In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 16-527V
(to be published)

* *	Chief Special Master Corcoran
GARY SCHILLING, *	
*	Filed: March 17, 2022
Petitioner, *	
v. *	
SECRETARY OF HEALTH *	
AND HUMAN SERVICES, *	
Respondent. *	
* *	

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

Ronalda E. Kosh, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On April 29, 2016, Gary Schilling filed a petition for compensation pursuant to the National Vaccine Injury Compensation Program 42 U.S.C. §§ 300aa-10 to -34 (2012) (the "Vaccine Program").[2] (ECF No. 1) ("Pet."). He alleged that he experienced reactivation of a latent varicella zoster virus ("VZV") infection, leading to shingles and associated encephalomyelitis, due to his receipt of an influenza ("flu") vaccine on October 16, 2013. Pet. at 1.

The parties agreed that the matter could be reasonably resolved on the papers, setting a

[1] This Decision shall be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012)). **This means that 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 Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen 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 whole Decision will be available to the public. *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 Section 300aa of the Act (but will omit the statutory prefix).

schedule for briefing the issue. Joint Status Report, dated Mar. 19, 2021 (ECF No. 52). Having reviewed the record, all expert reports and associated literature, and the briefs filed by both sides, I hereby deny entitlement. Petitioner has not preponderantly demonstrated that the flu vaccine can cause reactivation of a latent VZV infection, or did so in his case.

I. Factual Background

Mr. Schilling was born on July 21, 1951, and was thus 62 years old when he received the flu vaccine on October 16, 2013. Ex. 1 at 1; Ex. 3 at 1; Ex. 11 at 8–10. His prior medical history was significant for coronary artery disease, gout, and deep venous thrombosis. *See* Ex. 11 at 8–58. However, Petitioner alleges he was for the most part in good health at the time. Ex. 12 (Declaration of Gary Schilling) at 1. The records contain no evidence of any out-of-the-ordinary reaction to the vaccination, although Petitioner recalls that the injection site was immediately red and warn, and that he not long thereafter began to feel pain down his legs, plus hand tremors. *Id.* at 3.

Over a month later—on November 22, 2013—Petitioner presented to his primary care physician, Dr. Vjiay Malhotra, complaining of a "new painful rash [in his] right hip and groin area" that he reported began two days earlier. Ex. 11 at 8–10. He did not at this time mention any alleged prior vaccine reaction. Dr. Malhotra noted that Mr. Schilling had a "classical Pox marked rash [on the] right hip and groin area due to herpes [z]oster." *Id.* The assessment was a new onset of herpes zoster and Dr. Malhotra prescribed Famvir, Percocet, and Lidex cream. *Id.* He instructed Petitioner to follow-up in two weeks if needed. *Id.*

On November 26, 2013, Petitioner returned to Dr. Malhotra complaining of trouble voiding and weakness in his legs. Ex. 11 at 5–7. Dr. Malhotra sent him to the emergency department at Windber Medical Center ("Windber") for urological and neurological evaluations. *Id.* While at Windber, Petitioner reported that he was unable to urinate normally since that morning. Ex. 3 at 4–10. The onset of shingles six days earlier was reported. *Id.* Petitioner was diagnosed with urinary retention, a Foley catheter was placed, and he was discharged at his own request. *Id.*

The next day (November 27, 2013) Petitioner called the emergency department at Windber requesting results of lab tests from the day before. Ex. 3 at 10–11. Mr. Schilling now reported increased weakness in both of his legs, stating that he could "barely walk," and had been experiencing constipation for three days. *Id.* As a result, he was advised to return to the emergency department immediately due to the potential of death or disability. *Id.* Petitioner declined, insisting instead that he wait until Dr. Malhotra was available to discuss his symptoms. *Id.*

A nurse from the emergency department called Dr. Malhotra to notify him of Petitioner's current condition, and Dr. Malhotra called Petitioner and advised him to go to the emergency department at Conemaugh Valley Memorial Hospital ("Conemaugh"). Ex. 3 at 11; Ex. 5 at 1. Later

that morning, EMS responded to a call from Petitioner's residence reporting that he was found lying on the floor of the hallway inside the door of his home, having fallen while trying to put on his shoes. Ex. 5 at 1–3. He reported his shingles diagnosis on November 22, 2016, weakness in his legs, and his hospital visit at Windber due to his inability to urinate. *Id.* Petitioner was transported to Conemaugh. *Id.*

Upon admission to Conemaugh on November 27, 2013, Mr. Schilling gave a history of shingles, urinary retention, inconsistent periodic weakness in his lower extremities that increased over time, radicular symptoms, and paresthesias of the forefeet. Ex. 4 at 40–43, 56–58. He also noted difficulty standing and walking but denied pain. *Id.* at 40–43. A physical exam showed an area of dried vesicular lesions on the buttocks that appeared to be resolving, reduced strength in the lower extremities at 4/5, and diminished patellar and ankle reflexes. *Id.* MRIs of the thoracic and lumbar spine revealed increased signal attenuation consistent with a myelitis-type process. *Id.* Following evaluation and a neurology consultation with neurologist Dr. Daniel Orozco, myelitis secondary to varicella zoster infection was suspected. Ex. 4 at 284. A cerebral spinal fluid ("CFS") analysis as well as brain and cervical MRIs were ordered, and Petitioner was started on Acyclovir, a medication specific for herpes virus infections. *Id.* at 16–18, 25, 40–43, 57, 284.

CSF results revealed normal glucose, a white blood cell count of 165, and elevated protein level of 175.9. Ex. 4 at 16–18, 47. The impression was multilevel extensive transverse myelitis ("TM")[3], and additional testing and blood work were ordered. *Id.* at 16–18. The brain MRI showed evidence of diffuse chronic small vessel deep white matter ischemic changes, although changes of demyelination were also a consideration. *Id.* at 63–66. The cervical MRI showed areas of diffuse abnormal signal hyperintensity involving the brainstem and entire cervical cord. *Id.*

On November 29, 2013, petitioner was transferred to the University of Pittsburgh Medical Center ("UPMC") with possible zoster-related myelitis and neuromyelitis optica. Ex. 4 at 25, 160–65; Ex. 6 at 5. Upon admission, his strength and presentation had improved. Ex. 6 at 5. An NCS/EMG was mildly abnormal but did not show any convincing evidence of demyelinating polyneuropathy. *Id.* Despite a test that was negative for varicella zoster virus in the CSF, Petitioner continued to be treated with Acyclovir "given the high clinical suspicion of VZV encephalomyelitis." *Id.* Petitioner's Vitamin B12 deficiency was also deemed as likely contributory to petitioner's myelopathy, although it did not explain the full presentation, and vitamin treatments were also proposed. *Id.*

While at the hospital, attending neurologist Dr. Islam Zaydan noted improved strength in Petitioner's lower extremities and a resolution of diplopia. Ex. 6 at 20–22. Dr. Zaydan also

[3] Transverse myelitis is "myelitis in which the functional effect of the lesions spans the width of the entire cord at a given level." *Transverse Myelitis*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=91212&searchterm=transverse%20myelitis (last visited Mar. 9, 2022).

specifically noted that the flu vaccine Petitioner had received one month before his presenting zoster-like rash was "likely unrelated." *Id.* By December 3, 2013, Petitioner was able to urinate for the first time since his admission. *Id.* at 5–7. The next day, he was discharged to the University of Pittsburgh Rehabilitation Center ("UPRC") with the diagnosis of varicella encephalitis and encephalomyelitis. Ex. 7 at 13.

On admission to UPRC, Mr. Schilling was noted to have a macrocytic anemia that was attributed to B12 deficiency. Ex. 7 at 4–7. Petitioner tolerated more than three hours of daily therapy for at least five to six days a week without any significant interruption. *Id.* His diplopia, fatigue, and bladder function improved, and he was moderately independent for self-care and wheelchair mobility. *Id.* By December 21, 2013, Petitioner was discharged home, and he received in-home care until the middle of January 2014. *Id.* at 5–6; Ex. 8 at 1–27.

