In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: October 8, 2025

JENNIFER POWELL,	*	PUBLISHED
Petitioner,	*	No. 20-1726V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Rheumatoid Arthritis ("RA"); Causation-in-Fact; Significant Aggravation.
Respondent.	*	

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

DECISION[1]

On December 1, 2020, Jennifer Powell ("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 flu vaccinations administered on December 1, 2017 and October 16, 2018 "were the cause-in-fact of and/or aggravated" Petitioner's chronic rheumatoid arthritis ("RA"). Petition at ¶¶ 4, 23, 31 (ECF No. 1). Respondent argued against compensation, stating that "compensation is not appropriate in this

[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.

case" and "this case should be dismissed." Respondent's Report ("Resp. Rept.") at 1, 29 (ECF No. 24).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards,[3] the undersigned finds that Petitioner has failed to provide preponderant evidence that her flu vaccines caused her RA and thus has not satisfied her burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

I. ISSUES TO BE DECIDED

Diagnosis is not at issue; the parties agree Petitioner has RA. Joint Pre-Entitlement Submission ("Joint Sub."), filed Aug. 12, 2024, at 1-2 (ECF No. 90). The parties stipulate that Petitioner received flu vaccines on December 1, 2017 and October 16, 2018. Id. at 1. They agree that on March 6, 2018, Petitioner's rheumatologist noted "[i]mpression [n]o dramatic swelling but her pattern is certainly consistent with RA." Id. (quoting Pet. Ex. 4 at 34). Further, they agree that on July 5, 2018, Petitioner's rheumatologist diagnosed her with RA. Id.

Facts in dispute include the onset of Petitioner's RA. Joint Sub. at 2. Causation is also in dispute. Id. Specifically, the parties disagree as to whether the flu vaccine administered on December 1, 2017 caused Petitioner to develop RA pursuant to the Althen prongs. Id.

The parties also dispute whether Petitioner had "a significant worsening of her RA symptoms" after she received the flu vaccine on October 16, 2018, which they agree would be governed by Loving.[4] Joint Sub. at 2; see Loving v. Sec'y of Health & Hum. Servs., 86 Fed. Cl.

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[3] 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).

[4] Loving sets forth six elements for an off-Table significant aggravation case:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a 'significant aggravation' of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significant worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving v. Sec'y of Health & Hum. Servs., 86 Fed. Cl. 135, 144 (2009).

135, 144 (2009). However, the parties' experts do not address significant aggravation or the Loving factors.[5] See Petitioner's Exhibits ("Pet. Exs.") 9, 37, 42 (failing to mention "significant aggravation" or "Loving"); Resp. Exs. A, C, E-F (same). Therefore, the undersigned does not undertake a Loving analysis or make any finding relative to whether the flu vaccine of October 16, 2018 caused a significant aggravation of Petitioner's RA.

Even if the undersigned had addressed the question of significant aggravation, the outcome here would have been the same since the Loving factors incorporate the Althen prongs.[6] See Loving, 86 Fed. Cl. at 143 ("[T]he appropriate framework for determining whether a petitioner has made a *prima facie* showing that she has a compensable significant-aggravation off-Table claim . . . [includes] the three-part test articulated in Althen."); W.C. v. Sec'y of Health & Hum. Servs., 704 F.3d 1352, 1357 (Fed. Cir. 2013) (holding that "the Loving case provides the correct framework for evaluating off-table significant aggravation claims"); see also, e.g., Heller v. Sec'y of Health & Hum. Servs., 162 Fed. Cl. 621, 636 (2022) ("[T]he standards for assessing Althen prongs 1-3 also apply to Loving prongs 4-6.").[7] Therefore, because the undersigned finds Petitioner failed to carry her burden as to Althen, Petitioner also cannot carry her burden under Loving.

II. BACKGROUND

A. Medical Terminology

RA is a "chronic inflammatory joint disease characterized by a distinctive pattern of bone and joint destruction." Resp. Ex. A, Tab 2 at 2.[8] "The classic presentation of [RA] is a

[5] Arguments by counsel, without expert opinion, does not constitute evidence and are insufficient. The undersigned is unable to render findings relative to Loving in this case without expert opinion evidence.

[6] The three Althen prongs, which are also elements 4, 5, and 6 of Loving, require "(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.

[7] The parties agree. See Pet. Supportive Brief for Pet Mot. ("Pet. Br."), filed Aug. 12, 2024, at 4 (ECF No. 88) (noting "Loving prong four is covered [by] [the] discussion concerning Althen prong one," "Loving prong five is covered [by] [the] discussion concerning Althen prong two," and "Loving prong six is covered [by] [the] discussion concerning Althen prong three"); Resp. Response to Pet. Mot. ("Resp. Response"), filed Nov. 19, 2024 (ECF No. 95) (agreeing "[t]he Loving test [] incorporates and requires a petitioner to prove the three Althen factors").

[8] Gabriel J. Tobón et al., The Environment, Geo-epidemiology, and Autoimmune Disease: Rheumatoid Arthritis, 9 Autoimmun. Revs. A288 (2010).

symmetric small joint synovitis[9] of the hands and feet," although the illness may present with synovitis of the elbows and knees. Pet. Ex. 39 at 24.[10] 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," antibodies to citrullinated proteins, rheumatoid factor, abnormal inflammatory markers (erythrocyte sedimentation rate ("ESR") and C-reactive protein ("CRP")), and duration of symptoms of six weeks or greater. Id. at 24, 25 tbl.92.1.

The disease course can vary following onset and may be characterized as "progression, stable course with or without flares, or remission." Pet. Ex. 39 at 24. The disease incidence in North America and Northern Europe "range[s] from 20-50 cases per 100,000 population." Resp. Ex. A, Tab 2 at 2. The cause is not known, although environmental and genetic risk factors may play a role. Pet. Ex. 40 at 1.[11] These risk factors include genetics, age, gender, smoking, infections, dietary factors, and genetic and environmental interactions. Resp. Ex. A, Tab 2 at 3-4. Genetics "contribute 50% to 60% of the risk of developing RA." Id. at 3. The two genes most frequently associated with RA include HLA-DRB1, a "gene in the major histocompatibility complex," and PTPN22, a "tyrosine-phosphatase gene."[12] Id. Relative to environmental factors, "smoking has by far the strongest association with RA." Id. There are a number of infections that have been reported to induce RA, however, the question of whether infectious organisms induce RA "remains controversial" because the evidence "remains inconclusive." Id. at 3-4.

B. Procedural History

Petitioner filed her petition on December 1, 2020, along with an affidavit, and followed by medical records[13] in March and April 2021. Petition; Pet. Exs. 1-7. This case was assigned to the undersigned in September 2021. Notice of Reassignment dated Sept. 10, 2021 (ECF No. 16). Respondent filed his Rule 4(c) report, arguing against compensation, on February 7, 2022. Resp. Rept. at 1.

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[9] Synovitis is "inflammation of a synovial membrane; it is usually painful, particularly on motion, and is characterized by a fluctuating swelling due to effusion within a synovial sac." Synovitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=48576 (last visited Sept. 25, 2025).

[10] Katherine P. Liao, Rheumatoid Arthritis: Classification and Epidemiology of Rheumatoid Arthritis, in Rheumatology 747 (Marc C. Hochberg et al. eds., 7th ed. 2019).

[11] Zahra Bagheri-Hosseinabadi et al., *MTHFR* Gene Polymorphisms and Susceptibility to Rheumatoid Arthritis: A Meta-Analysis Based on 16 Studies, 39 Clin. Rheumatol. 2267 (2020).

[12] For an explanation of these two genes, see Resp. Ex. A, Tab 2 at 3.

[13] Medical records were filed throughout litigation.

On June 6, 2022, Petitioner filed an expert report from Dr. Omid Akbari. Pet. Ex. 9. On October 4, 2022, Respondent filed expert reports from Dr. John T. Bates and Dr. Karen L Law. Resp. Exs. A, C. Petitioner filed an expert report from Dr. David Axelrod on February 20, 2023. Pet. Ex. 37.

Thereafter, pursuant to a request from the parties, the undersigned held a Rule 5 conference. Rule 5 Order dated June 21, 2023 (ECF No. 50). The undersigned preliminarily found no dispute as to diagnosis (here, RA). Id. at 2. The undersigned noted issues with onset as there was a conflict between Petitioner's affidavit and medical records. Id. Lastly, the undersigned indicted she was unable to provide her preliminary findings as to causation. Id.

Petitioner filed a supplemental expert report from Dr. Akbari on August 7, 2023. Pet. Ex. 43. On May 5, 2024, Respondent filed responsive expert reports from Dr. Bates and Dr. Law. Resp. Exs. E-F.

The parties were unable to informally resolve this matter and requested to resolve entitlement through a ruling on the record. Resp. Status Rept., filed Feb. 16, 2024 (ECF No. 70); Joint Status Rept., filed June 4, 2024 (ECF No. 78). Petitioner filed his motion for a ruling on the record along with a supporting brief on August 12, 2024. Pet. Motion for Ruling on the Record ("Pet. Mot."), filed Aug. 12, 2024 (ECF No. 87);[14] Pet. Br. Respondent filed his response on November 19, 2024. Resp. Response. Petitioner did not file a reply.

This matter is now ripe for adjudication.

[14] In Petitioner's motion, counsel stated, "[w]ere the Special Master to opine that Petitioner is not entitled to compensation for either the causation-in-fact or significant aggravation claim, the Petitioner requests a live hearing before the Special Master." Pet. Mot. at 1. However, the parties requested to resolve entitlement through a ruling on the record. See Joint Status Rept., filed June 4, 2024. Further, the Vaccine Act and Rules encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. § 12(d)(2)(D); Vaccine Rule 3(b)(2) ("The special master is responsible for . . . endeavoring to make the proceedings expeditious, flexible, and less adversarial, while at the same time affording each party a full and fair opportunity to present its case and creating a record sufficient to allow review of the special master's decision."); Vaccine Rule 8(d) ("The special master may decide a case on the basis of written submissions without conducting an evidentiary hearing."). The decision to rule on the record in lieu of hearing has been affirmed on appeal. Kreizenbeck v. Sec'y of Health & Hum. Servs., 945 F.3d 1362, 1366 (Fed. Cir. 2020); see also Hooker v. Sec'y of Health & Hum. Servs., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided cases on the papers in lieu of hearing and that decision was upheld). The undersigned is not required to hold a hearing in every matter, no matter the preferences of the parties. And here, to hold a hearing after this case was submitted on the papers would not fairly resolve this matter and would further prolong the proceedings.

C. Factual History

1. Medical History[15]

Petitioner was thirty-five years old when she received the first flu vaccination at issue in this case on December 1, 2017. Pet. Ex. 5 at 100.

a. Medical History Prior to December 1, 2017 Vaccination

Before her December 1, 2017 flu vaccination, Petitioner had a significant medical history. On September 20, 2013, Petitioner presented to her primary care provider ("PCP"), Jane Ragland, M.D. Pet. Ex. 7 at 21-24. Petitioner requested a prescription for antidepressants and reported that she was prescribed Zoloft in the past, that she stopped taking it, and that she wanted to start taking medication for depression again. Id. at 23. Petitioner also complained of neck pain that began a few months prior that "ha[d] not gotten better" and occurred with movement. Id. On examination, Dr. Ragland noted Petitioner had tenderness in her neck muscles and decreased range of motion ("ROM"). Id. at 24. Dr. Ragland diagnosed Petitioner with depression, neck muscle spasm, and a lip cyst. Id.

Petitioner returned to Dr. Ragland on September 28, 2013. Pet. Ex. 7 at 40. Petitioner reported painful swelling in the back of her neck. Id. She also complained of feeling groggy in the morning and reported taking muscle relaxants at night. Id. Examination revealed Petitioner's neck was not as tight as it had been during the previous visit, and there was no swelling. Id.

On September 10, 2014, Petitioner received a flu vaccination in her right deltoid, with no reported adverse effects. Pet. Ex. 3 at 65. From September 4, 2014 to April 2, 2015, Petitioner presented for prenatal care. Id. at 12-56. Petitioner received a tetanus, diphtheria, and acellular pertussis ("Tdap") vaccination on February 25, 2015, with no reported adverse effects. Id. at 19, 64. Petitioner gave birth to a son via an elective caesarean section on April 7, 2015. Id. at 67-70. Petitioner's post-operative and postpartum courses were unremarkable. Id. Petitioner's

[15] This medical history is taken from Respondent's Responsive Brief as the undersigned finds it accurately reflects the medical records. See Resp. Response at 3-20. The undersigned has made significant edits, deleted less relevant entries, and included additional information.

summary noted a prior history of methylenetetrahydrofolate reductase ("MTHFR") deficiency.[16] Id.

On May 15, 2015, Petitioner saw Dr. Ragland complaining of pain from the middle-to-lateral right side of her chest, with some radiation to her back. Pet. Ex. 7 at 120-23. Her pain worsened with inspiration. Id. at 22. Dr. Ragland diagnosed Petitioner with pleurisy. Id. at 23.

The following year, on September 26, 2016, Petitioner called her PCP, reporting frequent headaches, which sometimes improved with rest. Pet. Ex. 7 at 160-61. Tylenol and ibuprofen were not alleviating the headaches. Id. at 161. Petitioner was instructed to keep a headache log for a week. Id. at 160-61. Petitioner received a flu vaccination on October 4, 2016, with no reported adverse effects. Pet. Ex. 2 at 1.

On October 28, 2016, Petitioner presented to Dr. Ragland. Pet. Ex. 7 at 169-72. She was back at work and reported having anxiety issues and becoming upset easily. Id. at 170. Petitioner shared her history of depression, explaining that she had taken Prozac in the past, but it did not help her very much. Id. Petitioner also complained of heart palpitations, and she felt like she was in a "constant state of crisis." Id. She had been experiencing headaches intermittently for the prior six months, and the headaches generally consisted of pain behind the right eye occurring multiple times a week. Id. Petitioner also stated she was not sleeping well, and she had "lots of stress with work." Id.

