential showing (i.e., epidemiologic studies) to satisfy her burden, special masters shall still consider and weigh the evidence in the record, including the epidemiological studies filed. See § 13(b)(1) (indicating the special master shall consider all materials in the record); Capizzano, 440 F.3d at 1325-26; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1149 (Fed. Cir. 1992) (finding "epidemiological studies are probative medical evidence relevant to causation" and "considerable weight [is] due to epidemiological studies in the absence of direct evidence of actual causation"). And after weighing the submitted evidence, the undersigned finds the evidence does not preponderate in Petitioner's 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."). The undersigned finds the totality of the medical literature evidence presented demonstrates no association between the flu vaccine and the development of RA.

Second, Petitioner's expert did not offer his opinions using the preponderant evidence standard. That is, Dr. Axelrod did not offer his causation opinions to a more likely than not standard. The few times that he quantified his opinion, he used words suggesting possibility, not more likely than not. See Pet. Ex. 22 at 12, 14 (referencing Xia et al. and stating they "showed that [flu] [HA] could inhibit binding of type II collagen to the T-cell receptor, suggesting a possible role of [flu] [HA] in the activation of autoreactive T-cells[] in [RA]"); Pet. Ex. 22 at 14 ("It is not inconceivable that [Petitioner] has these antibodies in his synovial tissue, without being able to detect them in his serum."); Pet. Ex. 53 at 6 (regarding "damage to the synovia, it is possible that the elevated cytokine levels caused damage"); Resp. Ex. 53 at 10 ("Perhaps the limited probability that similar sequences will align with the central position of autoreactive T-cell receptors contributes to the safety of the vaccines."). Opinions based on possibilities are insufficient to prove causation. See Waterman, 123 Fed. Cl. 564, 573-74; Moberly, 592 F.3d at 1322; de Bazan, 539 F.3d at 1351.

Lastly, the undersigned's finding that Petitioner has failed to provide preponderant evidence of causation is consistent with case law regarding vaccination (including but not limited to the flu vaccine) and RA. See, e.g., Hock v. Sec'y of Health & Hum. Servs., No. 17-168V,

2020 WL 6392770, at *23-25 (Fed. Cl. Spec. Mstr. Sept. 30, 2020) (discussing numerous reasoned Program decisions that have dismissed petitions alleging the flu vaccine can cause RA); Aultman v. Sec'y of Health & Hum. Servs., No. 21-1802V, 2025 WL 2401983, at *22 (Fed. Cl. Spec. Mstr. July 11, 2025) (listing reasoned decisions that have not found any covered vaccine can cause RA); Maxwell v. Sec'y of Health & Hum. Servs., No. 17-1367V, 2025 WL 1291642, at *29-30 (Fed. Cl. Spec. Mstr. Mar. 26, 2025) (discussing reasoned decisions that have rejected petitioners' theories that a vaccine can cause RA).

In Hock, the Chief Special Master found the petitioner's theory was not "sufficiently reliable" to show that the flu vaccine can cause RA. Hock, 2020 WL 6392770, at *1. There, petitioner's expert proposed a three-phase theory: an initial innate response with rapid secretion of cytokines, followed by "bystander activation," and ending with molecular mimicry. Id. at *5-7. The special master in Moran rejected the petitioner's theory that the flu vaccine can cause RA via molecular mimicry, finding it was not a sound and reliable theory. Moran v. Sec'y of Health & Hum. Servs., No. 16-538V, 2021 WL 4853544, at *22-30 (Fed. Cl. Spec. Mstr. Oct. 4, 2021). And the special master in Maxwell found the petitioner did not provide preponderant evidence to support his theory that the flu vaccine can cause an abnormal innate immune response in the joints. Maxwell, 2025 WL 1291642, at *23-27.

Recently, the undersigned issued a dismissal decision in a flu/RA case. Powell v. Sec'y of Health & Hum. Servs., No. 20-1726V, 2025 WL 3443590, at *31-34 (Fed. Cl. Spec. Mstr. Oct. 8, 2025). Petitioner's expert in Powell described six immunological concepts by which the flu vaccine can cause RA. Id. The undersigned found that none of the mechanisms reached the level of preponderant evidence required, and thus, due to the lack of a sound and reliable theory under Althen prong one, among other reasons, Petitioner's case was dismissed. Id.

The undersigned acknowledges there is one case where a petitioner was found entitled to compensation in a flu vaccine/RA case; however, the undersigned finds this case is not instructive because it applies a different standard (plausibility) and the facts and circumstances are distinct from this present case. See Campbell v. Sec'y of Health & Hum. Servs., 97 Fed. Cl. 650 (2011).