On January 31, 2014, Petitioner saw neurologist Dr. Jose Avila for a "post- hospitalization visit for encephalomyelitis, likely secondary to VZV." Ex. 10 at 8–12. He now reported a persistent burning sensation on the soles of his feet that he thought he had been experiencing before hospitalization by that had become worse. *Id.* On exam, he had decreased sensation on the left more than the right and vibration on the soles of his feet, but his strength and motor function was normal throughout, and overall his recovery was deemed excellent. *Id.* Dr. Avila also observed that "[e]ven though the CSF VZV PCR was negative I still believe that VZV encephalomyelitis was the likely Dx [diagnosis] given the clinical scenario." *Id.*

Several months later, on May 12, 2014, petitioner presented to Dr. Natasa Miljkovic, a specialist in physical medicine and rehabilitation, for a follow up after being hospitalized at the spinal cord injury inpatient rehabilitation unit. Ex. 9 at 1–4. Petitioner's exam showed normal strength, and he was deemed "improved with near complete recovery after [v]aricella encephalitis." *Id.* Petitioner also saw Dr. Avila again later that same month. Ex. 10 at 1–6. He again reported a burning pain in his legs and feet, occurring mostly when he used his treadmill or stationary bike. *Id.* His exam showed new hypoesthesia and paresthesia in a length-dependent fashion, suspected to be a progression of the sensory motor polyneuropathy. *Id.* Dr. Avila offered repeat testing and petitioner declined. Dr. Avila deemed Petitioner's recovery excellent overall, although he proposed additional medication for neuropathic symptoms. *Id.*

From this point through the end of the summer of 2016, Petitioner returned to Dr. Malhotra for follow-up visits regarding his myelitis. Ex. 14 at 1–54. Petitioner consistently reported difficulty ambulating and numbness in his lower extremities. *Id.* Petitioner received Vitamin B12 injections at every visit and was encouraged to keep taking Flomax and Neurontin and to continue home rehabilitation. *Id.* It appears that Petitioner continues to receive treatment from Dr. Malhotra. *See,* e.g., Ex. 19.

II. Expert Reports

A. *Petitioner's Expert: Lawrence Steinman, M.D.*

Dr. Steinman submitted two expert reports on behalf of Petitioner. Report, dated Mar. 26, 2017, filed as Ex. 15 (ECF No. 26-1) ("First Steinman Rep."); Report, dated Jan 3, 2018, filed as Ex. 17 (ECF No. 35-1) ("Second Steinman Rep.").

As shown in his CV, Dr. Steinman received his B.A. from Dartmouth College and his M.D. from Harvard Medical School. Ex. 16 at 2 (ECF No. 26-2) (Dr. Steinman's Curriculum Vitae ("Steinman CV")). He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past forty-one years (thirty-seven years at the time of filing). *Id.* Dr. Steinman has also published over four hundred peer-reviewed publications on immunology, neurology, and autoimmune disease. *Id.* at 5–37. He has special expertise in the study of immunology, having several articles published on the issues. *Id.* Dr. Steinman is part of the American Association of Immunologists and the Clinical Immunology Society, with patents in the field and many papers on the topic. *Id.* at 3. Dr. Steinman has insight into many demyelinating issues, caring for "hundreds of adults and children with various forms of inflammatory neuropathy, transverse myelitis, acute disseminated encephalomyelitis (ADEM), neuromyelitis optica (NMO) and multiple sclerosis (MS)." First Steinman Rep. at 2.

First Report

Dr. Steinman at the outset indicated that he felt it possible to establish that the flu vaccine could (a) trigger a VZV reactivation, and then (b) lead to Petitioner's injury (which at the time he characterized as TM), either alone or in concert with Petitioner's shingles.[4] First Steinman Rep. at 2. TM, he explained, is attributable to inflammation in the spinal cord, and is closely related to encephalomyelitis (which features inflammation in the brain *and* spinal cord). *Id.* at 8. Here, Dr. Steinman noted, Mr. Schilling showed signs of encephalomyelitis, but the greatest initial damage was to his spinal cord, leading Dr. Steinman to characterize the injury as TM. *Id.* TM's potential causes remain uncertain, although Dr. Steinman noted it could be triggered by viral infection, "as a complication of syphilis, measles, Lyme disease, and some vaccinations, including those for chickenpox and rabies." *Id.* at 8, 12.[5]

[4] As noted below, Petitioner contends that Dr. Steinman deemed TM and encephalomyelitis as on the same spectrum, distinguished primarily based on the situs of the demyelination, and hence used the terms interchangeably in describing Petitioner's injury—although the totality of the record best supports encephalomyelitis as the proper diagnosis.

[5] Dr. Steinman supported this contention with a direct quote from the same reference—*twice in his first report*. ***See*** First Steinman Rep. at 8, 12; Transverse *Myelitis Fact Sheet*, NATIONAL INSTITUTE OF NEUROLOGICAL DISORDERS AND STROKE, https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Transverse-Myelitis-

In addition, Dr. Steinman noted, medical science had observed that a reactivated VZV infection could result in TM as well as encephalomyelitis. First Steinman Rep. at 27; O. Abramsky & D. Teitelbaum, *The Autoimmune Features of Acute Transverse Myelopathy*, 2 Annals Neurology 36 (1977), filed as Ex. 15, Tab W (ECF No. 26) ("Abramsky"); A. Pohl-Koppe et al., *Myelin Basic Protein Reactive Th2 T Cells are Found in Acute Disseminated Encephalomyelitis*, 91 J. Neuroimmunology 19 (1998), filed as Ex. 15, Tab X (ECF No. 26); K. Van Haren et al., *Serum Autoantibodies to Myelin Peptides Distinguish Acute Disseminated Encephalomyelitis from Relapsing—Remitting Multiple Sclerosis*, 19 Multiple Sclerosis 1726 (2013), filed as Ex. 15, Tab Y (ECF No. 26). This would occur as a result of "sensitization to myelin proteins" caused by the direct VZV reinfection. First Steinman Rep. at 27. The flu vaccine also could itself, directly, cause demyelinating injuries, whether TM or encephalomyelitis, via molecular mimicry (a mechanism discussed below).

Dr. Steinman's theory maintained that the most probable version of the flu vaccine at issue (from 2013–14) "can trigger an immune response to the axons and myelin surrounding the dorsal root ganglion" could activate a latent VZV infection, leading in turn to shingles. First Steinman Rep. at 8. It was likely, he maintained, that Mr. Schilling had a latent, inactive VZV infection "hiding" in his dorsal root ganglion—a myelinated nerve structure including "axons with efferent motor fibers and afferent cell bodies of sensory neurons." *Id.* The flu vaccine would have triggered an adaptive immune response that would in turn produce a cross-reaction against the myelin proteins, but also an initial, innate response that could harm the underlying dorsal root ganglion, through activation of "NF-kB"[6]—am immune system-associated protein complex involved in both gene transcription and cytokine release—which would then reactivate the otherwise-latent VZV. This "sequence of events" would in turn cause TM, and do so in a timeframe consistent with the innate and adaptive immune responses generally. *Id.*

Dr. Steinman then offered a number of items of literature to support the different aspects of his theory. First, he referenced some medical and scientific articles establishing that the dorsal root ganglion is "ensheathed" by myelin, and that the ganglion is also the location where inactive VZV is believed to exist in the body. First Steinman Rep. at 10–11 (references omitted). Second,

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Fact-Sheet#3234_3 (last visited Mar. 9, 2022). It is, unfortunately, this kind of cut-and-paste, sloppy report work that has come to characterize Dr. Steinman's written work in the Program.

[6] NF-kB stands for "nuclear factor kappa light polypeptide gene enhancer in B-cells inhibitor." I. Haralambieva et al, *Transcriptional Signatures of Influenza A/H1N1-Specific IgG Memory-Like B Cell Response in Older Individuals Vaccine*, 34 Vaccine 3993, 3996 (2016), filed as Ex. 15, Tab S (ECF No. 26). The NF-kB pathway is a "signal transduction pathway [that] is an important regulator of innate immunity and inflammation that is triggered by a wide variety of stimuli, including virus infection, tumor necrosis factor alpha (TNF-α), and other cytokines and pathogens." E. Sloan et al., *Varicella-Zoster Virus Inhibition of the NF-κB Pathway During Infection of Human Dendritic Cells: Role for Open Reading Frame 61 as a Modulator of NF-κB Activity*, 86 J. VIROLOGY 1193 (2012), filed as Ex. 15, Tab T (ECF No. 26).

he noted that a variety of nerve-associated molecules can cross-react with antigens in the relevant version of the flu vaccine. *Id.* at 11–12. In support, he made observations about the applicability of the mechanism of molecular mimicry (utilizing the same diagrams he has often included in his reports). *Id.* at 12–13. He also (as he has done many times before) performed online BLAST[7] searches to identify amino acid sequence homology between flu vaccine antigens and nerve protein components he maintained would be the situs of a cross-reactive autoimmune attack, finding enough (and in a sufficient chain of amino acids) for him to conclude that a cross-reaction due to mimicry was likely (even if it could not be established with scientific certainty). *Id.* at 13–24 (references omitted).[8]

Of course, showing homology alone between vaccine antigens and nerve component structures was not enough to establish how vaccination would result in VZV reactivation, so Dr. Steinman shifted to that very question. He proposed that NF-kB was a "master regulator of inflammation." First Steinman Rep. at 24; S. Youssef & L. Steinman, *At Once Harmful and Beneficial: The Dual Properties of NF-kB*, 7 Nature Immunology 901 (2006), filed as Ex. 15, Tab R (ECF No. 26) ("Youssef"). In support he offered a Mayo Clinic study that examined a comparable version of the flu vaccine administered in an elderly population. First Steinman Rep. at 26; *See* I. Haralambieva et al, *Transcriptional Signatures of Influenza A/H1N1-Specific IgG Memory-Like B Cell Response in Older Individuals Vaccine*, 34 Vaccine 3993, 3996 (2016), filed as Ex. 15, Tab S (ECF No. 26) ("Haralambieva").

