In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 22-101V

RUBY SHARMA DHITAL,	*	Chief Special Master Corcoran
Petitioner,	*	Filed: October 2, 2025
v.	*	Reissued for Public Availability: February 6, 2026
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Michael G. McLaren, Black McLaren, et al., PC, Memphis, TN, for Petitioner.

Catherine E. Stolar, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On February 2, 2022, Ruby Sharma Dhital filed a petition seeking compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] Petition (ECF No. 1) at 1. Petitioner alleged that her Eosinophilic Granulomatosis with Polyangiitis ("EGPA"), also known as Churg-Strauss syndrome, was allegedly caused-in-fact by her receipt of an influenza ("flu") vaccine on October 1, 2019. *Id.*

A two-day Entitlement Hearing was held in Washington, DC on October 22 and 24, 2024. Now, for the reasons set forth below, and based upon the record evidence, I deny entitlement. Petitioner has not shown she likely had EGPA—nor has she preponderantly demonstrated that her vasculitis-like condition, however properly diagnosed, could be worsened by the flu vaccine.

[1] Pursuant to Vaccine Rule 18(b), this Decision was initially filed on October 2, 2025, and the parties were afforded 14 days to propose redactions. The parties did not propose any redactions. Accordingly, this Decision is reissued in its original form for posting on the court's website.

[2] The Vaccine Program comprises Part 2 of the national Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) ("Vaccine Act" or "the Act"). Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

I. Factual History

Some pre-vaccination medical treatment encounters are relevant to the present action. In April 2019, Petitioner had seen her primary care provider ("PCP"), Dr. Alina Dobrita, for an annual exam and complained of right shoulder pain that started that morning, along with pain in left shoulder, foot, and ankle. Ex. 4 at 13-14. Blood tests revealed anemia,[3] and a urinalysis showed blood in her urine/stool. *Id*. at 43. Ten days later, on April 19, 2019, Petitioner presented to family medicine Advanced Practice Registered Nurse ("APRN") Kim Morgan for pain in both shoulders (left greater than right) for one week. Ex. 3 at 2, 10. Her weight at this visit was 135 pounds. *Id.* at 12. Petitioner was diagnosed with left shoulder bursitis. *Id.* at 2. Petitioner also had a pre-vaccination history of hypothyroidism. *See id.* at 32; Ex. 9 at 9.

Vaccination and Initial Symptoms

Ms. Dhital was twenty-seven years old when she received the flu vaccine at issue on October 1, 2019. Ex. 9 at 162. There is no record evidence of any immediate vaccine reaction. But twelve days later (October 13, 2019), Petitioner returned to see APRN Morgan for a cough and cold symptoms, and was diagnosed with an upper respiratory infection. Ex. 3 at 23, 26. At this appointment, Petitioner now weighed 120 pounds (fifteen pounds less than her weight approximately six months before). *Id.* at 26. The next day (October 14, 2019), Petitioner was seen by Dr. Dobrita for a worsening cough and flu-like symptoms, including chest pain, chills, fever, headaches, and joint pain. Ex. 4 at 18–19. On exam, Petitioner had a low-grade fever and low blood pressure. *Id.* at 19–20. Petitioner "appear[ed] ill" and had mild redness in her throat, with ulcers. *Id.* at 20. Petitioner was treated for presumed influenza. *Id.* at 18.

A few days later, on October 17, 2019, Petitioner followed up with Dr. Dobrita, now complaining of a continued cough, joint pain in the knees and shoulders, pain under her left rib cage, chest tightness, swelling in her ankles, decreased appetite, generalized weakness, and fatigue. Ex. 4 at 23. On exam, Petitioner had a slight fever, appeared ill, and had tenderness in her chest, elbows, and knees. *Id.* at 24. Petitioner was unable to take deep breaths, and Dr. Dobrita observed decreased breath sounds in the left lower lobe. *Id.* Dr. Dobrita opined that Petitioner's joint pain was associated with her recent flu infection. *Id.* at 22. An x-ray revealed two round opacities in Petitioner's right lung. *Id.* at 23. Her blood tests showed worsening anemia, increased levels of

[3] "Anemia is a condition that develops when a person's blood produces a lower-than-normal amount of healthy red blood cells." *What Is Anemia?*, National Heart, Lung, and Blood Institute, https://www.nhlbi.nih.gov/health/anemia (last visited Sep. 29, 2025).

eosinophils[4], an elevated "SCL-70" antibody level,[5] and antinuclear antibodies. *Id.* at 44, 46, 49. Petitioner was started on antibiotics. *Id.* at 23.

By the next day, Ms. Dhital's joint pain had worsened, and she was having a hard time walking, so her husband took her to the emergency room. Ex. 4 at 23. Petitioner's ER records revealed elevated hemoglobin, anemia, microcytosis (smaller-than-normal red blood cells), hypochromia[6], a high erythrocyte sedimentation rate ("ESR"), plus a red patchy rash on her arms and legs. Ex. 4 at 23. Dr. Dobrita's assessment was an inflammatory rheumatologic disorder versus reactive arthritis with pneumonia. *Id.*

On October 22, 2019, Petitioner met with rheumatologist Dr. Ioana Stanescu and complained of persistent hemoptysis (coughing up blood). Ex. 8 at 67. Her cough and joint pain were improving, but she still exhibited joint tenderness and her left elbow was swollen. *Id.* at 68, 73. She also had erythematous skin, and a papular rash along the back of her ankles and left calf. *Id.* at 73. Dr. Stanescu noted that Petitioner's clinical presentation "strongly suggest[ed] serum sickness"[7] with possible infectious etiology. *Id.* at 67. She did not have clinical signs of systemic sclerosis,[8] however, and Dr. Stanescu opined that Petitioner's positive SCL-70 antibody test result could reflect a transitory "immune response to [a] present infection." *Id.*

Progression in Symptoms and Hospitalization

The next day (October 23, 2019), Petitioner returned to the ER with worsening symptoms, including fever, cough, chest pain, rash, joint pain, shortness of breath, and unintentional weight loss. Ex. 9 at 8–9. On exam, Petitioner had an elevated heart rate, fever, left eye conjunctival injection (blood-shot eyes), tachycardia (heart beating faster than normal), diminished breathing sounds, swelling along her left hand and fingers, and multiple areas of focal erythematous lesions

[4] Eosinophils are a type of white blood cell that help the body fight infections and allergens. *Eosinophils*, Cleveland Clinic, https://my.clevelandclinic.org/health/body/23402-eosinophils (last visited Sep. 29, 2025).

[5] SCL-70 antibody, also known as topoisomerase I antibody, is an autoantibody that targets a protein called topoisomerase I. It is associated with the autoimmune disease scleroderma/ systemic sclerosis. A. Aggarwal, *Role of Autoantibody Testing*, 28 Best Practice & Research Clinical Rheumatology 907 (June 2015).

[6] Hypochromia refers to a qualitative impression that red blood cells have less color than normal when examined under a microscope—this is usually related to a reduced amount of hemoglobin in the red blood cells. Hypochromia, Medline Plus, https://medlineplus.gov/ency/article/003455.htm (last visited Sep. 29, 2025).

[7] "Serum sickness is an immune-complex-mediated hypersensitivity reaction that classically presents with fever, rash, polyarthritis or polyarthralgias. Serum Sickness." National Library of Medicine, https://www.ncbi.nlm.nih.gov/books/NBK538312/ (last visited Sep. 29, 2025).

[8] Systemic sclerosis (also known as "scleroderma") is an autoimmune disease that involves the buildup of fibrous tissue/collagen in the skin. Scleroderma, PennMedicine, https://www.pennmedicine.org/for-patients-and-visitors/patient-information/conditions-treated-a-to-z/scleroderma (last visited Sep. 29, 2025).

(skin redness and discoloration). *Id.* at 10. A CT chest exam showed numerous, patchy, nodular opacities throughout the left lung and right lung base, "favored to reflect multifocal pneumonia." *Id.* at 32. And an x-ray of Petitioner's lungs showed opacities and airspace consolidations consistent with pneumonia. *Id.* at 275.

Petitioner was admitted to the hospital, and later that day met with internal medicine physician Hnin Oo, M.D. Ex. 9 at 25. Dr. Oo took note of Petitioner's blood-shot eyes and joint tenderness, and recommended that she be seen by infectious disease and rheumatology for sepsis secondary to multifocal pneumonia, rash, joint pain, and positive SCL-70. *Id.* at 25–26, 28.

The following day, Ms. Dhital had a consultation with infectious disease specialist Michael Lawlor, M.D., who determined that Petitioner had an "inflammatory process going on, perhaps vasculitis, perhaps a connective tissue disease." Ex. 9 at 33. Dr. Lawlor did not believe Petitioner's condition had an infectious etiology. *Id.* Petitioner also met at this time with a rheumatologist Tejas Sheth, M.D., who noted that Petitioner had blood-shot eyes, swelling and tenderness in the left knee and both hands, and a rash on both shins and feet. *Id.* at 40, 44–45. Dr. Sheth observed that Petitioner's clinical presentation, including migratory polyarthralgia (joint pain) with inflammatory arthritis on exam and lower extremity rash in the setting of fever, was concerning for serum sickness "likely triggered by infection/influenza/antiviral/antibacterial therapy versus arthritis versus connective tissue disease process[.]" *Id.* at 41. Dr. Sheth also noted that Petitioner's positive SCL-70 result "in [the] absence of clinical features of scleroderma," was of "unknown clinical significance[,] especially in [the] presence of infection." *Id.* Dr. Sheth agreed with aggressive antibiotic therapy for multifocal pneumonia. *Id.*

On the evening of October 24, 2019, Petitioner met with pulmonologist Michael Perkins, M.D. Ex. 9 at 48. Petitioner and her husband reported that "the flu vaccination kicked off her acute presentation, and aside from some mild seemingly isolated arthralgias[,] she was largely asymptomatic prior to this event." *Id.* at 48–49. Dr. Perkins did "not feel particularly strongly that this [condition] [was] an overwhelming multifocal pneumonia," but did "favor an autoimmune process" of some sort. *Id.* at 49. Dr. Perkins ordered a bronchoscopy for the next day. *Id.*

The next day (October 25, 2019), Petitioner underwent the planned bronchoscopy, and it was performed by pulmonologist Jeffery Nascimento, D.O. Dr. Nascimento noted that Petitioner had bilateral ground-glass opacities (right lung greater than left), with underlying autoimmune versus rheumatologic process and hemoptysis. Ex. 9 at 86, 525–26. On this same day, Petitioner complained about her itchy rash to dermatologist Frank Santoro, M.D., who observed Petitioner had "significantly elevated ESR and remarkable eosinophilia[.]" *Id.* at 53–55. Dr. Santoro also noted that peripheral eosinophilia could be "seen with Churg [S]trauss vasculitis or parasitic disease, although [Petitioner's] skin finding[s] [were] more [consistent with] vasculitis." *Id.* at 54.