Petitioner had a behavioral medicine consultation with psychiatrist Brandon Riley, M.D., on January 20, 2017. Pet. Ex. 5 at 180-83. Her history of depression and anxiety was noted, as was her treatment with medication. Id. at 180. She had stopped taking her medication because she started to feel better. Id. Petitioner reported that her anxiety had increased recently. Id. Petitioner also described her belief that she had attention-deficit/hyperactivity disorder ("ADHD") when she was a child. Id. Dr. Riley's impression was that Petitioner had generalized anxiety disorder and panic disorder, and he prescribed Lexapro. Id. at 182. Petitioner returned to see Dr. Riley monthly for follow-up visits. Id. at 146-79. Dr. Riley prescribed Adderall for ADHD symptoms and adjusted the doses of her medications. Id.

[16] MTHFR deficiency is "a common, autosomal recessive, inborn error of folate metabolism caused by mutation in the MTHFR gene . . . , which encodes the enzyme. The chief biochemical finding is homocystinuria with normal levels of plasma methionine." Methylenetetrahydrofolate Reductase (MTHFR) Deficiency, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30976 (last visited Sept. 25, 2025). The MFHFR enzyme "synthesises 5-methyl-tetrhydrofolate, which acts at the methyl donor for remethylation of homocysteine to methionine." Pet. Ex. 11 at 1 (Y. Berkun et al., Methotrexate Related Adverse Effects in Patients with Rheumatoid Arthritis Are Associated with the A1298C Polymorphism of the MTHFR Gene, 63 Ann. Rheum. Dis. 1227 (2004)). The C677T polymorphism of this genetic mutation "results in decreased enzyme activity and hyperhomocysteinaemia." Id. Methotrexate, a drug given for RA, is contraindicated in those who have the C677T polymorphism. Id.

On November 22, 2017, Petitioner saw Julie Haygood, M.D. Pet. Ex. 5 at 139-43. She complained of a cough, sputum production, and shortness of breath, but she did not have any wheezing. Id. at 140. Petitioner's musculoskeletal examination was within normal limits. Id. at 141. Dr. Haygood diagnosed Petitioner with acute respiratory infection, and possible recurrent pertussis, bronchitis, or atypical pneumonia. Id. at 142. A chest X-ray did not demonstrate any acute disease. Id. at 199. Petitioner returned to Dr. Haygood November 30, 2017. Id. at 134-38. She was still coughing a lot, especially at night, had chills, and a subjective fever. Id. at 135. Petitioner also reported night sweats for several months and having unintentionally lost fifty pounds in the previous few months. Id. Petitioner's cough originally started in April 2017, but had been lingering over the previous few weeks. Id. She reported tiring easily, heavy menses, and that she was planning to have a uterine ablation in January 2018. Id. Dr. Haygood placed Petitioner back on antibiotics and referred her to pulmonology. Id. at 137-38.

b. December 1, 2017 Flu Vaccination and Post-Vaccination Medical History

As noted above, on December 1, 2017, Petitioner received a flu vaccination in her right deltoid. Pet. Ex. 5 at 100.

Eleven days post-vaccination, on December 12, 2017, Petitioner presented to Dr. Haygood. Pet. Ex. 5 at 123-27. She complained of several days of fever, chills, productive cough, and facial pain associated with postnasal discharge, frontal headaches, and eye pain. Id. at 124. Petitioner's son had also been sick. Id. Petitioner was feeling fatigued and was dizzy when standing. Id. Examination revealed cervical lymphadenopathy with redness and swelling in her nasal passages. Id. at 125. Dr. Haygood diagnosed Petitioner with cough with fever, acute sinusitis, normocytic anemia, and MTHFR heterozygous deficiency. Id. at 126-28. Dr. Haygood ordered antibiotics and made referrals for pulmonology and hematology/oncology. Id. Chest X-ray revealed no acute disease. Id. at 198.

Petitioner's initial hematology/oncology consultation for complaints of bruising was on December 13, 2017 with Kamran Shahid, M.D. Pet. Ex. 5 at 115-22. Petitioner's review of systems was positive for fatigue, weight loss, and easy bruising but no joint pain, swelling, or redness. Id. at 116-17. She also denied decreased range of motion. Id. at 117. Examination revealed palpable cervical lymph nodes on the right side of Petitioner's neck. Id. at 118. Musculoskeletal examination revealed no tenderness or swelling. Id. at 119. Dr. Shahid's impression was that Petitioner bruised easily but the cause was uncertain. Id. at 122. Laboratory ("lab") results revealed elevated white blood cell count, which was thought to be related to her previous pertussis infection and her recent infection that was treated with azithromycin. Id. Dr. Shahid directed Petitioner to follow up in four weeks. Id. A neck computed tomography ("CT") scan to evaluate Petitioner's cervical lymph nodes was performed December 18, 2017. Id. at 196-97. The study did not identify any abnormally enlarged lymph nodes, but the right lobe of her thyroid was enlarged and she had several low-density nodules typical of a multinodular goiter. Id.

Petitioner had a pulmonology consult for her chronic cough on December 19, 2017 with Richard Kronenberg, M.D. Pet. Ex. 5 at 109-12. Medical problems included acute respiratory

infection, and Petitioner was noted to be taking amoxicillin which had been prescribed by Dr. Haygood on December 12, 2017. Id. at 109-110. Petitioner reported her cough had not gone away, although there was no wheezing or whooping, but it was chronic and occurred throughout the day. Id. at 110-11. Her pulmonary examination and pulmonary function tests were normal. Id. at 111. Dr. Kronenberg noted that the etiology of Petitioner's cough was "obscure," but that it may be due to irritation in her airways from her prior pertussis infection. Id. He prescribed Symbicort and directed Petitioner to follow up in two weeks. Id. at 111-12. A chest X-ray did not show any acute cardiopulmonary process. Id. at 195.

On December 21, 2017, Petitioner underwent an endometrial biopsy, which was positive for hepatitis C virus antibody. Pet. Ex. 8 at 6-7.

One week later, on December 27, Petitioner reported that her entire household had a stomach virus and she requested a prescription for Phenergan topical gel for nausea and vomiting. Pet. Ex. 7 at 227.

Moving forward to 2018, Petitioner returned to see Dr. Kronenberg on January 2, 2018 for "[p]ossible flu." Pet. Ex. 5 at 104-07. Since her visit two weeks prior, Petitioner had "developed flu-like symptoms with fever[,] muscle aches[,] and malaise." Id. at 105-06. Dr. Kronenberg noted Petitioner appeared acutely ill. Id. at 106. His assessment was "[p]ossible [flu]. Persistent nonproductive cough of uncertain etiology." Id. Petitioner was prescribed Tamiflu. Id.

In early January 2018, Petitioner discussed her positive hepatitis C virus antibody test at length with Dr. Ragland. Pet. Ex. 7 at 229-30. On January 3, Petitioner emailed Dr. Ragland, stating she did not fall into the risk category for hepatitis C and did not know how she could have gotten hepatitis C, if she actually had it. Id. at 229. Dr. Ragland responded, stating she was "hoping" for a false positive test. Id. On January 9, 2018, Petitioner emailed Dr. Ragland advising that her hepatitis C viral load test was negative. Id. at 229-30. Petitioner sent the negative result to Dr. Ragland on January 10, 2018. Id. at 230. Dr. Ragland stated that they could consider that Petitioner had a false positive test. Id.

Petitioner returned to see Dr. Shahid for follow-up on January 11, 2018. Pet. Ex. 5 at 90-99. During that appointment, Dr. Shahid reviewed the results of Petitioner's lab testing and CT scan. Id. at 90, 95-98. The CT showed an incidental finding of "[t]hyroid gland enlargement." Id. at 98. Based on Petitioner's testing, Dr. Shahid ruled out lymphoproliferative or myeloproliferative disorder. Id. at 90, 98. Due to the thyroid gland enlargement, the plan was for Petitioner to have a thyroid ultrasound and thyroid function tests. Id. at 98. Petitioner did not have any musculoskeletal complaints, and her musculoskeletal examination was within normal limits. Id. at 92-93. A January 18, 2018 thyroid ultrasound showed a multinodular goiter in the right lobe. Id. at 193-94.

To address her thyroid scan results, Petitioner saw endocrinologist Christina Bratcher, M.D., on January 24, 2018. Pet. Ex. 5 at 65-69. Review of systems was positive for appetite change, fatigue, fever, night sweats, weight loss, chills, diplopia without blurred vision, vertigo, sore throat, cough, dysphagia, constipation, joint pain, headache, heat and cold intolerance, and

bruising. Id. at 67. On examination, Dr. Bratcher observed an enlarged thyroid. Id. at 68. Musculoskeletal examination was not documented. See id. at 67-68. Dr. Bratcher noted the thyroid ultrasound showed multinodular goiter of the right thyroid lobe. Id. at 66. Diagnoses included thyroid nodule and nontoxic multinodular goiter. Id. at 69. Thyroid biopsy was performed. Id.

Dr. Shahid saw Petitioner on January 25, 2018. Pet. Ex. 5 at 45-51. Review of symptoms did not document complaints of joint pain, joint swelling, or decreased range of motion. See id. at 47. Physical examination revealed no abnormalities of the musculoskeletal system (no tenderness or swelling). Id. at 49. Dr. Shahid directed Petitioner to follow up with him as needed and to continue seeing her endocrinologist, Dr. Bratcher. Id. at 50.

On January 29, 2018, Petitioner saw Dr. Riley. Pet. Ex. 5 at 52-55. During this visit, her medication dosages were adjusted. Id. at 54. Dr. Riley diagnosed Petitioner with major depressive disorder in full remission, generalized anxiety disorder, and ADHD. Id.

Also on January 29, 2018, Petitioner had an ultrasound-guided thyroid biopsy, which revealed a benign nodule. Pet. Ex. 5 at 202.

On February 2, 2018, Petitioner saw Dr. Ragland. Pet. Ex. 7 at 243-46. Petitioner reported that she had the flu one month earlier, had gotten better, and then she became sick again the previous Wednesday night (January 31, 2018). Id. at 243. Petitioner had "fever, aches, cough, [and] congestion" and "hurt[] all over." Id. She was "more short[] of breath this time. The max temp was 102. She fe[lt] worse than when she recently had [the] flu." Id. Petitioner's rapid flu test and rapid strep test were both negative. Id. at 246. Review of systems was positive for fever, sore throat, cough, and muscle problems. Id. at 244. She appeared ill, but had a normal physical examination, including a normal musculoskeletal examination. Id. at 245. Dr. Ragland opined Petitioner probably had a viral infection. Id. at 245-46. Also on February 2, Petitioner sent an email to medical assistant, Amanda Westbrook, reporting that she had a urinary tract infection. Id. at 256. Petitioner was asked to bring a urine sample to the office for testing. Id. at 256-57.

In an addendum dated February 23, 2018, Dr. Haygood noted Petitioner requested Family and Medical Leave Act ("FMLA") paperwork to be completed because of her time off work frequently due to illnesses. Pet. Ex. 5 at 128. Petitioner described her history of recent illnesses and work up for health conditions, including her illness with pertussis the previous year and her recent illness with acute bronchitis. Id. Dr. Haygood noted that she had referred Petitioner to gastroenterology because of her positive hepatitis C antibody test result, but Dr. Haygood stated that Petitioner's ribonucleic acid ("RNA") was normal. Id. Petitioner "also mentioned morning stiffness/ joint aches" in her email. Id. Dr. Haygood ordered autoimmune testing, including antinuclear antibody ("ANA") and RA testing. Id.

On February 28, 2018, Petitioner had a urinalysis that was positive for *Escherichia* ("*E.*") *coli*. Pet. Ex. 5 at 41. Dr. Haygood referred Petitioner to rheumatology because she had a positive rheumatoid factor. Id. at 41-42.

On March 6, 2018, Petitioner presented to rheumatologist Glen Graves, M.D. Pet. Ex. 4 at 31-34. Petitioner reported she was diagnosed with pertussis in May 2017, and since then, she had "not felt well off and on." Id. at 31. Petitioner further reported that she has gotten sick every two weeks and has the MTHFR gene mutation. Id. She also complained of "joint pain in bilateral hands, ankles, hip[s], and knees." Id. "She state[d] [her] joint pain started a few years ago and ha[d] increased." Id. The prior week, "her hands were swollen and tingling," and these symptoms come and go. Id. Petitioner rated her daily joint pain as 4/10 in severity but noted that at times it could increase to 10/10. Id. Petitioner indicated that over-the-counter ibuprofen helped the pain "a little bit." Id. She also had chronic fatigue that worsened in the evening. Id. Adderall helped her fatigue, but when it wore off, she "c[ould] hardly stay awake." Id.

Relevant family history indicated her maternal grandmother has RA and her mother has osteoarthritis and fibromyalgia syndrome. Pet. Ex. 4 at 31. Review of systems was positive for fever, weight loss, dry mouth, cool extremities, hair loss, back pain, bone/joint symptoms, muscle pain, and neck stiffness. Id. at 33. Physical examination did not show active synovitis in Petitioner's upper or lower extremities, but she did have mild proximal interphalangeal tenderness and fibromyalgia tender points. Id. Dr. Graves noted that a visual overview of all of Petitioner's extremities was normal; her shoulders, elbows, hips, ribs, pelvis, knees, ankles, cervical spine, thoracic spine, and lumbar spine all appeared normal; and she did not have any edema. Id. at 33-34.

Dr. Graves diagnosed Petitioner with "[p]ain in hand," noting that although Petitioner did not have dramatic swelling in her hands, the pattern of swelling was consistent with RA, and the plan was to start Petitioner on prednisone. Pet. Ex. 4 at 34. Dr. Graves also diagnosed Petitioner with "[s]tiffness of unspecified joint," and noted that this was "certainly a clue to possible inflammatory disease," but that he would need check the results of Petitioner's lab testing. Id. Dr. Graves noted Petitioner's positive hepatitis C antibody test but negative titer, and he added that having hepatitis C could cause a false positive rheumatoid factor. Id.

On March 7, 2018, Petitioner saw Dr. Riley. Pet. Ex. 5 at 37-40. Dr. Riley noted that Petitioner recently started prednisone, had an elevated rheumatoid factor, and exhibited some signs of RA. Id. at 37. In the review of systems, Petitioner denied joint pain and muscle aches. Id. at 38. Dr. Riley adjusted Petitioner's Adderall dosage. Id. at 39-40.

