In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-0954V

(to be published)

* *	Chief Special Master Corcoran
KEVIN MCGUINNESS, *	
*	
Petitioner, *	Dated: October 20, 2021
*	
v. *	
*	
SECRETARY OF HEALTH AND *	
HUMAN SERVICES, *	
*	
Respondent. *	
*	
* *	

Ronald Craig Homer, Conway Homer, P.C., Boston, MA, for Petitioner.

Claudia Gangi, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On July 17, 2017, Kevin McGuinness filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Program").[2] ECF No. 1. Petitioner alleges that a pneumococcal conjugate vaccine (bearing the "Prevnar-13" tradename) administered to him on July 21, 2014, caused him to develop seronegative rheumatoid arthritis ("RA"). An entitlement hearing in the matter was held in Washington, D.C. on April 13, 2021.

Having reviewed the record, all expert reports and associated literature, and listened to the experts who testified at the hearing, I hereby deny an entitlement award. As discussed in greater

[1] This Decision will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet**. As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Ruling's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire Decision will be available to the public in its current form. *Id.*

[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) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

detail below, Petitioner has not preponderantly established that the pneumococcal vaccine can cause rheumatoid arthritis, or that it did so to Mr. McGuinness in the relevant timeframe.

I. Fact History

Vaccination and Onset of Symptoms

Petitioner's medical history was significant for sleep apnea, coronary artery disease, hyperlipidemia (not otherwise specified), benign hypertension, mild allergic asthma, colonic polyps, gallstones, and an angiomyolipoma of the left kidney. Ex. 2 at 61–62. He also smoked two packs of cigarettes a day for thirty years, but had quit in 1992. *Id.* at 62. Petitioner had no prior history of joint pain. *Id.* at 61–62.

On July 21, 2014, Mr. McGuinness (who was 67 at the time) received the pneumococcal vaccine from his Primary Care Physician ("PCP"), Jeffrey Wartman, M.D., at Bloomingdale Medical Associates ("BMA") in Riverview, Florida. Ex. 1 at 1. Mr. McGuinness has maintained that "shortly after the vaccination, [he] lost all mobility in everyone joint and muscle in [his] body." Ex. 9 at 1.

Mr. McGuinness returned to Dr. Wartman on August 8, 2014, complaining of a two-week history of pain in multiple joints (which if accurate would put onset on July 25, 2014—four days post-vaccination. Ex. 2 at 55. He stated that his knees and wrists were most problematic, and the pain made him feel like he had sand in his joints. *Id.* He also reported morning stiffness in his back that lasted about twenty minutes. *Id.* Mr. McGuinness noted that swimming helped, but he was also taking Aleve for the pain. *Id.* Petitioner had recently taken a trip to Long Island, N.Y., and thought he was possibly exposed to Lyme disease because it was common in that area, but had no lesions or tick bites. *Id.*

Dr. Wartman assessed arthralgias (meaning joint pain) in multiple areas of Petitioner's body, and concluded that Mr. McGuinness's reaction was likely due to the pneumococcal vaccine and not related to Lyme disease, as there was no evidence of joint swelling or tenderness, and no target lesions. Ex. 2 at 58. The physical examination did not reveal evidence of synovitis, but it did indicate symmetric arthropathy without any joint swelling. *Id.* Dr. Wartman did, however, order a Lyme disease antibody test despite his doubts, because of Mr. McGuinness's physical symptoms and prior travel to a place where the disease was prevalent. *Id.*

A month later, on September 2, 2014, Mr. McGuinness saw Dr. Wartman again, for treatment of persistent joint pain that had ebbed and flowed over the past month. Ex. 2 at 50. He stated that in the beginning, his pain was consistently in all of his joints, but now the pain bounced from one joint to another. *Id.* at 52. Mr. McGuinness's Lyme disease antibody test came back negative, and his physical exam revealed that he had no fever, dizziness, joint swelling, or skin

lesions. *Id*. Dr. Wartman assessed arthralgias once more, speculating a second time that Mr. McGuinness had experienced a reaction to the pneumococcal vaccine. *Id*. Though Mr. McGuinness tested negative for Lyme disease, Mr. Wartman recommended Doxycycline, and offered a lab test for a rheumatoid factor, but the test came back negative. *Id.* at 53, 115.

That same month, Mr. McGuinness had his first appointment with rheumatologist David Sikes, M.D., at Florida Medical Clinic on September 15, 2014. Ex. 2 at 146. Mr. McGuinness told Dr. Sikes that two days after receiving the pneumococcal vaccine he had woken up and "couldn't get out of bed." *Id.* During his visit, Mr. McGuinness complained of pain in his shoulders, hips, and groin. He reported that he had severe pain in his right wrist stating it was a 7/10 on the pain scale. *Id.* He reported morning stiffness for two to three hours a day and that his pain was worse when first waking up. *Id.* Over-the-counter medication helped with his pain, and a steroid allowed him to engage in leisure activities like golf. *Id.*

On physical examination, Mr. McGuinness displayed swelling in both wrists and middle finger metacarpophalangeal ("MCP") joint on his left hand. Ex. 2 at 148–49. He again tested negative for a rheumatoid factor, as well as C-reactive protein, and ANA.[3] *Id.* at 149. Dr. Sikes identified several possible diagnoses, including RA, but deemed it unlikely Petitioner had Lyme disease. *Id.* at 50. Dr. Sikes prescribed ten milligrams of Prednisone as a therapeutic trial. *Id.*

The following week (on September 24, 2014), Mr. McGuinness returned to Dr. Sikes. Ex. 4 at 7. He now reported that he almost felt like he was back to feeling 100 percent, and he had no joint pain, muscle stiffness, or a loss of function. *Id.* However, upon physical examination, Mr. McGuinness now displayed persistent joint swelling. *Id.* at 9–10. Dr. Sikes continued to prescribe fifteen milligrams of Prednisone and ordered a musculoskeletal ultrasound. Ex. 7 at 10. The ultrasound revealed active inflammation, with synovitis in Petitioner's right wrist and synovial thickness above normal limits in his joints. Ex. 4 at 12.

A month later, on October 20, 2014, Mr. McGuinness had a follow-up appointment with Dr. Sikes. Ex. 4 at 14. Mr. McGuinness continued to have peripheral joint pain and stiffness and a physical examination showed continued swelling in the middle finger MCP joint of the right hand and wrist. *Id.* at 14–16. Dr. Sikes's working diagnosis was RA, based upon Mr. McGuinness's active inflammation and joint pain. *Id.* at 14. Dr. Sikes prescribed a continuation of Prednisone with a gradual tapering off in the coming weeks, and beginning twenty milligrams of Methotrexate injections weekly. *Id.* at 14, 17–18, 19–22.

[3] C-reactive protein and ANA are markers for inflammation. P. Venables et al., *Clinical Manifestations of Rheumatoid Arthritis*, UpToDate 1, 7 (2014), filed as Ex. 12, Tab A on December 13, 2018 (ECF No. 21-2).