The intent of Haralambieva was to assess flu vaccine efficacy in the elderly (a group for whom immunosenescence, or an impaired immune response, diminished the protective effect of vaccination) by focusing on the role "influenza-specific memory B cells" would play in encouraging a "rapid secondary immune response to pathogenic challenge" (and thereby to suggest ways in the future of improving vaccines in turn). Haralambieva at 3993. Secondarily, Haralambieva observed that "NF-kB cell signaling" also played a role in the process (by stimulating cytokines and chemokines), and that memory B cell response due to vaccination stimulated this signaling. *Id.* at 3997–98. Haralambieva thus says nothing about NF-kB's pathologic potentiality—but Dr. Steinman nevertheless maintained it revealed a possibility that a

[7] Basic Local Alignment Search Tool ("BLAST") is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, https://blast.ncbi nlm nih.gov/Blast.cgi (last visited Mar. 11, 2022). A BLAST search involves review of an online database to "compare[] nucleotide and protein sequences, to search for a homology between the [] vaccine and [the body's myelin basic protein]." *Montgomery v. Sec'y of Health & Hum. Servs.*, No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019).

[8] This aspect of Dr. Steinman's opinion constituted a third of his entire first report. I do not, however, discuss it in detail, or cite the many items of literature or BLAST search data offered in its support, because (a) it is largely identical (if not precisely) to the majority of written reports Dr. Steinman has offered in the Program over the past several years, and (b) my resolution of this case does not turn on whether sufficient homology between vaccine antigens and nerve structures has been established.

comparable flu vaccine *might* activate this aspect of the immune response generally. First Steinman Rep. at 26.

In addition, Dr. Steinman argued, NF-kB could activate type 1 interferon—a cytokine that plays a role in inflammatory and immunoregulatory processes. First Steinman Rep. at 27; Youssef at 902. The herpes zoster gene, however, appears to maintain latency in the body in part by encouraging down-regulation of a specific cytokine important to "the type 1 interferon pathway." First Steinman Rep. at 27; E. Sloan et al., *Varicella-Zoster Virus Inhibition of the NF-κB Pathway During Infection of Human Dendritic Cells: Role for Open Reading Frame 61 as a Modulator of NF-κB Activity*, 86 J. Virology 1193 (2012), filed as Ex. 15, Tab T (ECF No. 26) ("Sloan").

As a result, the stimulation (via vaccination) of the NF-kB complex could have "tipped the balance" by eliminating the latency effects of the VZV, thereby causing its reactivation. First Steinman Rep. at 27; N. El Mjiyad et al., *Varicella-Zoster Virus Modulates NF-kB Recruitment on Selected Cellular Promoters,* 81 J. Virology 23, 13092–104 (2007), filed as Ex. 15, Tab U (ECF No. 26) ("El Mjiyad"). But although El Mjiyad did observe from its study results the manner in which active VZV infections might defeat the immune response mounted against them (specifically by inhibition of NF-kB binding), the study's authors seem focused *not* on how VZV's latency is maintained (or potentially undone by other immune stimuli), but instead how to control "infection progression" during a VZV infection's initial stages—noting that the interference with NF-kB processes particularly inhibited the cytokine response to skin lesions associated with herpes. El Mjiyad at 13092, 13102. El Mjiyad thus does *not* stand for the conclusion suggested by Dr. Steinman, even if it did show some relationship between VZV and NF-kB.

Dr. Steinman concluded his first report by addressing the remaining two prongs under the test set forth by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.,* 418 F.3d 1274, 1278 (Fed. Cir. 2005). He mainained the second, "did cause" prong was met, although in so concluding Dr. Steinman mainly relied on the timeframe in which Petitioner's injuries arose, along with the fact that he could identify no other causal explanations. First Steinman Rep. at 27–28. The third prong was also satisfied, Dr. Steinman opined, because onset of neurologic symptoms within five weeks of vaccination (with shingles first presenting approximately four to five weeks after vaccination) was a "well accepted" timeframe for an aberrant immune response. *Id.* at 28.[9]

[9] The sole independent support Dr. Steinman offered to defend this onset timeframe was an article frequently cited in Program cases, but which is specific to GBS and says nothing about the expected timeframe for encephalomyelitis secondary to reactivated VZV. *See* L. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program*, *United States, 1976–77*, 110 J. Epidemiology 105 (1979), filed as Ex. 15, Tab Y (ECF No. 26).

Second Report

Dr. Steinman's second report attempted to address issues brought up by Respondent's expert, with a particular focus on her arguments regarding the sufficiency of molecular mimicry as a causal theory. *See generally* Second Steinman Rep. at 1–3. He began by observing that autoimmune diseases were the product of a confluence of different factors, including "genetic susceptibility, host environment, environmental factors, etc." *Id.* at 1. As a result, the "commonality of mimics" in nature could not by itself undermine his causation theory, since an individual's susceptibility to an autoimmune cross-reaction due to mimicry would in turn be impacted by factors specific to that person. The "value of identifying linear sequential mimics" remained, in that it helped establish a first step toward an autoimmune process that might be due to a vaccine. *Id.*

Next, Dr. Steinman defended the sufficiency of his proposed specific amino acid sequences. He explained the criteria he uses in conducting BLAST searches, noting that he derived them from reliable scientific guidelines as well as his own immunologic expertise. Second Steinman Rep. at 1–2. He also emphasized that reliable literature established that homologic sequences in which five of 12 amino acids, or four of 11, were shared by antigen and host structure had been found sufficient to trigger experimental encephalomyelitis—even without specifically-identical ordinal sequences. *Id.* at 2. And he denied that shared chains of three amino acids in sequence were minimally necessary to spark a cross-reaction between autoantibodies and self. *Id.* at 2–3.

Regarding Dr. Collins's argument that the Petitioner had offered little in the way of literature showing that the proposed autoimmune cross-reaction would occur outside of an animal model and to a real person, Dr. Steinman objected that any scientific study sufficient to prove the contention would require unethical experimentation that would not likely be approved or permitted. Second Steinman Rep. at 3. Otherwise, however, Dr. Steinman's supplemental report did not go into further detail in defending his attempt to link the flu vaccine to VZV reactivation—whether or not the vaccine *also* contributed, directly or otherwise, to Petitioner's encephalomyelitis. Rather, he simply repeated, in block-quote form, his prior breakdown of the causal sequence he had outlined in his first report, along with a second reiteration of his conclusions that his showing satisfied the *Althen* prongs. *Id*. at 3–4.

B. *Respondent's Expert: Kathleen L. Collins, M.D., Ph.D.*

Dr. Collins submitted two expert reports on behalf of Petitioner. Report, dated as July 15, 2017, filed as Ex. A (ECF No. 31-1) ("First Collins Rep."); Report, dated as Feb. 20, 2018, filed as Ex. U (ECF No. 37) ("Second Collins Rep.").

As shown in her CV, Dr. Collins received her B.A. from Wellesley College and her M.D. and Ph.D. from John Hopkins, University School of Medicine. Ex. B at 2 (ECF No. 31-2). (Dr. Collin's Curriculum Vitae ("Collins CV")). She then had postdoctoral training in Internal Medicine at the Brigham and Women's Hospital, Infectious Disease Clinical Fellow, Research Fellow at Harvard University, and a Postdoctoral Fellowship at the Massachusetts Institute of Technology. *Id.* Dr. Collins was a professor of Microbiology and Immunology at the University of Michigan's Medical School. *Id.* She has conducted several studies on HIV, with publication of additional studies pending. *Id.* at 5–7. She also currently has graduate students and postdoctoral fellows she is training. *Id.* at 10. Dr. Collins has given several presentations on the topic and similar topics. *Id.* at 14–17. She has published several articles and reviews/book chapters on the topics of HIV, treatment of HIV, infection, and reactivation. *Id.* at 18–23.