Two weeks later, on March 23, 2018, Petitioner followed up with Dr. Haygood. Pet. Ex. 5 at 30-34. Petitioner reported that prednisone did not alleviate her joint pain or the swelling in her hands and feet, it made her ADHD worse, and it caused anxiety and heart palpitations. Id. at 31. Petitioner had discontinued prednisone the prior week. Id. Petitioner reported that she had developed nasal congestion and felt like her chest was "on fire." Id. Petitioner also reported a sore throat, increased fatigue, and nasal and postnasal drainage over the past week, but she denied any facial or dental pain. Id. Petitioner reported having a fever of up to 101.4°F last Wednesday and Thursday nights, which had resolved. Id. Petitioner also reported having a cough with green sputum for the past week. Id. Petitioner's review of systems was positive for joint pain and swelling. Id. at 32. Dr. Haygood's impression was an upper respiratory infection. Id. at 34.

At her next visit with rheumatology, on April 3, 2018, Petitioner complained of morning stiffness in both of her hands, with edema that lasted for three-to-four hours. Pet. Ex. 4 at 27. Petitioner was not taking any medication for her stiffness currently, as she was unable to tolerate prednisone due to heart palpitations. Id. Petitioner's review of systems was now negative for pain. Id. at 29. On examination, Petitioner did not have any active synovitis or edema. Id. Her diagnosis remained "[p]ain in unspecified joint." Id. Petitioner's lab testing revealed an elevated rheumatoid factor, and it was noted that she had a "rheumatoid pattern of pain." Id. The plan was to start Petitioner on sulfasalazine and for her to continue taking diclofenac. Id.

On April 5, 2018, Petitioner spoke by telephone with Dr. Haygood. Pet. Ex. 5 at 35. Petitioner reported her right cervical lymphadenopathy had increased, but that she did not have any pain. Id. It was further noted that Dr. Graves diagnosed Petitioner with RA, and that Petitioner was having some improvement with the sulfasalazine. Id. Dr. Haygood advised that she placed a consult with immunology to determine if a possible immunodeficiency was the cause of Petitioner's frequent infections. Id.

Petitioner returned to see her rheumatologist, Dr. Graves, on July 5, 2018. Pet. Ex. 4 at 22-26. Petitioner complained of increased pain in her neck, hands, hips, knees, feet, and ankles, which had worsened over the previous week. Id. at 22. Petitioner also complained of swelling in her hands and feet, for which she had been taking diclofenac, but it had not helped her pain. Id. Petitioner's review of systems was positive for fatigue, fever, generalized weakness, insomnia, hair loss, bone/joint symptoms, and neck stiffness. Id. at 24. On examination, Petitioner did not have active synovitis, fibromyalgia tender points, or edema. Id. at 25.

As of July 5, 2018, Petitioner's diagnosis was listed as RA, and the plan was for her to stop taking sulfasalazine and start taking methotrexate. Pet. Ex. 4 at 25. Hepatitis C was also listed as one of Petitioner's diagnoses, although Dr. Graves noted that her viral load was negative. Id. Dr. Graves also noted that a positive hepatitis C antibody test could cause a false positive rheumatoid factor, but that Petitioner's levels were quite high with a negative cyclic citrullinated peptide[17] antibody test. Id. Dr. Graves also diagnosed Petitioner with cervicalgia. Id.

c. October 16, 2018 Flu Vaccination and Post-Vaccination Medical History

On October 16, 2018, Petitioner received a flu vaccination in her left deltoid. Pet. Ex. 7 at 286.

On October 23, 2018, one week after vaccination, Petitioner presented for a follow-up rheumatology appointment with Dr. Graves. Pet. Ex. 4 at 17-21. She complained of increased bilateral pain in her hands, wrists, shoulders, ankles, and feet "for the past couple of weeks." Id.

[17] Cyclic citrullinated peptide is "a synthetic, citrulline-containing peptide with a cyclic structure, used in assays for [RA]; the presence of antibodies to this peptide is highly specific for [RA]." Cyclic Citrullinated Peptide, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=97140 (last visited Sept. 25, 2025).

at 17. Petitioner also complained of a productive cough, fever, chills, muscle pain, gastrointestinal pain, and diarrhea for the past two days. Id. Examination revealed metacarpophalangeal joint synovitis. Id. at 20. Dr. Graves's impression was RA, and because Petitioner was "doing poorly" on methotrexate, he changed her medication to Enbrel injections. Id. Dr. Graves also prescribed azithromycin for Petitioner's upper respiratory symptoms. Id.

On November 15, 2018, Petitioner saw PCP Alison Collins, D.O. Pet. Ex. 7 at 292-95. Petitioner complained of swelling on the left side of her face. Id. at 292. Petitioner reported that the swelling started as a small knot one week prior, and that it had been spreading since then. Id. She further reported it hurt to swallow, that she could not open her mouth, and that the pain was becoming severe and was starting to radiate down her neck. Id. Petitioner had started receiving Enbrel injections three weeks earlier. Id. Petitioner had tenderness to palpation over the left submandibular area and pain and swelling up to her left temple and under the left side of her mandible. Id. at 295. Dr. Collins diagnosed Petitioner with sialadenitis[18] and directed her to go to the emergency department ("ED") for further evaluation and treatment. Id. Later that day, Petitioner presented to the ED. Id. at 302-10. Examination showed that Petitioner had spasm of the jaw muscle and tooth decay. Id. at 305. She was diagnosed with sialadenitis and prescribed antibiotics. Id. at 309.

One week later, on November 21, 2018, Petitioner called her PCP reporting that she was having a difficult time sitting because of pain due to shingles on her right buttock. Pet. Ex. 7 at 375. Dr. Collins recommended Petitioner present to urgent care to obtain a prescription for antiviral medication. Id. Petitioner decided not to go to urgent care because she had a follow-up appointment scheduled on November 23. Id. On November 23, Petitioner saw Dr. Collins. Id. at 379. Petitioner complained of a rash on her right buttock that had been present for two days with tingling pain and no itching. Id. It was noted that an ear, nose, and throat ("ENT") doctor, or otolaryngologist, believed that the etiology of Petitioner's sialadenitis was Sjögren's disease.[19] Id. Petitioner planned to follow up with ENT and rheumatology. Id. Dr. Collins's assessment was sialadenitis and herpes zoster without complication. Id. at 382.

Moving to 2019, Petitioner presented to the ED on January 14, 2019. Pet. Ex. 7 at 400. Petitioner reported that she was assaulted the previous night by her ex-boyfriend. Id. Petitioner lost consciousness for "'seconds' multiple times." Id. Petitioner complained of neck pain, that it hurt to swallow, headache, dizziness, visual changes, and nausea/vomiting. Id. On examination,

[18] Sialadenitis is the "inflammation of a salivary gland." Sialadenitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=45680 (last visited Sept. 25, 2025).

[19] Sjögren syndrome is "a symptom complex of unknown etiology, usually occurring in middle-aged or older women, marked by the triad of keratoconjunctivitis sicca with or without lacrimal gland enlargement, xerostomia with or without salivary gland enlargement, and the presence of a connective tissue disease, usually [RA] but sometimes systemic lupus erythematosus, scleroderma, or polymyositis. An abnormal immune response has been implicated." Sjögren Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=111409 (last visited Sept. 25, 2025).

Petitioner had tenderness to her trachea and the paraspinal muscles around her neck, as well as diffuse anterior wall tenderness. Id. at 401. Petitioner's musculoskeletal examination revealed normal ROM, no edema, and no tenderness. Id. at 402. She was diagnosed with a closed head injury, anterior neck pain, and multiple thyroid nodules. Id. at 405.

On January 17, 2019, Petitioner saw Dr. Riley. Pet. Ex. 5 at 18-22. Petitioner stated that she was diagnosed with RA in April 2018, and she felt like her depression and anxiety had worsened since then. Id. at 19. Petitioner did not believe that her medications for her mental health issues had been working for "some time." Id. Petitioner also discussed her relationships, noting that she had been involved in tumultuous relationships in the past. Id. Dr. Riley noted Petitioner had "very significant mood lability," physical and mental difficulties working, and that she was currently working from home. Id. Dr. Riley adjusted Petitioner's medication. Id. at 21.

On January 24, 2019, Petitioner saw rheumatology Nurse Practitioner Ranie Whatley. Pet. Ex. 4 at 11-16. Petitioner complained of pain in all of her joints, muscle pain in her neck and right shoulder, swelling in her hands and feet, and numbness and tingling in her hands and toes. Id. at 11. She also reported hair loss since she started taking methotrexate. Id. Petitioner's diagnosis remained RA with a positive rheumatoid factor. Id. at 14. Petitioner was instructed to continue receiving Enbrel injections because she had significant improvement while taking it. Id. Petitioner was also diagnosed with right shoulder bursitis. Id. at 15.

On January 29, 2019, Petitioner exchanged emails with Dr. Ragland. Pet. Ex. 7 at 442. Petitioner wrote that since November 2018, she had been unable to take her RA medications due to repeat upper respiratory infections and other medical issues. Id. She also mentioned Dr. Riley's recent diagnoses and discussed the medications she was taking for those conditions. Id. Petitioner reported that her ability to work had been negatively impacted because she had just started retaking her RA medications the prior week and was now on new medications prescribed by Dr. Riley. Id. Thus, Petitioner requested Dr. Ragland complete short-term disability paperwork. Id. Dr. Ragland told Petitioner that she would complete the paperwork if she could review records from Petitioner's rheumatologist and psychiatrist since Dr. Ragland had not been managing the issues for which Petitioner was trying to get short-term disability. Id.

Petitioner returned to see Dr. Riley on February 19, 2019. Pet. Ex. 5 at 14-17. Her mood had stabilized, and she had less lability and anger. Id. at 15. She also reported sleeping better, but did not feel refreshed, which Petitioner believed was due to her RA. Id. Work was her largest stressor. Id. Petitioner was scheduled to start psychotherapy sessions. Id. at 17. Dr. Riley noted that Petitioner's ADHD was not controlled and that she would restart Adderall. Id. Petitioner returned to see Dr. Riley on March 29, 2019. Id. at 7. Petitioner reported that she was doing better, although she still had a lot of stressors, and she still felt that her medications wore off before lunchtime. Id. at 8. Dr. Riley adjusted Petitioner's medication. Id. at 10.

On October 28, 2019, Petitioner called her PCP, reporting a recent exposure to the flu. Pet. Ex. 7 at 444-45. Petitioner's niece tested positive for the flu, and Petitioner had cold-like symptoms and fever that started the previous night. Id. at 445. Petitioner requested a prescription for Tamiflu, which Dr. Ragland provided. Id.

At her next rheumatology appointment on June 24, 2020, Petitioner reported that she had lost her health insurance, and because of that, had not had recent visits. Pet. Ex. 4 at 6. Her last Enbrel injection was in October 2019. Id. Petitioner reported pain all over that was constant, varied in severity, and worsened with increased activity. Id. Petitioner's diagnosis remained RA with a positive rheumatoid factor. Id. at 9. The plan was for Petitioner to restart Enbrel injections after obtaining updated lab testing. Id.

Additional records have been filed but are not summarized, as they are not relevant to the issues in dispute.

2. Petitioner's Affidavit

Petitioner averred that "[w]ithin 45 minutes after receiving [her] flu shot" on December 1, 2017, she "began having trouble standing, and every joint in [her] body felt as though it [were] on fire. [She] had difficulty moving and was unable to fully extend [her] joints." Pet. Ex. 1 at ¶¶ 2-3. And "[w]ithin several hours," she developed a fever. Id. at ¶ 3. The following day, she was unable to move or walk and called out of work. Id.

On December 12, 2017, Petitioner saw Dr. Haywood. Pet. Ex. 1 at ¶ 4. She "was still struggling with pain in [her] body and a fever that had lasted several days." Id. She also had "a hard cough," fatigue, and dizziness. Id. Petitioner was prescribed an antibiotic (Augmentin) and hydrocodone cough syrup. Id. The next day, she saw Dr. Shahid complaining of night sweats and easy bruising. Id. at ¶ 5. Testing was "negative for lymphoproliferative disorder, but myelodysplastic disorders could not be ruled out." Id. No complaints of joint pain were made at these visits. See id. at ¶¶ 4-5; Pet. Ex. 5 at 115-27.

On December 18, 2017, Petitioner underwent a CT scan of her neck that revealed a multinodular goiter. Pet. Ex. 1 at ¶ 6. The next day, she saw Dr. Kronenberg, complaining of a cough that "had not subsided and was progressing." Id. at ¶ 7. Joint pain was not mentioned at this visit. See id.; Pet. Ex. 5 at 109-12.

The following month, on January 2, 2018, Petitioner returned to Dr. Kronenberg, complaining of "mild fever, body aches, cough, and a general feeling of illness." Pet. Ex. 1 at ¶ 8. Later that month, on January 24, Petitioner reported "symptoms of diplopia, vertigo, sore throat, tachycardia, worsening cough, dysphagia, worsening joint pain, headache, heat and cold intolerance, worsening bruising, and anxiety." Id. at ¶ 11. Petitioner began speaking to her psychiatrist in January 2018 about her "heightened anxiety and depression due to [her] symptoms." Id. at ¶ 13.

In March 2018, Petitioner first saw rheumatologist Dr. Graves. Pet. Ex. 1 at ¶¶ 15, 17. Petitioner complained of "bilateral joint pain in [her] hands, ankles, hips, and knees." Id. at ¶ 17. She also reported chronic fatigue and insomnia due to her severe pain. Id. At that visit, on March 6, Dr. Graves stated Petitioner's "pattern of pain was consistent with [RA] and possible inflammatory disease." Id. He prescribed prednisone; however, Petitioner discontinued this medication due to side effects. Id. at ¶ 15. She returned to rheumatology on April 3, where it was noted that she had elevated rheumatoid factor. Id. at ¶ 19.

Petitioner continued to see various health care professionals in 2018. Pet. Ex. 1 at ¶¶ 20-31. On October 16, 2018, Petitioner received a flu vaccine. Id. at ¶ 26. She returned to rheumatologist Dr. Graves on October 23, 2018, one week later, complaining of "increased joint pain . . . over the past couple of weeks." Id. at ¶ 27.

Petitioner maintained that she did not experience any of these symptoms prior to her December 1, 2017 vaccination, and these symptoms continue as of the date she executed her affidavit, December 1, 2020. Pet. Ex. 1 at ¶ 32, 8.