Treatment History: End of 2014 to the Present

From November 2014 to June 2016, Mr. McGuinness had numerous follow-up appointments with Dr. Sikes. Ex. 4 at 24, 29, 35, 41, 49, 58, 64, 74, 82, 88, 93, 98, 104, 107, 112, 119. Mr. McGuinness's symptoms fluctuated in this time period, often as a result of changes in his medication. *See, e.g.*, Ex. 2 at 41, 43–46 (continuing joint pain and stiffness coming off Prednisone and starting Methotrexate on November 17, 2014); *id.* at 36, 38 (reporting significant improvement on July 13, 2015); *id.* at 25–28 (reporting a migratory pattern of his RA with no improvement on Methotrexate or Leflunomide on January 14, 2016). His joint pain would also flair up after the benefit of RA-specific treatments waned. Ex. 4 at 119. Petitioner, however, found the medication gave him fatigue. Ex. 3 at 7–10.

In July 2016, Mr. McGuinness returned to his PCP, Dr. Wartman, for a routine follow-up appointment. Ex. 2 at 18. He now reported that he had experienced rheumatologic problems for the past two years, attributing them to the pneumococcal vaccine. *Id*. That summer, he continued to experience some worsening joint pain in his knees and right hand, which he felt might be a medication side-effect in part. Ex. 2 at 112–13. Dr. Sikes prescribed switching from Actemra to 1000 milligrams of Orencia infusions weekly. Ex. 4 at 140–43. The working diagnosis for Petitioner's symptoms remained seronegative rheumatoid arthritis. Ex. 4 at 115.

The start of 2017 saw Petitioner continuing to suffer from RA symptoms, and treaters maintained Methotrexate while adjusting other medications based on their demonstrated responsiveness or Petitioner's ability to tolerate them. *See, e.g.*, Ex. 4 at 173–76, 179–82; Ex. 2 at 11.[4] Changes in the infused medications he received did demonstrably improve how he felt. Ex. 4 at 187–90.

An ultrasound performed in late February 2017 revealed low disease activity, but some synovial thickening in the joints of Petitioner's hands and wrists. Ex. 4 at 193. RA symptoms continued to wax and wane throughout the year, and Petitioner did not at this time ever test positive for autoantibodies commonly associated with RA, thus confirming his seronegative diagnosis. Ex. 4 at 206–10. Petitioner continued, however, to attribute his medical problems throughout the last three years to the pneumococcal vaccine he had received on July 21, 2014. Ex. 9 at 3.

On May 1, 2019, Mr. McGuinness had a routine six-month follow-up with his PCP, Dr. Wartman. Ex. 15 at 5. Mr. McGuinness now reported that he had occasional wrist stiffness, but since stopping biologics had not experienced side effects, and otherwise expressed the view that his RA symptoms were resolved or in remission—an opinion shared by Dr. Wartman. *Id.* at 5, 8.

[4] Notably, the record establishes Petitioner received a second pneumococcal vaccine in the winter of 2017, but records do not indicate any symptom flares or other reaction. Ex. 2 at 15.

That assessment remained as of July 3, 2020 (Ex. 14 at 9), and no subsequent medical records have been filed in this case.

II. Witness Testimony

A. Petitioner's Expert

1. *Samar Gupta, M.D.* – Dr. Gupta, a board-certified rheumatologist, was a non-treating medical expert who submitted an expert report and testified for the Petitioner in support of the contention that the pneumococcal vaccine can cause RA. *See generally* Tr. at 4–75. Report, dated Nov. 11, 2018, filed as Ex. 12 (ECF No. 31-1) ("Gupta Rep.").[5]

Dr. Gupta completed his medical residency from Wayne State University in Michigan, and obtained his Rheumatology Fellowship Degree from the University of Michigan, Ann Arbor. *See* Curriculum Vitae, filed Dec. 13, 2018 (ECF No. 31-8) ("Gupta CV") at 2. He is currently an Associate Professor of Medicine at the University of Michigan School of Medicine and a Section Chief for the VA Rheumatology Clinical Services and Medical Education. Gupta CV at 2. He is also the clinical director of VA rheumatology clinical services in Ann Arbor, and he directs the medical education program at the VA. Tr. at 6. Overall, Dr. Gupta monitors about 500 patients yearly. *Id.* at 7. His clinical area of expertise is in inflammatory arthritis, which specifically includes rheumatoid arthritis and psoriatic arthritis. *Id.* at 6. However, Dr. Gupta has never published any papers regarding vaccines or the immune system. Tr. at 44–46.

Dr. Gupta began with an overview of RA's characteristics and presentation. Tr. at 11; Gupta Rep. at 3. As he explained, RA is a chronic autoimmune disease that can involve other areas of the body besides the joints. Tr. at 11–13; Gupta Rep. at 3. It typically progresses from one joint to another, in a bilateral and symmetric pattern. *Id.* Most of the time RA involves the small rather than the large joints. Tr. at 12. Common symptomatic features of RA include morning stiffness, fatigue, and weight loss. *Id.* at 11–12. Dr. Gupta admitted RA's onset can be insidious and can long precede outward clinical manifestation, although he also noted (from an article cited by Respondent) that "acute onset reflecting immediate immune perturbation is also possible." J. Smolen et al., *Rheumatoid Arthritis*, 4 Nature Rev.'s: Disease Primers 1, 5 (2018), filed as Ex. C, Tab 2 on Mar. 9, 2021 (ECF No. 65-1).

RA can further be differentiated into two categories: seronegative and seropositive. Tr. at 13–14. A seropositive RA diagnosis is based on testing positive for the rheumatoid factor ("RF") or anticyclic citrullinated peptide ("anti-CCP") antibodies, whereas a seronegative RA diagnosis

[5] Dr. Gutpa's report also proposed an alternative theory that he later retracted, which relied on the causal impact of the vaccine's aluminum adjuvant triggering an autoimmune cascade (a causal mechanism usually referred to by the acronym "ASIA" (autoimmune/inflammatory syndrome induced by adjuvants)). Tr. at 37, 54–55. I thus do not herein consider the parts of his report addressing ASIA as a mechanistic explanation.

is a clinical profile consistent with RA but without evidence of the antibodies' presence. *Id.* Both antibodies are understood to be drivers of the autoimmune processes resulting in RA symptoms. *Id.* at 14–15. Dr. Gupta articulated that seronegative versus seropositive RA are not distinguishable as a general matter but noted that a seropositive RA diagnosis suggests a more aggressive disease course, allowing medical providers to better predict prognosis and thus propose the most effective treatments. *Id.* at 15. Diagnosis of RA is based on laboratory and radiological exams (x-rays, ultrasounds, and MRIs) plus the clinical evidence. *Id.* at 12–13. He added that there are two sets of criteria for diagnosing RA—1987 criteria developed for recruiting patients for clinical research trials, and 2010 criteria used for classifying RA in a more clinical fashion. *Id.*

Dr. Gupta then went on to discuss the causes of RA, which he admitted are largely still unknown. There is evidence that a genetic predisposition in RA patients is a factor, although not all individuals possessing a predisposition actually go on to develop RA. Tr. at 30; A. Sharma et al., *Vaccination as a Triggering Agent for the Development of Rheumatoid Arthritis*, Int. J. Rheum. Dis. e8, e8 (2011), filed as Ex. 19 on Mar. 2, 2021 (ECF No. 58-1) ("Sharma"). Additionally, there are risk factors, like smoking, that can make patients more likely to develop RA. Tr. at 30–31. Here, Mr. McGuinness had been a regular smoker in the past, although Dr. Gupta noted that the record suggested Petitioner had quit 20 to 30 years before vaccination, thus reducing its potential explanatory significance. *Id.* at 39–40. He also noted the risk ratio of smokers versus nonsmokers as 1.5, which he deemed low. *Id.* at 40.