First Report

Dr. Collins begins her opinion with a review of the pertinent medical facts in the case. First Collins Rep. at 1–2. She then offered her own explanations of TM, VZV, and other medical concepts relevant herein. *Id.* at 2–3. In particular, she noted that VZV causes shingles secondarily, after its reactivation in the body (where the virus, subsequent to an initial infection, "hides" in the sensory nerves). *Id.* at 3. Older age is an important risk factor, with reactivation usually deemed "influenced by age-related loss of immune response that normally maintains the virus in a latent state." *Id*; M. Albrecht, *Clinical Manifestations of Varicella-Zoster Virus Infection: Herpes Zoster*, UpToDate (June 7, 2016) (M. Hirsch & J. Mitty eds.), filed as Ex. F (ECF No. 31-6) ("Albrecht"). Neurologic complications are common, with 10–15 percent of patients also developing postherpetic neuralgia. *Id.* at 3. And of particular relevance herein, TM often developed secondarily to an VZV reactivation days to weeks following rash onset. Albrecht at 4–5; O. Devinsky et al., *Herpes Zoster Myelitis*, 114 Brain 1181 (1991), filed as Ex. D (ECF No. 31-4) (in study of 13 people with VZV and myelitis, leg weakness and sensory abnormalities were common presenting symptoms).

After summarizing Dr. Steinman's opinion, Dr. Collins outlined her several bases for deeming the theory unpersuasive and medically/scientifically reliable. First Collins Rep. at 4. First, she noted that although molecular mimicry as an explanatory mechanism for certain autoimmune disease processes had reliable support when invoked in connection with some diseases, "it is not sufficient to show that amino acids are similar between two proteins" to establish that a pathologic process was likely, since homologies were common in nature but did not inerrantly lead to disease. *Id.*; Committee to Review Adverse Effects of Vaccines, Board on Population Health and Public Health Practice, ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY 61 (K. Stratton et al. eds.) (2012), filed as Ex. M (ECF No. 32-3) ("Stratton"). Indeed, autoantibodies can be found in healthy individuals, and could also be the product of a disease *process* rather than the cause. First Collins Rep. at 4; N. Rose, *Overview of Autoimmunity*, UpToDate (May 22, 2015) (P. Schur & P.

Romain eds.), filed as Ex. G (ECF No. 31-7). Additional evidence (for example, of "demonstration of local binding of antibody with activation of the complement cascade, activation of the appropriate co-stimulatory T-cell signals and cytokines, and/or involvement of other pathogenic effector mechanisms in a biologically relevant tissue site") was required to deem molecular mimicry likely causal. First Collins Rep.at 4; Stratton at 61.

More specifically, Dr. Collins questioned whether Dr. Steinman's report established sufficient amino acid chain homology between flu vaccine components and elements of myelin basic protein. First Collins Rep. at 4. He had, in her view, merely established homology based on five of eleven amino acids, but had also allowed that disease incidence would diminish with greater difference in sequence or identity. *Id.* at 5. In her reading, the experimental models that supported some of Dr. Steinman's contentions actually showed "the importance of having a relatively high rate of consecutive, identical amino acids," along with total identity—and regardless, Dr. Steinman's homology contentions were not specific to circumstances involving reactivation of VZV. *Id.* Dr. Collins also noted that the BLAST searches Dr. Steinman had performed to link flu vaccine antigens to nerve components did not even meet his own standards for what constituted sufficient homology, while (again) not also demonstrating any reliable science showing that the homologous antigens were capable of demonstrably causing demyelinating disease. *Id.*

Second, Dr. Collins challenged Dr. Steinman's assertions that the immune response to the flu vaccine could result in reactivation of VZV, manifesting initially as shingles. First Collins Rep. at 6. The literature Dr. Steinman relied upon for this part of his opinion, like Haralambieva, did not support his argument, since all it did was analyze the effect of B cell responses to a vaccine antigen—not show that the flu vaccine could cause VZV reactivation. Indeed, Dr. Collins found no independent support for this contention in her own research in preparing her opinion. *Id.* The idea that VZV latency involved downregulation of type 1 interferon (thus allowing for the possibility that *upregulation* of that cytokine due to stimulation of NF-kB could cause reactivation) was also deficient support for causation, she maintained, noting that (a) the article referenced by Dr. Steinman for this proposition, Sloan, involved a different herpes zoster gene than he had cited, and (b) there was a lack of reliable proof that mere activation of the relevant interferon pathways was likely to reverse latency. *Id.* The article offered for that secondary proposition, El Mjiyad, was specific in observing that the VZV's impact of NF-kB activation helped the initial infectious process in replicating in the skin—*not* that latency itself (which would occur after the suppression of the first infectious process) was later reversible through vaccine stimulation of NF-kB activation. *Id.*

Dr. Collins deemed the other aspects of Dr. Steinman's theory to be unreliable or evidentiarily deficient. She noted, for example, that items of literature like Abramsky did not address how VZV reactivation could sensitize myelin proteins sufficient to cause some kind of demyelinating disease. First Collins Rep. at 6. At most, Dr. Steinman had acknowledged that VZV

reactivation *itself* was associated with TM, but was not also shown to involve immune stimulation due to a vaccine as co-factor. *Id.* at 7. Nothing credible, in her opinion, had been offered to establish that a flu vaccine would act as the "lynchpin" resulting in both VZV reactivation and subsequent central nervous system neuropathies. *Id.* Rather, the well-understood association between shingles and TM and/or encephalomyelitis was a far more persuasive explanation for Petitioner's injury than the vaccine coincidentally received prior to onset. *Id.* at 7–8.

Finally, Dr. Collins proposed alternative factors that she characterized as more likely causal than vaccination under the circumstances. First Collins Rep. at 7. Petitioner's age, for example, was itself explanatory, since "Zoster incidence increased 10-fold when comparing children less than 10 with persons aged 80 to 89 years." *Id.*; R. E. Hope-Simpson, *Postherpetic Neuralgia*, 25 J. Royal College General Practitioners 571 (1975), filed as Ex. P (ECF No. 32-6) ("Hope-Simpson"). Indeed, other literature suggested that approximately 50 percent of people who live to 85 years will have an episode. K. Schmader, *Herpes Zoster in Older Adults*, 32 CLINICAL INFECTIOUS DISEASES 1481 (2001), filed as Ex. Q (ECF No. 32-7) ("Schmader"). Further, the article noted that "[t]he incidence of HZ increases sharply among patients aged ~50–60 years and continues an upward course in the decades >60 years." Schmader at 1482. This is crucial since Petitioner was 62 when he experienced the relevant case of shingles.

Another possible contributing factor, in Dr. Collins's view, was the Vitamin B12 deficiency that the record revealed Petitioner was experiencing. First Collins Rep. at 8. A B12 deficiency can result in neurologic problems because it can impact myelin formation. *Id.* A neuropathy attributable to such a vitamin deficiency can manifest with leg tingling or burning—as it did with Petitioner. *Id;* S. Schrier, *Etiology and Clinical Manifestations of Vitamin B12 and Folate Deficiency*, UpToDate (Dec. 17, 2014) (W. Mentzer & J. Timauer eds.), filed as Ex. J (ECF No. 31-10). And in fact, Petitioner's doctors included B12 injections as part of his treatment, in order to maintain normal levels. Ex. 6 at 6.

Second Report

Dr. Collins second report reacted to Dr. Steinman's objections to her first report. Second Collins Rep. at 2. She repeated prior contentions about Petitioner's history and the nature of shingles, emphasizing that (a) the record corroborated that he had experienced VZV reactivation, (b) TM was a known reactivation complication (meaning that Petitioner's shingles best explained his neuropathic symptoms), and (c) that his established Vitamin B12 deficiency was a more reliable and credible cause of these same symptoms than vaccination—especially given the lack of other scientific or medical evidence linking the flu vaccine to shingles or its secondary symptoms. *Id.* at 2–3.

Dr. Collins then devoted several pages of her second report to disputing Dr. Steinman's

arguments about the sufficiency of evidence relied upon for his molecular mimicry causation theory. Dr. Steinman's contention about the multi-factorial nature of autoimmune disease (which in turn, he implied, made it difficult to study or identify causal factors) was undercut by some large-scale epidemiologic studies showing no association between, say, the flu vaccine and TM. Second Collins Rep. at 2; R. Baxter et al., *Acute Demyelinating Events Following Vaccines: A Case-Centered Analysis*, 63 CLINICAL INFECTIOUS DISEASES 1456 (2016), filed as Ex. E (ECF No. 31-5) ("Baxter").[10] Thus, such studies had "sufficient power to tease out individual variables" that Dr. Steinman proposed prevented bigger-picture conclusions.