D. Expert Reports

1. Petitioner's Expert, Dr. David Axelrod[20]

a. Background and Qualifications

Dr. Axelrod is a clinical immunologist board certified in internal medicine, adult rheumatology, and allergy and immunology. Pet. Ex. 37 at 1. He received his M.D. at the 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. Pet. Ex. 38 at 1. He then completed an internal medicine residency at University of Toronto School of Medicine and William Beaumont Hospital. Id. 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. 37 at 1. Dr. Axelrod has authored or co-authored several publications. Pet. Ex. 38 at 3-4.

b. Opinion

Dr. Axelrod described RA as an "autoimmune disease that may result in destruction of synovial joints, with resultant severe disability and premature mortality." Pet. Ex. 37 at 9. He explained that "[j]oint damage is rarely apparent in the early stages of disease and 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.

[20] Dr. Axelrod submitted one expert report. Pet. Ex. 37.

Citing the 2010 ACR/EULAR diagnostic criteria,[21] Dr. Axelrod explained that those patients who have a score less than six out of ten do not have definite RA, but if the illness worsens, reassessments may show that the criteria are "fulfilled cumulatively over time." Pet. Ex. 37 at 9-10 (citing Pet. Ex. 39 at 25 tbl.92.1). Reviewing Petitioner's medical records, Dr. Axelrod noted that Petitioner's February 2018 lab studies showed a positive rheumatoid factor, negative Immunoglobulin G ("IgG") anti-cyclic citrullinated peptide antibody, negative ANA screen, negative IgG antineutrophil antibody, and normal ESR. Id. at 6 (citing Pet. Ex. 5 at 228-30, 242). When Petitioner first saw Dr. Graves on March 6, 2018, Dr. Graves noted that she did not have active synovitis, but he documented proximal inter-phalangeal joint tenderness, and thought her presentation was consistent with RA. Id. (citing Pet. Ex. 4 at 31-39). He also prescribed prednisone for two weeks. Id. (citing Pet. Ex. 4 at 34).

Dr. Axelrod reviewed Petitioner's visits to Dr. Graves on April 3, 2018 and July 5, 2018. Pet. Ex. 37 at 7. On April 3, Dr. Graves documented Petitioner's "increased pain in both hands, with morning stiffness and swelling lasting [three-to-four] hours." Id. (citing Pet. Ex. 4 at 27). Again, he noted Petitioner had no active synovitis, but found Petitioner had a pattern of pain consistent with RA and prescribed Sulfasalazine, "a treatment for mild to moderate [RA]." Id. (citing Pet. Ex. 4 at 27-30). At the next appointment with Dr. Graves on July 5, 2018, he documented "increased pain at her neck, hands, hips, knees, feet[,] and ankles, worse over the previous week, with swelling of her hands and feet, despite Diclofenac and Sulfasalazine." Id. (citing Pet. Ex. 4 at 22-25). Again, he noted no active synovitis. Id. (citing Pet. Ex. 4 at 25). On that date, "Dr. Graves diagnosed [Petitioner] with [RA]." Id. Dr. Axelrod opined that Petitioner had joint pain in her hands, wrists, shoulders, ankles, and feet, a positive rheumatoid factor, and symptoms lasting longer that six weeks, which satisfied the 2010 ACR/EULAR diagnostic criteria for RA. Id. at 9-10.

He further explained that "[t]he cause of [RA] is not known." Pet. Ex. 37 at 11. He noted "a possible mechanism . . . is exposure to a pathogen," although "no single pathogen or group of pathogens ha[ve] been defined" as causal. Id. Infections thought "to cause or contribute" to RA includes Epstein-Barr virus, Herpesvirus, Parvovirus, Retroviruses, Hepatitis

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[21] Dr. Axelrod included the criteria in his report:

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	

Pet. Ex. 37 at 9 (citing Pet. Ex. 39 at 25, tbl.92.1).

B and C, and fungal infections, among others. Id. RA has also been associated with parainfluenza virus, corona virus, and metapneumovirus. Id.

Regarding Petitioner's history of infections and the question of alternative causes of her RA, Dr. Axelrod noted that although she had a known history of Herpesvirus, Petitioner had not had a "breakout" in the 12 years prior to the onset of morning stiffness and joint pain.[22] Pet. Ex. 37 at 11. As for other infections, Dr. Axelrod acknowledged Petitioner's cough that was treated with antibiotics on November 22, 2017, but he opined that if the etiology was bacterial, the antibiotic would have "cleared the infection before she developed an immune response that might have resulted in [RA]." Id. Further, he noted that testing for flu A and B and blood cultures were negative. Id. at 12. Next, Dr. Axelrod addressed Petitioner's cough documented on November 30, 2017. Id. This time, Dr. Haygood did not prescribe an antibiotic or diagnose a viral infection. Id. Further, Dr. Axelrod opined that in December 2017, Petitioner "did not have evidence of Hepatitis C infection." Id. In conclusion, Dr. Axelrod opined that there is no evidence of an alternative cause, and that the only "new exposure" prior to the onset of RA was the flu vaccination administered December 1, 2017. Id.

Petitioner filed an "evidentiary brief" on Dr. Axelrod's medical literature. See Pet. Evidentiary Brief Concerning Propositions of Medical Literature Utilized in Doctor Axelrod's Expert Report ("Pet. Axelrod Evidentiary Br."), filed Aug. 12, 2024 (ECF No. 86). In it, Petitioner stated that Dr. Axelrod cited to a textbook to "discuss the possibility for RA development subsequent to exposure to a foreign pathogen, with the caveat that no single pathogen or group of pathogens is definitively classified as a cause for RA." Id. at 2 (emphasis omitted) (citing Pet. Ex. 41).[23]

Regarding all three Althen prongs, Dr. Axelrod deferred to Petitioner's other expert, Dr. Akbari. See Pet. Ex. 37 at 10, 12.

Dr. Axelrod did not offer opinions about whether the flu vaccine Petitioner received on October 16, 2018 caused a significant aggravation of her RA. See Pet. Ex. 37.

In conclusion, Dr. Axelrod opined that Petitioner's diagnosis is RA, and there is no evidence of any other cause, other than the flu vaccination administered to her on December 1, 2017. Pet. Ex. 37 at 13.

[22] Dr. Axelrod did not cite to the records or provide foundational support for the assertion that Petitioner had not had an outbreak of Herpesvirus in the 12 years prior to onset of her RA. See Pet. Ex. 37.

[23] Andrew P. Cope, Rheumatoid Arthritis, in Clinical Immunology: Principles and Practice 705 (Robert R. Rich et al. eds., 5th ed. 2019).

2. Petitioner's Expert, Dr. Omid Akbari[24]

a. Background and Qualifications

Dr. Akbari is a professor of immunology and professor of medicine at the University of Southern California, Keck School of Medicine. Pet. Ex. 33 at 2. He received a Ph.D. in cellular and molecular immunology at the National Institute for Medical Research in London, United Kingdom. Id. at 1. Thereafter, he completed a postdoctoral fellowship at Stanford University. Id. Dr. Akbari's research activities focus on the "[r]ole of [i]mmune cells in health and disease," including "[i]mmune tolerance and immune dysregulation" and the "[r]ole of [i]mmune cells in viral and neurological disease." Id. at 5-7. Dr. Akbari serves as an associate editor and reviewer on several journals. Id. at 4-5. He has authored or co-authored numerous publications. Id. at 10-17. None focus on RA. See id.

b. Opinion

i. Althen Prong One

Dr. Akbari purported to show "numerous ways in which the scientific evidence supports the biological mechanism by which an immune stimulated response to the flu vaccination is able to trigger immune cells that cause inflammatory disease such as RA." Pet. Ex. 9 at 12. His initial report generally discuss five topics, including the role of Th17 cells and regulatory T lymphocytes (Tregs) in the induction of RA, inflammasome pathways triggered by adjuvants and toxins, MTHFR mutations, and host susceptibility.[25] See id. at 3-11. In his second expert report, Dr. Akbari also discussed molecular mimicry. See Pet. Ex. 43 at 9-11. Each of these subjects are discussed in turn.

Dr. Akbari began with a general discussion of the role of T helper cells, or Th17 cells, noting that these cells "secrete interleukin (IL)-17" which induces inflammation that plays a role

[24] Dr. Akbari submitted two expert reports. Pet. Exs. 9, 43. Portions of Dr. Akbari's reports address concepts not related to the immediate question of whether the flu vaccine can cause RA or the other Althen prongs. For example, in his second expert report, Dr. Akbari generally discusses vaccine safety, the study of vaccine adverse reactions, the difficulty investigating rare adverse effects of vaccines, etc. See Pet. Ex. 43 at 14-18. Due to lack of relevance, these sections are not summarized.

[25] The last subject discussed by Dr. Akbari in his first expert report relates to host susceptibility. See Pet. Ex. 9 at 10-11. In his second expert report, he revisited this topic. See Pet. Ex. 43 at 20-21. His discussion of host susceptibility, and the papers cited, do not directly relate to the issues here, and therefore, are not discussed.

in the cause of "many autoimmune diseases."[26] Pet. Ex. 9 at 3-4. He asserted research of RA patients has shown "increased levels of Th17 cells[] in their peripheral blood and joints." Id. at 4. To support this proposition, he cited a paper by Zambrano-Zaragoza et al.[27] Id. (citing Pet. Ex. 32). They stated that mice studies suggest that "Th17 cells may participate in the production of autoantibodies that can induce arthritis." Pet. Ex. 32 at 5. They also noted that Th17 "frequencies were found to be increased in patients compared to controls." Id. While noting that an increase in Th17 cells has been reported in RA patients, the authors did not discuss vaccinations, or describe whether vaccinations generally, or the flu vaccination specifically, played a role in causing RA in humans.

The next article cited by Dr. Akbari to support his comment that patients with RA have increased levels of Th17 cells, Egan et al.,[28] does not reference patients with RA. See Pet. Ex. 15. In its introductory paragraph, the authors stated that Th17 cells "have been shown to mediate models of autoimmune diseases [], including . . . collagen-induced arthritis."[29] Id. at 1. They also stated that IL-17 has "potent proinflammatory effects that may promote the development of inflammatory arthritis." Id. But the article does not provide support for the proposition that human patients with RA have increased levels of Th17 cells in their serum or joints.

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[26] In his second expert report, Dr. Akbari discussed pathogenetic and non-pathogenic Th17 cells, asserting that "[p]athogenic Th17 cells are associated with inflammatory and autoimmune diseases such as RA." Pet. Ex. 43 at 7. He explained that the balance between the pathogenic and non-pathogenic Th-17 cells is important for the maintenance of a healthy immune system. Id.

[27] José Francisco Zambrano-Zaragoza et al., Th17 Cells in Autoimmune and Infectious Diseases, 2014 Int'l J. Inflam. 1. This article is also cited as Petitioner's Exhibit 74. Zambrano-Zaragoza et al. provided the following frame of reference: CD4+ T cells initiate immune responses "by providing help to other cells and taking on a variety of effector functions during immune reactions." Pet. Ex. 32 at 1. After stimulation with an antigen, "naïve CD4+ T cells activate, expand, and differentiate into different effector subsets called T helpers." Id. One of these helpers is Th17, which is "characterized by the production of distinct cytokines and effector functions. Th17 cells have been identified as one of the major pathogenic Th cell populations underlying the development of many autoimmune diseases . . . ." Id. The IL-17 family includes IL-17A and IL-17F, and these cytokines may be proinflammatory, but they also "perform diverse immunoregulatory roles during infection." Id. at 2. The focus of the discussion appears to be infection. Vaccination was not mentioned.

[28] Paul J. Egan et al., Promotion of the Local Differentiation of Murine Th17 Cells by Synovial Macrophages During Acute Inflammatory Arthritis, 58 Arthritis & Rheum. 3720 (2008).

[29] Collagen-induced arthritis is experimental form of arthritis induced in mice using complete Freund's adjuvant and type II collagen. David D. Brand et al., Collagen-Induced Arthritis, 2 Nat. Protocs. 1269 (2007), https://doi.org/10.1038/nprot.2007.173.

Egan et al. described an animal study where methylated bovine serum albumin[30] was injected into knee joints to induce inflammatory arthritis. Pet. Ex. 15 at 1. Th17 cells were found in the "inflamed synovium at the peak of disease." Id. The researchers suggested that the "differentiation of Th17 cells [was] maintained at the site of tissue inflammation by the local cytokine milieu produced by synovial macrophages." Id. at 2. However, they noted that "[t]he ability of local antigen-presenting cells to induce Th17 cells may [] depend on the initial stimulus, the cytokines produced, and the local environment." Id. at 9. Thus, the authors advised that the findings were limited to the methods employed in the study. See id. at 8-9. The authors did not suggest the findings could be extrapolated to the effects of vaccination in humans.

After asserting that Th17 and IL-17 play a role in inducing RA, Dr. Abkari then suggested that the flu vaccine increases the levels of these T helper cells and related cytokines. Pet. Ex. 9 at 6. Dr. Akbari explained that "flu infection or immunization with flu vaccine increases the level of IL-17 and Th17 cells. This observation is supported by the fact that significantly higher levels of IL-17 [were] detected in the serum, after flu infection or administration of flu vaccine." Id. In support, he cited two studies. Id. The first, by Bermejo-Martin et al.,[31] described the immune response following a severe pandemic flu infection, not vaccination. See Pet. Ex. 48. Moreover, the notable findings in the study were that in flu infections, Th17 cytokine responses were an "early distinctive hallmark[] of severe respiratory compromise." Id. at 8. The findings are not relevant to flu vaccination or to RA. The second article, by Lin et al.,[32] makes an introductory reference to IL-17 being associated with "tissue destruction" in "models of autoimmune diseases such as arthritis;" however, the focus of the paper is the protective role of IL-17 in vaccine-induced immunity against infections. Pet. Ex. 58 at 1.

Although Dr. Akbari referenced Th17 cells and their cytokine progeny (which he identified as IL-17A, IL-17F, and IL-22), he did not offer medical literature or other foundational evidence that flu vaccination increases these cells or that these cells play a role in triggering RA after administration of a flu vaccination.

The second focus of Dr. Akbari's opinions relates to regulatory T lymphocytes (Tregs). See Pet. Ex. 9 at 7-8, 11; Pet. Ex. 43 at 11-13. Dr. Akbari explained that Tregs are "key in preventing excessive immune responses and autoimmunity." Pet. Ex. 9 at 7. "A balance in the levels of Treg and T effector cells [Teffs] maintains the homeostatic and disease free state." Id.