There is also evidence, Dr. Gupta proposed, that environmental agents—including vaccines—can trigger RA. Tr. at 31–32; Gupta Rep. at 4; E. Giat & M. Lidar, *Vaccinations in Rheumatoid Arthritis*, Vaccines & Autoimmunity 233, 234 (2015), filed as Ex. 12, Tab E on Dec. 17, 2018 (ECF No. 32-1) ("Giat & Lidar"); Ray et al., *Vaccine Safety Datalink Team. Risk of Rheumatoid Arthritis Following Vaccination with Tetanus, Influenza and Hepatitis B Vaccines Among Persons 15-59 Years of Age*, Vaccine 6591, 6596 (2011), filed as Ex. 20 on Mar. 2, 2021 (ECF No. 58-2) ("Ray"). Dr. Gupta admitted there is no direct scientific proof that vaccines can cause RA, but opined that there are enough case reports to support an association between the two generally. Tr. at 30–32. In Dr. Gupta's opinion, the pneumococcal vaccine could cause RA as well. *Id.* at 30–31.

Dr. Gupta relied on molecular mimicry as a mechanistic explanation for how the pneumococcal vaccine could result in RA. Tr. at 35. The pneumococcal vaccine contains antigens comprised of sequences that may mimic homologous sequences at the situs of RA's attack (synovial tissues), resulting in a cross-reaction by the antibodies generated in response to the vaccine. Tr. at 35; Gupta Rep. at 4–5. The cross-reaction would continue and become chronic, even after the vaccine itself has cleared from the body. Tr. at 35–36. Dr. Gupta deemed molecular mimicry a theory accepted by medical science, and constituted a strong explanation for what could occur in an autoimmune process absent other reliable explanations. Tr. at 138–39.

Dr. Gupta did not, however, identify any homology (e.g., identity of amino acid sequences) between antigens of the pneumococcal vaccine and protein components of joint tissues attacked in RA, and he did not offer any literature or other medical or scientific evidence connecting the vaccine (or even the wild bacterial infection version) to RA. Tr. at 44–46. Rather, he mostly referenced some case reports involving injury after vaccination (which he deemed legitimate proof for rare kinds of occurrences like vaccine injuries). Tr. at 32, 35, 139.

One such article involved a different autoimmune illness—idiopathic thrombocytopenic purpura ("ITP")[6]—which Dr. Gupta still deemed relevant in showing how molecular mimicry would function in driving a pneumococcal vaccine-caused autoimmune injury. Tr. at 32, 58; S. Gupta & D.C. Brennan, *Pneumococcal 13-Valent Conjugate Vaccine (Prevnar 13)-Associated Immune Thrombocytopenic Purpura in a Renal Transplant Recipient: A Case Report*, Transplantation Proceedings 262, 263 (2016), filed as Ex. 12, Tab C on Dec. 13, 2018 (ECF No. 31-4) ("Gupta & Brennan").[7] Dr. Gupta posited that if this phenomenon could happen in other autoimmune diseases, it is possible that the same mechanism occurs in the development of RA. Tr. at 32–33.[8] He also referenced a chapter of a textbook discussing the theory of molecular mimicry in the context of RA and vaccination. Giat & Lidar at 234. But Dr. Gupta admitted that Giat & Lidar actually concluded that "vaccines likely play an *insignificant* role in the pathogenesis of RA." *Id.* at 235 (emphasis added).

Dr. Gupta also described an alternative mechanism for causation—nonspecific activation of the immune system. Tr. at 36. As he proposed, a patient predisposed to RA who received the pneumococcal vaccine (which is highly antigenic) could experience nonspecific inflammation and immune reactivity that could cause the patient to get clinical RA. *Id.* at 36–37. Dr. Gupta did not specify whether this reaction would be the product of the initial, innate immune response or the subsequent adaptive response (which would inherently be specific to the vaccine itself), although the "inflammation" he referenced would more likely occur as part of the innate/immediate response. Regardless, he offered only one piece of medical literature to support his contention, Giat & Lidar, and it offered little direct or independent proof in support as it only proposed nonspecific immune system activation as a theory. Giat & Lidar at 234. The literature did not

[6] Idiopathic purpura is "a type of thrombocytopenic purpura [commonly known as ITP] that is not directly associated with any definable systemic disease but often follows a systemic infection; it has been found to be an autoimmune condition, caused by antigens against platelets, resulting in ecchymoses, petechiae, and other bleeding." *Dorland's Illustrated Medical Dictionary* 901 (33rd ed. 2020) ("*Dorland's*").

[7] The author of this article is not the same person as Dr. Gupta.

[8] Another case report offered by Dr. Gupta involved a patient who developed polyarthritis after getting a different vaccine - the rabies vaccine. Tr. at 34; Sharma at e8.

explain the relevant timeframe of when this reaction would take place or how this reaction explains chronic conditions like RA. *Id.*

Next, Dr. Gupta addressed the medical record, noting how he felt it supported the conclusion that the pneumococcal vaccine could have caused Mr. McGuinness's RA. There was no evidence that Mr. McGuinness ever had RA prior to vaccination (even though the insidious pathology of the condition allowed for that possibility). Tr. at 37–38, 74. He deemed this significant, adding that since Mr. McGuinness had consistently tested negative for the autoantibodies most commonly associated with RA, it was unlikely Petitioner had a strong disposition to develop RA pre-vaccination. *Id.* at 38–39, 41. Thus, the post-vaccination start of clinical symptoms was to Dr. Gupta evidence of the vaccine's causal role. Indeed, one of Petitioner's treating physicians, Dr. Wartman, had proposed a connection between the vaccine and his initial symptoms on two occasions close-in-time to onset: August 8, 2014, and September 2, 2014. *Id*. at 38–39; Ex. 2 at 54, 59.

The record, Dr. Gupta maintained, further substantiated that Mr. McGuinness's RA onset likely began two weeks after receiving the vaccine (although in his expert report he had stated onset began within the longer timeframe of four weeks). Tr. at 16, 49, 74; Gupta Rep. 4. But in so opining Dr. Gupta blurred the timeframe a bit, acknowledging an association between the nonspecific, malaise-like complaints Petitioner reported experiencing immediately after the vaccine (but which he maintained resolved within 24 to 48 hours) and the "true" manifestations of RA two weeks later. Tr. at 51. To connect these symptoms, Dr. Gupta proposed that 20 percent of vaccinated patients complain of muscle pain occurring usually within 7-14 days of receiving the pneumococcal vaccine, and 10 percent of vaccinated patients experience limited arm movement as well.[9] Over time, Petitioner's joint pain worsened to joint swelling (which Dr. Gupta said is more indicative of RA) by September 15, 2014—eight weeks after vaccination. Tr. at 137; Ex. 2 at 148–49. However, on cross examination Dr. Gupta admitted that there was no evidence that Mr. McGuinness's initial pain dissipated immediately after vaccination (and thus could arguably be connected to his purported onset two weeks later). Tr. at 54.