Dr. Santoro raised interstitial or palisaded granulomatous dermatitis[9] as an alternative possible diagnosis. *Id.*

On October 27, 2019, Dr. Sheth recommended that Petitioner commence an empiric trial of IV Solumedrol, given her "worsening pulmonary status." Ex. 9 at 107. Later that day, Petitioner's transbronchial biopsy results revealed diffuse alveolar hemorrhage[10] with eosinophils and capillaritis. *Id.* at 247–48. The reviewing pathologist noted that "due to the abundance of eosinophils, Churg-Strauss disease should be considered." *Id.* at 248. In an addendum authored the same day, Dr. Sheth stated that "per [his] recent conversation with Dr[.] Nascimento," Petitioner's preliminary transbronchial pathology was "consistent with EGPA in setting of findings of capillaritis eosinophil infiltrate and [diffuse alveolar hemorrhage]." *Id.* at 111. Dr. Sheth recommended proceeding with pulse steroids for three days. *Id.*

The following day, Dr. Sheth met with Petitioner, who reported "dramatic improvement" following the first dose of pulse steroids, with improved breathing and no pain in her joints or hemoptysis. Ex. 9 at 111. She still had a lower extremity rash and small lesions on her left palm and elbow. *Id.* at 129. Dr. Sheth's assessment was "likely EGPA." *Id.* at 128. Petitioner also again saw Dr. Santoro, who noted that she did not have a history of atopy (hypersensitivity to environmental allergens), which would be highly atypical for EGPA patients, but did have a history of eosinophilia and elevated immunoglobulins. *Id.* at 132. He further noted that Petitioner's skin pathology correlated with her lung pathology. *Id.* Dr. Santoro's assessment was systemic vasculitis in the small and medium blood vessels. *Id.*

On October 29, 2019, Petitioner met with another rheumatologist, John Vischio, M.D., who noted that her EGPA diagnosis was supported by the transbronchial biopsy results, skin biopsies that were consistent with vasculitis, a history of peripheral eosinophilia and elevated IgE, inflammatory polyarthritis, and elevated ESR and CRP. Ex. 9 at 158. Petitioner was started on Rituxan IV the next day (a medication used for autoimmune diseases, including different forms of polyangiitis). *Id.* Petitioner also received a red blood cells transfusion and iron. *Id.* at 158, 162. The next day, internist April Goller, D.O., observed that Petitioner had microcytic anemia that "look[ed] like anemia of chronic disease." *Id.* at 179.

[9] Granulomatous dermatitis is a rare inflammatory skin condition associated with systemic immune-mediated diseases. *Granulomatous Dermatitis*, DermNet, https://dermnetnz.org/topics/granulomatous-dermatitis (last visited Sep. 29, 2025).

[10] Diffuse alveolar hemorrhage ("DAH") is a syndrome that involves damage to the small blood vessels that supply the lungs. DAH results in recurrent or persistent bleeding in the lungs and is most often caused by an autoimmune disorder. *Diffuse Alveolar Hemorrhage*, Merck Manual, https://www.merckmanuals.com/home/lung-and-airway-disorders/autoimmune-disorders-of-the-lungs/diffuse-alveolar-hemorrhage (last visited Sep. 29, 2025).

Treatment After Hospitalization

On November 1, 2019, Petitioner was discharged from the hospital with an EGPA diagnosis. Ex. 9 at 19. At the time of discharge, however, Petitioner's ANCA[11] and Proteinase-3 ("PR3")[12] antibody tests were still pending. *Id.* at 23. These tests ultimately revealed somewhat increased levels of ANCA, but high levels of PR3 antibodies. *Id.* at 219–20. Petitioner's SCL-70 antibody levels also remained elevated. *Id.* at 222.

On November 4, 2019, Petitioner followed up with Dr. Stanescu. Ex. 8 at 60. On exam, Petitioner displayed an erythematous, popular rash on the back of her ankles and left calf, oral thrush, and a small ulcer on her tongue. *Id.* at 66. Her inflammatory arthritis had resolved. *Id.* at 60. The next day, she saw nephrologist Jeffrey Laut, M.D., who noted that Petitioner had "recently developed respiratory failure with hemoptysis, with a final diagnosis of Eosinophilic Granulomatosis or Churg-Straus Syndrome." Ex. 7 at 19. Dr. Laut added that "this all started with a flu-like syndrome as a prodrome, and a flu vaccine a week before symptoms started." *Id.* His assessment included eosinophilic vasculitis and acute glomerular disease.[13] *Id.*

A November 27, 2019, X-ray showed near-complete resolution of Petitioner's previous infiltrates. Ex. 8 at 97. A few weeks later, on December 17, 2019, Petitioner met with pulmonology Physician Assistant Sarah Fontaine and reported improved breathing. Ex. 6 at 13. Her pulmonary function tests were normal. *Id.* at 15. PA Fontaine's assessment was Churg-Strauss syndrome with lung involvement, but she noted that Petitioner was "asymptomatic respiratory-wise." *Id.* at 17.

By January 8, 2020, Petitioner was down to 15 mg of prednisone a day and had no more rashes, joint pain, fatigue, fever, flank pain, hematuria (blood in the urine), or hemoptysis. Ex. 8 at 47. Dr. Stanescu now noted that petitioner's C-ANCA was positive, and the diagnosis now proposed was granulomatosis with polyangiitis ("GPA"), with renal involvement. *Id.* Later that same month, she saw an ophthalmologist, who diagnosed her with scleritis. Ex. 15 at 6.

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[11] "Anti-neutrophil cytoplasm antibodies [("ANCA")] are autoantibodies that target a type of human white blood cell called neutrophils, which are important in health for fighting infection partly through the release of toxic substances that destroy bacteria." *Anti-Neutrophil Cytoplasm Antibodies (ANCA)*, Vasculitis UK, https://www.vasculitis.org.uk/about-vasculitis/what-is-anca (last visited Sep. 29, 2025). As discussed below, there are a number of different types of "ANCA-associated Vasculitis," including EGPA.

[12] Proteinase 3 ("PR3") is a protein. Patients with ANCA-associated vasculitis often test positive for autoantibodies against PR3. *Anti-Neutrophil Cytoplasm Antibodies (ANCA)*, Vasculitis UK, https://www.vasculitis.org.uk/about-vasculitis/what-is-anca (last visited Sep. 29, 2025).

[13] Glomerular disease causes damage to a person's kidneys. The disease attacks glomeruli—tiny filters in the kidneys where the blood is cleaned. Damaged glomeruli can allow proteins and sometimes red blood cells to leak into a person's urine. *Glomerular Disease*, National Institute of Diabetes and Digestive and Kidney Diseases, https://www.niddk.nih.gov/health-information/kidney-disease/glomerular-disease (last visited on Sep. 29, 2025).

Petitioner again saw Dr. Stanescu in the late summer and fall of 2020. Dr. Stanescu agreed that Ms. Dhital was likely experiencing "smoldering nephritis" requiring Rituxan. Ex. 8 at 21 (August 2020 encounter). But Dr. Stanescu also continued to embrace the modified diagnosis of "GPA /ANCA associated vasculitis with renal involvement." *Id.* at 14 (October encounter).

On March 11, 2021, Petitioner followed up with Dr. Stanescu and reported that she felt "very well." Ex. 8 at 7–8. She complained of intermittent headaches, burning on the bottom of her feet, and knee pain prior to her menses, but denied fatigue, shortness of breath, cough, sinus pressure, congestion, runny nose, blood in her urine, joint pain, and rashes. *Id.* at 8. She was now diagnosed with granulomatosis with polyangiitis, with renal involvement. *Id.* at 7, 13, 93.

By May 11, 2021, Petitioner was receiving Rituxan every six months, and her renal function was normal. Ex. 7 at 2. Dr. Laut observed that Petitioner was "dramatically better" at this point, and she did not appear to have "active" glomerular disease. *Id.* at 2, 4. Dr. Laut was in favor of lowering her Rituxan dose, while beginning a very low dose of an ACE inhibitor. *Id.* at 4. No documents for subsequent periods were filed.

II. Witness Testimony

A. Petitioner's Fact Witnesses

1. *Ms. Dhital* – Petitioner testified at the hearing. *See generally* Tr. 7–28. She began her testimony by recalling the day she received the flu vaccine (October 1, 2019). *Id.* at 11. She did not experience any immediate reaction other than slight soreness in her arm. *Id.* at 12. She had received the flu vaccine prior in her life, but not since, because her doctors have now advised her to avoid it. *Id.* at 13–14. She also had never previously been diagnosed with any serious illnesses and had no history of asthma. *Id*. at 12–14. At most, she had experienced a mild, nonspecific joint pain in April 2019, but testified that the pain went away on its own after a couple days. *Id.* at 16. When asked about her 10–15-pound weight loss in the interim period, Petitioner explained that she had been exercising and had engaged in intermittent fasting in order to lose weight postpartum. *Id.* at 16–17.

Petitioner then discussed her health in the weeks following the relevant vaccination. Tr. at 17. She testified that she developed a sore throat, congestion, and a cough one to two weeks post-vaccination. *Id.* By October 13, 2019, her symptoms had not dissipated, so she went to urgent care, where she received a prescription. *Id.* at 18. But her symptoms thereafter continued to worsen, so she visited her PCP with her husband. *Id.* at 19. By this time, she was bedridden, so her husband took her everywhere. *Id.* at 20. At this visit with her PCP, she took a flu virus test and was prescribed Tamiflu. *Id.* at 21.