Other literature further substantiated the conclusion that VZV reactivation, or some other kind of infectious process, best explained subsequent neuropathic symptoms, without a "need for concomitant vaccination" as an explanatory factor. One, a retrospective analysis, observed that 21 percent of 33 TM cases had experienced infections within a month prior of neurological symptoms, most being upper respiratory or gastroenteritis. D. Jeffery et al., *Transverse Myelitis: Retrospective Analysis of 33 Cases, with Differentiation of Cases Associated with Multiple Sclerosis and Parainfectious Events*, 50 ARCH. NEUROLOGY 532, 534 (1993), filed as Ex. W (ECF No. 37-2) ("Jeffrey"). Jeffrey also noted prior literature (not filed in this case) which observed that 37 percent of 52 TM patients reported a preexisting infection. Jeffrey at 532. In a more on-point (if older) item of literature, nine cases of TM were observed to have developed within 5–16 days after manifestation of a varicella rash. J. McCarthy & J. Amer, *Postvaricella Acute Transverse Myelitis: A Case Presentation and Review of the Literature*, 62 Pediatrics 202, 202, 203 (1978), filed as Ex. Y (ECF No. 37-4) ("J. McCarthy & J. Amer").

Dr. Collins concluded with the argument that Dr. Steinman still had not established sufficient amino acid homology to establish that a cross-reaction based on mimicry was likely. Second Collins Rep. at 3–5. She questioned whether Dr. Steinman's own literature supported a likelihood of an autoimmune attack based on only four common amino acids out of an 11-chain sequence, noting as well that true sequential homology was dramatically less likely for fewer than four consecutive identical amino acids. *Id.* at 3–4. And she emphasized the fact that because millions received the flu vaccine each year—with the same homology between vaccine components and self-nerve protein components deemed sufficient for a cross-reaction present in every case—the very "experiment" of vaccination itself disproved the likelihood of vaccines being causal of the injuries at issue, since injury happened so rarely. *Id.* at 5.

[10] Baxter, Dr. Collins noted, considered "18.9 million trivalent and quadrivalent inactivated influenza vaccines as well as monovalent H1N1 influenza vaccinations given from 1 January 2007 until 31 December 2012" out of 64 million vaccine doses in total, but observed only 67 cases of TM within nine months of vaccination—with no frequency increase whether the exposure window was 5–28 days or 2–42 days. Second Collins Rep. at 2; Baxter at 1458. Looking at the results of the flu vaccine at TM there were 38 cases studied who received the vaccine compared with 969,316 of the comparison risk set, the vaccinated had a 7.9% exposure interval and the comparison set a 6.2% exposure interval. Baxter at 1458. This returned a *P* Value of .98, in all returning an adjusted odds ratio of one. *Id.*

III. Procedural History

The claim was initiated in April 2016, with medical records being filed several times throughout the case. Respondent filed his Rule 4(c) Report on November 9, 2016. ECF No. 18. The parties next each filed two expert reports, as explained, and the case was then reassigned to me on March 2, 2021. Order, dated Mar. 2, 2021 (ECF No. 50). After a status conference the parties were ordered to file a joint status report to set deadlines for briefing a ruling on the record. Scheduling Order, dated Mar. 12, 2021. After the filing of the Joint Status Report (ECF No. 52) the schedule was set. Scheduling Order, dated Mar. 19, 2021. Both parties filed briefs in support of their positions. Petitioner's Motion, dated May 25, 2021 (ECF No. 54) ("Mot."); Response, dated Sept. 9, 2021 (ECF No. 56) ("Opp."); Reply, dated Sept. 23, 2021 (ECF No. 57). This case is now ripe for resolution.

IV. Parties' Arguments

A. *Petitioner*

Mr. Schilling maintains that he has demonstrated that the flu vaccine he received caused a reactivation of a latent VZV infection, leading to shingles plus encephalomyelitis (whether or not the vaccine directly caused the neurologic symptoms *as well as* the reactivation, or merely caused reactivation *leading* to those symptoms).[11] Mot. at 20–21.

Dr. Steinman has proposed that "an adaptive immune response to myelin proteins and proteins on the [nerve] axons, involving molecular mimicry, resulted in inflammation, myelin destruction and axonal injury." Mot. at 20-21; First Steinman Rep. at 8. This autoimmune cross-reaction against the nerves therein caused the inactive HSV to manifest clinical symptoms, leading to other CNS injury. He also maintains that the flu vaccine has the capacity to trigger encephalomyelitis *independent* of VZV reactivation (First Steinman Rep. at 29), although Petitioner ultimately seems to embrace the conclusion that his shingles was likely related in some form to *both* vaccination and his subsequently-diagnosed encephalomyelitis.

In support of his theory, Dr. Steinman observed that animal models have shown reactivity between foreign antigens and self structures occurring due to the existence of amino acid homology. Mot. at 23. Such homology is not, Dr. Steinman acknowledged, sufficient for a cross-reaction to occur—but Dr. Steinman's BLAST searches established sufficient homology between vaccine components and nerve structures to increase its likelihood. *Id.* at 24; First Steinman Rep. at 16–23. In addition, the homologic nerve structures (myelin and axonal molecules) are within

[11] Although Dr. Steinman's reports seem to define Petitioner's injury as TM, Respondent notes that Petitioner does not ultimately maintain he suffered from TM. Opp. at 12. Petitioner maintained in his brief that Dr. Steinman did not distinguish between TM and encephalomyelitis—even though the latter (*not* TM) was the diagnosis Petitioner ultimately received and is thus the more precise characterization for Petitioner's post-shingles injury. Mot. at 20 n.9.

the spinal cord, so demyelination centered therein is capable of causing disease. Mot. at 25–26.

Petitioner also maintains he has established the "did cause" *Althen* prong. Prior to vaccination he was a "generally healthy adult male," but immediately post-vaccination experienced aching and pain in his legs along with tremors in his hands. Mot. at 30. In November 2013, Petitioner subsequently experienced "a painful rash in the right groin, hip and buttocks, which was diagnosed as shingles." *Id.* at 31; Ex. 11 at 8–10; Ex. 12 at 2. Then, roughly six days after that, he had neurological complaints with "urinary retention and bilateral leg weakness, and was ultimately diagnosed with encephalomyelitis." Mot. at 30; Ex. 3 at 4–7; Ex. 11 at 5–7. And Respondent had not in Petitioner's estimation established VZV reactivation *by itself* as an alternative explanation for Petitioner's injury (with vaccination being a coincidental factor). Mot. at 35. It was simply more likely that the flu vaccine (a) triggered VZV reactivation, and then (b) *either* separately caused TM/encephalomyelitis directly, *or* indirectly was causal due to reactivation (since either process could cause that kind of neurologic injury). *Id.* at 36–37.

Finally, Petitioner also argued the timing element was met. His neurological symptoms appeared within six weeks of the vaccine, which Petitioner deemed "an appropriate timeframe for an immune-mediated process" that could not be attributed to coincidence. Mot. at 32, 33–35.

On Reply, Petitioner reiterated the contention that Dr. Steinman had established that a limited amount of specific homology was sufficient for a cross-reaction. Reply at 3-4; First Steinman Rep. at 13–14. Petitioner also attacked Respondent's reliance on the Baxter study, arguing that it only shows statistical associations or lack thereof, without ruling out the reliability of mechanisms of disease that might be experienced in the limited number of individuals in the population who are injured by a vaccine. Reply at 5. Indeed, Respondent's expert conceded aspects of Petitioner's theory, mostly disputing whether sufficient homology had been demonstrated. *Id.* at 6.

B. *Respondent*

Respondent contests Petitioner's success in meeting his causation showing. Regarding the "can cause" prong, he maintains that "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." Opp. at 13 (*citing McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019)). And in this case, as Dr. Collins noted, there was no evidence of a reliable connection between the flu vaccine and varicella reactivation leading to TM or encephalomyelitis. Opp. at 13. Dr. Collins also persuasively established that Dr. Steinman's homologies were insufficient based on *his own data,* with some items of literature showing that the sequence numbers proposed by Dr. Steinman did not in fact lead to cross-reactivity. *Id.* at 13–

14. In addition, as stated by Dr. Collins, the "mere presence of antibodies may be the result, not the cause, of the disease process," and thus even when autoantibodies were produced due to molecular mimicry this did not mean the antibodies in question would always be causal of subsequent disease. *Id.* at 14.