As evidence for the proposed onset, Dr. Gupta highlighted three of Petitioner's doctor's visits from the summer of 2014. Tr. at 16. The August 8, 2014 visit (occurring less than three weeks post-vaccination) revealed Petitioner's complaints of persistent morning stiffness—a classification criterion under the 1987 diagnostic guidelines. *Id.* at 17. Petitioner at this time also reported swelling, though it was not then visible to the physician, which indicates the disease was not yet chronic. *Id.* at 19. And Petitioner's PCP, Dr. Wartman, also noted that this was likely a reaction to the vaccine. *Id.* at 17.

[9] *See* Prevnar 13 Package Insert, *available at* https://www.fda.gov/downloads/BiologicsBloodVaccines/Vaccines/ApprovedProducts/UCM574852.pdf.

By the time of the September 2, 2014 visit, Petitioner was experiencing continued pain that had begun migrating to different joints, although his presentation overall had yet to manifest the full spectrum of RA. Tr. at 19–20. Then, on September 15, 2014 (now eight weeks after the vaccine), Petitioner reported a history of multiple swollen joints and joint pain—all indicative of inflammatory arthritis. *Id.* at 21. His exams also revealed swelling of the middle finger MCP joint and swelling over the joint line on both his wrists. *Id.* This visit was also important because an ultrasound was ordered to help diagnose whether Mr. McGuinness had RA, and it supported that conclusion, along with a Vectra DA test, which indicated elevated levels of inflammation. *Id.* at 21, 29; Gupta Rep. at 4. Petitioner was thus correctly diagnosed with seronegative RA (a conclusion that Dr. Gupta noted was shared by Respondent's experts). Tr. at 27–28.

Dr. Gupta did not believe there was an alternative cause for Mr. McGuinness's RA. Tr. at 41. No other serological tests for alternative arthritic diseases came back positive—and although Petitioner had displayed some clinical features of psoriasis or irritable bowel syndrome ("IBS"), Dr. Gupta discounted these diseases. *Id.* at 40. He explained that psoriatic arthritis can look like RA, and the rheumatoid factor is not usually present, thus making it facially comparable in some respects to Petitioner's presentation. *Id.*; *See Dorland's Illustrated Medical Dictionary* 154 (33rd ed. 2020) ("*Dorland's*"). However, psoriatic arthritis typically includes skin rashes and distal joints of the fingers, which Mr. McGuinness did not experience. Tr. at 40. Dr. Gupta similarly ruled out IBS because even though Petitioner displayed symptoms such as altered bowel habits, he had undergone diagnostic testing from colonoscopies that did not confirm it. *Id.* at 41; *Dorland's* at 1805. And although Respondent's expert deemed some references to diarrhea in the record significant (*Id.* at 24), Dr. Gupta denied it was reflective of RA, adding that the medical records mentioning this symptom observed its independent character. *Id.* Otherwise, Dr. Gupta argued, an inflammatory form arthritis associated with IBS has a different symptomatology than what Petitioner experienced. *Id.* at 25.

Two additional pieces of literature were brought up on cross examination. Dr. Gupta admitted that one, the Ray article, acknowledged the need for larger studies to confirm more definitively if there was a risk of RA in association with *any* vaccine. Tr. at 65; Ray at 6596. But Dr. Gupta objected that a large-scale study was difficult because the circumstances of any vaccine injury were generally rare. Tr. at 66. Contrary to Dr. Gupta's position, however, Ray had found no statistically significant association between exposure to at least one of the vaccines at issue (the hepatitis B vaccine) and the development of RA. Ray at 6596. There was only a possible association between RA and the flu vaccine in the initial cohort analysis, but an extended case-control analysis and the addition of more data points did not demonstrate this increase. *Id.*

The other piece of literature discussed at this time was filed by Respondent, and related to a study considering immunological provocations of common vaccines in causing RA. C.

Bengtsson et al., *Common Vaccinations Among Adults Do not Increase the Risk of Developing Rheumatoid Arthritis: Results from the Swedish EIRA Study*, Annals Rheumatic Diseases 1831, 1832 (July 5, 2010) (ECF No. 54-1) ("Bengtsson") (looking for possible associations between vaccination and the risk of developing RA using data from a Swedish population-based RA case-control study). Dr. Gupta agreed that Bengtsson stood for the proposition that most vaccines do not increase the risk of RA in individuals with established risk factors. Bengtsson at 1833.

B. Respondent's Experts

1. *Robert Lightfoot, M.D., –* Dr. Lightfoot, a board-certified rheumatologist, testified on behalf of Respondent, and submitted a single expert report. *See generally* Tr. at 75–105; Report, dated Feb. 19, 2019, filed as Ex. A (ECF No. 34-1) ("Lightfoot Rep."). Dr. Lightfoot discussed Petitioner's disease presentation and medical history, as well as other aspects of his evidentiary showing.

Dr. Lightfoot obtained his medical degree in microbiology and immunology from Vanderbilt University School of Medicine. Lightfoot First Rep. at 1; Dr. Lightfoot Curriculum Vitae, filed as Ex. B (ECF No. 34-2) ("Lightfoot CV") at 1. He was trained in Internal Medicine at Columbia-Presbyterian Medical Center in New York City, New York, and at Vanderbilt University Hospital in Nashville, Tennessee. Lightfoot Rep. at 1. He was also trained in Rheumatology at Columbia-Presbyterian Medical Center. Lightfoot First Rep. at 1; Lightfoot CV at 1. Currently, he is a partially-retired Professor of Medicine at the University of Kentucky, where he has been teaching for the past thirty-three years. Tr. at 77. Dr. Lightfoot had a regular clinic of inpatient and outpatient services, along with a teaching clinic with rheumatology fellows. *Id.* at 78. He monitors approximately 800 to 1,000 patients yearly, through his previous private clinic (and now his fellows clinics). *Id.* He has also authored approximately 45 to 50 peer-reviewed articles on the topic of rheumatology. *Id.* at 79.

Dr. Lightfoot began broadly by defining RA as a common disease that involves many joints. Tr. at 80. He characterized it as a chronic, inflammatory arthritis that tends not to remit without medication. *Id.* He also explained that even if RA patients using medication go into remission, it is common for them to continue to exhibit considerable thickened MCP joints. Lightfoot Rep. at 7. Early RA can present atypically, and serological evidence indicates that antibodies associated with it can be present in the body years before the onset of symptoms. *Id.*

Dr. Lightfoot agreed with Dr. Gupta that under the relevant classification criteria, Petitioner's working diagnosis was properly deemed seronegative RA. Tr. at 81–82, 95-97; Lightfoot Rep. at 5. One criterion particularly relevant herein (in addition to the absence of serologic evidence) was the fact that Mr. McGuinness suffered from none of the other illnesses (gout, spondylarthritis, psoriatic arthritis, and inflammatory bowel disease) often associated with RA. *Id.* at 93; Lightfoot Rep. at 6. However, unlike Dr. Gupta, Dr. Lightfoot found that Petitioner's

medical history made it hard to tell whether Mr. McGuinness truly had a non-rheumatoid-originating form of RA, although the record does not state otherwise. Tr. at 93.