Petitioner eventually went to the ER, due to her worsening symptoms. Tr. at 22. She testified that she had a fever and a weakness level of 10 (on a scale from 1–10). *Id.* The next several weeks were a blur for Petitioner, but she remembers being diagnosed with EGPA by Dr. Stanescu, a rheumatologist. *Id.* at 23. Since her diagnosis, Petitioner sees Dr. Stanescu every three months for follow-ups. *Id.* at 24. She has been managing her autoimmune disease with diet, exercise, and regular bloodwork checkups. *Id.*

Petitioner concluded her testimony by explaining that she has changed drastically since her diagnosis. Tr. at 25. She used to be a dancer, but can no longer tolerate the strain of such an endeavor, and cannot teach her daughter to dance. *Id.* at 25–26. She lacks confidence and hesitates when making decisions. *Id.* at 26. She is also very weak—she cannot walk long distances without tiring out. *Id.* Her life has been turned "upside down" since she was diagnosed. *Id.* at 27.

2. *Rupesh Parajuli* - Mr. Parajuli is Petitioner's husband, and he also testified at the hearing. *See generally* Tr. 29–47. As he recalled, Petitioner was healthy prior to the vaccination. *Id.* at 33. They had been together since 2012, and he was confident that Petitioner would have confided in him if she was having any health issues. *Id.* at 34. When asked about Petitioner's weight loss in the period up to vaccination, he provided the same explanation as she did—they were eating healthier and intermittent fasting after she gave birth to their son. *Id.* at 35.

Mr. Parajuli recalled that Petitioner developed congestion and a very deep, persistent cough about one week after vaccination. Tr. at 31–32. After the initial urgent care visit, he took her to their PCP twice. *Id.* at 32. Mr. Parajuli recalled that Petitioner's cough progressively worsened every day after her initial visit to her PCP. *Id.* at 37. She would cough all night long and disrupt the children's sleep. *Id.* She had become bedridden during this time, so he had to carry her everywhere. *Id.* at 38.

Since the vaccine, Mr. Parajuli has observed Petitioner to lack physical strength. Tr. at 40. Mr. Parajuli had to take care of everything on his own for a while, which was very difficult. *Id.* at 41. He testified that Petitioner was deeply saddened by the fact that she had to stop breastfeeding her son after she developed this autoimmune disease. *Id.* at 41–42. Petitioner now gets bloodwork done every three months and is monitored closely by Dr. Stanescu. *Id.* at 42.

B. Petitioner's Experts

1. *Dr. M. Eric Gershwin* - Dr. Gershwin testified at trial for Petitioner, and prepared two reports in this case. Report, dated Jan. 16, 2023, filed as Ex. 17 (ECF No. 19-1) ("First Gershwin Rep."); Supplemental Report, dated Sep. 18, 2023, filed as Ex. 27 (ECF No. 30-1) ("Second Gershwin Rep.").

Dr. Gershwin is a Distinguished Professor of Medicine in the Division of Rheumatology/Allergy and Clinical Immunology at the University of California Davis School of Medicine. Curriculum Vitae, dated Jan. 26, 2023 (ECF No. 19-2) at 1. Before his current role, he served as the Chief of the same division for nearly forty years. *Id.* Dr. Gershwin received his medical degree from Standford University, and then completed his residency at Tufts-New England Medical Center thereafter. *Id.* He triple board-certified with boards in internal medicine, allergy-immunology, and rheumatology. *Id.* at 2. Dr. Gershwin has treated hundreds of patients in his over 50-year medical career with vasculitis, including EGPA. First Gershwin Rep. at 1. Dr. Gershwin serves as an editor for several autoimmunity and allergy journals, and his research has led to the publication of over a thousand peer-reviewed articles. *Id.* 1–2.

Dr. Gershwin began with an overview of EGPA/Churg-Strauss syndrome. He deemed it the rarest form of pulmonary vasculitis, affecting only 1–3 people per million. Tr. at 56. Two cardinal features distinguish it from other forms of pulmonary vasculitis: 1) Churg-Strauss is defined by eosinophilic infiltration into affected tissues, and 2) with rare exceptions, Churg-Strauss occurs only in patients with bronchial asthma. First Gershwin Rep. at 7. Dr. Gershwin explained that different types of pulmonary vasculopathies have an enormous degree of overlap. Tr. at 68. He also noted that very little is known about the molecular etiology of Churg-Strauss. *Id.* at 112. While there is no single antibody that is pathognomonic of the disease, ANCA autoantibodies are often identified in serum testing for people with Churg-Strauss/EGPA. *Id.* at 68, 113; First Gershwin Rep. at 6.

There are several distinct phases in the development of EGPA. First Gershwin Rep. at 7. The disease starts with a prodromal/quiescent phase. Tr. at 54. Ms. Dhital was likely in this phase during the period of time pre-vaccination when she lost weight, had unexplained blood in her urine, and was iron-deficient. *Id.* at 54, 114–15. Next, EGPA enters a phase characterized by eosinophilia and atopic symptoms like asthma. *Id.*; First Gershwin Rep. at 7. These first two phases can span years. Tr. at 54. Finally, patients experience a sudden clinical exacerbation and enter the vasculitic phase, which often involves life-threatening systemic vasculitis of the medium and small blood vessels. *Id.*; First Gershwin Rep. at 7.

Petitioner's clinical course, Dr. Gershwin maintained, reflected the progression of these phases, and was thus consistent with a Churg-Strauss/EGPA diagnosis. She had gone to urgent care 12 days after vaccination with complaints of a cough, congestion, and a runny nose. Tr. at 61. The next day, Petitioner saw her PCP for treatment of a worsening cough. *Id.* at 62. At this appointment, she was also found to have a fever, an oral ulcer, and cervical lymphadenopathy. *Id.* Petitioner took a rapid flu test, which came back weakly positive, but Dr. Gershwin opined that she likely did not have an influenza infection, consistent with the view of some of her subsequent treaters. *Id.* at 63.

During this same appointment, Petitioner's PCP observed chest opacities on x-ray, which suggested the presence of inflammation in Petitioner's lungs. Tr. at 64. An October 17th test also revealed increased blood eosinophils, which would increase when a person is experiencing allergies or infection. *Id.* Dr. Gershwin emphasized that eosinophilia is a hallmark for Churg-Strauss. *Id.*; First Gershwin Rep. at 7. The testing also revealed mild to moderate anemia. Tr. at 65. In the days that followed, Petitioner tested positive for certain autoantibodies and for inflammation biomarkers, leading treaters to seek a rheumatologic referral. *Id*. at 66. At this time, Petitioner was experiencing joint pain and was having a very hard time walking, and had developed a rash on her arms and legs. *Id.* at 66–67. Dr. Gershwin deemed these symptoms—multi-organ involvement including the lungs, anemia, arthritis, rash—to be consistent with Churg-Strauss syndrome/EGPA. *Id.* at 65–67. Although on October 22nd Dr. Stanescu suggested that Petitioner might be experiencing serum sickness (which results in inflammation but is not a form of autoimmune disease), Dr. Gershwin distinguished that from EGPA. *Id.* at 70–71.

On October 23rd, when Petitioner presented to the ER, her treaters shifted away from an infection theory, and began to consider the possibility that Petitioner's condition was immunological—and vasculitic in nature. Tr. at 71–73. In the discharge summary from that day, the treaters noted that Petitioner's eosinophils were higher—at almost 1,300 (normal range is 500). *Id.* at 72. Other tests also revealed increased inflammation. *Id.* A skin biopsy showed vasculitis, which is consistent with EGPA. *Id.* at 73. Four days later, on October 27, 2019, a bronchoscopy test revealed eosinophilic infiltrates, which Dr. Gershwin explained is "confirmatory" and "extremely consistent" with Churg-Strauss. *Id.* at 74. Two days later, on October 29th, Petitioner was given a "working diagnosis" of Churg-Strauss/EGPA by several treaters. *Id.* at 76.

Respondent's rheumatologic expert, Dr. Mehrdad Matloubian, had specifically emphasized several features from Petitioner's medical history inconsistent with an EGPA/Churg-Strauss diagnosis: her lack of asthma, the fact that she had lower eosinophilia levels than expected, and the fact that she tested positive for anti PR3 antibodies (which would be more characteristic of another form of pulmonary vasculitis: GPA). Tr. at 91. But Dr. Gershwin noted independent support for the conclusion that Petitioner's specific form of disease was an *atypical* but recognized variant of Churg-Strauss patients who are positive for PR3, since they also display less frequent active asthma and peripheral neuropathy, along with more frequent manifestations on the skin, and lower eosinophil counts. *Id.* at 92; T.E. King, *Clinical Features and Diagnosis of Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss)*, UpToDate 1, 7–8 (2023), filed as Ex. A, Tab 5 (ECF No. 27-6) ("King") (noting differences in clinical manifestations between ANCA-positive and negative patients). Petitioner did not file the study that King relies upon for this contention, however—and the data cited clearly indicates that only a very small percentage of the alleged variant form of EGPA featured positive PR3 levels (approximately two percent of a pool of 734 patients). King at 7–8. Dr. Gershwin nevertheless maintained that Petitioner's presentation was

"very typical of patients with Churg-Strauss who are PR3-ANCA positive," which also usually did not involve an asthma prodromal period. Tr. at 92, 94.

Dr. Gershwin generally opined that the flu vaccine had likely accelerated Petitioner's Churg-Strauss Syndrome/EGPA, causing her to move rapidly into the vasculitic (and more clinically-evident) phase. Tr. at 53; First Gershwin Rep. at 4. He was thus of the view that Petitioner already had the disease before she was vaccinated. Tr. at 77. The flu vaccine did not *cause* Petitioner's autoimmune disease, but instead "unmasked" it. *Id.* at 77–78, 110; First Gershwin Rep. at 4.