Although decisions of other special masters are not binding, the undersigned finds the above cases instructive and follows the reasoning of her colleagues. 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).

Overall, the undersigned finds that here, Petitioner's proffered concepts do not constitute sound and reliable causal theories. Thus, 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. 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 his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Since Petitioner failed to prove Althen prong one, it follows that he cannot prove Althen prong two. However, even if Petitioner had proven Althen prong one, the undersigned finds an additional reason why Petitioner has failed to provide preponderant evidence that there is a logical sequence of cause and effect showing Petitioner's flu vaccine caused his RA.

The undersigned bases her finding on the lack of support by Petitioner's treating physicians. Petitioner's treating physicians did not offer opinions in his medical records associating his RA with his flu vaccination. In cases with such evidence, it can be considered in an analysis of Althen prong two. See 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)).

There is one reference where Dr. Chimata noted in the history that Petitioner had received a flu vaccine before he presented with symptoms of RA. However, this notation reflects merely a temporal association. Dr. Chimata did not offer any opinion to suggest vaccine causation. Isaac v. Sec'y of Health & Hum. Servs., No. 08-601V, 2012 WL 3609993, at *26 (Fed. Cl. Spec. Mstr. July 30, 2012) ("A treating physician's recognition of a temporal relationship does not advance the analysis of causation.").

Regarding alternative causes, the undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding petitioner does not bear the burden of eliminating alternative independent potential causes). However, it is reasonable to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379; see also Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 963 (Fed. Cir. 2023) ("Such contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the

law."); Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case."). Here, the record indicates two other possible causes: (1) medical record documentation of a sore throat prior to his illness and (2) Petitioner's history of antibiotic use, as described by Dr. Matloubian. However, the undersigned finds Dr. Matloubian did not opine that he held either of these opinions to a preponderant evidence standard. Thus, the undersigned does not find evidence of an alternative cause for Petitioner's RA.

Regardless, for the reasons stated above, the undersigned finds that Petitioner failed to satisfy his burden under Althen prong two.

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 term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame 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 1239, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see also Pafford, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. See, e.g., Veryzer, 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that [P]etitioner must posit a medical theory causally connecting the vaccine and injury").

Since Petitioner failed to prove Althen prong one, it follows that he cannot prove Althen prong three. In addition, there are independent reasons why the undersigned finds Petitioner failed to prove Althen prong three.

Petitioner offered two theories of causation, each having a different medically acceptable time frame within which onset would be expected to occur. In support of his cytokine theory, Dr. Axelrod cited Hayden et al., who showed that when humans are infected with the flu A virus, they release cytokines IL-6 and TNF-α in the first three days. He also cited studies showing that cytokines IL-6 and TNF-α peak a day after vaccination and up to seven days, although Yamaguchi et al. noted increased proinflammatory cytokines (IL-1 β and TNF-α) within 24 to 48 hours.

Both experts note there are inconsistencies in the record regarding onset of Petitioner's symptoms. Petitioner's affidavit placed onset eight days after vaccination. The medical records show that Petitioner requested a prescription for Motrin approximately 14 days after vaccination. Other medical records indicate Petitioner had widespread pain approximately November 15, four days after vaccination. And Dr. Chimata's history referenced onset began four weeks prior or one to two months prior, which would place onset between October 10 and November 12, 2019, which ranges pre-vaccination to one day after vaccination.

Dr. Axelrod opined that onset of either four days or eight days is consistent with a secondary adaptive immune response, but this opinion is premised on research related to booster vaccinations. There is no foundational evidence that the flu shot administered to Petitioner was a booster vaccine. Therefore, the undersigned does not find this opinion to be supported by the evidence.

In the alternative, based on Dr. Chimata's note on December 10, 2019, Dr. Axelrod opined that Petitioner had experienced pain for four weeks, which would place onset a day or two after vaccination. He opined this time frame was consistent with an innate immune response and "symptoms expected from the elevated cytokines." Pet. Ex. 22 at 21. However, the undersigned did not find that Petitioner proved his Althen prong one theories by preponderant evidence, thus, this opinion, or any other opinion offered by Dr. Axelrod as to Althen prong one is insufficient to support Althen prong three.

Therefore, the undersigned finds that Petitioner failed to provide preponderant evidence of Althen prong three.

V. CONCLUSION

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

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that his flu vaccine caused his RA. Therefore, Petitioner is not entitled to compensation, and the petition must be dismissed.

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