Dr. Lightfoot offered several comments about Petitioner's purported onset. As a general matter, he deemed the contention that Mr. McGuinness's RA was associated with his receipt of the pneumococcal vaccine, since symptoms began not long thereafter, a *post hoc* fallacy, arguing that the temporal association alone did not establish a causal relationship. Tr. at 94; Lightfoot Rep. 7. Dr. Lightfoot in fact did not find any evidence in the record that Petitioner's RA was attributable to the vaccine. Tr. at 88. As a general matter, in Dr. Lightfoot's experience RA could begin pathologically years prior to clinical manifestations. *Id*. And a process involving a purported immune response involving the production of antibodies triggered by vaccination would take at least one to two weeks after immunization. *Id.* at 94. Yet in this case, Petitioner had informed treaters on August 8, 2014, that he was experiencing myalgia, muscle aches, joint aches, *a few days* post-vaccination—far too soon to reflect an immune-caused response. *Id.* at 96.

Dr. Lightfoot allowed that the lack of subjective complaints, plus objective evidence of swelling during this August visit, did not preclude the onset of RA at this time, since the disease's pathology is slow to progress. Tr. at 96. But its onset in fact was likely far sooner, and close in time to the vaccination, given the insidious nature of RA and the record in this case. Lightfoot Rep. at 8. This was too soon after vaccination to associate onset with the vaccine.

Besides addressing such matters, Dr. Lightfoot also contested whether there was any reliable evidence linking the pneumococcal vaccine to RA in the first place. Tr. at 88. A molecular mimicry process, he noted, would be dependent on an antibody cross-reaction with self amino acid chains found in the joints and their synovia, but the pneumococcal vaccine's antigens are polysaccharides,[10] carbohydrates composed of sugar molecules rather than amino acids. *Id.* at 89. Additionally, Dr. Lightfoot cited Bengtsson, which found a group of adults who received a vaccine five years before onset did not see an increased risk of RA thereafter. Bengtsson at 1832. However, Dr. Lightfoot is not an immunologist, and he otherwise deferred to Dr. He on such matters.

Petitioner's prior smoking habits were also mentioned by Dr. Lightfoot as a risk factor for RA. Tr. at 98. However, certain items of literature (some filed by Respondent) seemed to place limits on the degree of risk in individuals who had ceased smoking at some point. *See* G. Firestein & I. McInnes, *Immunopathogenesis of Rheumatoid Arthritis*, 46 Immunity Rev. 183, 185 (2017), filed as Ex. C, Tab 1 on Feb. 19, 2019 (ECF No. 36-1) ("Firestein"). Firestein, for example, offered some data revealing that the risk from smoking *declined* beginning ten years after the date of quitting. Firestein at 185. Dr. Lightfoot also admitted it was possible that smoking was in fact mainly a relevant risk factor for *seropositive* RA patients (and thus not Petitioner). Tr. at 97, 101,

[10] Monosaccharides are simple sugars, so polysaccharides are "a carbohydrate that on hydrolysis yields a large number of monosaccharides (variously defined as five or more to eleven or more)." *Dorland's* at 1161, 1471.

105; *see also* A. Pratt & J. Isaacs, *Seronegative Rheumatoid Arthritis: Pathogenetic and Therapeutic Aspects*, 28 Best Prac. & Res. Clinical Rheumatology 651, 653 (2014), filed as Ex. C, Tab 3 on Feb. 19, 2019 (ECF No. 36-2) ("Pratt & Isaacs"). Besides smoking and gender, Dr. Lightfoot was otherwise not aware of any other specific environmental stresses bearing on the development of RA that would pertain to this claim. Tr. at 101–02.

2. *You-Wen He, M.D., PhD.* – Dr. He, an immunologist with specialties in studying innate immunity, adaptive immunity, vaccines, vaccine diseases, and cancer immunotherapy, testified on Respondent's behalf and prepared a written report, as well. *See generally* Tr. at 106–134; Report, dated February 11, 2019, filed as Ex. C (ECF No. 34-3) ("He Rep.").

Dr. He received his medical degree from The Fourth Military Medical University in Xian, China. Dr. He Curriculum Vitae, filed as Ex. D on February 19, 2019 (ECF No. 34-4) ("He CV") at 1. He obtained his Master of Science degree from the Institute of Microbiology and Epidemiology, Academy of Military Medical Sciences in Beijing, China, and his Ph.D. from the Department of Microbiology & Immunology, University of Miami School of Medicine in Miami, Florida. He CV at 1. He is currently working at Duke University Medical Center as a Professor of Immunology. He Rep. at 1; He CV at 1. Dr. He has held the position as course director of immunology and human disease for the last 8 years of the immunology program. Tr. at 107–08. Additionally, he currently serves as the Co-Principal Investigator for 4 clinical trials focusing on cancer immunotherapy. He Rep. at 1. He has also authored 130 peer-reviewed articles on the topic of immunology. Tr. at 108. His areas of specialty include innate immunity, adaptive immunity, vaccines, vaccine disease, cancer immunotherapy. *Id.* at 108. Dr. He is not board-certified in the U.S., however, because he did not go through a residency program. *Id.* at 108–09.

Dr. He began his testimony by defining "autoimmune disease" to mean an illness in which the immune system attacks its own tissue. Tr. at 110. But he disputed that Petitioner had offered a reliable or reputable theory for how any vaccine could trigger an autoimmune process resulting in RA. *Id*. at 111, 120; He Rep. at 4.

In particular, Dr. He questioned molecular mimicry's value as a potential mechanism in this case.[11] The core concept in molecular mimicry is that antigenic epitope similarity between a vaccine's antigens and human proteins allows for cross-reactivity between antibodies (created by the host's adaptive immune system in reaction to those antigens) and structures comprising self tissue. *Id.* at 111. But the mere possibility of cross-reactivity immune response due to amino acid

[11] Indeed, Dr. He reiterated later in his testimony that those working in the autoimmune disease field had been attempting to confirm the process of molecular mimicry since 1976, but unsuccessfully. Tr. at 130; Firestein at 183, 186. Otherwise, Dr. He contended that modern genomic thinking was evolving away from embracing molecular mimicry as explanatory of virtually any autoimmune process. *Id.* at 113.

sequential homology does not automatically lead to an autoimmune disease. *Id.* at 119, 126. The amount of homology/antigenic similarity occurring naturally, but without an autoimmune disease, was too high to conclude that the two were inexorably linked. *Id.* at 126. However, Dr. He admitted that cross-reactivity is widely understood to occur in the pathologic context of many autoimmune diseases. *Id*. at 129.

Moreover, Dr. He noted that molecular mimicry was a mechanism associated with an *adaptive* immune response—the secondary arm of the immune response that invariably took several days at a minimum, or longer, to begin producing antibodies. Tr at 119–20. A mimicking cross-reaction could implicate a variety of immune cells, including self-reactive T cells, B cells, and lymphocytes, and in Dr. He's view it would take between five days to one or two weeks after the introduction of an antigen into the body for a T cell and a self-reactive T lymphocyte to match. *Id.* at 113–15. This meant that the timeframe from vaccination to onset in Petitioner's case—around two to three days—was too short to be considered medically acceptable. *Id*. at 116. However, Dr. He admitted that if Dr. Gupta's timeframe of two weeks was accepted, this would be medically acceptable, though he maintained his contention that Mr. McGuinness' RA was not caused by molecular mimicry. *Id*. at 125.