The capacity of vaccination to elicit the production of a class of messenger immune cells—cytokines—constituted the driver of Petitioner's disease acceleration, in Dr. Gershwin's view. Churg-Strauss, he explained, is a cytokine-driven disease. Tr. at 88. Vaccines elicit pro-inflammatory cytokines, which are "signaling molecules." *Id.* at 78. According to one of Dr. Gershwin's cited articles, "variations in the balance between Th1 and Th2 cytokines at different disease stages could contribute to the distinct clinical course seen in patients with Churg-Strauss." B. Hellmich et al., *Proinflammatory Cytokines and Autoimmunity in Churg-Strauss Syndrome*, 1051 Ann. N.Y. Sci. 121, 121 (2005), filed as Ex. 22 (ECF No. 19-6) ("Hellmich"). Ms. Dhital was likely genetically predisposed to some form of autoimmunity, which meant she already had a dysregulated immune system. Tr. at 78–79, 89; First Gershwin Rep. at 8. The cytokines produced in reaction to the flu vaccine promoted dysregulation—either by producing more inflammation, or by changing the balance of cytokines. Tr. at 79, 89.

Dr. Gershwin acknowledged that Hellmich identified a specific cytokine, IL-5, as the most potent stimulator of eosinophils in Churg-Strauss syndrome. Tr. at 116; Hellmich at 121. But he deemed IL-5 to be less important in Petitioner's pathogenesis, arguing instead that general, cytokine-driven inflammation due to vaccination was what mattered for his causal theory. Tr. at 117. Dr. Gershwin cited a different article to support this contention, although on cross-examination he conceded that it did not stand for the proposition that the flu vaccine is capable of promoting a level of inflammation high enough to be "disease causing." Tr. at 121; N. Chatziandreou et al., *Macrophage Death Following Influenza Vaccination Initiates the Inflammatory Response that Promotes Dendritic Cell Function in the Draining Lymph Node*, 18 Cell Rep. 2427–41 (2017), filed as Ex. 25 (ECF No. 19-9) ("Chatziandreou"), at 2428 (study aimed at evaluating impact of flu vaccine on stimulation of cytokine-producing inflammatory pathway in lymph nodes, with the goal of improving vaccine immunogenicity).

Dr. Gershwin nevertheless maintained that the flu vaccine did not *need* to cause a pathological level of inflammation under his theory. Tr. at 121. Rather, the cytokines produced following receipt of the flu vaccine were pathological to Petitioner because of her (assumed) pre-existing difficulties with immune regulation. *Id.* Petitioner would have experienced a greater deal

of inflammation following vaccination *because* she had an autoimmune disease susceptibility, featuring immune dysregulation and cytokine imbalance. *Id.* at 122–23. This imbalance was further encouraged by the cytokines produced following the flu vaccine. *Id.* at 123. Dr. Gershwin also agreed that in Chatziandreou, cytokines provoked by a flu vaccine peaked and then abruptly decreased 24 hours post-vaccination. *Id.* at 124; Chatziandreou at 2432. But this, he argued, was consistent with his view in this case that the biological events that led to Petitioner's cytokine imbalance likely *began within the first 24 hours of vaccination*. Tr. at 125. (There is, however, no record evidence in this case that Petitioner experienced any close-in-time vaccine reaction and/or post-vaccination inflammatory response).

To more directly connect EGPA with vaccination, Dr. Gershwin offered a number of items of literature. *See* J. Sidney et al., *Epitope Prediction and Identification—Adaptive T Cell Responses in Humans*, 50 Seminars in Immuno. 1–27, filed as Ex. 55 (ECF No. 50-1) ("Sidney"). Sidney, however, is not specific to the kind of vasculitic injury at issue in this case, and only addresses more broadly the capacity of T cells to recognize antigenic peptide sequences, with some secondary discussion of how a better understanding of the recognition process might impact vaccine designs (in order to increase the likelihood of a robust and protective immune response). Sidney at 10–11. Dr. Gershwin also contended that a form of vasculitis that was drug-induced (effectively what he contended herein) would be clinically-indistinguishable from forms with different triggers, emphasizing that an association with different pharmaceutical interventions had been observed. Tr. at 80–81; M. Doyle & M. Cuellar, *Drug-Induced Vasculitis*, 2 Expert Opinion on Drug Safety 401–09 (2003), filed as Ex. 56 (ECF No. 50-2) ("Doyle"). Cell-mediated and humoral immunity appear to play important roles in the pathology of pulmonary vasculitis, regardless of its trigger, even though the exact pathological mechanisms of these diseases remain to be elucidated. Doyle at 401.

Doyle is a review article that discusses prior published studies evaluating instances in which vasculitic disorders have been associated with a variety of drugs or associated therapies. Doyle at 406. Most of the instances of post-vaccination vasculitis it addresses, however, were not the same as EGPA. Doyle at 406. Moreover, on cross-examination Dr. Gershwin conceded that the three case reports involving Churg-Strauss listed in Doyle involved the hepatitis B vaccine, rather than the flu vaccine, and that Doyle did not directly link the flu vaccine as potentially causal except in passing (with no citation to support the contention either). Tr. at 98–99, 103; Doyle at 406 ("[o]ther vaccines less commonly implicated in the development of vaccine-induced vasculitis include the influenza, tetanus, hepatitis A, and pneumococcal vaccines"). Dr. Gershwin also agreed that Doyle did not address exacerbation or acceleration of EGPA. Tr. at 100–01, 104.

Dr. Gershwin similarly admitted that case reports he had referenced involved the development of vasculitis after receipt of a *Covid* rather than flu vaccine. Tr. at 104–05; G. Fiorillo et al., *Leukocytoclastic Vasculitis (Cutaneous Small-Vessel Vasculitis) after COVID-19*

Vaccination, 127 J. of Autoimmunity 1–3 (2022), filed as Ex. 57 (ECF No. 50-3) ("Fiorillo"); I. Qaisar & K. Sunmboye, *A Case of Severe ANCA Associated Vasculitis After Covid-19 Vaccination*, 81 Annals of Rheumatic Diseases 1860 (2022), filed as Ex. 20 (ECF No. 19-4) ("Qaisar"). And it did not appear that the patient in Fiorillo ever developed any form of ANCA-associated vasculitis. Tr. at 106; Fiorillo at 1–2. Dr. Gershwin also admitted he could point to no case reports that showed an acceleration of Churg-Strauss/EGPA following administration of the flu vaccine, and that case reports alone are not robust proof of causation (although he argued they should still be given some evidentiary value). Tr. at 110–11. And he acknowledged that his causal theory could apply to many other vaccines, and would only be relevant for a person with a pre-existing compromised immune system, such as (presumably) Ms. Dhital. *Id.* at 137, 139.

2. *Dr. Jordan Fein* - Dr. Fein, a pulmonologist, authored one report in this case, and testified at the hearing. Fein Report, dated Apr. 8, 2024, filed as Ex. 28 (ECF No. 31-1) ("Fein Rep."). He opined, consistent with Dr. Gershwin, that the flu vaccine likely contributed to the acceleration of Petitioner's EGPA/ Churg-Strauss syndrome. Tr. at 245.

Dr. Fein earned his M.D. from the University of California, Davis School of Medicine. *See* Curriculum Vitae, filed June 11, 2024 (ECF No. 32-1) at 1. After Graduation, Dr. Fein attended University of California, Davis Medical Center for both his residency in Internal Medicine, and then his fellowship where he specialized in Pulmonary and Critical Care Medicine. *Id.* Dr. Fein is board certified in Interventional Pulmonology and has been practicing medicine for well over a decade. *See id.* He currently practices at Legacy Emanuel Medical Center and Legacy Good Samaritan Medical Center in Portland, Oregon, where he serves as the Chair of Medicine. *Id.* at 2.

Dr. Fein defined Churg-Strauss to be a chronic inflammatory disorder characterized by multisystem involvement, although it most commonly impacts the lungs. *Id.* at 241; Fein Rep. at 5. The disease consists of three phases—the prodromal phase, the eosinophilic stage, and the vasculitic phase. Tr. at 248. The prodromal phase, which is typically characterized by asthma and chronic rhinosinusitis, can last for three to ten years. *Id.* Dr. Fein noted, however, that one out of ten individuals with EGPA present without a clinical history of asthma (thus suggesting that asthma's absence does not wholly rule out the diagnosis). *Id.* at 249; Fein Rep. at 5.

Dr. Fein concurred with Dr. Gershwin that Petitioner was properly diagnosed with EGPA. While he acknowledged that there are no definitive lab tests that differentiate between the types of vasculitides, he noted that each form of the disease has distinctive features. Tr. at 259, 252. The presence of eosinophilia, he explained, is a "strong" differentiator between EGPA, on the one hand, and MPA and GPA on the other. *Id.* at 259. Petitioner's lung biopsy identified an increased amount of eosinophils, which immediately raised EGPA as a likely diagnosis. *Id.* at 256; Ex. 9 at 563. At the same time, the biopsy did not reveal evidence of necrotizing granulomas, which one would expect to see in a person with GPA. Tr. at 255; Ex. 9 at 563.

Dr. Fein also noted that the bronchoalveolar lavage performed on Ms. Dhital demonstrated normal bronchial mucosa and older blood in the airways. Fein Rep. at 7; Ex. 9 at 86. While the returned fluid from the lavage was blood-tinged, it was not progressively darker with each serial lavage. Fein Rep. at 7; Ex. 9 at 27. Diffuse alveolar hemorrhage ("DAH") (bleeding that is occurring from the alveolar space) is common in GPA. Tr. at 254. But Dr. Fein would have expected to see an increase in bloody return with each lavage performed on Petitioner had she been experiencing GPA—and since she displayed no frank bleeding, and nothing was visualized with the scope in the central airways as a cause for bleeding, GPA was undermined as a likely diagnosis. *Id.* at 253–55. (On cross, however, Dr. Fein admitted that Petitioner's lung biopsy had demonstrated "lung parenchyma *with diffuse alveolar hemorrhage*" (emphasis added), but clarified his view—that Petitioner had not suffered from *active* DAH, as evidenced by a proper bronchoalveolar lavage. *Id.* at 271.) In Dr. Fein's opinion, the findings of the bronchoscopy in conjunction with the pulmonary infiltrates on CT, peripheral blood eosinophilia, and elevated IgE levels were all confirmatory for EGPA. Fein Rep. at 7. This diagnosis was also endorsed on several occasions by Petitioner's treaters. Tr. at 256–57; 262; Ex. 9 at 158, 563.