Respondent also attacked Dr. Steinman's reliance on NF-kB protein complex/transcription factor, and its ability to impact inflammatory pathways, as something that could be stimulated by vaccination and thereby encourage VZV reactivation, arguing such contentions lacked evidentiary support. Opp. at 15. Dr. Steinman relied on Sloan for this proposition, but, as Dr. Collins pointed out, the HSV gene discussed therein (which Dr. Steinman noted helped maintain latency—and thus, in theory, could have its effects reversed by interaction with a vaccine-instigated immune process) is not equivalent to the one at issue *in this case*, and nothing else connected vaccination to reversal of HSV latency. *Id.* at 15–16. Although Petitioner need not have medical literature support to prevail, Respondent noted, whether "a theory is or is not supported by peer-reviewed medical literature can be a factor in assessing the credibility and reliability of expert witnesses." *Id.* at 16; *Perreira v. Sec'y of Health & Hum. Servs.*, 33 F.3d 1375, 1376 (Fed. Cir. 1994). Here, Dr. Steinman's theory lost credibility due to an absence of sufficient reliable support in the medical or scientific literature. Opp. at 17.

Respondent further maintained that the second *Althen* prong was not satisfied, questioning the very logic of associating the flu vaccine to VZV reactivation and subsequent symptoms when those symptoms could fully be explained by the fact of Petitioner's bout with shingles. Opp. at 18. Moreover, although Petitioner received his flu vaccine roughly a month prior to the rash appearing on his body, none of his treating physicians connected it as the cause, with one (Dr. Zayden) explicitly deeming the vaccine as "likely unrelated." *Id.*; Ex. 4 at 20–22. Instead, Petitioner's treating physicians associated the zoster infection/shingles as the best explanation of subsequent symptoms, given that neurologic illnesses like TM are a known complication. Opp. at 18–19. VZV reactivation thus adequately explained Petitioner's symptoms. *Id.* Reliable medical literature in fact associated reactivated zoster virus with neurological problems in individuals over 50 years of age. *Id.* at 20; Hope-Simpson at Fig. 1. Since Petitioner was 62 years old at the time of reactivation, he clearly fell within this at-risk category. Opp. at 20.

Finally, Respondent denied that Petitioner had adequately shown that the timeframe in which Petitioner experienced VZV reactivation and subsequent symptoms was medically acceptable. Mot. at 21–22. The timing of onset was no more than coincidental with vaccination. *Id.*

V. Applicable Law

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, petitioners must prove that: (1) they suffered an injury falling within the Vaccine Injury Table (i.e., a "Table Injury"); or (2) they suffered an injury actually caused by a vaccine (i.e., 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). 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., Inc. v. United States*, 6 Cl. Ct. 476, 486 (1984) (explaining that mere conjecture or speculation is insufficient under a preponderance standard). On one hand, proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). But on the other hand, 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 *Althen* decision: "(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." Each *Althen* prong requires a different showing and is discussed in turn along with the parties' arguments and my findings.

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.

However, the Federal Circuit has *repeatedly* stated that the first prong requires a preponderant evidentiary showing. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1360 (Fed. Cir. 2019) ("[w]e have consistently rejected theories that the vaccine only "likely caused" the injury and reiterated that a "plausible" or "possible" causal theory does not satisfy the standard"); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010). This is consistent with the petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted). If a claimant must *overall* meet the preponderance standard, it is logical that they be required also to meet each individual prong with the same degree of evidentiary showing (even if the *type* of evidence offered for each is different).

Petitioners may offer a variety of individual items of evidence in support of the first *Althen* prong, and are not obligated to 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). No one "type" of evidence is required. 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." *Andreu,* 569 F.3d at 1380. Nevertheless, even though "scientific certainty" is not required to prevail, the individual items of proof offered for the "can cause" prong must *each* reflect or arise from "reputable" or "sound and reliable" medical science. *Boatmon,* 941 F.3d at 1359-60.

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).

However, medical records and/or statements of a treating physician's views 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 also 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) (stating it is 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, 356–57 (2011), *aff'd without opinion*, 475 F. App'x. 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 also coincide 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.*, 2013 WL 1896173 (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 review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Law Governing Analysis of Fact Evidence*

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 are likely 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 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 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. *Evaluation of Expert Opinions*

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 Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors 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*., 844 F.3d 1322, 1328 (Fed. Cir. 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. Appx. 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").

E. *Disposition of Case Without Hearing*

I am resolving this claim on the papers, rather than by holding a hearing, in accordance with the parties' expressed preference. ECF No. 91. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.,* No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld).

ANALYSIS

I. Overview of Shingles

Although it is not disputed in this matter that Mr. Schilling experienced shingles, with subsequent neurologic symptoms (that in turn may or may not have been related to vaccination, depending on the version of Petitioner's theory that is considered), some discussion of the primary injury, and what it entails, is necessary to properly analyze the claim (since Petitioner argues *either* that the flu vaccine caused his shingles, or that it interacted with it to result in the secondary myelitis symptoms he experienced).

An initial VZV infection, commonly called "chickenpox," is caused "by human herpesvirus 3, usually affecting children, spread by direct contact or the respiratory route via droplet nuclei, and characterized by the appearance on the skin and mucous membranes of successive crops of typical pruritic vesicular lesions that are easily broken and become scabbed." Chickenpox, DORLAND'S MEDICAL DICTIONARY ONLINE https://www.dorlandsonline.com/dorland/definition?id=9096 (last visited Feb. 23, 2022). Initial symptoms occur within a 10–21-day incubation period, with fever, malaise, and an itchy rash. *Atlas of Pediatric Physical Diagnosis* 444 (5th ed. 2007). A VZV infection is usually self-limiting and mild, although severe and potentially-fatal complications may arise, such as secondary bacterial infections or neurologic impacts (encephalitis). *Id.* Varicella is especially dangerous to children and the immune-compromised. One form, "breakthrough" varicella, occurs in person who received the varicella vaccine but subsequently (42 days or more post-vaccination) incur a wild virus infection, and features a slightly different kind of presenting rash. *Infectious Diseases (Varicella-Zoster Virus)* in Nelson Textbook of Pediatrics 1581 (R. Kliegman et al., 20th ed. 2016) ("Nelson").

A varicella infection is commonly diagnosed from clinical indicia, such as the presence of

the rash commonly associated with it. Nelson at 1584. However, some laboratory testing can also confirm its presence. Evaluation of the virus's presence from direct testing of skin lesions/vesicles is most common, although strain identification can also assist in distinguishing the wild virus from vaccine-associated strains. *Id.* It is commonly treated with the antiviral drug Acyclovir. *Id.*

One feature of VZV that distinguishes it from other viral infections is its capacity for latency and subsequent reactivation. *Pearson v. Sec'y of Health & Human Servs.,* No. 16-9V, 2019 WL 3852633, at *15 (Fed. Cl. Spec. Mstr. 2019) ("varicella zoster reactivation is a relatively common ailment—approximately 1 million new cases are diagnosed annually in the United States, and 90% of these patients are immunocompetent"). After exposure to the virus in childhood, the immune system is usually able to eliminate it, but it often remains dormant/latent in two parts of the central nervous system: the ganglia adjacent to the spinal cord or base of the skull. Phillip S. LaRussa & Mona Marin, *Textbook of Pediatrics: Chapter 253 Varicella-Zoster Virus* 1579 (20th ed. 2016). Thereafter, the latent virus can reactivate in adulthood, causing a varicella zoster infection, or "shingles," which is characterized by a painful localized rash. *Id.* Although shingles usually resolves in a few weeks, it can have associated secondary symptoms, or lead in some cases to ongoing nerve pain or neuralgia. *Pearson,* 2019 WL 3852633, at *15. Only those who previously were infected with VZV can experience shingles, and it most commonly afflicts those over 50 (thus underscoring the fact that the virus's latency can be decades-long).

It remains unknown to medical science *why* VZV remains latent for such lengthy periods of time. However, it has been determined that during dormancy, infected nerve cells continue to manufacture viral proteins, suggesting that rather than true latency, the dormant period might better be characterized as a chronic, if extremely low-level, active infection. B. Grinde, *Herpesviruses: Latency and Reactivation—Viral Strategies and Host Response*, J. Oral Microbiology (2013) (available at https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3809354/). The immune system otherwise suppresses this ongoing, sub-clinical infectious process—consistent with the fact that shingles is more common in those whose immune systems are compromised (whether due to aging, receipt of medical treatments known to be immunosuppressive, or psychologic stress). *Shingles*, Mayo Clinic (Sept. 17, 2021), https://www.mayoclinic.org/diseases-conditions/shingles/symptoms-causes/syc-20353054 (last visited Mar. 14, 2022).