Dr. He raised other objections to the substantiation offered for Dr. Gupta's opinion. Case reports, he argued, carried minimal weight in proving causation. Tr. at 112. Dr. He also stated that he himself had been unable to identify a single case report involving the pneumococcal vaccine and rheumatoid arthritis. *Id.* And he questioned whether a "nonspecific" immune system reaction (an innate immune activation that release cytokines and other immediate and available immune cells in reaction to a threat) would persist for more than 24 to 48 hours, and thus could not be responsible for a chronic condition. Tr. at 124–25. Dr. He agreed on cross examination, however, that genetics accounted for a large percentage of the risk for RA. *Id*. at 122, 125; He Rep. at 4.

III. Procedural History

Mr. McGuinness filed his petition on July 17, 2017, and an amended petition on June 4, 2018. ECF Nos. 1, 23. By June 2018, Petitioner had filed all relevant medical records and a Statement of Completion. ECF No. 27. Respondent filed a Rule 4(c) report on August 13, 2018, contesting Petitioner's right to compensation. ECF No. 28. Petitioner then filed his expert report from Dr. Gupta on December 2018, with Respondent thereafter filing expert reports from Dr. Robert W. Lightfoot and Dr. You-Wen He. After the matter was transferred to me on July 20, 2020, I held a status conference with the parties and subsequently set the matter for a hearing, to be held on March 13, 2021. ECF No. 52. The trial occurred as scheduled, and the parties submitted post hearing briefs on August 30, 2021. ECF Nos. 70-71. The matter is now 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 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).[12] In this case, Petitioner does not assert a Table claim.

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*, 418 F.3d at 1278: "(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."

[12] 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 a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (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*, 121 Fed. Cl. at 245.

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 Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)). 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 Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 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. denied 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. denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *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.

C. *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. 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").

D. *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. RA and its Treatment in Vaccine Program Decisions

RA is a chronic, inflammatory autoimmune condition mainly affecting the synovial joints. Its causes are thought to be a mix of immune, genetic, and environmental factors, but it is ultimately of an unknown etiology. RA specifically involves an autoimmune attack on the synovial membranes of the joints, causing inflammation and progressive erosion and destruction of joint surfaces, such as cartilage and bone, along with deformity of affected joints. *Dorland's at* 154; P. Venables et al., *Clinical Manifestations of Rheumatoid Arthritis*, UpToDate 1, 1 (2014), filed as Ex. 12, Tab A on December 13, 2018 (ECF No. 21-2) ("Venables"). It is often characterized by abnormalities in the blood and synovial fluid, and is associated with specific antibodies, such as RF and anti-CCP. About 75-80 percent of patients with RA test positive for RF, anti-CCP, or both. Those patients are known as having seropositive RA because they have the presence of these antibodies. Patients that do not have these antibodies, but display clinical signs of RA, are known to have seronegative RA. Venables at 7.

There are a number of risk factors associated with the development of RA, and they are consistent with what the experts discussed at hearing. Smoking is the most widely recognized non-genetic risk factor, and is strongly linked with the presence of the anti-CCP antibodies often found in cases of seropositive RA. *See* Tr. at 31, 98, 102. Genetic factors are also associated with RA. *Id.* at 30, 102. It is undisputed in this case that Mr. McGuinness did not test positive for the RF or anti-CCP antibodies.

Program petitioners asserting RA as a vaccine injury have in only a few circumstances obtained entitlement awards.[13] *See, e.g.*, *H.J. v. Sec'y of Health & Hum. Servs.,* No. 11-301V, 2015 WL 6848357 (Fed. Cl. Spec. Mstr. Nov. 6, 2015) (petitioner established that her immune system was predisposed to autoimmune diseases such as RA, and that the Tdap vaccine significantly aggravated her pre-existing RA); *Campbell v. Sec'y of Health & Hum. Servs.,* 97 Fed. Cl. 650

[13] Decisions from different cases do not control the outcome herein, with only Federal Circuit decisions setting legal standards to which new claims must adhere. *Boatmon*, 941 F.3d at 1358–59; *Hanlon v. Sec'y of Health & Hum. Servs*., 40 Fed. Cl. 625, 630 (1998). Nevertheless, special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Hum. Servs*., 76 Fed. Cl. 328, 338–39 (2007) ("[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim") (emphasis added). They would thus be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions.

(2011) (petitioner successfully demonstrated that the flu vaccine could cause RA). This kind of claim has been more often unsuccessful. *See, e.g.*, *Moran v. Sec'y of Health & Hum. Servs.,* No. 16-538V, slip op. (Fed. Cl. Spec. Mstr. Oct. 4, 2021) (denying entitlement for a petitioner asserting the flu vaccine caused him to develop symptoms of RA after three days); *Monzon v. Sec'y of Health & Hum. Servs.*, No. 17-1055V, 2021 WL 2711289 (Fed. Cl. Spec. Mstr. June 2, 2021) (denying entitlement to a petitioner alleging Tdap vaccine caused her to develop RA after 10 days); *Hock v. Sec'y of Health & Hum. Servs.,* No. 17-168V, 2020 WL 6392770 (Fed. Cl. Spec. Mstr. Sept. 30, 2020) (denying entitlement for a petitioner alleging that he developed RA after receipt of the flu vaccine); *Suliman v. Sec'y of Health & Hum. Servs.,* No. 13-993V, 2018 WL 6803697 (Fed. Cl. Spec. Mstr. Nov. 27, 2018) (dismissing petition and denying entitlement for claim alleging the Tdap vaccine caused Petitioner to develop polymyalgia rheumatica and/or myositis); *Bean-Sasser v. Sec'y of Health & Hum. Servs.,* No. 13-326V, 2016 WL 1649355 (Fed. Cl. Spec. Mstr. April 5, 2016) (denying entitlement to a petitioner alleging the hepatitis B vaccine caused her to manifest symptoms of RA approximately 11 hours later).

These prior decisions provide helpful guidelines in understanding the specific contours of causal theories involving how a vaccine might result in RA. As I explained in *Hock*, other than the *Campbell* decision noted above, I could find no cases asserting that *any* vaccine could cause either the development of RF or anti-CCP antibodies associated with RA's chronicity.[14] Indeed, given what is known about RA (and in particular the fact that the presence of the antibodies closely associated with it often long precede onset of RA symptoms), it is highly unlikely a vaccine could *either* cause these autoantibodies to develop in a medically-reasonable timeframe, *or* spark an autoimmune process dependent upon them, such that a vaccine administered close in time to appearance of RA symptoms could be deemed causal. *See also Olson v. Sec'y of Health & Hum. Servs.,* No.13-439V, 2017 WL 3624085 at *5 (Fed. Cl. Spec. Mstr. July 14, 2017 (finding the HPV vaccine was not causal of RA), *mot. for review den'd,* 2017 WL 6809589 (Fed. Cl. Dec. 14, 2017), *aff'd,* 758 Fed. App'x. 919 (Fed. Cir. 2018).