Ms. Dhital, Dr. Fein proposed, was likely in the prodromal phase of her disease *prior* to receipt of the flu vaccine. Tr. at 259. This opinion was supported by the fact that she had experienced joint pain and unintentional weight loss—both systemic signs of EGPA. *Id.*; Fein Rep. at 6; Ex. 17 at 2. But her subsequent receipt of the flu vaccine accelerated the prodromal phase, causing her to essentially "blow through" the period in which she would have been expected to develop asthma. Tr. at 260, 263. The vaccine pushed her into the vasculitic phase within a few months of developing EGPA. *Id.* at 261.

With respect to possible explanations for Petitioner's EGPA, Dr. Fein ruled out an infectious process. He acknowledged that Petitioner's rapid flu test was weakly positive, but stated with confidence that Petitioner did not have a wild flu infection. Tr. at 251. Petitioner underwent a bronchoscopy procedure, which included a viral panel, and the PCR tests were negative for influenza. *Id.*; Ex. 9 at 238. In his experience, patients who have the flu and undergo a bronchoscopy procedure are typically positive for weeks following clinical improvement. Tr. at 251. Thus, Dr. Fein concluded that Petitioner's symptoms were more likely attributable to an autoimmune process. *Id.* at 251–52. Many of Petitioner's treaters seemed to share this opinion. Fein Rep. at 6.

On cross examination, Dr. Fein acknowledged that Dr. Stanescu had offered her impression of Churg-Strauss as a good diagnostic explanation for Petitioner's symptoms before she had access to Petitioner's ANCA results. Tr. at 265–66. But after Dr. Stanescu received the ANCA results, she began listing primary diagnoses *other* than EGPA, and ultimately seem to embrace GPA as the correct diagnosis. *Id.* at 267. Dr. Fein also agreed that a study offered by Respondent revealed

that 25 percent of people with GPA display mild to moderate eosinophilia. *Id.* at 267–68; M. Iudici et al., *Significance of Eosinophilia in Granulomatosis with Polyangiitis: Data from the French Vasculitis Study Group Registry*, 61 Rheumatology 1211–16 (2022), filed as Ex. E, Tab 5 (ECF No. 45-6) ("Iudici"), at 1215 (noting that "mild to moderate increase" of eosinophilia in blood testing is often evident in GPA patients at time of diagnosis, and stressing the need to identify a kind of non-EGPA class of "ANCA-negative, asthma-free patients with hypersinophilia and biopsy-proven vasculitis"). However, Dr. Fein deemed Iudici an outlier. Tr. at 269. He also clarified that he did *not* believe it was more likely for an individual with GPA to present *with* eosinophilia than it was for an individual with EGPA to present *without* asthma (despite his prior testimony that only 10 percent of EGPA patients present without asthma). *Id.* And he acknowledged that the presence of anti-PR3 antibodies (which were seen in Petitioner's history as well) is less common in patients with EGPA. *Id.*

Dr. Fein further admitted that the case reports he had referenced did not establish the existence of any specific causal relationship between EGPA and the flu vaccine. Tr. at 272–74; M. Jafarpour et al., *Eosinophilic Granulomatosis with Polyangiitis Following Flu Guard Influenza Vaccination: A Case Report*, 11 Clin. Case Rep. 1–5 (2023), filed as Ex. 40 (ECF No. 31-12) ("Jafarpour") (a patient who developed EGPA following administration of a flu vaccine had asthma for two years prior to disease development); T. Kobayashi et al., *An Atypical Case of Non-Asthmatic Eosinophilic Granulomatosis with Polyangiitis Finally Diagnosed by Tissue Biopsy*, 58 Internal Med. 871–75 (2019), filed as Ex. 38 (ECF No. 31-10) (no vaccination was reported in relation to the patient's development of EGPA).

C. Respondent's Experts

1. *Dr. Mehrdad Matloubian* – Dr. Matloubian, a rheumatologist, prepared two reports in this case, and testified at trial. Matloubian Report, dated Aug. 13, 2023, filed as Ex. A (ECF No. 27-1) ("First Matloubian Rep."); Matloubian Supplemental Report, dated Aug. 7, 2024, filed as Ex. C (ECF No. 41-1) ("Second Matloubian Rep.").

Dr. Matloubian holds both a M.D., and a Ph.D. from the University of California in Los Angeles. *See* Curriculum Vitae, filed Aug. 15, 2023 (ECF No. 27-19) ("Matloubian CV") at 1; First Matloubian Rep. at 1. Dr. Matloubian is a board-certified practicing rheumatologist, who continues to see and treat patients with complex autoimmune diseases on a limited basis. *See* Matloubian CV at 3. Most of his work revolves around virology research, which he has been engaged in for over twenty-five years. First Matloubian Rep. at 1. Dr. Matloubian specifically studies T cell and B cell responses to viruses and factors that regulate lymphocyte circulation and trafficking. *Id.* Dr. Matloubian's research has led him to publish numerous peer-reviewed articles regarding the innate and adaptive immune response to acute and chronic viral infections. *See* Matloubian CV at 10–14.

Dr. Matloubian opined that Petitioner suffered from an ANCA-associated vasculitis—but not EGPA specifically. ANCA-associated vasculitis, he explained, is an autoimmune disease that affects blood vessels. Tr. at 145. It is categorized mainly by the presence of ANCA autoantibodies, and its subtypes are distinguished by certain features, like the kind of blood vessels that are affected. *Id.* Based on the totality of Petitioner's presentation, which included anti-PR3 positivity, no history of asthma, scleritis, a tongue ulcer, renal involvement, and DAH, Dr. Matloubian concluded that Petitioner likely had GPA. *Id.* at 146.

Petitioner's overall course and clinical presentation, Dr. Matloubian maintained, was most consistent with GPA as the proper diagnosis. First, he found the absence of asthma significant. Tr. at 149. Between 90–100% of people diagnosed with EGPA have a prodromal state of asthma that lasts for an average of eight years. *Id.*; C. Comarmond et al., *Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss)*, 65 Arthritis and Rheumatism 270, 273 (2013), filed as Ex. 30 (ECF No. 31-2) ("Comarmond"). It would be highly unusual for EGPA to occur without also some evidence of asthma before. Tr. at 149.

Second, Dr. Matloubian highlighted aspects of Petitioner's presentation inconsistent with an EGPA diagnosis. For example, Petitioner had a tongue ulcer—a clinical feature of people who present with GPA, but not EGPA. Tr. at 149; John H. Stone, *Granulomatosis with Polyangiitis,* in Current Diagnosis & Treatment: Rheumatology 1, 5 (John H. Stone ed., 4th ed. 2021), filed as Ex. A, Tab 2 (ECF No. 27-3) ("Stone"). Petitioner's kidney involvement also supported a GPA diagnosis. Tr. at 150. On average, 80% of people with GPA have kidney involvement, compared to 22% of people with EGPA. *Id.*; Stone at 7. And Dr. Matloubian deemed significant Petitioner's DAH as supportive of a GPA diagnosis, considering that only 3–4% of EGPA patients develop DAH, whereas up to 45% of EGPA patients do. Tr. at 151; J. White & S. Dudley, *Eosinophilic Granulomatosis with Polyangiitis: A Review*, 22 Autoimmunity Rev. 1, 3 (2023), filed as Ex. 34 (ECF No. 31-6) ("White & Dudley"); L. Quartuccio, *Alveolar Hemorrhage in ANCA-Associated Vasculitis: Long-Term Outcome and Mortality Predictors*, 108 J. of Autoimmunity 1, 1 (2020), filed as Ex. 35 (ECF No. 31-7).

Applying the above factors, and other test results, to the indicia set forth by the American College of Rheumatology and the European Alliance of Associations for Rheumatology also yielded results inconsistent with the alleged EGPA diagnosis. Tr. at 154; P. Khoury, *Clinical Features and Diagnosis of Eosinophilic Granulomatosis with Polyangiitis (Churg-Strauss)*, UpToDate 1, 13–14 (2024), filed as Ex. 32 (ECF No. 31-4) ("Khoury"). Under recognized criteria referenced in Khoury, any score of six or above has a specificity of 99% for EGPA—but after applying those criteria to Petitioner, Dr. Matloubian came up with a total score of three points. Tr. at 154–55; Second Matloubian Rep. at 4; Khoury at 13–14. This moved the diagnostic needle toward GPA, in Dr. Matloubian's view. Tr. at 156.

Dr. Matloubian acknowledged that Dr. Stanescu's diagnostic opinion had at some points in Petitioner's disease course favored an EGPA diagnosis. But he did not find this surprising, because Dr. Stanescu's earlier embrace of EGPA as a diagnosis did not take into account the results of Petitioner's ANCA testing (which revealed PR3 positivity). Tr. at 158. In Dr. Matloubian's experience, different types of vasculitides can be categorized based on the existence/degree of ANCA involvement. *Id.* at 147. For example, more than 90% of people with GPA have positive ANCA, compared to 30–50% of EGPA patients. *Id.*; Khoury at 8. GPA also tends to be more associated with anti-PR3 positivity. Tr. at 147; Stone at 10 ("[t]he combination of a C-ANCA pattern by immunofluorescence and a positive PR3-ANCA by enzyme immunoassay has a high positive predictive value for GPA"). In contrast, the majority of EGPA patients display anti-MPO or P-ANCA antibodies. Tr. at 147; Khoury at 8 (finding that only 2–4% of people with EGPA have antibodies to PR3). By January 2020, after the results of Petitioner's ANCA testing were revealed, Dr. Stanescu stopped using the diagnosis of EGPA and settled on GPA. Tr. at 160.

Eosinophilia, Dr. Matloubian acknowledged, is highly predictive for EGPA. Tr. at 160. But eosinophilia *can* be seen in patients with GPA, so Petitioner's high eosinophil count did not *per se* discount a GPA diagnosis. *Id.* at 156; White & Dudley at 4. And although Petitioner had eosinophils in her lung biopsy, they were not also seen in her skin biopsy, further diminishing the significance of those findings. Tr. at 161.

Dr. Matloubian then turned to Petitioner's causation theory, which invoked the bystander activation hypothesis. Tr. at 162. But in Dr. Matloubian's view, the Institute of Medicine had reviewed this theory and found no evidence to support the conclusion that bystander activation or excessive cytokines play a role in alleged vaccine injuries. *Id.* at 162–63; Institute of Medicine, Adverse Effects of Vaccines: Evidence and Causality 70, 76 (K. Stratton et al. eds., 2012), filed as Ex. A, Tab 13 (ECF No. 27-14).