There is a Table claim for varicella vaccine-induced VZV reactivation, but not for reactivation attributable to the flu vaccine. *See* 42 C.F.R. § 100.3(a)(X)(C). I am aware of no cases in which a petitioner has successfully established that a vaccine *other* than varicella itself caused a VZV reactivation—although this issue has previously arisen. In an almost identical case, for example, a claimant alleged that the flu vaccine caused her TM. *Forrest v. Sec'y of Health & Hum. Servs.*, No. 14-1046V, 2019 WL 925495, at *1 (Fed. CL. Spec. Mstr. Jan. 28, 2019). Entitlement in this matter was denied based upon a timing issue, but also (specifically relevant in this case) because it appeared VZV reactivation better explained the petitioner's TM. *Id.* at *2, 9–10. In a

similar case, a petitioner alleged that the Tetanus-diphtheria-acellular pertussis ("Tdap") vaccine caused him to develop meningoencephalitis.[12] *Dunn v. Sec'y of Health & Hum. Servs.*, No. 16-1506V, 2020 WL 1243237, at *1 (Fed. Cl. Spec. Mstr. Feb. 19, 2020). Respondent, however, argued that a VZV reactivation was instead the cause of petitioner's injury, and the special master so found. *Id.*

II. Petitioner Has Not Carried His Burden of Proof[13]

A. *Althen Prong One*

This matter represents yet another Vaccine Program case in which Dr. Steinman was retained by a petitioner to offer up what appears on paper to be an extensive, science-intensive opinion on the subject of molecular mimicry—a reliable mechanistic theory for how autoimmunity *can* occur in some circumstances. *See generally Barone v. Sec'y of Health & Hum. Servs.*, No. 11-707V, 2014 WL 6834557, at *8–9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014). To that end, he has performed what he has previously deemed in other cases "in silica" experiments[14] (e.g., computer database research) to identify amino acid sequences shared in common between the antigens of the flu vaccine and a proposed situs for a cross-reactive autoimmune attack, in an effort to establish how a vaccine might instigate disease. Indeed, both experts devoted a not-insignificant portion of their reports to debating whether Dr. Steinman showed *enough* homology for a cross reaction to have occurred.[15]

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[12] Meningoencephalitis is also called cerebromeningitis and/or encephalomeningitis, being the "inflammation of the brain and meninges." *Meningoencephalitis*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=30351&searchterm=meningoencephalitis (last visited Mar. 14, 2022).

[13] My analysis considers only the *Althen* prongs that mainly inform my Decision, rather than all three. Regarding the timeframe prong, I note that Petitioner's onset of both VZV reactivation and subsequent initial neurologic symptoms occurred in the timeframe deemed medically acceptable by Dr. Steinman, and this issue was largely unrebutted by Dr. Collins. Dr. Steinman, however, relied in part upon Schonberger for this aspect of his opinion—an item of literature often invoked to defend the timeframe for post-flu vaccine *GBS* (and hence not directly relevant herein), although onset of neurologic symptoms in the third week of November was still within the six-week timeframe often deemed medically acceptable for demyelinating injuries. But acceptable onset for TM/encephalomyelitis after vaccination is different depending on whether the VZV reactivation is taken into account—for if it is, and if it is deemed the first "step" toward Petitioner's neurologic symptoms, then Dr. Steinman's theory of an innate stimulation of the NF-kB pathway to causing reactivation should have occurred far sooner than over 30 days post-vaccination, since an innate response to vaccination does not take weeks.

I do not attempt to resolve the above, however, because the claim fails on the other two *Althen* prongs as well as Respondent's successful "factor unrelated" showing.

[14] *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *6 n.6 (Fed. Cl. Spec. Mstr. Feb. 4, 2022).

[15] On this subject, Dr. Collins raised reasonable questions about whether Dr. Steinman's own literature fully supported his view that merely four common amino acids contained in a larger, eleven amino-acid chain—whether or not antigen and self component chains had any sequential identity as well, or sufficient amounts of it—was enough to spark,

However, this "battle of the experts" missed the forest for the trees, so to speak—and not just because, as Respondent notes (and which I have previously observed, now many times), establishing the potentiality of molecular mimicry *alone* is not enough to preponderantly establish causation. Opp. at 13; *McKown v. Sec'y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019). Rather, it was because this case involved the undisputed fact that Petitioner *first* developed a VZV reactivation, which treaters reasonably associated with Petitioner's subsequent myelitis symptoms. Ex. 4 at 25; 284; Ex. 6 at 5; Ex. 7 at 13; Ex. 10 at 8–12. Accordingly, the causation theory most pertinent to the circumstances of this case was whether the flu vaccine could *itself* cause VZV reactivation.

Petitioner made some effort to meet this challenge. Dr. Steinman's report does (albeit briefly—in the space of four to five pages of a 29-page report) attempt to opine as to how VZV reactivation might be triggered by vaccination. But ultimately the theory he offered on this subject was quite thin and not preponderantly supported by reliable evidence. He was unable to reference his own experience studying varicella reactivation, so he instead attempted to string together a number of smaller independent contentions. He noted, for example, that the situs where the VZV is believed to "hide" in the body, the dorsal root ganglion, is a nerve structure, and hence composed of the myelin he opines the flu vaccine can react against. But this hardly establishes that receipt of the vaccine likely causes a cross-reaction leading to reactivation, simply because *other* autoimmune injuries involve myelin attacks. In other contexts, like a flu-GBS claim, far more scientifically is understood about the autoantibody attack at the understood situs of the disease process, and hence cross-reactive potential *plus* this other evidence is enough to establish preponderance. Here, by contrast, there is far less understood about what can cause reactivation, reducing the value of Dr. Steinman's homology showing.

Otherwise, the best Dr. Steinman could do was to muster up some literature, like Youssef, suggesting that the NF-kB immune signaling pathway can cause upregulation of specific cytokines that the VZV infectious process succeeds in suppressing—concluding from this that the opposite was possible as well, and that vaccination could also stimulate this pathway to "un-suppress" a latent VZV infection. But the articles cited do not in the least stand for this speculative contention, and instead merely explore ways to make vaccination more effective in promoting immune memory, especially in elderly recipients. *See,* e.g., Haralambieva. They also seem more focused on the immune response to an active and progressing VZV infection, as opposed to what is relevant here—when the infection becomes latent, and what might in turn cause subsequent reactivation. Thus, articles like El Mjiyad make reliable scientific observations about how an active VZV infection will initially inhibit NF-kB activity—but *not* that stimulation of the same pathway later on can cause reactivation.

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through mimicry, a cross-reaction. Because, however, my Decision turns on other issues, I do not attempt to resolve who carried the day, preponderantly, on this ultimately non-dispositive issue.

Thus, little reliable or scientific evidence has been offered to show that the innate response to a flu vaccine would trigger reactivation. Indeed, it has not even been demonstrated that a wild viral flu infection (which would inherently be more stimulative of a potentially-aberrant immune response) could produce reactivation in the manner proposed. Dr. Steinman has simply done the same thing for which I have criticized experts in prior cases: attempting to flip what is known about the proper functioning of the immune system into a theory for how it can go awry. *See,* e.g., *Putman v. Sec'y of Health & Hum. Servs.*, No. 19-1921V, 2022 WL 600417, at *21 (Fed. Cl. Spec. Mstr. Jan. 31, 2022) (normal cytokine response to vaccination not shown to have capacity to aberrantly interact with intercurrent infection, resulting in juvenile idiopathic arthritis). Proving immune system "links" in a healthy process is only the start of a preponderant causation theory.

In the end, Dr. Steinman's theory is speculative and only plausible—and barely so at that. He has not preponderantly demonstrated that the flu vaccine is likely to cause VZV reactivation. Nor did he dispute that shingles alone is *incapable* of causing subsequent demyelination of the kind experienced by Petitioner—while Dr. Collins did reliably so demonstrate, with reference to a number of articles establishing that very point. *See,* e.g., J. McCarthy & J. Amer.