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[30] Methylated bovine serum albumin is used to induce arthritis in mouse models. See Pet. Ex. 15.

[31] Jesus F. Bermejo-Martin et al., Th1 and Th17 Hypercytokinemia as Early Host Response Signature in Severe Pandemic Influenza, 13 Crit. Care R201 (2009). This article was also filed as Petitioner's Exhibit 12.

[32] Yinyao Lin et al., Th17 Cytokines and Vaccine-Induced Immunity, 21 Semin. Immunopathol. 79 (2010). This article was also cited as Petitioner's Exhibit 24. Further discussion of this article is below. See infra Section II.D.4.b.

at 7 fig.1. He opined that vaccines stimulate Teffs, which when induced, cause "protection or pathology such as RA and joint inflammation." Id. at 7-8; Pet. Ex. 43 at 12. Dr. Akbari further opined that in those with a genetic predisposition (like Petitioner), vaccination causes "pathogenic Teff cells . . . to differentiate further, exert robust effector function[,] and cause severe injury." Pet. Ex. 9 at 8; Pet. Ex. 43 at 12. He cited a paper by Herrero-Fernández et al.[33] purporting to show that Treg/Teff imbalance was associated with a "higher inflammatory status in a cohort of elderly" vaccinees. Pet. Ex. 9 at 8; Pet. Ex. 43 at 12; Pet. Ex. 18 at 1.

The finding of the paper, however, was that "[a]ge related homeostatic dysregulation involving the proliferation of . . . T-cell subsets, including Tregs, was related to a limited responsiveness to the [flu] vaccination and a higher inflammatory status" in 60 subjects ages 61-98. Pet. Ex. 18 at 1-2, 5 fig.1. There was a "higher baseline frequency of total-Tregs in non-responders than responders," and this higher baseline "impaired the response to vaccination." Id. at 5, 7. This same group also had "a tendency towards higher levels of [] inflammation-related markers such as D-dimers, neutrophiles[,] or the NLR," but the authors did not attribute this finding to vaccination, (and it is not clear that these markers were only seen post-vaccination) instead questioning whether it was related to thymic dysfunction, aging, conditions that cause chronic systemic inflammation, or "the underlying mechanisms [related to] progression to death in elderly people." Id. at 7-8. The authors of the study do not suggest that lack of responsiveness to the vaccine or the increase inflammatory markers seen in the non-responsive group was caused by vaccination. Thus, the relevance of this paper is not clear.

The third subject discussed by Dr. Akbari related to adjuvants or toxins in vaccines and how these initiate inflammasomes.[34] Pet. Ex. 9 at 5-6. However, there is no evidence that the flu vaccination at issue here contained either adjuvants or toxins. See Pet. Ex. 43 at 2 n.1 (Dr. Akbari agreeing the flu vaccine does not contain any adjuvant). Thus, Dr. Akbari's opinions about the effects of adjuvants or toxins, or their ability to trigger inflammasome pathways are not discussed. In his second expert report, Dr. Akbari opined that even in the absence of adjuvants or toxins, vaccines (viral peptides) can activate inflammasome pathways. Id. at 1-2. He cited several articles in support of this opinion, but none support the conclusion that vaccine-induced inflammasome activation (inflammasome dependent cytokines like IL-b and IL-1) can cause RA.[35] See id.

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[33] I. Herrero-Fernández et al., Effect of Hemostatic T-Cell Proliferation in the Vaccine Responsiveness Against Influenza in Elderly People, 16 Immun. & Ageing 1 (2019). This article is also cited as Petitioner's Exhibit 54.

[34] Dr. Akbari explained that inflammasomes have three components: "a patterns recognition receptor [], an adaptor protein, and an effector molecule called caspase-1." Pet. Ex. 43 at 8. He described the process by which inflammasomes trigger inflammation, noting their role in neurological diseases, such as multiple sclerosis, and asserted they play a role in RA. See id. at 8-9.

[35] Dr. Akbari noted one study examined the role of cytokines IL-1, IL-6, and TNF-α in the induction of experimentally-induced demyelination; however, RA was not discussed. Pet. Ex. 43 at 5.

After discussing adjuvants and toxins, Dr. Akbari opined that the MTHFR genetic mutation had a "possible role" in the "etiology of [RA]." Pet. Ex. 9 at 8-9; see also Pet. Ex. 43 at 13, 19. Dr. Akbari cited a paper by Berkun et al. as reporting a "two-fold increase in [the] prevalence of RA in patients with MTHFR polymorphism." Pet. Ex. 9 at 8 (citing Pet. Ex. 11). This data point, however, does not appear to be referenced in the article. Even if the article did offer the statistic cited by Dr. Akbari, the study is not relevant as to whether the flu vaccination can cause RA.

In his discussion of the MTHFR genetic mutation and risk of disease following various vaccinations, Dr. Akbari referenced a study by Reif et al.[36] Pet. Ex. 9 at 9 (citing Pet. Ex. 35). In that study, a single nucleotide polymorphism of MTHFR (rs1801133) was associated with adverse events after smallpox vaccination. Pet. Ex. 35 at 3. The adverse events included "fever, rash, or reginal lymphadenopathy." Id. at 1. There was no mention in the article of RA, or RA as an adverse reaction to the smallpox vaccination.

Relative to the MTHFR mutation, Dr. Akbari explained that "the decreased number of [Tregs] [is] most likely [] a main factor resulting in a person's predisposition to develop autoimmunity." Pet. Ex. 9 at 11. He asserted that this "genetic variability may influence disease progression or delay [] onset as strong Tregs are able to inhibit dysregulated immune responses and control local pathogenic cells." Id. He pointed out that "[t]hese observations [] explain why some autoimmune diseases are acute whereas others [have] [] [a] more delayed onset." Id. These observations, however, do not appear to be relevant to explain how the flu vaccine can cause RA. And Dr. Akbari does not explain the relevance to this case.[37] He cited an article by Ohkura and Sakaguchi,[38] which described how genetic variations affecting Treg cell development and function (called "Tregopathies") can cause autoimmune diseases, including

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[36] David M. Reif et al., Genetic Basis for Adverse Events After Smallpox Vaccination, 198 J. Infect. Dis. 16 (2008) (also filed as Petitioner's Exhibit 27). In his expert report, Dr. Akbari cited to an article by Rubini et al. about MTHFR polymorphism in Italian patients associated with RA; however, this article was not filed. See Pet. Ex. 9 at 8; Pet. Ex. 27. Dr. Akbari also discussed two other papers, but neither appear to be relevant and are therefore not discussed. See Pet. Ex. 9 at 9 (citing Pet. Ex. 31 (Tomoyuki Yamaguchi et al., Control of Immune Responses by Antigen-Specific Regulatory T Cells Expressing the Folate Receptor, 27 Immunity 145 (2007) (also filed as Petitioner's Exhibit 71)); Pet. Ex. 22 (Makoto Kinoshita et al., Dietary Folic Acid Promotes Survival of Foxp3+ Regulatory T Cells in the Colon, 189 J. Immunol. 2869 (2012) (also filed as Petitioner's Exhibit 57))).

[37] In this section of his expert report, Dr. Akbari cited two articles, one by Glatigny et al., which does not appear to be relevant and is therefore not discussed. See Pet. Ex. 17 (Simon Glatigny et al., Integrin Alpha L Controls the Homing of Regulatory T Cells During CNS Autoimmunity in the Absence of Integrin Alpha 4, 5 Sci. Reps. 1 (2015)).

[38] Naganari Ohkura & Shimon Sakaguchi, Transcriptional and Epigenetic Basis of Treg Cell Development and Function: Its Genetic Anomalies or Variations in Autoimmune Diseases, 30 Cell Rsch. 465 (2020).

diabetes and RA. Pet. Ex. 25 at 1, 6. The article, however, does not address vaccine causation of RA.

In his second expert report, Dr. Akbari, in response to Respondent's expert, Dr. Law, discussed the concepts of "[s]hared epitopes and molecular mimicry after inflammasome induction."[39] Pet. Ex. 43 at 9. Dr. Akbari opined shared epitopes and homology "play a crucial role in the induction of inflammasomes . . . thereby triggering T cells capable of causing [RA]." Id. He referenced a paper by Kanduc et al.,[40] where the authors showed that "various viruses, including the [flu] virus, share peptides with the human proteome." Id. (citing Pet. Ex. 56). He surmised that "if a vaccine contains peptides that mimic human autoantigens, administering that vaccine has the potential to induce an autoimmune disorder, such as [RA], particularly in susceptible individuals." Id. Even acknowledging potential homologies, Dr. Akbari agreed that "most people do not develop autoimmune diseases." Id. at 10. Further, he explained that "molecular mimicry alone is insufficient to induce autoimmunity" due to immune regulatory factors, including Tregs. Id.

Dr. Akbari cited Yoshida et al.[41] as "evidence" of homology in the context of RA. Pet. Ex. 43 at 11 (citing Pet. Ex. 73). Yoshida et al. sampled the serum of 130 patients with RA and found that 78.5% had autoantibodies to citrullinated type II collagen.[42] Pet. Ex. 73 at 1. The authors concluded that "involvement of these autoantibodies in RA pathogenesis remains unclear." Id. Yoshida et al. did not discuss the flu vaccine or provide evidence suggesting the flu vaccine caused RA by triggering autoantibodies to type II collagen.

He also cited a paper by Frankild et al.[43] to suggest that "amino acid similarity [] is a predictive measure of cross-reactivity, as observed in [flu]-induced autoimmune diseases like [RA] resulting from molecular mimicry." Pet. Ex. 43 at 10 (citing Pet. Ex. 52). However, Dr.

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[39] Dr. Akbari also cited a paper by Rojas et al. related to "autoimmune tautology" and molecular mimicry. Pet. Ex. 43 at 10. He cited the paper for the proposition that the "[flu] vaccination has been associated with an approximately [three]-fold increase in the risk of [RA] following immunization with the H1N1 [flu] vaccine;" however, he did not provide a citation to the article, nor is it filed or identified in Petitioner's list of exhibits. Id.

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

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

[42] The authors explained that "[RA] is associated with citrullination, which is the conversion of arginine residues into citrulline residues. . . . Citrullinated proteins have been found in the synovium of RA patients." Pet. Ex. 73 at 1. Of note, Petitioner had a negative cyclic citrullinated peptide antibody test. See Pet. Ex. 4 at 25.

[43] 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).

Akbari's reliance on the article is misplaced as it does not support the conclusion that RA is induced by flu infection or vaccination. The authors studied cytotoxic T cell cross reactivity. See Pet. Ex. 52. RA was not discussed. See id. The authors did not study RA, did not address whether T cell cross-reactivity was applicable to RA, or otherwise suggest that the flu infection or the flu vaccine plays any role in causing RA.

In further support of causation, Petitioner submitted an "evidentiary brief" on Dr. Akbari's medical literature. Pet. Evidentiary Br. Concerning Propositions of Medical Literature Utilized in Doctor Akbari's Expert Reports ("Pet. Akbari Evidentiary Br."), filed Aug. 12, 2024 (ECF No. 85). In this supporting brief, Petitioner explained her supporting medical literature, using the word "might" 16 times. See id. For example, citing to Symmons and Chakravarty,[44] Petitioner stated that "[i]n genetically susceptible individuals, the immune stimulation induced by a vaccine might cause a disruption in immune regulation, leading to the activation of inflammatory pathways that contribute to RA." Id. at 16 (citing Pet. Ex. 28). Describing MTHFR mutations, Petitioner cited the study by Bermejo-Martin et al. and stated that while the study describes the immune response to flu infection, "it provides insights relevant to understanding how similar responses might be triggered by vaccines." Id. at 24 (citing Pet. Ex. 48). Next, Petitioner stated that the study by Frankild et al. is "relevant for understanding how [flu] vaccines might trigger autoimmune reactions through molecular mimicry." Id. at 29 (citing Pet. Ex. 52). Lastly, by way of example, referring the paper by Tang et al.,[45] Petitioner stated that "[t]he authors investigated how the components of the flu vaccine might activate the inflammasome." Id. at 38 (citing Pet. Ex. 66).

ii. Althen Prongs Two and Three

Regarding Althen prong two, Dr. Akbari opined that Petitioner "developed RA[] as a result of the flu vaccination administered on December 1, 2017." Pet. Ex. 9 at 1. He further opined that Petitioner's "onset of symptoms following the flu vaccine support that the most logical sequence of cause and effect . . . is that the flu vaccine administered on December 1, 2017 resulted in the onset of RA." Id. at 12.

Dr. Akbari also opined that the time frame between Petitioner's flu vaccination and onset of her RA is consistent with the literature. Pet. Ex. 9 at 12; Pet. Ex. 43 at 23. In reaching this conclusion, he noted that 11 days after vaccination, on December 12, 2017, Petitioner saw Dr. Haygood, and complained of "fever, chills, cough, facial pain, and fatigue for several days." Pet. Ex. 43 at 23 (citing Pet. Ex. 5 at 123-27). Dr. Akbari also noted that Dr. Haygood diagnosed cough and fever and prescribed antibiotics. Id. Approximately two months later, on February 27, 2018, Petitioner tested positive for rheumatoid factor and was referred to rheumatology. Id. According to Dr. Akbari, most adverse reactions begin with "a type of hypersensitivity associated with the induction of inflammasome" which "increase[] over time." Id. He opined

[44] D.P.M. Symmons & K. Chakravarty, Can Immunisation Trigger Rheumatoid Arthritis?, 52 Ann. Rheum. Dis. 843 (1993). This article was also filed as Petitioner's Exhibit 65.

[45] Weichun Tang et al., Post-Vaccination Serum Cytokines Levels Correlate with Breakthrough Influenza Infections, 13 Sci. Reps. 1 (2023).

that "[t]he timing between receipt of the vaccine and development of inflammasome related reaction and subsequent development of RA by molecular mimicry is an appropriate timeframe for the immune-mediated mechanism described" in his reports. Id.

In support of the temporal association between vaccination and onset, Dr. Akbari cited Basra et al.,[46] a case report of a patient who "developed symptoms of RA shortly after receiving the swine flu vaccine." Pet. Akbari Evidentiary Br. at 1 (citing Pet. Ex. 10). "This temporal association suggests that the vaccine might have played a role in triggering the autoimmune response." Id. at 2.