More specifically, Dr. Matloubian contested Petitioner's interpretation of her disease course, and the argument that she experienced the prodromal phase of EGPA prior to her receipt of the flu vaccine. Tr. at 163. He noted that her weight loss (which Dr. Gershwin maintained was evidence supporting that phase) was intentional, as reflected in actual witness testimony from Petitioner and her husband, and thus could not constitute evidentiary support for a prodromal EGPA phase. *Id.* He also surmised that Petitioner's hematuria could be attributed to her period, noting that findings from April 2019 were cloudy (rendering their value as a good specimen). *Id.* at 163–67. And although Petitioner's blood tests from April 2019 revealed that Petitioner was anemic, her platelet counts were normal, which would be very unusual in a person who was purportedly experiencing a systemic inflammatory response. *Id.* at 164. Dr. Matloubian was otherwise unaware of any convincing evidence that immunization can essentially cause acceleration of the prodromal stage in individuals who are eventually diagnosed with EGPA. *Id.* at 168.

Dr. Matloubian further challenged the aspects of Petitioner's theory specific to the flu vaccine. He emphasized, for example, that the non-adjuvanted flu vaccine Petitioner received is one of the weakest vaccines from an immunogenic standpoint—it does not induce significant immune responses and cytokine production. Tr. at 168. After a person receives this vaccine, they may at worst experience mild symptoms, like a sore arm and a brief fever, but in general this vaccine is much more tolerable than an adjuvanted one. *Id.* at 169. And Petitioner never experienced any sort of reaction immediately following immunization. *Id.* at 170.

Dr. Matloubian disagreed with Petitioner's contention that the flu vaccine's localized response in the axillary lymph nodes could subsequently result in an immune response in the pulmonary lymphatics (where some EGPA-associated symptoms might later manifest). Tr. at 171–75. He agreed that "an influenza vaccine can induce an inflammatory response within a regional lymph node," but the "regional" node implicated by vaccination would be only the axillary lymph node, located in the armpit. First Matloubian Rep. at 15. Studies had actually shown that the immediate immune response to a peripherally-administered vaccine was in this particular regional lymph node. *Id.* at 15; Tr. at 171. By contrast, the "lung-draining" lymph nodes are located in the chest, and do not access directly the deltoid muscle. First Matloubian Rep. at 15. As a result, it was highly unlikely an immunization received in the arm could rapidly impact a different organ—as would be required here if in fact vaccination was triggering an aberrant response resulting in EGPA-specific symptoms. And Dr. Matloubian rejected the contention that Chatziandreou found otherwise. Chatziandreou at 2437 (animal study involving vaccination of mice in their footpad, with evaluation of "popliteal lymph node"[14] (located peripherally, behind a mouse's knee)).

Dr. Matloubian offered several items of literature to support his contention about what lymph nodes would be most immediately impacted by vaccination. A. Nawwar et al., *Bilateral Avid Axillary Nodes on FDG PET/CT Due to Concurrent Booster COVID-19 Immunization and Seasonal Influenza Vaccination*, 47 Clinical Nuclear Med. 712, 713 (2022), filed as Ex. A, Tab 14 (ECF No. 27-15) ("Nawwar") (evidence of "moderately avid axillary nodes" after receipt of COVID booster and flu vaccine); S. Norihisa et al., *Axillary Lymph Node Accumulation on FDG-PET/CT After Influenza Vaccination*, 26 Annals of Nuclear Med. 248–252 (2012), filed as Ex. A, Tab 15 (ECF No. 27-16) (flu vaccine can result in transient lymph node inflammation, most immediately seen in axilla); A. Trivedi, *The Lymphatic Vasculature in Lung Function and Respiratory Disease*, Frontiers in Med. 1, 2 (2023), filed as Ex. A, Tab 16 (ECF No. 27-17) (lung lymphatics drain to thoracic lymph nodes).

[14] Popliteal lymph nodes are "lymph nodes embedded in the fat of the popliteal fossa, comprising superficial and deep groups; their efferent vessels accompany the femoral vessels to the deep inguinal lymph nodes." Popliteal lymph nodes, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=93362 (last visited Sep. 30, 2025).

Dr. Matloubian also contended that a figure in Dr. Gershwin's supplemental report demonstrated the low likelihood that vaccine-induced cytokines would cause acceleration in autoimmune diseases. Tr. at 176; Second Gershwin Rep. at 2.[15] Dr. Gershwin maintained in this second report that Dr. Matloubian had "refused to credit the concept that cytokines released following vaccination will have a significant affect [sic] on an ongoing immune response," and he purported that the figure demonstrated how cytokines are produced shortly after immunization and circulate for a few days. Second Gershwin Rep. at 2; But Dr. Matloubian deemed the figure only established "a normal response to vaccine antigens and producing antibodies to antigens"—not that cytokines produced in response to a peripherally-administered vaccine would impact lymph nodes elsewhere in the body. Tr. at 176, 177; Nawwar at 2; *see also* C. Herve et al., *The How's and What's of Vaccine Reactogenicity*, 4 NPJ Vaccines 1, 3–4 (2019), filed as Ex. A, Tab 11 (ECF No. 27-12) (cytokine increase and associated inflammatory stimuli short-lived after vaccination).

Epidemiology filed in this case further suggested that vaccination did not cause or encourage EGPA or GPA, in Dr. Matloubian's view. Tr. at 183. One such article was a systematic review of all literature regarding the temporal association between vasculitis and vaccination. *Id.* at 184; C. Bonetto et al., *Vasculitis as an Adverse Event Following Immunization – Systematic Literature Review*, 34 Vaccine 6641–51 (2016), filed as Ex. A, Tab 9 (ECF No. 27-10) ("Bonetto"). Bonetto's authors determined that most of the higher-quality studies had found no causal association between vaccination and subsequent development of vasculitis. Bonetto at 6641. Another study of 230 patients with ANCA-associated Vasculitis observed no increase in relapse after receipt of the flu vaccine. *Id.* at 6643. Bonetto concluded that the flu vaccine was safe for patients with ANCA-associated Vasculitis. *Id.* Dr. Matloubian deemed this study to be particularly informative, since it was more than a single case report or case series. Tr. at 186. Dr. Matloubian also cited an article finding no increase in disease flares after receipt of the flu vaccine in patients with GPA. Tr. at 187; J. Westra et al., *Vaccination of Patients with Autoimmune Inflammatory Rheumatic Diseases*, 11 Nature Rev. Rheumatology 135, 141 (2015), field as Ex. A, Tab 10 (ECF No. 27-11) ("Westra").

On cross examination, Dr. Matloubian admitted that he did not know if Petitioner was predisposed to ANCA-associated vasculitis. Tr. at 194. He acknowledged that she had experienced an autoimmune thyroid disease, but could not conclude that this made her more likely to incur other autoimmune diseases or vasculitidies. *Id.* Dr. Matloubian also reviewed the diagnoses of EGPA that were offered by several of Petitioner's treaters throughout her disease course and agreed that these preliminary diagnoses were reasonable based on the information that treaters had at the time (even if ultimately inaccurate). *Id.* at 212–20.

[15] Dr. Gershwin's second report provides no citation for the source of this figure, and it does not appear that any article containing it was ever filed in this case.

2. *Dr. Frederic F. Little* - Dr. Little is an immunologist/pulmonologist, and he authored one report in this case and testified at the hearing. Little Report, dated Sep. 10, 2024, filed as Ex. E (ECF No. 45-1) ("Little Rep.").

Dr. Little earned his M.D. from Tufts University School of Medicine in Boston, MA, before undergoing his residency and fellowship at the University of California, San Francisco. *See* Curriculum Vitae, filed Aug. 16, 2024 (ECF No. 41-2) ("Little CV") at 1. After completing post-graduate training, Dr. Little became board-certified in Pulmonary Diseases, Critical Care Medicine, and Allergy and Immunology, and has worked in these specialized fields for over twenty years. *Id.* at 2; Little Rep. at 1. Dr. Little has a research background in basic and clinical immunology and has published articles focusing on basic and clinical immunology of allergic responses in mice and humans. Little Rep. at 1. Dr. Little also has experience evaluating and treating patients with EGPA and AAV. *Id.* Currently, Dr. Little serves as the Clinical Associate Professor of Medicine at Boston University School of Medicine in Boston, MA. Little CV at 1.

Dr. Little opined, consistent with Dr. Matloubian, that a diagnosis of GPA best fit Petitioner's presentation, acute hospitalization, and care. Tr. at 282. Although Dr. Stanescu began with a working diagnosis was EGPA, her *final* impression was GPA, and Dr. Little testified that this evolution in thinking could be explained by the fact that not all of Petitioner's test results were initially available to the treating physicians. *Id.* at 283–85. Dr. Little agreed with the final diagnosis of GPA because Petitioner developed symptoms that are "not atypical" to it, including hemoptysis (caused by DAH) and findings on skin biopsy of a leukocytoclastic vasculitis with the absence of eosinophils on that biopsy. *Id.* at 285–86; Little Rep. at 9. Furthermore, Petitioner had a progression of nephritis, which is one of the known vasculitic complications of GPA. Tr. at 286; Little Rep. at 9. And finally, Petitioner experienced tongue ulcers and scleritis (inflammation in the whites of her eyes), both of which are commonly seen in patients with GPA. Tr. at 286; Little Rep at 10. (Dr. Little later acknowledged on cross, however, that Petitioner's tongue ulcer could have been coincidental. Tr. at 327).

Dr. Little disputed, by contrast, that Petitioner likely had EGPA, as evidenced by the absence of many of its common features—including a prolonged prodromal phase featuring asthma. Tr. at 287–89. While Dr. Gershwin had attempted to identify specific "symptoms" from the medical record as evidence that Petitioner was in the prodromal phase of her illness in the months leading up to vaccination, Dr. Little considered most of them to have alternative explanations. *Id.* at 289. For example, Petitioner had directly attributed (in live witness testimony) her joint pain to over-exercising, or her weight loss to eating healthy after she had her second child. *Id.* Furthermore, Petitioner's blood testing in April 2019 did not reveal elevated eosinophils, which suggested that there was no evidence of a Type 2 immune overdrive typically seen in EGPA as part of its prodrome. *Id.* at 289–90, 294. And Dr. Little did not deem Petitioner's hematuria to be

significant, since it is difficult to determine the cause of blood in a woman's urine, especially if she is menstruating. *Id.* at 291–92.