I acknowledge that Dr. Steinman has also proposed, albeit somewhat in passing, that the flu vaccine could independently cause TM and/or encephalomyelitis. There is far more preponderant support for this proposition.[16] But the theoretical capacity of the flu vaccine to cross-react with myelin proteins and produce autoimmune-driven demyelination is the answer to a question that the case itself does not ultimately pose. Petitioner himself seemed to recognize this—since even in enumerating alternative causation theories, he has maintained that the flu vaccine *first* caused VZV reactivation, regardless of what *then* prompted his encephalomyelitis. *See,* e.g., Mot. at 20–21. He does not decouple his shingles. And even if Petitioner's theory had solely focused on the relationship between the flu vaccine and his subsequent encephalomyelitis, my acceptance of such a more truncated theory would still run up against the fact of his intervening shingles (as discussed below), preventing an entitlement determination in his favor.[17]

[16] TM (and other comparable acute demyelinating conditions) has in the Program been repeatedly seen as associated with the flu vaccine. *See*, e.g., *Spayde v. Sec'y of Health & Hum. Servs.*, No. 16-1499V, 2021 WL 686682 (Fed. Cl. Spec. Mstr. Jan. 27, 2021); *Davis v. Sec'y of Health & Hum. Servs*., No. 07-451V, 2010 WL 1444056, at *13 (Fed. Cl. Spec. Mstr. Mar. 16, 2010) (explaining how the flu vaccine can cause demyelinating conditions). And the concept of molecular mimicry itself as a mechanism to explain how a vaccine or other wild virus antigenic stimulus might provoke an autoimmune disease is itself a reliable concept – when applied correctly to specific kinds of disease processes. At the same time, there are some decisions going the other way. *See,* e.g., *Caves v. Sec'y of Health & Hum. Servs.*, No. 07-443V, 100 Fed. Cl. 119 (2011) (affirming decision of special master dismissing flu vaccine-TM case).

[17] I give some limited weight to Dr. Collins's citation to Baxter, which undermines the conclusion that the flu vaccine can cause TM at all. Baxter has been criticized for its methodology (although I deem some of those criticisms to elevate form over substance, based upon a "needle in a haystack" search for weakness in what otherwise stands as a fairly reliable and comprehensive large-scale epidemiologic report). *See J. v. Sec'y of Health & Hum. Servs.*, No. 16-864V, 155 Fed. Cl. 20 (July 20, 2021). And it is of course always true that *no* epidemiologic study can "disprove" the possibility of vaccine causation. But this does not mean epidemiologic studies are inadmissible evidence, to be ignored

B. *Althen Prong Two*

As noted, the fact of Petitioner's shingles is uncontroverted—and it cannot be concluded from this record that its reactivation was likely vaccine-triggered. At bottom, his shingles best explains his subsequent myelitis based on the record before me. Certainly no treaters embraced Petitioner's theory, other than to note he had received a flu vaccine prior to his injuries, with one treater expressly discounting any vaccine association. Ex. 4 at 16–18; Ex. 6 at 5–7; 14. There is also nothing in the record except for the temporal relationship between vaccine and injury to connect the two—the kind of association long recognized in the Program as insufficient proof of a causal relationship. *Caves v. Sec'y of Health & Hum. Servs.*, No. 07-443V, 100 Fed. Cl. 119, 141–42 (2011). Petitioner's personal recollection of post-vaccination malaise is not uncommon, but the record does not amplify this into anything that would suggest he was undergoing a vaccine-caused immune process leading to VZV reactivation. Finally, Petitioner's medical history reveals an additional explanation for some of his symptoms—a Vitamin B12 deficiency that treaters took seriously enough to attempt to address—and this evidence also undermines his claim, because it was not adequately addressed or rebutted, even if I cannot find that it stands as a preponderant explanation for his injury.

C. *Alternative Cause/Factor Unrelated*

Even if a petitioner establishes the elements necessary for entitlement, Respondent may overcome that showing by preponderantly demonstrating that the injury is "due to factors unrelated to the administration of the vaccine." Section 13(a)(1)(B). The Vaccine Act provides that "factors unrelated to the administration of the vaccine," are those "which are shown to have been the agent ... principally responsible for causing the petitioner's illness, disability, injury, condition or death." Section 13(a)(2)(B).

Here, the record provides strong evidence of a factor unrelated—Petitioner's intervening VZV reactivation. There is no dispute in this case that shingles can lead to neurologic harm akin to what Petitioner experienced, and Petitioner's treaters deemed that association to have the most explanatory power. Therefore, in this case, even if I had focused only on the capability of the flu vaccine to cause TM or encephalomyelitis, I would still have found that Respondent carried his burden of preponderantly establishing an alternative cause. Of course, because I have already noted that Petitioner's main *Althen* prong one effort (which attempted to link the flu vaccine to VZV reactivation) failed, the burden of proof never shifted in the first place. *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1358 (Fed. Cir. 2010) ("[Petitioner] never established a prima facie

out of hand or given no weight simply because vaccine injuries are uncommon. *King v. Sec'y of Health & Hum. Servs.*, No. 03-584V, 2010 WL 892296, at *74 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) ("special masters have routinely found that epidemiologic evidence, and/or other medical journal articles, while not dispositive, should be considered in evaluating scientific theories").

case, so the burden (and attendant restrictions on what 'factors unrelated' the government could argue) never shifted").[18]

III. This Case Was Appropriately Decided on the Papers

In ruling on the record, I am choosing not to hold a hearing. Determining how best to resolve a case is a matter that lies generally within my discretion, and although the parties have not objected to this method of adjudication, I shall explain why a hearing was not required.

Prior decisions have recognized that a special master's discretion in deciding whether to conduct an evidentiary hearing "is tempered by Vaccine Rule 3(b)," or the duty to "afford[] each party a full and fair opportunity to present its case." *Hovey*, 38 Fed. Cl. at 400–01 (citing Rule 3(b)). But that rule also includes the obligation of creation of a record "sufficient to allow review of the special master's decision." *Id*. Thus, the fact that a claim is legitimately disputed, such that the special master must exercise his intellectual faculties in order to decide a matter, is not itself grounds for a trial (for if it were, trials would be required in every disputed case). Special masters are expressly empowered to resolve fact disputes without a hearing—although they should only so act if a party has been given the proper "full and fair" chance to prove their claim.

This matter was appropriately resolved on the papers rather than via a hearing. The primary facts relevant to my Decision are not in contention and did not require fact witness testimony. Both experts' reports were mostly comprehensible in their written form. And since the experts' focuses strayed a bit from the primary issues in contention, it was not difficult to parse out the sections of the reports most relevant to the case's resolution. Dr. Steinman's opinion devoted substantial time to establishing points about molecular mimicry and homology that

[18] In addition, I do not find that Petitioner otherwise established that the flu vaccine was a "substantial factor" in *either* his VZV reactivation or encephalomyelitis. In what is sometimes in the Program referred to as a *Shyface* analysis, petitioners can in some cases prove that a vaccine was a substantial factor in causing their injury, even though other causal factors exist, with no one primary cause that can be identified. *Martin v. Sec'y of Health & Hum. Servs.*, No. 17-250V, 2020 WL 4815840, at *33 (Fed. Cl. Spec. Mstr. July 17, 2020), *citing Shyface,* 165 F.3d at 1352–53. Thus, in a case where more than one potential explanations for injury had support, and one could not be preponderantly demonstrated as more likely than another, a vaccine could still be found causal if it played *some* role in the disease process.

The record in this case does not permit me to ignore the evidence of Petitioner's shingles, or the high likelihood that his subsequent neurologic symptoms were a by-product of it, rather than something independent—and nothing in Dr. Steinman's opinion establishes the contrary. Thus, even if it is equally true that shingles *or* the flu vaccine can cause TM/encephalomyelitis, the evidence *in this case* does not suggest that Petitioner's shingles was not *more likely* causal. No treaters or expert (other than perhaps Dr. Steinman) opined or proposed that the vaccine was to blame. Also, Petitioner's neurologic symptoms began far closer-in-time to his shingles onset (both of which happened in the second half of November 2013) than his vaccination on October 16th—so if temporality receives any weight at all, it favors shingles. By contrast, there is *no* evidence from the date of vaccination to the time of Petitioner's likely shingles onset over a month later (November 20, 2013 (Ex. 11 at 8–10)) that the vaccination did anything to Petitioner other than stimulate the kind of expected post-vaccination malaise. More alarming reactions he may have alleged are not corroborated by the contemporaneous record.

proved less relevant (if at all) to my disposition of the matter—and by comparison he offered far less on the question of whether and how the flu vaccine could cause VZV reactivation. The interests of justice favored my chosen manner of resolution over holding a trial to hear from the two experts—a trial that would have prolonged the matter's conclusion by six to eight additional months. This case is now nearly six years old, so expediting my determination was a reasonable consideration.

CONCLUSION

Petitioner has not met her burden of proof and is therefore not entitled to an award of damages. 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.[19]

IT IS SO ORDERED.

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

[19] 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.