Dr. Akbari did not offer opinions in support of the question of whether Petitioner experienced any significant aggravation of her RA after her flu vaccination on October 16, 2018. See Pet. Exs. 9, 43.

3. Respondent's Expert, Dr. Karen L. Law[47]

a. Background and Qualifications

Dr. Law is board certified in rheumatology and internal medicine. Resp. Ex. C at 1; Resp. Ex. D at 2. She received her M.S. from Brown University. Resp. Ex. D at 2. Thereafter, she completed an internal medicine residency and a fellowship in rheumatology at Emory. Id. She is a clinical rheumatologist and a professor of medicine in the rheumatology division at Emory University School of Medicine, as well as the associate vice chair of education and program director for the internal medicine residency at Emory. Id.; Resp. Ex. C at 1. Dr. Law "ha[s] experience in evaluating a spectrum of rheumatic diseases, including [RA], and [she] ha[s] published in the areas of [RA] and other rheumatic diseases." Resp. Ex. C at 1. She "estimates that [she] ha[s] treated over 500 [] patients with [RA] over the course of [her] career." Id. Dr. Law is also the editor and author of the 2014 textbook, *Rheumatology Board Review*, and she has authored or co-authored other publications on rheumatology. Id.; Resp. Ex. D at 15-17.

b. Opinion

i. Althen Prong One

Dr. Law opined that there is "no confirmed causal relationship" between the flu vaccine and RA. Resp. Ex. C at 9. Further, she explained that Dr. Akbari's mechanistic theory is not supported by published data and "presents an overly simplified version of possible causality" based on "one potential pathway of inflammation," without regard to multiple other "cell signaling pathways" of the immune system. Id.

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[46] Gurjot Basra et al., Rheumatoid Arthritis and Swine Influenza Vaccine: A Case Report, 2012 Case Reps. Rheumatol. 1. This article was also filed as Petitioner's Exhibit 47.

[47] Dr. Law submitted two expert reports. Resp. Exs. C, F.

She explained that RA is a common type of "inflammatory, autoimmune arthritis," affecting 1% of the population. Resp. Ex. C at 5. RA occurs more commonly in women with onset between the ages of 35 to 50. Id. The typical clinical presentation is greater than six weeks of "inflammatory arthritis in the wrists" and joints of the fingers, in a symmetrical pattern, progressing to morning stiffness, swelling, redness, warmth, decrease range of motion, and pain. Id. Without treatment, the process can lead to destruction of the joints along with systemic symptoms. Id. Diagnosis is made after a physical examination and positive laboratory studies evidencing inflammation, including elevated rheumatoid factor, anticyclic citrullinated antibody, and inflammatory markers. Id. Although RA is "mediated by immune complex deposition and inflammation in the lining of the small and medium-sized joints," the cause is not known. Id. Dr. Law noted "many genetic and environmental factors may contribute" to the disease. Id. She also emphasized that many of the genetic mutations relevant to RA have not been identified. Resp. Ex. F at 3. Similarly, there are "multiple unknown molecular mechanisms that contribute to the development of [RA]." Id.

Next, Dr. Law critiqued the causal theories advanced by Dr. Akbari. First, as for the "shared epitope" theory[48] advanced by Dr. Akbari, Dr. Law criticized his reliance on Basra et al. Resp. Ex. C at 5 (citing Pet. Ex. 10). As Dr. Law noted, Basra et al. specifically explained that a "causal relationship has not been confirmed." Id. (quoting Pet. Ex. 10 at 2). However, Dr. Akbari cited the article for the reported "temporal association," and thus, it does not appear that he inappropriately characterized this particular paper. See Pet. Ex. 9 at 6.

Regarding the role of Th17 and Tregs in RA, Dr. Law explained that Dr. Akbari "present[ed] widely accepted data" about the finding of elevated Th17 cells in synovial macrophages in animal and human models of RA. Resp. Ex. C at 6. While Dr. Law agreed these cells play a role in RA, she disagreed that there is any "specific pathway leading directly from vaccination to the development of [RA]" implicating these cells. Id. She opined Dr. Akbari's opinions "deliberately ignore[] the varied roles these cells play in . . . autoimmunity" as well as their other responses to "many environmental stimuli, including vaccination." Id.

Dr. Akbari also opined that Petitioner's MTHFR gene mutation increased her risk for RA post-vaccination, which Dr. Law observed is unsupported by any published studies. Resp. Ex. C at 7; see Pet. Ex. 9 at 8-12. Dr. Law explained MTHFR mutations have only noted a "possible link" and such link is "independent of any exposure to vaccination." Resp. Ex. C at 7. She gave an example of Dr. Akbari improperly citing an article (Reif et al.) for the general proposition that systemic adverse effects of vaccination in individuals with the MTHFR mutation may include RA, where the article did not mention RA. Id. (citing Pet. Ex. 35). Instead of RA, the adverse reactions after the smallpox vaccine in that study were identified as fever, lymphadenopathy, and rash. Id. (citing Pet. Ex. 35 at 1).

Dr. Law further criticized Dr. Akbari's discussion of alum adjuvants in vaccines, since there is "no published data supporting the hypothesis that alum adjuvant stimulation of the

[48] Dr. Law referred to this theory as the "shared epitope" theory, but the Basra et al. authors referred to it as an "antigen specific" process whereby the "vaccine products share epitope mimicry." Pet. Ex. 10 at 2.

immune system" causes unfavorable outcomes after vaccination in RA patients or that it is associated with the development of RA.[49] Resp. Ex. C at 7.

Lastly, Dr. Law observed that it does not appear that Dr. Akbari has any particular experience related to RA and "its pathogenesis or immunologic basis, nor has he had experience diagnosing or treating [RA] or associated rheumatic conditions." Resp. Ex. C at 8.

In summary, Dr. Law opined that Dr. Akbari's theory is not supported by published data. Resp. Ex. C at 9; Resp. Ex. F at 4. "No vaccine safety events linking the [flu] vaccine and RA have been reported or confirmed by vaccine surveillance programs to date." Resp. Ex. F at 4. Further, Dr. Law noted that Dr. Akbari's theory of "inflammasome-mediated pathogenesis" conflicts with "decades of scientific research suggesting multiple genetic and environmental factors, rather than one specific exposure" as the cause of RA. Id. The idea that one specific exposure could induce RA is inconsistent with "current scientific opinion" which views the case of RA to be based on a "multifactorial model of pathogenesis." Id.

ii. Althen Prongs Two and Three

Dr. Law opined that Dr. Akbari failed to provide any "logical sequence of cause and effect for the development of [RA] following [flu] vaccination [] supported by established data." Resp. Ex. C at 8. That is, he failed to identify any "specific biological mechanism connecting [the] [flu] vaccination to [RA]." Id.

Further, while the cause of RA is not known, Dr. Law agreed that Petitioner's MTHFR gene mutation does "increase [her] likelihood of developing [RA], independent of vaccination status." Resp. Ex. C at 9. Dr. Law did not opine that the increased risk posed by the MTHFR mutation, more likely than not, caused Petitioner's RA. See id. Instead, she explained that "many genetic and environmental factors may contribute" to the cause of RA. Id.

Regarding a temporal association, Dr. Law explained that the onset of Petitioner's joint symptoms is not clear. Resp. Ex. C at 6 n.1. Although Petitioner reported joint pain on January 24, 2018, she also reported that her joint pain began "a few years ago" during her first visit to her rheumatologist on March 6, 2018. Id. (quoting Pet. Ex. 4 at 31) (citing Pet. Ex. 5 at 65-69). In her affidavit, she averred that her joints felt like they were "on fire" within 45 minutes of vaccination. Id. (quoting Pet. Ex. 1 at ¶ 3). And in the weeks following vaccination, Petitioner described "fever, myalgia, and fatigue, but she did not specifically report joint pain, swelling, [or] morning stiffness," symptoms characteristic of RA. Id. at 8. Dr. Law noted the first documented complaint of joint pain was on January 24, 2018, "more than [seven] weeks after vaccination." Id.

Dr. Akbari referenced only one published report of RA following the flu vaccination, from Basra et al., where onset occurred one week after vaccination. Resp. Ex. C at 5-6, 8 (citing Pet. Ex. 10). Here, the records first note joint pain on January 24, 2018, seven weeks after vaccination. Id. at 6, 8. Dr. Law opined that seven weeks "is far beyond a medically appropriate

[49] There is no foundational evidence that the flu vaccine at issue in this matter contained alum.

timeframe to infer causation." Id. at 8. Although Dr. Akbari opined that Petitioner's symptoms occurred within "an appropriate timeframe," Dr. Law observed that he failed to provide supportive evidence to support an onset of seven weeks, especially given his theory based on an "inflammasome-related reaction." Resp. Ex. F at 2-3.

4. Respondent's Expert, Dr. John T. Bates[50]

a. Background and Qualifications

Dr. Bates is a "broadly trained immunologist" working as an assistant professor in the Department of Cell and Molecular Biology and in the Department of Medicine at the University of Mississippi Medical Center. Resp. Ex. A at 1; Resp. Ex. B at 2. He received his Ph.D. in microbiology from the University of Alabama. Resp. Ex. B at 1. Thereafter, he completed two post-doctoral fellowships, the first at Wake Forrest University School of Medicine, where he studied "the innate immune response to vaccine adjuvants, namely flagellin," and the second at Vanderbilt University School of Medicine, "where [he] worked on a number of B-cell and antibody-related projects ranging from design of vaccine immunogens to testing anti-viral vaccines in non-human primate models." Resp. Ex. A at 1; see also Resp. Ex. B at 1. Dr. Bates is a member of professional organizations, including the American Association of Immunologists and International Society for Vaccines, he has reviewed various manuscripts for journals, he has served as an ad hoc member of four different NIH study sections related to vaccines and immunology, and he has authored or co-authored numerous publications. Resp. Ex. A at 1; Resp. Ex. B at 2-8.

b. Opinion

Dr. Bates opined that the essence of Dr. Akbari's theory is that the flu immunization administered to Petitioner on December 1, 2017, "triggered an imbalance in [Petitioner's] immune system which compromised the function of immunoregulatory cells of her immune system." Resp. Ex. A at 5. He further opined "Dr. Akbari speculate[d] that such immunological imbalances are the product of inflammasome activation and a subsequent Th17-skewed CD4+ T cell response, confounded by a shortage of regulatory CD4+ T cells." Id. Although Dr. Bates agreed that Th17 CD4+ T cells are involved in inflammatory and autoimmune conditions, he noted that many details of Petitioner's RA do not support Dr. Akbari's causal injury. Id.

First, Dr. Bates disagreed that induction of inflammasome pathways by adjuvants or components of viruses (in the vaccine) result in the production of cytokines that contribute to the manifestation of RA. Resp. Ex. A at 5 (citing Pet. Ex. 9 at 6). Dr. Bates explained that fever and lassitude often "accompany vigorous immune responses that have no association with RA." Id. Further, most people who experience these symptoms do not develop RA. Id. Instead, the symptoms of fever and lassitude are "self-resolving and characteristic of a normal, vigorous innate immune response." Id.

[50] Dr. Bates submitted two expert reports in this matter. Resp. Exs. A, E.

Next, regarding Dr. Akbari's theory that adjuvants and toxins played a casual role, Dr. Bates researched the vaccine given to Petitioner, and based on the lot number, determined she received the Fluzone Quadrivalent seasonal flu vaccine, which does not contain adjuvants or toxins. Resp. Ex. A at 5-6. In response, Dr. Akbari opined that even if the flu vaccine did not contain an adjuvant, it could still stimulate inflammasomes. Resp. Ex. E at 1 (citing Pet. Ex. 43 at 1-4). In support of this opinion, Dr. Akbari cited Crooke et al.[51] Id. (citing Pet. Ex. 43 at 1-4; Pet. Ex. 51).

Dr. Bates distinguished Crooke et al., explaining that the "paper does not directly address [Dr. Akbari's] point." Resp. Ex. E at 1 (citing Pet. Ex. 51). Dr. Bates stated that in Crooke et al., the authors "measured the production of inflammasome-dependent cytokines in cultured human cells, which is not the same as measuring *in vivo* activation of the inflammasome following vaccination." Id. (citing Pet. Ex. 51). Further, Dr. Bates noted that the Crooke et al. paper was "confusing" because the authors discussed cell response after vaccination and cell response *in vitro* at the same time. Id. (citing Pet. Ex. 51). Another point made by Dr. Bates was that Crooke et al. found that post-vaccination cytokine levels were similar to baseline levels, and thus, they did not seem to have any immediate effect on inflammasome activity. Id. (citing Pet. Ex. 51 at 4). Due to this lack of response, an experimental adjuvant, Resiquimod (R848)[52] was used. Id. Based on these reasons, Dr. Bates concluded that flu vaccines without an adjuvant does not stimulate inflammasome-dependent cytokines. Id. at 2.

Dr. Bates also discussed Chatziandreou et al.,[53] another article cited by Dr. Akbari to support his opinion that the flu vaccination activated the inflammasome pathway. Resp. Ex. E at 3-4 (citing Pet. Ex. 43 at 3-4; Pet. Ex. 49). Dr. Bates explained that while cytokine IL-1α levels were elevated, this cytokine is not considered to be dependent on inflammasome. Id. at 3. The authors stated they were studying "an 'inflammasome-independent' event," which Dr. Bates noted was a "signaling pathway that does not involve the inflammasome." Id. at 3-4 (quoting Pet. Ex. 49 at 2). For these reasons, Dr. Bates concluded that Dr. Akbari's opinion that the "flu vaccine [] activates the inflammasome" is not supported by the literature in this case. Id. at 4.

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[51] Stephen N. Crooke et al., Inflammasome Activity in Response to Influenza Vaccination Is Maintained in Monocyte-Derived Peripheral Blood Macrophages in Older Adults, 2 Front. Aging 1 (2021).

[52] Resiquimod has "potential immune response modifying activity;" "[d]ue to its immunostimulatory activity, this agent may potentially be useful as a vaccine adjuvant." Resiquimod, PubChem, Nat'l Ctr. for Biotech. Info., Nat'l Libr. Med., https://pubchem.ncbi.nlm.nih.gov/compound/Resiquimod (last visited Oct. 6, 2025).