Reactive arthritis is also a recognized arthritic syndrome, distinguishable from seronegative RA, and it has also been the subject of prior Program claims. *See,* e.g. *Wyatt v. Sec'y of Health & Human Servs.,* 144 Fed. Cl. 531 (Fed. Cir. 2019); *Campbell v. Sec'y of Health & Human Servs.,* 90 Fed. Cl. 369 (Fed. Cir. 2009). Reactive arthritis is characterized by joint pain and swelling triggered by an infection in another part of the body. *Dorland's* at 154. Reactive Reiter's syndrome is a type of reactive arthritis where an autoimmune reaction, usually in response to bacterial infection, occurs. *Dorland's* at 1816; *Gearin v. Sec'y of Health & Human Servs.,* No. 07-0737V, 2008 WL 2009736, at *1–2 (Fed. Cl. Spec. Mstr. January 31, 2008). Some authorities

[14] Additionally, the other case that granted entitlement, *H.J.,* involved a claim of significant aggravation, which has not been asserted herein. *H.J.*, 2015 WL 6848357, at *8.

now consider this symptom complex to be more appropriately classified as reactive arthritis and not distinguished or named separately, however. *Id.*

There are a handful of cases discussing how a vaccine could cause reactive arthritis, and they seem to allow for a greater possibility of causation. *See, e.g.*, *Frazer v. Sec'y of Health & Human Servs.,* No. 17-1229V, 2019 WL 4741745, at *4 (Fed. Cl. Spec. Mstr. August 9, 2019); *Suliman*, 2018 WL 6803697, at *30. The reliability of a potential association between a vaccine and reactive arthritis is facially greater from the outset, especially when the timeframe is close. *Campbell,* 97 Fed. Cl. at 653. However, this form of arthritis is generally not chronic, but is instead understood to be a transient infectious reaction—and hence a claim alleging this more limited form of arthritic injury is not likely to be able to meet the Act's six-month severity requirement. *Wyatt,* 144 Fed. Cl. at 537–38. And there is no case law discussing how a reactive arthritis triggered by vaccination could transmute into the kind of classic, seronegative RA that Mr. McGuinness was diagnosed with.

The pneumococcal vaccine is a sterile suspension of saccharides of the capsular antigens of *Streptococcus pneumoniae* bacteria. *See* Prevnar 13 Package Insert, *available at* https://www.fda.gov/downloads/BiologicsBloodVaccines/Vaccines/ApprovedProducts/UCM574852.pdf. The specific form of vaccine at issue in this matter, Prevnar-13, is the conjugated version—meaning the pneumococcal vaccine combines with the polysaccharides and the protein carrier CRM197 to elicit a T cell dependent immune response, which in turn provide the needed signals for maturation of the B cell response, and hence enhances the immune response.[15] This is in direct contrast to unconjugated vaccines, in which immunity is induced exclusively through B cell antibody production reacting to the vaccine's antigens directly (and are thus "T cell independent"). D. Musher, *Pneumococcal Vaccinations in Adults*, UpToDate 1, 17 (2018), filed as Ex. C, Tab 6 on February 19, 2019 (ECF No. 36-5).

II. Petitioner Has Not Carried His Burden of Proof[16]

A. *Althen Prong One*

Petitioner has not preponderantly established a reliable theory of causation that the pneumococcal vaccine can cause RA.[17] Dr. Gupta proposed a theory of molecular mimicry,

[15] A conjugated version of a vaccine uses an "antigen produced by coupling a hapten to a protein carrier molecule through covalent bonds; when it induces immunization, the resultant immune response is directed against both the hapten and the carrier." *Dorland's* at 1985.

[16] I address the *Althen* prongs herein in order of their significance to my decision, rather than in the order they are typically presented.

[17] Dr. Gupta's alternative ASIA theory for adjuvant-induced autoimmunity was retracted as a theory of causation. But this was wise – for ASIA has never been deemed a medically-reliable causation in any prior Program cases. *See generally Morris v. Sec'y of Health & Human Servs.,* No. 12-415V, 2016 WL 3022141, at * 12 (Fed. Cl Spec. Mstr.

arguing that the pneumococcal vaccine's antigens could mimic homologous sequences on the synovial tissue resulting in cross-reactivity. But although he described the theory correctly, and how it might apply to *other* autoimmune diseases or vaccines, he did not offer reliable evidence to support his overall contention *in this context*.

In particular, Petitioner does not provide an answer as to *how* this cross-reactivity functions for the relevant disease pathogenesis, especially in the context of a seronegative case of RA (where there is no evidence that the kinds of antibodies commonly associated with RA were present to begin with). *Monzon*, 2021 WL 2711289, at *22 ("[w]hat evidence establishes that an intercurrent inflammatory process of any kind (brought on by a wild infection or vaccine) might interact with separate RA risk factors, and how might this happen?"). Indeed (and unlike other kinds of vaccines), Petitioner faces additional obstacles herein in explaining how molecular mimicry would work with respect to a vaccine that is *intended* to provoke a T cell (rather than B cell-producing antibody) response. And adding further to the complexity, the pneumococcal vaccine presents antigens to the immune system that are *not* comprised of protein made up of amino acid sequences.

The fact that molecular mimicry has scientific reliability as a general matter does not mean its mere invocation satisfies the "can cause" prong. I have repeatedly noted in other cases that petitioners cannot simply refer to molecular mimicry as a mechanistic explanation for *certain* autoimmune disease processes and expect that to suffice for *all* autoimmune diseases. *See, e.g.*, *McKown v. Sec'y of Health & Hum. Servs.,* No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (citing *Devonshire v. Sec'y of Health & Hum. Servs.,* No. 99-031V, 2006 WL 2970418, at *15 (Fed. Cl. Spec. Mstr. Sept. 2006)) ("[b]ut merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the mechanism to the injury and/or vaccine in question") (emphasis in original), *mot. for review den'd,* 76 Fed. Cl. 452 (2007)).

Here, Dr. Gupta could not marshal sufficient scientific or medical proof that molecular mimicry reliably explains how a vaccine could result in seronegative RA. He did not identify any homology between the vaccine's antigens and the protein components of joint tissues that attack RA, did not offer any literature connecting the pneumococcal vaccine and RA, nor could he reference his own professional research into the matter. At most, Dr. Gupta relies on case reports—none of which involve the pneumococcal vaccine or RA injury—to support his theory. Giat & Lidar at 234; Gupta & Brennan at 263; Sharma at e8. But case reports are well understood in the Program to be weak evidence (although they have some value nonetheless). *See, e.g.*, *Campbell*,

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Apr. 1, 2016) (discussing lack of reliability of ASIA theory); *Rowan v. Sec'y of Health & Human Servs.,* No. 10-272V, 2014 WL 7465661, at * 16 (Fed. Cl. Spec. Mstr. Dec. 8, 2014), *mot. for review den'd*, 2015 WL 3562409 (Fed. Cl. May 18, 2015); *D'Angiolini v. Sec'y of Health & Human Servs.,* No. 99-578V, 2014 WL 1678145, at *60 (Fed. Cl. Spect. Mstr. Mar. 27, 2014), *mot. for review den'd*, 122 Fed. Cl. 86 (2015), *aff'd*, 645 F. App'x 1002 (Fed. Cir. 2016).

97 Fed. Cl. at 668 ("[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value ... [but] the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary weight."). Although Dr. Gupta has clinical expertise and familiarity with RA, his research into the pneumococcal vaccine is limited, as he has never published any papers regarding vaccines or the immune system.