From the above, Dr. Little concluded that Petitioner did not experience a prodromal EGPA phase, but instead had presented acutely with the vasculitic phase, which is far more common in GPA. Tr. at 293. He testified that he was unaware of any medical literature that suggests patients with EGPA can "skip" the asthmatic and eosinophilic phases and go right into the vasculitic phase. *Id.* And even if Petitioner's nonspecific symptoms *were* prodromal, they were much more consistent with the prodromal phase of GPA. *Id.* at 314; Little Rep. at 10.

Dr. Little identified other aspects of Petitioner's presentation that were inconsistent with EGPA. For example, he noted that Petitioner's eosinophilia count was "on the low side" for someone with EGPA. Tr. at 294. He also highlighted the fact that about 25% of people with GPA can have eosinophilia above a normal range. *Id.* at 295; Iudici at 1212. Dr. Little then explained that only 5–10% of EGPA patients present without asthma. Tr. at 296; Little Rep. at 9; Comarmond at 273. Therefore, Dr. Little maintained, the chance of presenting with eosinophilia in GPA was higher than the chance of presenting *without* asthma in EGPA. Tr. at 296.

Petitioner's immunoglobulin counts were also unsupportive of EGPA, in Dr. Little's view. Tr. at 297. IgE is the "allergic antibody" that mediates the body's allergic responses. *Id.* Because EGPA is a Type 2 immune overdrive system of unclear pathogenesis, it features hyper-eosinophilia caused by an IL-5 elevation and increased class switching to the production of IgE caused by interleukin-4, which is another Type 2 cytokine. *Id.*; Little Rep. at 9. Dr. Little testified that a range of 500–700 kU/L IgE would be expected alongside eosinophilia, but Petitioner's IgE level was at lower range (151). Tr. at 297; Ex. 9 at 254.

Next, Dr. Little discussed ANCA antibodies. In his experience, most people with EGPA do not display positive ANCA levels—and if they do, it is typically the *P-ANCA* rather than PR3 antibodies (which was what Petitioner possessed). Tr. at 298; Little Rep. at 6. In fact, only 2% of individuals with EGPA test positive for any PR3 antibodies. Tr. at 299; J. Fijolek & E. Radzikowska, *Eosinophilic Granulomatosis with Polyangiitis – Advances in Pathogenesis, Diagnosis, and Treatment*, 10 Frontiers in Med. 1, 16 (2023), filed as Ex. 60 (ECF No. 50-6) ("Fijolek & Radzikowska"). In contrast, PR3 is seen in 80–90% of people with GPA. Tr. at 299; Fijolek & Radzikowska at 16.

Petitioner also suffered from DAH, which is rare in EGPA while being much more common in GPA. Tr. at 301, 304; R. Da Silva & P. Adhikari, *Granulomatosis with Polyangiitis Presenting with Diffuse Alveolar Hemorrhage: A Systematic Review*, 14 Cureus 1, 1 (2022), filed as Ex. E, Tab 3 (ECF No. 45-4) ("Da Silva") (DAH appears in 5–15% of GPA patients); King at 3 (DAH is rare in EGPA patients). Dr. Little allowed that Petitioner had "old blood" in her airways,

which could not be explained by the frequency of her coughing, since there was no evidence of inflamed airways or active bleeding. Tr. at 302. But in the absence of endobronchial lesions on imaging, and given the lung biopsy results showing "diffuse acute alveolar hemorrhage," there was in his view no plausible explanation other than DAH for Petitioner's hemoptysis. Little Rep. at 12. Dr. Little reached this conclusion despite the fact that there was no visual change in lavage fluids taken from Petitioner. Tr. at 303.

Petitioner's radiologic findings were consistent with acute GPA. Tr. at 306. Dr. Little pointed to the consolidative opacity in Petitioner's right upper lobe and other nodules on Petitioner's lungs and explained that they were consistent with GPA central mass-like opacities. *Id.* at 307–09; Ex. 9 at 32. He acknowledged that patients with GPA typically have large cavitary masses on a CT scan, but surmised that Petitioner's opacities would have likely cavitated over time. Tr. at 309. Dr. Little also pointed out that pleural effusion (fluid between the chest wall and inner lining of the lung) is never found in GPA, but can be seen in EGPA—and there was no pleural effusion on Petitioner's scan. *Id.* at 310.

Towards the end of his testimony, Dr. Little addressed the clinical classification criteria for GPA and EGPA accepted by the American College of Rheumatology and the European Alliance for Rheumatology. Tr. at 311; Little Rep. at 8. While Dr. Little acknowledged that these diseases do not possess definitive diagnostic criteria, the criteria such organizations have developed are generally accepted as valid for distinguishing between EGPA and GPA. Tr. at 312. After applying the criteria to Petitioner's case, Dr. Little concluded that Petitioner met the criteria for GPA (with a score of 6), but did not meet the criteria for EGPA (with a score of 3). *Id.* at 312–13; Little Rep. at 9.

Dr. Little concluded that the flu vaccine could not have accelerated Petitioner's injury. Tr. at 313. Petitioner never experienced a prodromal phase, nor did she have EGPA. *Id.* at 314. And Dr. Little cited an epidemiological study to support his conclusion that there is no known causal connection between vaccines and comparable autoimmune rheumatic conditions. *Id.* at 315; G. Nakafero et al., *Association Between Inactivated Influenza Vaccine and Primary Care Consultations for Autoimmune Rheumatic Disease Flares: A Self-Controlled Case Series Study Using Data from the Clinical Practice Research Datalink*, 78 Annals of Rheumatologic Diseases 1122, 1124–25, filed as Ex. E, Tab 7 (ECF No. 45-8) (finding no significant association between the flu vaccine and primary care consultations for vasculitis and non-infective fever).

III. Procedural History

This matter was initiated in February 2022. It was assigned to a different special master, but reassigned to me in February 2023, after the filing of Respondent's Rule 4(c) Report contesting entitlement. The parties filed the expert reports referenced above, and the matter went to hearing

in October 2024. The parties filed post-hearing briefs through April 2025, after which time the matter became ripe for resolution.

IV. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly ex rel. Moberly v. Sec'y of Health & Hum. Servs*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[16] There is no Table injury consistent with EGPA or GPA.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate 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)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

[16] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or even a generally accepted medical theory. *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs.*, 107 Fed. Cl. 280, 245 (2012).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Cerrone v. Sec'y of Health & Hum. Servs.*, 146 F.4th 1113, 1121 (Fed. Cir. 2025) (arguing that *Althen* prong one requires only a showing of plausibility "understates the burden [a petitioner] bears under the first factor in the *Althen* formulation"); *Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions

and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical 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 particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Elements of Significant Aggravation*

Where a petitioner alleges significant aggravation of a preexisting condition, the *Althen*

test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *Loving v. Sec'y of Health & Hum. Servs*., 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Servs.*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (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 significantly 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, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the *Loving* test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.,* 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, A Petitioner need not demonstrate an *expected* outcome, but merely that her current-post vaccination condition was "worse" than pre-vaccination. *Sharpe*, 964 F.3d at 1081. It is not entirely clear what "worse" actually means. *Sharpe* clearly does not obligate claimants to offer an expected disease trajectory, and subsequent Circuit decisions have stressed that the degree of worsening need not be shown to have risen to a catastrophic level. *Osenbach v. Sec'y of Health & Hum. Servs*., No. 2024-1663, 2025 WL 2387944, at *4 (Fed. Cir. Aug. 18, 2025) (petitioners are not obligated to demonstrate that a given vaccine "wholly alter[ed]" an allegedly-aggravated disease course) (citations omitted). And a claimant may make out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish alternative cause, after the burden of proof has shifted to Respondent). *Id.* at 1083.

C. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). 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." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven 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"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also 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), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.,* 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) 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) ("like 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*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

D. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See*, *e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. Sep. 10, 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

E. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed

every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. Petitioner Did Not Likely Have EGPA

As the parties agreed, EGPA is a rare autoimmune disorder characterized by inflammation of blood vessels (vasculitis) and the presence of eosinophils. King at 1; Comarmond at 271. As King notes EGPA commonly presents with asthma and sinus or nasal symptoms, as well as peripheral neuropathic symptoms. King at 2. It usually proceeds thorough three phases: a prodromal phase (where asthma and rhinitis are its clinical manifestations), the eosinophilic phase (when the eosinophils are infiltrating organs systemically), and a vasculitic phase (which can be life-threatening, but also often involves evidence of skin issues). *Id.* at 2, 3. "Granulomas," or aggregations of immune cell macrophages that form in response to chronic inflammation, are often observed in the skin lesions or other organs. *Id.* at 3. Many EGPA patients test positive for ANCA antibodies; it is not known whether those antibodies are specifically pathogenic for EGPA. Comarmond at 275, 278; *Id.* at 5.

GPA is a distinguishable diagnosis, involving different criteria and indicia, despite its overlap with EGPA. As noted in Stone, GPA is "one of the most common forms of systemic vasculitis," and while its cause is unknown, "a response to an inhaled antigen" is believed possibly involved since GPA also features some respiratory issues. Stone at 1, 2. It can also involve clinical manifestations of oral ulcers/gum inflammation, scleritis of the eyes, and DAH. *Id.* at 2–7. And renal involvement, while not usually seen early on, often manifests as the disease progresses. *Id.* at 7. ANCA positivity is common as well, with antibodies to PR3 the most often-identified in cases of GPA. *Id.* at 9, 10; R. Falk & P. Merkel, *Clinical Spectrum of Antineutrophil Cytoplasmic Autoantibodies,* UpToDate (T. Post, ed. 2023), filed as Ex. A Tab 1 (ECF No. 27-2) ("Falk & Merkel"), at 4–5. Identification of this autoantibody is less common in EGPA cases. King at 7; Falk & Merkel at 6.