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

Next, Dr. Bates examined Dr. Akbari's assertion that patients with RA have "increased levels of Th17 cells[] in their peripheral blood and joints," and "[t]his increase in Th17 cells "is believed to be involved in the pathogenesis of RA." Pet. Ex. 9 at 4. In response, Dr. Bates explained that Dr. Akbari failed to show that the flu vaccination is associated with "detrimental levels of IL-17." Resp. Ex. A at 6. He noted Bermejo-Martin et al., an article cited by Dr. Akbari to support his opinion, reported IL-17 levels following the 2009 pandemic flu virus infection, not vaccination.[54] Id. (citing Pet. Ex. 48). Dr. Bates explained that an immune response to active infection is "significantly different" than the response following vaccination with an inactivated flu virus. Id. Dr. Bates noted that Dr. Akbari's theory, when "considered in light of . . . the Bermejo-Martin paper, may be applicable to [Petitioner's] [flu] infection in January 2018. However, it is not applicable to [Petitioner's] vaccination against [flu] in December 2017." Id.

Moreover, Dr. Bates opined that Dr. Akbari's reliance on Lin et al. is also problematic. Resp. Ex. A at 6 (citing Pet. Ex. 58). Lin et al. described the role of Th17 cells in producing cytokine IL-17 after flu vaccination.[55] Pet. Ex. 58 at 1. The authors acknowledged that IL-17 plays a role "in tissue destruction associated with models of autoimmune diseases such as arthritis, multiple sclerosis, and colitis." Id. However, the focus of the paper was on how Th17 cells, and their progeny, including IL-17, work to create vaccine-induced immunity against infectious diseases. Id. Lin et al. reviewed relevant studies and stated that "[c]ollectively, [] these reports [] demonstrate that IL-17 has a critical role in vaccine-induced immunity against bacterial infections . . . . Therefore, targeting IL-17 is an area of active research and likely to impact future vaccine design against bacterial infections." Id. at 4. In addition to bacteria, the authors examined the potential for Th17 vaccine-induced immunity against fungus, viruses, and parasites as well as how to use Th17 cytokines in conjunction with adjuvants. Id. at 4-7. The authors do not discuss the flu vaccine at issue here, nor whether it can induce production of Th17 cells that cause or contribute to the induction of RA.

Further, Dr. Bates noted that the vaccine described in Lin et al. is an "experimental DNA-based vaccine,"[56] which he opined is "fundamentally different" from the seasonal flu vaccine because the nature of the immune responses is different in DNA vaccines. Resp. Ex. A at 6 (citing Pet. Ex. 58). Dr. Bates explained that the seasonal flu vaccine uses inactivated viral

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[54] As noted by Dr. Bates, this paper examined Th1 and Th17 cytokines following infection with the pandemic flu virus. See Pet. Ex. 48. The study did not examine cytokines following vaccination or after the seasonal flu vaccination. See id.

[55] In Lin et al., the authors suggested IL-17 plays a critical role in "protective vaccine-induced immunity against infectious diseases" and may be "a good predictor of vaccination success." Pet. Ex. 58 at 1-2.

[56] Lin et al. described "DNA booster vaccines expressing *M. tuberculosis* Ag85A in the adjuvant vaxfectin induced higher levels of IL-17 . . . when compared to unvaccinated . . . groups." Pet. Ex. 58 at 6-8. This vaccine was a DNA vaccine that contained an adjuvant. See id. at 4 tbl.1. The undersigned agrees with Dr. Bates that Dr. Akbari's reliance on this information is misplaced.

antigens to induce an immune response, whereas DNA vaccines stimulate production of "antigens by cells of the immunized individual." Id. Due to the differences between the vaccines, Dr. Bates asserted Dr. Akbari's comments about the vaccine in Lin et al. cannot be extrapolated to this case. Id.

Dr. Bates next noted another paper cited by Dr. Akbari, from Symmons and Chakravarty, suffered from a similar lack of relevance problem. Resp. Ex. A at 6 (citing Pet. Ex. 28). Symmons and Chakravarty (1993) conducted a literature review of post-vaccination arthritis cases and concluded that the Rubella live virus vaccine was associated with arthritis. Pet. Ex. 28 at 1-2. The authors suggested a prospective study was warranted to explore an association between tetanus toxoid and RA. Id. As noted by Dr. Bates, the authors did not reach any such conclusions about the seasonal flu vaccine at issue here. Resp. Ex. A at 6.

In contrast, Dr. Bates cited Ray et al.,[57] a large study of one million Kaiser patients that found no association between hepatitis B, tetanus, and flu vaccinations and RA. Resp. Ex. A at 7 (citing Resp. Ex. A, Tab 1). The study employed a retrospective chart review with cohort and case-control analysis of ages 15 to 59 from 1997 to 1999. Resp. Ex. A, Tab 1 at 1. A "possible association" between the flu vaccination and RA was seen in the ranges of 180 and 365 days of vaccination in the cohort analysis, but in the larger case-control analysis this association was not seen. Id.

In addition to distinguishing the medical literature cited by Dr. Akbari based on a lack of relevance, Dr. Bates identified facts that weigh against Dr. Akbari's theories. Resp. Ex. A at 7. For example, Dr. Bates explained that Petitioner received numerous vaccinations prior to her receipt of the flu vaccination on December 1, 2017, and Petitioner did not experience any adverse effects even though she has a MTHFR mutation. Id. Citing Reif et al., Dr. Akbari opined that Petitioner's MTHFR mutation increased her risk of adverse reactions to vaccination. Pet. Ex. 9 at 9 (citing Pet. Ex. 35). But the Reif et al. study involved the smallpox vaccine, which contained a live virus, unlike the flu vaccine here. Resp. Ex. A at 7; Pet. Ex. 35. Dr. Bates explained that a live virus vaccine may be contraindicated in those who are able to receive inactivated viral vaccines, like the flu vaccine at issue here. Resp. Ex. A at 7. Moreover, Dr. Bates noted that Petitioner received the smallpox vaccine in 2006, and there was no mention of any adverse reaction to it, or any other vaccines she received, in her records. Id. (citing Pet. Ex. 5 at 59).

In addition to the risk factor associated with Petitioner's MTHFR mutation, both Dr. Akbari and Dr. Bates agreed that there are other contributing risk factors for RA, including

[57] 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).

infectious triggers,[58] such as infection with the flu virus. Resp. Ex. A at 7-8; Pet. Ex. 9 at 11. Other risk factors include family history, female gender, history of smoking, and depression. Resp. Ex. A at 8. Given her many risk factors, Dr. Bates opined it was not surprising that she developed RA at age 35. Id. He especially noted Petitioner's history of chronic infections, including Fifth's disease (documented in a February 2018 addendum) and flu infection (documented on January 2, 2018). Id. (citing Pet. Ex. 5 at 104-07, 128).

Specific to Fifth's disease, Dr. Bates noted that it is caused by Parvovirus B19, which is "one of the most frequent causes of viral arthritis." Resp. Ex. A at 8 (citing Resp. Ex. A, Tab 7 (noting "[i]nfection with parvovirus B19 causes several clinical syndrome [including []fifth disease" and listing RA as a complication post-parvovirus B19 infection during pregnancy);[59] Resp. Ex. A, Tab 8 ("Parvovirus B19 . . . [is] among the most common causative agents of viral arthritis.")];[60] see also Pet. Ex. 28 at 2 ("Parvoviruses are [] capable of inducing both an acute and a chronic arthritis. . . . Some patients with early RA have serological evidence of recent parvovirus infection.").

Dr. Akbari also opined that the flu virus can be a trigger for RA, which suggested to Dr. Bates that Petitioner's flu illness in January 2018 was "a potential cause of her RA." Resp. Ex. A at 8 (citing Pet. Ex. 9 at 11). However, Dr. Bates observed that Dr. Akbari did not address Petitioner's "intervening infection with [flu] on January 2, 2018" as "an infectious potential trigger for RA," despite identifying the flu virus as a trigger. Id. (citing Pet. Ex. 9 at 11).

Regarding the time frame between vaccination and onset of RA, Dr. Bates raised several concerns. Resp. Ex. A at 7-9. At her initial visit March 6, 2018 with her rheumatologist Dr. Graves, Petitioner reported that her joint pain began "a few years ago and ha[d] increased." Id. at 7 (quoting Pet. Ex. 4 at 31). She also reported that she had not felt well since her pertussis

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[58] Dr. Bates discussed the infections suffered by Petitioner prior to her vaccination. See Resp. Ex. A at 2-4. Of note, Dr. Bates summarized

> In February [2017], [Petitioner] suffered from a urinary tract infection ([Pet.] Ex. 5 at 208). In May [2017], [Petitioner] tested positive for antibodies against *Bordetella pertussis* ([Pet.] Ex. 5 at 162). In October, [Petitioner] presented with a "left labial abscess" ([Pet.] Ex. 7 at 196-200). On November 22, 2017, [Petitioner] was diagnosed with "[a]cute respiratory infection-possible recurrent pertussis, bronchitis or atypical pneumonia," and she had an elevated white blood cell count ([Pet.] Ex. 5 at 142 and [Pet.] Ex. 5 at 213). On November 30, 2017, one day prior to her immunization, [Petitioner's] review of systems included "fever, night sweats, weight loss, chills, [and] nosebleed." ([Pet.] Ex. 5 at 136).

Id. at 4.

[59] L.D. Rogo et al., Human Parvovirus B19: A Review, 58 Acta Virologica 199 (2014).

[60] Vikas Sharma & Aman Sharma, Infectious Mimics of Rheumatoid Arthritis, 36 Best Prac. & Rsch. Clin. Rheumatol. 101736 (2022).

infection in May 2017. Id. (citing Pet. Ex. 4 at 31). Dr. Bates opined that if Petitioner's symptoms began years or months before her December 2017 flu vaccination, then her RA was not caused by her flu vaccination. Id. He also referenced a medical record from 2013, where Petitioner complained of "arthralgia" in her review of systems and neck joint pain that had been "waxing and waning." Id. at 8 (quoting Pet. Ex. 7 at 40). Dr. Bates opined that these entries in Petitioner's medical records, especially Dr. Graves' March 2018 note reporting that her joint pain began a few years earlier, did not support a temporal association between her flu vaccination and the onset of her RA. Id. at 8-9.

In conclusion, Dr. Bates opined that Dr. Akbari failed to provide a "credible medical theory" associating the flu vaccination with Petitioner's RA. Resp. Ex. E at 5. Dr. Bates opined "it is highly unlikely" that Petitioner's flu vaccination on December 1, 2017 caused her to develop RA. Resp. Ex. A at 8; Resp. Ex. E at 5.

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

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

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

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

IV. ANALYSIS

A. Onset

Petitioner received the flu vaccination on December 1, 2017. The first mention of joint pain in her medical records was January 24, 2018, when she saw Dr. Bratcher. Review of systems was positive for "joint pain." Pet. Ex. 5 at 67. Although joint pain was documented, there was no description of the location of the pain, whether it was in symmetric joints, and swelling of the joints was not documented on physical examination (musculoskeletal examination was not documented). The following day, January 25, Petitioner saw Dr. Shahid. Dr. Shahid did not document joint pain in the review of systems. Physical examination did not reveal abnormalities of the musculoskeletal system, and no joint pain or swelling was noted at this visit. Petitioner's musculoskeletal examination was also normal when documented by Dr. Ragland on February 2, 2018.

An addendum, dated February 23, 2018, from Dr. Haygood,[61] referenced an email she received from Petitioner, presumably that day. Dr. Haygood noted Petitioner had complained of "morning stiffness/ joint aches." Pet. Ex. 5 at 128. Dr. Haygood ordered lab work for RA that day, and Petitioner's lab results dated February 27, 2018 revealed a positive rheumatoid factor test.

When Petitioner saw rheumatologist Dr. Graves on March 6, 2018, he took a very thorough history. Petitioner reported that her joint pain began "a few years ago." Pet. Ex. 4 at 31. At that visit, Petitioner reported bilaterial joint pain in her "hands, ankles, hip[s], and knees." Id. The prior week, "her hands were swollen" and she reported pain levels of 4/10 up to 10/10.

[61] Dr. Axelrod interprets Dr. Haygood's addendum to be an addition to the note referable to Petitioner's visit on December 12, 2017. The undersigned does not agree with Dr. Axelrod's' interpretation, and instead, finds that Dr. Haygood's addendum reflects receipt of an email, on or about February 23, 2018, from Petitioner requesting paperwork for her FMLA form. Presumably, in that email, Petitioner complained of morning stiffness and joint aches. This interpretation is consistent Petitioner's affidavit which states that on February 23, 2018, she sent an email to Dr. Haygood. Dr. Haygood responded to Petitioner's email and ordered autoimmune labs, which were done February 27, 2018.

Id. Dr. Graves observed that Petitioner did not have dramatic swelling in her hands, or active synovitis, but that her presentation was consistent with RA.

Petitioner executed an affidavit on December 1, 2020, which was filed with her petition on December 1, 2020. In it, Petitioner avers that "[w]ithin 45 minutes after receiving [the] flu shot" on December 1, 2017, "every joint in [her] body felt as though it [were] on fire. [She] had difficulty moving and was unable to fully extend [her] joints." Pet. Ex. 1 at ¶¶ 2-3. Petitioner's first reference in her affidavit to bilateral joint pain was on March 6, 2018, when she stated she complained of "bilateral joint pain in [her] hands, ankles, hips, and knees." Id. at ¶ 17. And Dr. Graves told her that her "pattern of pain was consistent with [RA] and possible inflammatory disease." Id.

In his explanation of the ACR/EULAR diagnostic criteria, Dr. Axelrod explained that patients can initially present with symptoms that do not fulfill the diagnostic criteria, but over time may show that the criteria are met through additional reassessments. Dr. Axelrod noted that at Petitioner's first visit to Dr. Graves on March 6, 2018, her February 27, 2018 lab studies included a positive rheumatoid factor, negative cyclic citrullinated peptide antibodies, and normal ESR. When she saw Dr. Graves on March 6, 2018, he noted that she did not have active synovitis, but he documented proximal inter-phalangeal joint tenderness, he thought her presentation was consistent with RA, and he prescribed prednisone for two weeks. Dr. Graves reached a "diagnosis" of RA on July 5, 2018, when Petitioner complained of "increased pain at her neck, hands, hips, knees, feet[,] and ankles, worse over the previous week, with swelling of her hands and feet, despite Diclofenac and Sulfasalazine." Pet. Ex. 4 at 22-25.