Dr. Gupta's alternative theory of causation—nonspecific innate reaction—also had fatal flaws. He contended that a patient given the pneumococcal vaccine who was already predisposed to RA could experience immune reactivity due to an immediate release of cytokines in response to vaccine stimulation of the immune system. However, as Dr. He noted, this reaction would last for only 24 to 48 hours, and thus cannot explain Petitioner's chronic condition *thereafter*. And the injury Petitioner incurred was not properly understood to be reactive arthritis, which arguably could be triggered simply by an aberrant innate immune response. I thus find that Petitioner has not offered a plausible causation theory, based on reliable scientific evidence.

B. *Althen Prong Three*

The experts disagreed on precise onset, with Drs. Lightfoot and He favoring a two to three-day post-vaccination onset, while Dr. Gupta proposed a two-week onset. The record better preponderantly supports Respondent's position that Petitioner's onset was close-in-time to his vaccination.[18] Petitioner stated that shortly after vaccination, he lost all mobility in his joints and muscles. Ex. 9 at 1. Then, at his first post-vaccination doctor's visit (on August 8, 2014 visit), he reported experiencing myalgia, muscle aches, and joint aches for *the past two weeks*. Ex. 2 at 55. This corroborates the conclusion that his symptoms occurred a few days after vaccination. Petitioner's subsequent visits showed he experienced continued, migrating pain that eventually showed visible signs of swelling—all of which further supports the determination that onset was days after vaccination but progressed, and worsened, over time.

Petitioner's expert, by contrast, relied on a timeline less substantiated by the record, and which unpersuasively distinguished between Petitioner's immediate symptoms and manifestation of RA two weeks later. Tr. at 51. Dr. Gupta thus emphasized the first reported evidence of Petitioner's joint swelling, even though other indicators—like the joint pain Mr. McGuinness experienced 2-3 days after vaccination—are also signs of RA onset. Also, there is no evidence that Petitioner's initial pain dissipated over the following visits, but instead built in a progressive

[18] In addition, Petitioner did not sufficiently rebut the possibility that onset might have *predated* vaccination entirely. As Dr. Lightfoot persuasively explained, arthritis can occur even years before the underlying illness presents itself. However, this possibility was not preponderantly supported by Respondent either—especially since Petitioner's seronegative RA rules out the conclusion (unlike other cases) that the anti-CCP or RF antibodies thought to drive seropositive RA could have been present long before onset. *Monzon*, 2021 WL 2711289, at *21.

manner. Petitioner's own expert even noted that RA's course tends to be progressive. Gupta Rep. at 3.

Based on a finding that onset began only a few days after vaccination, Petitioner did not succeed in establishing that his RA began in a medically acceptable timeframe under his causation theory. *See Monzon,* 2021 WL 2711289, at *24 (a two to three-day onset for classic RA was not medically acceptable). Even if I had accepted Petitioner's causation theory, it would have taken more than a few days post-vaccination to produce the antibodies that could initiate an autoimmune cross-attack. A fast, somewhat non-specific reaction close-in-time to vaccination might establish onset of a *different* injury, reactive arthritis—but that is not the injury at issue in this case. *Keja v. Sec.'y of Health & Hum. Servs.*, No. 17-1511, 2021 WL 1736816, at *20 (Fed. Cl. Spec. Mstr. Apr. 2, 2021) (serum sickness reaction to vaccine could manifest within a day of vaccination); *Hock,* 2020 WL 6392770, at *25 (one-day post-vaccination transient reaction).

By contrast, an onset beginning about two weeks after vaccination, as Petitioner alleges, is consistent with his most-favored theory (adaptive immune response mediated by molecular mimicry). The time it would take for the antigens that are immunologically similar to the host's antigens to induce a cross-reactive autoimmune response requires a longer onset. Indeed, Dr. He allowed that a two-week onset under this theory could be reasonable. Tr. at 125. However, because the pneumococcal vaccine has not been shown herein to likely cause RA in the manner alleged, a favorable finding on the fact of onset would not aid Petitioner.

C. *Althen Prong Two*

Because Petitioner cannot meet *Althen* prongs one and three, I need not evaluate his success in preponderantly establishing that Mr. McGuinness's receipt of the pneumococcal vaccine was in fact likely responsible for his injury. But I will briefly address what the evidence and expert reports said about the matter.

Petitioner has placed too much weight on the temporal association between receipt of the vaccine on July 21, 2014, and Petitioner's subsequent onset and diagnosis. Tr. at 51, 74. But (as Dr. Lightfoot noted) this type of temporal association alone cannot establish such a causal relationship—a concept well-understood in the Program. Lightfoot Rep. 7; *Moberly*, 592 F.3d at 1323 ("a proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury.") (quoting *Grant*, 956 F.2d at 1148); *de Bazan*, 539 F.3d at 1352 ("the proximate temporal relationship prong requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact."); *Pafford*, 451 F.3d at 1358 (finding that the Special Master properly required evidence of a temporal relationship between the vaccine and petitioner's injury).

The record evidence of what occurred *between* vaccination and Petitioner's evolving symptoms also does not suggest the vaccine was causal. At most (and assuming Petitioner's initial symptoms did not reflect his RA onset), the pneumococcal vaccine may have triggered the common, post-vaccination "malaise" distinguishable from Petitioner's later, more RA-like symptoms. *See e.g.*, *Hock*, 2020 WL 6392770, at *28; *Monzon*, 2021 WL 2711289, at *20. But what remains missing are pieces of proof that, under Petitioner's causation theory, should have *also* been reflected in the medical records, such as a distinguishable timeframe for which Petitioner felt better between the post-vaccination "malaise" and the beginning of Petitioner's more classic RA symptoms. Alternatively, if (as Respondent seemed to propose) the temporal spectrum of Petitioner's symptoms reveals a related progressive series leading to his subsequent RA diagnosis, then the medical record otherwise offers thin support for the conclusion that the vaccine had anything but a temporal association with the illness.

Admittedly, Petitioner herein was able to show some treater support for the "did cause" prong, in the form of Dr. Wartman's proposal at various points in the late summer of 2014 that the vaccine might be related to Petitioner's symptoms. Ex. 2 at 52, 58. However, I am not bound to accept a treater opinion, especially one that is facially speculative like the present. *Snyder*, 88 Fed. Cl. at 746 n.67. And Dr. Wartman never suggested that the pneumococcal vaccine was linked to Petitioner's later RA *diagnosis*, and no other subsequent doctors, including specialists like rheumatologist Dr. Sikes, suggested an association, even having the benefit of a larger medical history to consider.[19]

[19] I do find that Petitioner successfully rebutted the contention that his history of smoking provided a potential explanation for his seronegative RA. Petitioner smoked two packs of cigarettes a day for thirty years until 1992. Ex. 2 at 61–62. This would help establish an alternative cause to Petitioner *if* his diagnosis had been seropositive RA. But Respondent's own medical literature stated that although smoking is considered a major risk factor for seropositive RA, it "plays a lesser, and perhaps negligible, role in the induction of seronegative RA." Pratt & Isaacs at 653. Respondent also cited to medical literature that the risk factor of smoking declines back to normal after stopping for 10 years. Firestein at 185.

CONCLUSION

A Program entitlement award is only appropriate for claims supported by preponderant evidence. Here, Petitioner has not made such as showing. Petitioner is therefore not entitled to compensation.

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.