Thus, EGPA and GPA can be distinguished in multiple ways. For example, GPA usually presents with granulomatous inflammation in the respiratory tract—such as sinusitis, nasal polyps, and lung disease—and can feature renal involvement. Stone at 1–2, 7. Whereas EGPA presents with asthma, rhinosinusitis, skin lesions, and is more likely to involve risk of allergic reactions and gastrointestinal issues. Khoury at 1, 5, 15–17. Laboratory findings are also unique to each disorder. GPA patients test positive for PR3-ANCA antibodies more often than EGPA patients. Stone at 9–

10; Khoury at 7–8, 15–16. But EGPA has been found to be more closely associated with antibodies other than PR3-ANCA, and patients with EGPA routinely show high levels of eosinophils in the blood. Khoury at 7–8, 15–16. Further, the clinical features of EGPA generally occur in sequential phases, where GPA does not have the same defined, phasic progression. Khoury at 2, Stone at 1.

Here, although both sides can highlight clinical or testing factors supporting their favored diagnosis, numerous facts gleaned from the medical record are *better supportive* of GPA (or some kind of ANCA-associated vasculitis) as Petitioner's proper diagnosis. First, many important test results and clinical features seen in Petitioner's medical history are more consistent with GPA. In particular, Petitioner tested positive for the PR3 ANCA antibody commonly associated with GPA. Fijolek & Radzikowska at 16; Ex. 9 at 219–20. She developed clinical manifestations of GPA, like mouth ulcers and her later renal involvement, as confirmed by Dr. Stanescu. Ex. 8 at 14, 47. She also displayed some factors that less-robustly support GPA, such as her DAH, given that the experts for both sides pointed out reasons to either elevate or reduce the significance of this finding (although I found Dr. Little's contentions about the medical record discussion of DAH slightly more persuasive). Ex. 9 at 128, 248; Da Silva at 1.

But then there is a second, more powerful consideration: the lack of evidence Petitioner ever experienced any prodromal phase of her disease course, featuring asthma. Petitioner was *clearly not suffering from long-standing and persistent chronic respiratory issues prior to her vaccination*—even though Petitioner's experts acknowledged that this is a common feature of EGPA. First Gershwin Rep. at 7 ("with rare exceptions CSS occurs only in patients with bronchial asthma"). Indeed, her presentation was quite acute (and that fact, coupled with its temporal vaccine association, seem to have at certain points to have persuaded treaters that the vaccine played a role in Petitioner's disease). The contention that Petitioner's EGPA went through a pre-vaccination, prodromal phase (consistent with what is known about the condition) was particularly poorly substantiated, with experts like Dr. Gershwin almost *assuming* she was prodromal, despite evidence to the contrary. Petitioner's own testimony at hearing attributed her weight loss (which could be somewhat-nonspecific evidence of vasculitic problems) *to her own efforts*, yet her experts posited (as if they had not heard her testify at all) that it reflected the disease process at work.

Petitioner's experts strained elsewhere to explain away features of her presentation that were inconsistent with EGPA. Thus, in order to get around the serologic findings in the record of PR3 antibodies more strongly associated with GPA than EGPA, Dr. Gershwin proposed there existed an "atypical" form of EGPA that just so happened to be consistent with Petitioner's history, but which also featured higher titers of this particular antibody—even though the authority he cited for this proposition was not only an unfiled reference in *another* filed article, but did not stand for this proposition. King at 7–8; Tr. at 92, 94.

This is not to say that no evidentiary factors favor Petitioner's proposed diagnosis. The testing levels for eosinophils clearly supported the diagnosis, as Respondent's experts tacitly admitted. *See, e.g.*, Little Rep. at 8 (acknowledging presence of sufficient blood eosinophils in

calculating diagnostic EGPA "score"); Khoury at 13–14. In addition, Petitioner can point to treater views embracing EGPA. *See, e.g.*, Ex. 9 at 158. At the same time, however, treaters who had the benefit of consideration of the greater arc of Petitioner's course—in particular, Dr. Stanescu—ultimately moved away from the EGPA diagnosis, after they were able to factor in indicia like PR3 antibody levels and Petitioner's later course. Ex. 8 at 14, 47. I give those views more weight than initial treater diagnostic supposition.

Overall, then, the evidence preponderates against Petitioner's proposed diagnosis of EGPA. It does not matter, however, whether my determination is ultimately correct—since EGPA is *not* more likely vaccine-caused than GPA, and (as discussed below) the argument that vaccination could "unmask" EGPA in its prodromal phase—as both Petitioner's experts contend—was not preponderantly established.

II. Petitioner Has not Carried her Burden of Proof to Establish Significant Aggravation

Regardless of the proper diagnosis for Petitioner's illness, it has not been preponderantly shown in this case that the flu vaccine could unmask *any* form of ANCA-associated vasculitis. Since all *Loving* prongs (including those that correspond to *Althen*) must be satisfied, this is sufficient basis for denying entitlement.

A foundational obstacle to Petitioner's significant aggravation theory is the fact that she cannot demonstrate she already had EGPA pre-vaccination (as would be required under *Loving* prong one). As noted, Petitioner's personal history is not consistent with the finding that she was in the prodromal EGPA phase (more often than not evidenced with a long history of asthma) before vaccination—a critical feature of EGPA. King at 2 ("[a]sthma is the cardinal clinical feature of EGPA and is preset in more than 90 percent of patients").[17] Thus, there was no disease process in existence *to aggravate*—and any conclusion to the contrary lacks evidentiary support. This prevents *Loving* prong one from being established.

But even if it had been demonstrated not only that Ms. Dhital already was developing EGPA, but clearly so (i.e., that she had been asthmatic for many years), I would not be able to find on this record that it has been preponderantly established that the flu vaccine is capable of worsening this form of vasculitis, or speeding up the disease process through its known phases.

[17] Petitioner cannot evade the absence of this evidence by arguing that her disease was "atypical" or an exception to the rule. The Program resolves questions of fact by applying a "more likely than not" standard—and given what is known about EGPA, it is clearly "more likely than not" that most EGPA patients would have been asthmatic before their disease progressed. To stress that Petitioner's actual presentation means she likely fits into the 10 percent (according to King) of patients who are not employs circular, conclusory reasoning. And this kind of reasoning is especially unpersuasive in the context of a disease that Petitioner's experts have admitted is *itself* very rare to begin with. Tr. at 56.

First, there has been little offered that would generally suggest any vaccine-EGPA association. As noted in literature filed in this case, EGPA's general etiology remains unknown. *See, e.g.*, King at 16. This, therefore, is not a case in which an infectious process comparable to a specific vaccine can be the basis for a causal theory (and a wild infection certainly has not been linked to hastening EGPA's disease process).

Second, the flu vaccine itself has not otherwise been specifically linked to EGPA. Doyle, which Dr. Gershwin cited as relevant proof that vaccines constitute a form of pharmaceutical drug-like intervention that could in turn lead to some kinds of vasculitic conditions (as opposed to exacerbation of EGPA), only directly discussed case reports involving a distinguishable vaccine. Doyle at 406. Other case reports (a kind of evidence usually given low probative weight in Program decisions)[18] similarly involved different vaccines. *See, e.g.*, Fiorillo, Qaisar (both involving COVID vaccines). And the one case report specific to the flu vaccine, Jafarpour, involved a person who clearly had asthma long before vaccination, and thus was clinically distinguishable. By contrast, Respondent's experts invoked some larger-scale epidemiologic studies undercutting the contention that vaccination could be problematic in the context of an existing vasculitic condition. *See generally* Bonetto, Westra.

In addition, the theory Petitioner's experts proposed, as well as its specific mechanism (encouragement of a proinflammatory context due to cytokine upregulation) was overbroad and too general to deem preponderantly established. One article Dr. Gershwin seemed to rely on to establish the centrality of cytokines in encouraging EGPA, Hellmich, identified a particular cytokine that he later denied was specifically significant to his theory—and he contended in effect that the level of inflammation need not be obviously pathologic for his theory to "work," but instead that what was relevant was the overall cytokine balance. Tr. at 87, 121. He even maintained that it did not matter what vaccine was at issue. *Id.* at 137–38.

But Respondent's experts, like Dr. Matloubian, persuasively demonstrated not only that vaccine-induced cytokine upregulation would not be long-standing,[19] but also that the peripheral situs of vaccination implicated a lymph node *other* than the respiratory-oriented nodes more likely

[18] *See, e.g., Wagner v. Sec'y of Health & Hum. Servs.*, No. 17-1388V, 2019 WL 3297509, at *6 (Fed. Cl. May 8, 2019) ("case reports, a kind of evidence long deemed of low probative value in the Program"); *Campbell v. Sec'y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) ("[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value compared particularly to formal epidemiological studies"); *Bast v. Sec'y of Health & Hum. Servs.*, No. 01-565V, 2012 WL 6858040, at *38 n.104 (Fed. Cl. Dec. 20, 2012) ("[c]ase reports generally carry limited weight on the issue of causation").

[19] Petitioner's actual medical history is also not on all fours with this theory, as it would need to be under *Loving* prong 5 (to prove the flu vaccine "did cause" the unmasking of Petitioner's preexisting EGPA). Over ten days passed from the time of vaccination on October 1, 2019, and Petitioner's first visit to treaters (for what looks in retrospect solely to have been an intercurrent infection). This timeframe is not consistent with a person with a preexisting illness, coupled by a propensity for autoimmunity, seeing their immune response thrown out of proportion post-vaccination, since it is hardly acute enough temporally to associate the two.

relevant to the initial symptoms of EGPA. Tr. at 171–75; First Matloubian Rep. at 15; Chatziandreou at 2437. Thus, in this case (like many others) all the Petitioner seeks to do is to convert the known, *expected* immune-stimulative effect of a vaccination into something pathologic. *See*, *e.g.*, *Godfrey v. Sec'y of Health & Hum. Servs.*, No. 10-565V, 2015 WL 10710961, at *11–13 (Fed. Cl. Spec. Mstr. Oct. 27, 2015), *mot. for review den'd*, 146 Fed. Cl. 70 (2016) (theory that vaccine could promote cytokine upregulation was insufficient to establish injury in question because no reliable scientific evidence supported proposition that cytokine upregulation was pathogenic). It is not likely the flu vaccine could unmask a preexisting case of EGPA in the manner proposed.

CONCLUSION

Because Petitioner has not met her burden of proof, she is not entitled to compensation on this claim. In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[20]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[20] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.