Regarding Petitioner's affidavit testimony about having a response 45 minutes after vaccination, feeling like "her joints were on fire," the undesigned notes that none of the experts opined that this response was a manifestation of the onset of her RA. Without an opinion by a treating physician or expert that Petitioner's response was a manifestation of RA, the undersigned does not find it evidences the onset of RA. Further, Petitioner's medical records do not document this response. After her flu vaccination, Petitioner was seen by many different health care providers over the course of the following months, and none of these providers documented the response she described in her affidavit as occurring 45 minutes after vaccination.

Medical records generally "warrant consideration as trustworthy evidence." Cucuras, 993 F.2d at 1528. However, greater weight is typically given to contemporaneous records. Vergara v. Sec'y of Health & Hum. Servs., No. 08-882V, 2014 WL 2795491, at *4 (Fed. Cl. Spec. Mstr. May 15, 2014) ("Special Masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recorded in later medical histories, affidavits, or trial testimony."); see also Doe/70 v. Sec'y of Health & Hum. Servs., 95 Fed. Cl. 598, 608 (2010). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Cucuras, 993 F.2d at 1528. Here, the contemporaneous records are silent regarding any signs or symptoms described by Petitioner in her affidavit.

Therefore, the undersigned does not find Petitioner's affidavit testimony regarding her recitation of the events on the day of vaccination to be persuasive evidence. See Vergara, 2014 WL 2795491, at *4 ("[T]estimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." (quoting Murphy, 23 Cl. Ct. at 733)); see also Doe/70, 95 Fed. Cl. at 608 ("Given the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law.").

Taking the whole of Petitioner's clinical course into account, the undersigned finds that she had joint pain for "a few years" before she saw Dr. Graves on March 6, 2018. A reasonable interpretation of the phrase "a few years" is more than one year, but maybe two or three years. The records again noted joint pain on January 24, 2018. However, joint pain on review of systems, without more, is not preponderant evidence of RA. More specific to RA, and based on the opinions of Dr. Axelrod, as well as the medical records of Dr. Haygood and Dr. Graves, the undersigned finds that Petitioner complained of morning stiffness and joint pain in an email to Dr. Haygood on February 23, 2018. She had a positive rheumatoid factor serology on February 27, 2018. On March 6, 2018, she reported involvement of her hands, hips, knees, feet, and ankles. As explained by Dr. Axelrod, and documented by Dr. Graves, these symptoms are consistent with RA. Therefore, the undersigned finds the first manifestation specific to RA occurred on February 23, 2018, when she complained of morning stiffness and joint pain in an email to Dr. Haygood, which caused Dr. Haygood to order lab work up for autoimmune conditions, including RA.

B. Causation

1. Althen Prong One

Under Althen Prong One, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 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 v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("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. There are several reasons for this finding.

First, Dr. Akbari describes six immunological concepts, drawn from general immunology principles and various clinical studies, but none of the concepts individually or when joined together constitute a mechanism to explain how the flu vaccine can cause RA. For example, his discussion about Th17 cells and IL-17 does not address basic questions relevant to the pathogenesis of RA. Dr. Akbari provides an overview of Th17 cells, explaining that these cells produce cytokines, induce inflammation, and assiss in the maintenance of immune system homeostasis. But he does not explain from start to finish what and how the flu vaccine induces Th17 cells upon vaccination, what happens after vaccination in the body relative to these cells, how these cells travel to the joints, what part of the joints are affected, or how these cells induce joint pain and destruction. He also does not explain how this process becomes chronic. In summary, he introduces concepts pulled from the literature but does not build the framework required to support a mechanism of causation.

In the conclusion of his first expert report, Dr. Akbari writes that he has "shown numerous ways in which the scientific evidence supports the biological mechanism by which an immune stimulated response to the flu vaccination is able to trigger immune cells that cause inflammatory disease such as RA." Pet. Ex. 9 at 12. While it may not be inappropriate to explore six alternative ways that a vaccine could cause RA, Petitioner's approach of identifying a handful of theories unsupported by literature or other evidence reduces the persuasiveness of the opinions offered. See, e.g., Baron v. Sec'y of Health & Hum. Servs., No. 14-341V, 2019 WL 2273484, at *17 (Fed. Cl. Spec. Mstr. Mar. 18, 2019) ("Although Petitioners . . . do not need to provide the specific components of the mechanism by which the vaccine[] at issue can cause [the alleged injury], they do need to propose something more than taking a vague 'kitchen sink' approach and listing eleven mechanisms that have been previously submitted in the Program for claims of vaccine-caused injury with various degrees of success. Petitioners have listed many possibilities but have not identified a sound and reliable explanation that can be applied to the vaccines and injury in this case.").

Second, Dr. Akbari offered an erroneous opinion which the undersigned finds also undermined his credibility and persuasiveness. In his initial report, he discussed the addition of adjuvants and toxins to the vaccine. However, the flu vaccine at issue here does not contain adjuvants or toxins, which Dr. Akbari agreed with in this second expert report after Dr. Bates pointed this out. See Pet. Ex. 43 at 1-2, 2 n.1 (Dr. Akbari agreeing the flu vaccine does not contain any adjuvant). Thus, it appears Dr. Akbari was not aware of the components of the flu vaccine when he issued his first report. An immunology expert who offers opinions about vaccine causation is expected to have a working knowledge of vaccines in general, as well as the vaccine at issue. This includes a general understanding of the components of the vaccine, including whether it contains an adjuvant to induce an immune response. Dr. Akbari's discussion of adjuvants in his first expert report suggests that he did not know that the vaccine did not contain an adjuvant. This lack of knowledge diminishes the persuasiveness of his opinions.

Third, Dr. Akbari mischaracterizes the medical literature he cites which further adversely affects his credibility and the reliability of his opinions. A number of examples are cited above, during the summary of his expert opinions and discussion of the medical articles. See supra

Section II.D.2.b. Without reviewing all of the examples set forth above, several are summarized here. He cited Egan et al. for the proposition that Th17 cells play a role in inducing RA. Egan et al., however, discusses experimental collagen-induced arthritis in animal models and describes a study where methylated bovine serum albumin is used in animals to induce inflammatory arthritis. While the study does offer important information about Th17 cells in acute inflammatory arthritis in the context of animal models, the authors do not suggest that its findings are comparable to the effects of the flu vaccination in humans or that their study simulates vaccination. Dr. Akbari failed to identify the distinction between the study and the context at hand.

Another illustration is Dr. Akbari's use of Bermejo-Martin et al. to support his opinion that the flu vaccination increases the Th17 and IL-17 levels. Bermejo-Martin et al. described an immune response to a severe pandemic form of flu infection, not vaccination. Further, the Th17 response was described as severe respiratory distress, not an inflammatory response of the joints that occurs in RA. Again, in his discussion, Dr. Akbari did not describe the limitations of the study.

Next, Dr. Akbari cited to papers related to infections, not vaccinations, suggesting that the immune responses to both were the same. See Pet. Ex. 9 at 6 ("[F]lu infection or immunization with flu vaccine increases the level IL-17 and Th17 cells"). Dr. Akbari did not acknowledge or identify the limitations of the studies he cited, differentiate between infection and vaccination, or provide foundational evidence to bridge the gap between experimentation and preponderant proof of causation. "An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of Health & Human Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280).

Fourth, there is a lack of evidence that the flu vaccine can cause RA. Petitioner cites only one case report (Basra et al.) of RA following the flu vaccination. While case reports merit some evidentiary weight, they do not constitute strong supportive evidence of vaccine causation, and alone cannot meet the preponderance of the evidence standard. See Campbell v. Sec'y of Health & Hum. Servs., 97 Fed. Cl. 650, 668 (2011); Caves v. Sec'y of Health & Hum. Servs., No. 07-443V, 2010 WL 5557542, at *14 (Fed. Cl. Spec. Mstr. Nov. 29, 2010) (finding a petitioner's "reference to case reports [did] not help her meet her burden of demonstrating a persuasive and reliable theory causally connecting the [] vaccine to [the injury]"), mot. for review den'd, 100 Fed. Cl. 119 (2011), aff'd, 463 F. App'x 932 (Fed. Cir. 2012); Muchnik ex rel. Muchnik v. Sec'y of Health & Hum. Servs., No. 90-703V, 1991 WL 217673, at *4 (Fed. Cl. Spec. Mstr. Oct. 10, 1991) ("For the petitioner to establish causation in fact by a preponderance of the evidence in any given case requires something more than case reports . . . .").

Additionally, Respondent filed several large studies which do not show an association between the flu vaccine and RA. For example, in Ray et al., the authors conducted a study involving medical chart review of approximately one million patients with RA from 1997 to 1999. The authors 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 in their larger case-control analysis, no association was seen.

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 evidence presented demonstrates no association between the flu vaccine and the development of RA.

Fifth, in support of causation, Petitioner submitted an evidentiary brief regarding the medical literature cited by Dr. Akbari. In this brief, Petitioner repeatedly used the word "might" in relation to causation. According to Merriam-Webster dictionary, "might" is "used to say something is possible." Might, Merriam-Webster Dictionary, https://www.merriam-webster.com/dictionary/might (last visited Oct. 7, 2025). Similarly, Cambridge Dictionary indicates "might" is "used to express the possibility that something will happen or be done, or that something is true although not very likely." Might, Cambridge Dictionary, https://dictionary.cambridge.org/us/dictionary/english/might (last visited Oct. 7, 2025). Counsel's interpretation of the medical literature is consistent with that of the undersigned. The articles offer possible concepts which might be relevant. Possibilities, however, are insufficient. 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, for example, 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 more recently, 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.

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 these cases instructive. 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 supported by medical or scientific facts, research, or any other reliable evidence. Moreover, the concepts are not developed and are speculative and conclusory in nature. When evaluating whether petitioners have carried their burden of proof, special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. denied, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. See id.

In summary, Petitioner has failed to offer a sound and reliable medical theory in support of her claim. 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 her burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

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

First, Petitioner's treating physicians did not offer opinions in her medical records associating her RA with her flu vaccinations. 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)). Here, there is no such supportive evidence.

Second, there is evidence of risk factors and alternative causes for Petitioner's RA. Petitioner's most significant risk factor was her MTHFR mutation. The experts generally agreed that this risk factor made Petitioner more susceptible to developing the disease, though they did not opine that the risk factor alone more likely than not caused her RA. See Pet. Ex. 9 at 8-9 (Dr. Akbari discussing the "possible role" of the MTHFR genetic mutation in the "etiology of [RA]"); Resp. Ex. A at 7-8 (Dr. Bates listing numerous risk factors Petitioner had for developing RA, including the MTHFR polymorphism); Resp. Ex. C at 7, 9 (Dr. Law agreeing that Petitioner's MTHFR gene mutation does "increase [her] likelihood of developing [RA], independent of vaccination status," but not opining that the increased risk was more likely than not the cause of Petitioner's RA).

Regarding an alternative cause, however, Dr. Bates identified parvovirus and flu infections as potential causes of Petitioner's RA. Both Dr. Bates and Dr. Akbari cited literature supporting parvovirus as a common cause of arthritis; however, Dr. Akbari did not discuss parvovirus, or its relation to Fifth's disease. See Resp. Ex. A, Tabs 7-8; Pet. Exs. 9, 28, 43. Dr. Akbari agreed that the flu infection can be a trigger for the illness. See Pet. Ex. 9 at 11 ("Additional infectious potential triggers include . . . [flu] virus."). But he failed to discuss or acknowledge Petitioner's flu infection in January 2018. See Pet. Exs. 9, 43.

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 causes, parvovirus and flu illnesses. The undersigned finds it reasonable to consider these potential causes of Petitioner's condition in determining that Petitioner has failed to provide preponderant evidence of prong two.

Accordingly, the undersigned finds that Petitioner failed to satisfy her 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").

Petitioner, through Dr. Akbari, offered six different theories of causation, describing how "the flu vaccine can provoke innate and adaptive immunity." Pet. Ex. 9 at 1. But Dr. Akbari did not provide an appropriate timeframe relative to each of his theories. As a general principle, innate immune responses occur more quickly than adaptive immune responses.[62] However, Petitioner did not provide such time periods here.

For example, Dr. Akbari did not opine as to an appropriate onset timeframe for his theory about the role of inflammasome and Th17 cells in the induction of RA. But see Resp. Ex. F at 2-3 (Dr. Law opining that Dr. Akbari failed to provide supportive evidence to support an onset of

[62] See Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57, 57-58 (Kathleen Stratton et al. eds., 2012).

seven weeks here, especially given his theory based on an "inflammasome-related reaction," when the only supportive literature was Basra et al., who documented a case report of an onset of one week) (citing Pet. Ex. 10)). The same is true for Dr. Akbari's other five theories. Instead, Dr. Akbari opined that "onset of disease occurred within an appropriate time period after vaccination to implicate the vaccination as a cause of her illness." Pet. Ex. 9 at 1-2. He concluded that Petitioner's development of RA was consistent with the "cause and effect" of the flu vaccination. Id. at 12.

To the extent that Dr. Akbari opines Petitioner's onset was 11 days post-vaccination because this was her first health care appointment after her December 1, 2017 flu vaccination, the undersigned does not agree. As Dr. Akbari documents, Petitioner complained of "fever, chills, cough, facial pain, and fatigue for several days" during this visit and was diagnosed with a cough and fever. Pet. Ex. 43 at 23. No complaints of joint pain were made during this visit. Nor were physical examination findings consistent with RA documented. According to Dr. Akbari, most adverse reactions begin with "a type of hypersensitivity associated with the induction of inflammasome" which "increase[] over time." Id. However, neither of his reports discuss this "hypersensitivity" reaction, or mention "hypersensitivity" again.

In summary, Petitioner offered no expert opinions as to the specific onset or range of onset of her RA. Nor did Petitioner provide expert opinions explaining how the causal theories offered were consistent with that time frame.

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

V. CONCLUSION[63]

The undersigned extends her sympathy to Petitioner for the pain and suffering that she experienced due to her 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 her flu vaccine caused her 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

[63] Due to the sensitive nature of Petitioner's medical and psychiatric history, Petitioner is encouraged to seek redaction of her name to her initials in accordance with Vaccine Rule 18(c).